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The European Research Journal

Aim and Scope

The European Research Journal (EuRJ) is an international, independent, double-blind peer reviewed, Open Access and online publishing journal, which aims to publish papers on all the related areas of basic and clinical medicine.

Editorial Board of the European Research Journal complies with the criteria of the International Council of Medical Journal Editors (ICMJE), the World Association of Medical Editors (WAME), and Committee on Publication Ethics (COPE).

The journal publishes a variety of manuscripts including original research, case reports, invited review articles, technical reports, how-to-do it, interesting images and letters to the editor. The European Research Journal has signed the declaration of the Budapest Open Access Initiative. All articles are detected for similarity or plagiarism. Publication language is English. The journal does not charge any article submission or processing charges.

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burken

Dear colleagues,

We are pleased to invite you to the 5th Uludağ Cardiology Summit to be held at Grand Karina Hotel Uludağ between 6–9 March 2025.

This year's congress will feature the latest developments in cardiology with sessions that include both theoretical and practical presentations. Developments in the field of cardiology will be discussed in the presence of expert speakers. During the Congress; it is aimed to evaluate developments in diagnosis and treatment spectrum in the light of current approaches. Research and projects in the field of cardiology can be presented and experiences can be shared with oral presentation sessions.

The success of the congress will be possible with your active participation. Looking forward to meeting in the winter of 2025 and having a successful, productive congress…

Prof. Dr. Sümeyye GÜLLÜLÜ
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Doç. Dr. Mustafa Adem YILMAZTEPE
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Scientific Programme

Bilimsel Program

06 MART 2025, PERŞEMBE		
	A Salonu	B Salonu
15:30 - 16:30	AÇILIŞ	
	HİPERTANSİYON **Oturum Başkanları: Mehmet Akif Düzenli, Nizamettin Toprak**	
16:30 - 16:42	Yeni Hipertansiyon Kılavuzunun Getirdikleri **Fatih Sinan Ertaş**	
16:42 - 16:54	Yaşam Tarzı Değişiklikleri ve Medikal Tedavi **Barış Güngör**	
16:54 - 17:06	İzole Diastolik Hipertansiyonu Tedavi Edelim mi? **Hatice Selçuk**	
17:06 - 17:18	Girişimsel Tedaviler **Regayip Zehir**	
17:18 - 17:30	Tartışma	
19:30 - 21:30	Akşam Yemeği	

07 MART 2025, CUMA		
	A Salonu	B Salonu
	KAPAK HASTALIKLARI HER YÖNÜYLE TRİKÜSPİT YETERSİZLİĞİ **Oturum Başkanları: Saide Aytekin, Recep Demirbağ**	**Sözel Bildiri Oturumu - 1** **Oturum Başkanı: Ayşen Ağaçdiken, Tunay Şentürk**
09:00 - 09:12	Triküspit Kapak Yetmezliği Değerlendirmesi **Ebru Özpelit**	Atrioventriküler Tam Bloğa Neden Olan Miyokardit **Emrah Kaya, Yakup Han Yılmaz** (Kütahya SBÜ)
09:12 - 09:24	Triküspit Kapak Yetersizliğinde Girişim Zamanlaması **Bahar Pirat**	Kronik Böbrek Yetmezliği ile Kardiyak Elektrofizyolojik Denge İndeksi Arasındaki İlişki **Ahmet Yıldırım, Adnan Duha Cömert** (Bursa Şehir H.)
09:24 - 09:36	Perkütan Triküspit Kapak Girişimleri **İsmail Ateş**	Primer Elektriksel Kardiyak Hastalıklarda Yapay Zeka: ChatGPT'nin Klinik Karar Destek Aracı Olarak Değerlendirilmesi **Muhammet Geneş** (Sincan EAH)
09:36 - 09:48	Cerrahi Tedavi Seçenekleri **Mustafa Tok**	Non-Valvüler Atriyal Fibrilasyon Hastalarında Serebrovasküler Olaylar ile Kan Belirteçleri Arasındaki İlişki **Murat Bilgin** (Yalova Özel Aktif H.)
09:48 - 10:00	Tartışma	Kolonoskopinin Atriyal Parametreler Üzerine Etkisi **Mustafa Sekmen, Seyit Ali Volkan Polatkan** (Uludağ Üni.)

10:00 - 10:20	Kahve Molası	
10:20 - 11:00	**En İyi Sözlü Bildiri Oturumu** **Oturum Başkanları: Mehmet Demir, Sümeyye Güllülü** Transkateter Aort Kapak İmplantasyonu Yapılan Hastalarda TAPSE/PASP Oranının Prognostik Önemi **Duygu İnan, Ayşe İrem Demirtola Mammadli, Anar Mammadli** (Çam ve Sakura Şehir H.) Ferritin-CRP Oranı: Düşük Ejeksiyon Fraksiyonlu Kalp Yetersizliğinde Mortalite Riski İçin Yeni Bir Gösterge **Çetin Alak, Şükrü Ciriş, Furkan Fatih Yurdalan, Fazıl Çağrı Hunutlu, Tunay Şentürk** (Uludağ Üni.) Diabetes Mellitus Tip 2 Hastalarında C-reaktif Protein/ Albumin Oranı (CAR) İle Mortalite Arasındaki İlişkinin Değerlendirilmesi **Selim Aydemir, Sidar Şiyar Aydın, Eda Özcan** (Erzurum Şehir H.) Akut Koroner Sendromlu Hastalarda İntravenöz Nitrogliserin Tedavisinin Klopidogrel'in Antiplatelet Etkileri Üzerindeki Etkisi: Pilot Çalışma **Hasan Arı, Mehmet Melek, Alper**	**Sözel Bildiri Oturumu - 2** **Oturum Başkanları: Dilek Yeşilbursa, Mustafa Yılmaz** Prostat Kanserinin Sık Görülmeyen Komplikasyonu Vaka Takdimi; Bıyık Şeklinde İntrakardiyak Trombüs **Özgen Şafak** (Balıkesir Üni.) Kalp Yetmezliği Semptomları ile Başvuran Sol Atrial Dev Miksoma Olgusu **Çağlar Alp, Zehra Uyan Ulaş, Saliha Barin, Muhammed Karadeniz** (Kırıkkale Üni.) Ameliyat Öncesi Torasik BT'de Tesadüfi Kardiyak Olmayan Radyolojik Bulgular Kalp Cerrahları için Bir Engel midir? **Özgür Barış, Törehan Özer** (Kocaeli Üni.) Persistan Sol Superior Vena Cava ile At Nalı Böbrek İlişkisi **Batuhan Özbaş** (Merkezefendi Devlet H.)

	Karakuş, Selma Arı, Ahmet Tütüncü, Tansin Bozat (Bursa Yüksek İhtisas EAH)	
11:00 - 11:20	Kahve Molası	
	KALP YETERSİZLİĞİ **Oturum Başkanları: Osman Akın Serdar, Birhan Yılmaz**	**Sözel Bildiri Oturumu - 3** **Oturum Başkanları: Tahsin Bozat, Davran Çiçek, Yusuf Ata**
11:20 - 11:32	EF'si Düzelen Kalp Yetersizliği Hastasını Nasıl Yönetelim? **Yüksel Çavuşoğlu**	SYNTAX Skorunun Perkütan Koroner Girişim ile Tedavi Edilmiş Yaşlı Hastalarda Mortaliteye Etkisi **Murat Çimci, Damla Raimoglou, Bilgehan Karadağ** (İstanbul Üni-Cerrahpaşa)
11:32 - 11:44	Kalp Yetersizliği Hastasında Unuttuklarımız **Hakkı Kaya**	
11:44 - 11:56	Kalp Yetersizliği CRT-D mi? İleti Sistemi Pacing? **Ahmet Vural**	Rüptüre Sinüs Valsalva Anevrizmasının Perkütan Kapatılması **Ertuğrul Yencilek, Birce Elif Bayır, İbrahim Halil Uygun, Saadet Demirtaş İnci, İbrahim Hakan Güllü, Mustafa Mücahit Balcı, Hatice Tolunay** (Etlik Şehir H.)
11:56 - 12:08	Mekanik Dolaşım Desteği: Hangi Hastaya Hangi Teknik? **Erman Pektok**	
12:08 - 12:30	Tartışma	Orta Dereceli Sol Ana Koroner Lezyonlarında Kullanılabilecek Trigliserit Glikoz İndeksi, İntravasküler Ultrasonografi Çalışması **Bilal Mete Ülker, Ömer Furkan Demir, Fahriye Vatansever Ağca** (Bursa Yüksek İhtisas EAH)

		Kalsifik Sol Ana Koroner Arter Lezyonuna Rotasyonel Aterektomi ile Girişim **Ömer Faruk Kahraman, Hasan Arı, Seray Yazgan** (Bursa Yüksek İhtisas EAH) Perikardiyosentez Sonrası Gelişen İyatrojenik Pnömoperikardiyum **Selvi Oztas, Selma Kenar Tiryakioğlu, Oğuz Kağan Şentürk, Sevil Güçlü** (Bursa Şehir H.) Visseral Adipozite İndeksinin Aterosklerotik Kardiyovasküler Hastalığı Olmayan Postmenopozal Kadınlarda Yüksek Kardiyovasküler Riski Öngörmedeki Tanısal Performansı **Onur Yıldırım, Ajar Koçak** (Liv Ankara H.)
12:30 - 14:00	Öğle Yemeği	
14:00 - 14:40	**UYDU SEMPOZYUM - 1** **Oturum Başkanı: Alparslan Birdane** **Jardiance' ın Gücüyle Dahası Var** Konuşmacılar: Alparslan Birdane, Abdulmecit Yıldız	Boehringer Ingelheim
14:40 - 15:00	Kahve Molası	
	GİRİŞİMSEL KARDİYOLOJİ **Oturum Başkanları: Vedat Aytekin, Cevat Kırma**	**Sözel Bildiri Oturumu - 4** **Oturum Başkanları: Ufuk Aydın, Mehmet Melek, Sinem Özyılmaz**
15:00 - 15:12	İlaç Kaplı Balonlar, Kime, Ne Zaman? **Korhan Soylu**	

15:12 - 15:24	OCT IVUS FFR Klinik-Kılavuzlar Arasındaki Klinik Uygulama **Bilgehan Karadağ**	Fibrotik Mitral Darlığı Olan Hastada Sol Atrium İçinde Trombüs **Canan Aydoğan** (Konya Numune H.) Asendan Aort Grefti Psödoanevrizmasının "Konar VSD Oklüder" Cihazı ile Perkütan Kapatılması **İbrahim Halil Uygun, Hatice Feyza Dilek, Ezgi Polat Ocaklı, İbrahim Hakan Güllü** (Etlik Şehir H.) Ciddi Mitral Darlığı ve Kalp Yetersizliği ile Birlikte G6PD Eksikliği: Yönetilmesi Zor Bir Vaka **Bilal Mete Ülker, Ayşe Dilara Balyimez, Mehmet Taşci, Mustafa Kani Gözcü, Esra Akpınar, Hasan Arı, Mehmet Melek** (Bursa Yüksek İhtisas EAH) Koroner Anjiyografi Sırasında Sıyrılan Stentin Düşük Profilli Balon Desteğiyle Lezyona Yerleştirilmesi **Çağlar Alp, Muhammed Ensar Eker, Muhammed Karadeniz** (Kırıkkale Üni.) Genç ACS Hastalarında Ejeksiyon Fraksiyonunu Tahmin Etmede Beyaz Kan Hücresi Düzeylerinin Rolü **Murat Demirci** (Marmara Üni.) Tip 2 Diabetes Mellituslu Hastalarda GLP-Agonistlerinin Sol Atriyum Volumu ve Boşalma Fonksiyonları Üzerine Etkileri
15:24 - 15:36	SCAD Tanı Tedavi Yaklaşımları **Can Yücel Karabay**	
15:36 - 15:48	Koroner Kalsifik Lezyonlarda Tedavi Seçenekleri **Altuğ Çinçin**	
15:48 - 16:00	Tartışma	

		Dogac Oksen (Altınbaş Üni.)
16:00 - 16:20	Kahve Molası	
16:20 - 17:00	**UYDU SEMPOZYUM - 2** **Oturum Başkanı: Korhan Soylu** Boston Scientific **Modern PCI'da Gelişmeler: Gör-Hazırla-Tedavi Et Stratejileri ile Optimum Sonuçlar** Konuşmacılar: Hakan Erkan, Korhan Soylu	
17:00 - 17:20	Kahve Molası	
	GÖRÜNTÜLEME **Oturum Başkanları: Serdar Küçükoğlu, Elif Eroğlu**	**Sözel Bildiri Oturumu - 5** **Oturum Başkanları: Mehmet Ali Astarcıoğlu, Fahriye Vatansever**
17:20 - 17:28	Mitral Yetmezliği Değerlendirmede Zorlu Vaka Örnekleri **Omaç Tüfekçioğlu**	Malign Perikardiyal Efüzyonda İntraperikardiyal Bleomisin Uygulaması **Canan Aydoğan, Hüseyin Tezcan** (Konya Numune H.)
17:28 - 17:36	Aort Yetmezliği Değerlendirmede Kardiyak MR **Ahmet Barutçu**	Akut Koroner Sendrom Tablosuyla Gelen Hipereosinofili Sendromu Öntanısı Olan Hastaya Yaklaşım **Cem Utku Yeşilkaya, Mustafa Yılmaz, Hakan İskender** (Kulu Devlet H.)
17:36 - 17:44	Aort Darlığı Değerlendirmesinde Zorlu Vaka Örnekleri **Selcen Yakar Tülüce**	
17:44 - 17:52	Protez Kapak Hastalıklarının Değerlendirilmesi **Zübeyde Bayram**	Trabectedin ile İlişkili Miyokardit **Safa Oktay, Doğan Ormancı, Seray Yazgan, Ayşe Dilara Balyimez, Ömer Faruk Kahraman, Mehmet Melek, Hasan Arı** (Bursa Yüksek İhtisas EAH)
17:52 - 18:00	Tartışma	

		Nadir Bir Vaka Akut Q Ateşi **Selma Kenar Tiryakioğlu , Uğur Demirpek, Emre Sefa Düz, Gülşah Sefer** (Bursa Şehir H.) Antegrad Transseptal Yaklaşımla Retrograd Transmitral Paravalvüler Kaçak Kapatılması **Birce Elif Bayır, Ertuğrul Yencilek, İbrahim Halil Uygun, Ayşenur Özkaya İbiş, Gürkan İş, İbrahim Hakan Güllü** (Etlik Şehir H.)
19:30 - 21:30	Akşam Yemeği	

08 MART 2025, CUMARTESİ		
	A Salonu	B Salonu
	KORONER ARTER HASTALIĞI **Oturum Başkanları: Mustafa Kemal Erol, Abdurrahman Oğuzhan**	**Sözel Bildiri Oturumu - 6** **Oturum Başkanları: Mustafa Adem Yılmaztepe, İrfan Barutçu**
09:00 - 09:12	Yeni CCS Kılavuzu Neler Getirdi? **Oğuz Yavuzgil**	Akut Koroner Sendromlu Hastalarda Beslenme Durumunun, Visseral Yağlanmanın ve Böbrek Fonksiyonunun Santral Arteriyel Sertliğe Etkisi **Esra Akpınar, Ömer Furkan Demir, Nazife Nur Özer Şensoy, Ayşe Dilara Balyimez, Bilal Mete Ülker, Tolga Doğan** (Bursa Yüksek İhtisas EAH) Kolondan Kalbe: Enterococcus Faecalis Enfektif Endokarditi ve Kolorektal Neoplazi **Burak Yaman, Çetin Alak, Furkan Fatih Yurdalan, Dilek Yeşilbursa, Bülent Özdemir** (Uludağ Üni.) Spontan Koroner Arter Diseksiyon Yönetimi **Seray Yazgan, Doğan Ormancı, Muhammet Nusret Akın, Davut Davutoğlu, Hasan Arı** (Bursa Yüksek İhtisas EAH) Kolesterol Kristallerinin İzinde: Tanı Labirentinde Bir Yolculuk **Burak İbrahimoğlu, Samatar**
09:12 - 09:24	Refrakter Anjinalı Hastaya Yaklaşım **Taner Şen**	
09:24 - 09:36	Kronik Koroner Sendromlarda Antiagregan ve Antikuagulan Tedaviler **Özgen Şafak**	
09:36 - 09:48	Koroner Arter Hastalığında Residüel Risk Azaltıcı Tedavi Yaklaşımları **Meral Kayıkçıoğlu**	
09:48 - 10:00	Tartışma	

		Adam, Çetin Alak, Dilek Yeşilbursa (Uludağ Üni.) Radial Arter Girişimlerinde Radial Arter Spazmına Anatomik Varyasyonların Etkileri **Bedrettin Boyraz** (Bursa Medicana H.) Türkiye'de Sigara Kullanım Dinamikleri: Başlama Nedenleri, Sürdürme Motivasyonları ve Nikotin Bağımlılık Düzeyleri Üzerine Bir Analiz **Şenol Coşkun, Akın Torun** (Kocaeli Üni.)
10:00 - 10:20	Kahve Molası	
10:20 - 11:00	**UYDU SEMPOZYUM - 3** **Oturum Başkanı: Mehmet Birhan Yılmaz** **Kararın Fark Yaratsın: Forziga Tedavisi** Konuşmacılar: Mehmet Birhan Yılmaz, Hakkı Kaya	AKILCI İLAÇ KULLANIMI **Oturum Başkanı: Ahmet Tütüncü** **Konuşmacı:** Alper Karakuş
11:00 - 11:20	Kahve Molası	
	PULMONER HİPERTANSİYON **Oturum Başkanları: Cihangir Kaymaz , Mehmet Akbulut**	KARDİYAK YOĞUN BAKIM HEMŞİRELİĞİ - 1 **Oturum Başkanları: Selma Tiryakioğlu, Hemşire Sevil Tecir, Öznur Acar**

11:20 - 11:30	7. Dünya sempozyumunun Getirdikleri **Bahri Akdeniz**	Atriyal Fibrilasyon ve Hemşirelik Bakımı **Hemşire Esma Aydoğdu**
11:30 - 11:40	Nonkardiyak Cerrahiye Gidecek Hastaların Yönetimi **Barış Kaya**	Kalp Yetmezliği ve Hemşirelik Bakımı **Uzm. Hemşire Seher Kantar**
11:40 - 11:50	Romatolojik Hastalıklarda Pulmoner Hipertansiyon **Murat Meriç**	Yoğun Bakım Ünitelerinde Sık Görülen Enfeksiyonlar ve Kanıta Dayalı Uygulamalar **Hemşire Kamile Hakim**
11:50 - 12:00	Konjenital Kalp Hastalıklarında Pulmoner Hipertansiyon **Necip Ermiş**	Romotolojik Hastalarda Kardiyak Tutulum **Hemşire Tuğba Navruz**
12:00 - 12:30	Tartışma	Tartışma
12:30 - 14:00	Öğle Yemeği	
14:00 - 14:40	**UYDU SEMPOZYUM - 4** **Oturum Başkanı: Serdar Küçükoğlu** janssen | PHARMACEUTICAL COMPANIES OF Johnson&Johnson **PAH Tedavisinde Güncel Yaklaşımlar** Konuşmacılar: Cihangir Kaymaz, Mehmet Akbulut, Serdar Küçükoğlu	KARDİYAK YOĞUN BAKIM HEMŞİRELİĞİ - 2 **Oturum Başkanları: Şeyda Günay Polatkan, Selma Arı, Eylem Paslı Gürdoğan**
14:00 - 14:08		Koroner Yoğun Bakım Ünitesinde Antitrombolitiklerin Kullanımı ve Dikkat Edilmesi Gerekenler **Hemşire Gökhan Kalkan**

14:08 - 14:16		Koroner Yoğun Bakım Ünitesinde EKO ile Değerlendirenin Önemi **Hemşire Merve Çetin**
14:16 - 14:24		Kalp Hastalıklarında Psikolojik Problemler **Hemşire Hatice Özyıldırım**
14:24 - 14:32		Kalp Tamponadı ve Hemşirelik Bakımı **Hemşire Tuğba Navruz**
14:32 - 14:40		Tartışma
14:40 - 15:00	Kahve Molası	
	HER TELDEN KARDİYOLOJİ **Oturum Başkanları: Aytül Belgi, Mehmet Ertürk**	KATETER LABORATUVARI HEMŞİRELİĞİ - 1 **Oturum Başkanları: Bülent Özdemir, Ercan Karabey**
15:00 - 15:08	Ramazan ve Kalp Hastalıkları **Çiğdem İleri Doğan**	Elektrofizyolojik Çalışmalarda Salon Hazırlığı ve Hemşirelik Yaklaşımı **Hemşire Doğukan Dülger**
15:08 - 15:16	Takatsubo Türk Uzlaşı Raporu **Servet Altay**	Perkütan ASD Kapama Vakalarında Hasta ve Salon Hazırlığı **Hemşire Gamze Gelen**
15:16 - 15:24	Kalp Hastalıkları ve AI **Nurgül Keser**	Kalıcı Kalp Pili Enfeksiyonlarını Önlemede Anjio Hemşireliği **Hemşire Nurullah Kaya**
15:24 - 15:32	Çevre Kirliliği, Mikroplastik ve Kalp Hastalıkları **Emine Gazi**	Primer PCI Vakalarında Anjio Hemşireliği

		Hemşire Muhammed Hüseyin Dağ
15:32 - 15:40	Akut Pulmoner Emboli Tedavisi **Hacer Ceren Tokgöz**	Tartışma
15:40 - 15:48	Kardiyolojide PET' in Kullanımı **Emrah Erdoğan**	
15:48 - 15:56	Dikişsiz Kapaklarda Sorunlar **Mesut Engin**	
15:56 - 16:20	Tartışma	
16:20 - 16:30	Kahve Molası	
	AF GÜNCELLEMESİ **Oturum Başkanları: Sedat Köse, Fethi Kılıçaslan**	KATETER LABORATUVARI HEMŞİRELİĞİ - 2 **Oturum Başkanları: Aysel Aydın Kaderli, Zülfiye Tay Göven, Dudu Biler**
16:30 - 16:42	Yeni AF Kılavuzunda Neler Değişti? **Nihal Akar**	FFR Vakalarında Salon ve Malzeme Hazırlığı **Hemşire Sümeyye Gedik**
16:42 - 16:54	AF' de Farmakolojik Tedavi **Kutay Vurgun**	Hemodinami Laboratuvarında İABP kullanımı **Perfüzyonist Eşe Hanım Karaoğlu**
16:54 - 17:06	AF' de Girişimsel Tedavi Seçenekleri **Taylan Akgün**	Radyasyon Güvenliği **Radyoloji Teknikeri Hasan Ali Kiremitçi**
17:06 - 17:18	LAA Oklüzyonu **Levent Şahiner**	Tartışma
17:18 - 17:30	Tartışma	

	EDİTÖRLER İLE GENÇ KARDİYOLOGLAR BULUŞUYOR **Moderatör: Hasan Arı**	
17:45 - 18:45	Konuşmacılar: **Bilgin Timuralp** **Şenol Yavuz** **Servet Altay**	
19:30 - 21:30	Akşam Yemeği	

09 MART 2025, PAZAR		
	A Salonu	B Salonu
	GİRİŞİMSEL İŞLEMLERDE KOMPLİKASYON YÖNETİMİ **Oturum Başkanları: Engin Bozkurt, Halil İbrahim Kurt**	**Sözel Bildiri Oturumu - 7** **Oturum Başkanları: Vedat Koca, İbrahim Baran**
09:00 - 09:12	Olgularla Koroner Komplikasyon **Nihat Kalay**	Akut Koroner Sendromda Şok Indeksinin Prognostik Rolü **Sibel Çatalkaya, Eda Özcan** (Bursa Sehir H.)
09:12 - 09:24	Olgularla Yapısal Komplikasyon **Hakan Uçar**	TCBI Kalp Yetmezliği Hastalarında Mortalite ve Hastane Yatış Sürelerini Öngörebilir mi? **Alkame Akgümüş, Ali Duygu, Ahmet Balun** (Onyedi Eylül Üni.)
09:24 - 09:36	Olgularla Pacemaker ve Ablasyon Komplikasyon **Hasan Koca**	STEMI mi, Yoksa Bir Yanılsama mı? CPR Sonrası Pnömomediastinumun Beklenmedik Kardiyak Yansıması **Kaan Turhan, Harun Şenocak, Çetin Alak, Dilek Yeşilbursa** (Uludağ Üni.)
09:36 - 09:48	Olgularla Periferik Komplikasyon **Ertan Vuruşkan**	ST-Segment Yükselmesi Olmayan Miyokard İnfarktüsü Hastalarında Hastane İçi Majör Kardiyak Olayların Belirlenmesinde Yeni İnflamatuar Markırların Yeri; Yeni Bakış Açısı **Oğuzhan Baran** (Kayseri Şehir H.)
09:48 - 10:00	Tartışma	

		Yüksek Riskli Biyoprotez Kapak Disfonksiyonu Hastasında Mitral Valve-in-Valve Prosedürü **Betül Sarıbıyık Çakmak, Çağla Akçay Ürkmez, Ahmet Can Çakmak** (Sakarya Üni.) Yüksek Yoğunluklu Lipoprotein Kolesterol-Monosit Oranı ile Koroner Arter Hastalığı Arasındaki İlişki **Zeki Çetinkaya** (Fethi Sekin Şehir H.)
10:00 - 10:20	Kahve Molası	
	KARDİYOLOJİ'DE 2024 YILI GÜNCELLEMESİ **Oturum Başkanları: Ali Aydınlar, Ethem Kumbay**	**Sözel Bildiri Oturumu - 8** **Oturum Başkanları: Hasan Arı, Hasan Ali Barman**
10:20 - 10:28	2024'de Aritmiler **Erol Tülümen**	Perkütan Patent Foramen Ovale Kapatmada Tek Merkez Deneyimi: Kısa Dönem Klinik Sonuçlar **Fatih Koca, Ece Çelebi Coşkun** (Bursa Yüksek İhtisas EAH) Akrep Sokması İle İlişkili Myokardit Olgusu **Gökhun Akkan** (Nazilli Devlet H.) Antiagregan Tedaviler Non-Obstrüktif Koroner Arterlerde İskemi Durumunda Majör Olumsuz Kardiyovasküler Olayları Azaltabilir mi?
10:28 - 10:36	2024'de Koroner Arter Hastalığı **Muhammet Gürdoğan**	
10:36 - 10:44	2024'de Kapak Hastalıkları **Fatih Güngören**	
10:44 - 10:52	2024'de Girişimsel Kardiyoloji **Hakan Güllü**	
10:52 - 11:00	2024'de Kalp Yetersizliği **Ahmet Çelik**	
11:00 - 11:08	2024'de Görüntüleme **Demet Menekşe Gerede Uludağ**	
11:08 - 11:20	Tartışma	

		Aykut Demirkıran, Cihan Aydın, Ferhat Perk, Şeref Alpsoy (Namık Kemal Üni.) Non-ST Elevasyonlu Miyokard İnfarktüsü İle Başvuran Hastalarda Saphenous Ven Grefti Perkütan Koroner Girişimi Sonrası No-Reflow Fenomeninin Tahmininde Beslenme İndeksleri **Ahmet Karaduman, Cemalettin Yılmaz** (Koşuyolu Yüksek İhtisas EAH) Spor Kardiyolojisi Perspektifinden; Elit Porcularda Overuse Sendromunu Öngörmede Kalp Hızı Değişkenliği Analizi **Akın Torun, Şenol Coşkun** (Göztepe Medical Park H.) Voleybol Sultanlar Liginde Kalp Hızı ve Toparlanma Zamanının Servis Başarısına Etkisi **Akın Torun** (Göztepe Medical Park H.) Koroner Bypass Greft - Native Damar Anastomoz Yeri Darlıklarında Nasıl Bir Strateji İzleyelim? **Tolga Memioğlu, Mehmet İnanır** (Abant İzzet Baysal Üni.) Asemptomatik Karotid Arter Darlığı Olan Hastalarda Ekokardiyografi ve Yüzey Elektrokardiyografi Kullanılarak Atrial Fibrilasyon Gelişme Riskinin
		Değerlendirilmesi **Yücel Yılmaz, Oğuzhan Baran** (Kayseri EAH)
11:20 - 11:40	KAPANIŞ	

5th Uludag Cardiology Summit

March 6-9, 2025, Bursa, Türkiye

5. Uludağ Kardiyoloji Zirvesi

6-9 Mart 2025, Bursa, Türkiye

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MEETING ABSTRACTS

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MEETING ABSTRACTS

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OP-01.
A Case of Myocarditis Associated With a Scorpion Sting

Gökhun Akkan

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Introduction: Scorpion stings are commonly encountered in regions with hot climates, especially during the summer months. While most are harmless, some cases of envenomation present with local or systemic symptoms. Locally, pain, increased temperature, and paresthesia may occur at the sting site, while systemic effects include circulatory disorders, myocarditis, pulmonary edema, and neurological complications. The most common cause of death following a scorpion sting is cardiovascular complications. The development of cardiovascular symptoms is attributed to increased catecholaminergic activity, reduced myocardial perfusion due to microvascular spasm, and the direct toxic effects of the venom on myocardial fibers. Severe toxic effects are more frequently observed in children and the elderly.

Case Report: A 70-year-old female patient presented to the emergency department with pain, swelling, and redness in her right big toe after being stung by a yellow-colored scorpion. She had no history of chronic illness or coronary artery disease. Her electrocardiogram (ECG) showed normal sinus rhythm, and her blood pressure was measured at 130/80 mmHg. Initial laboratory tests revealed a troponin I level of 148 ng/L, with no other abnormal values. Upon follow-up, her troponin I level increased to 464 ng/L, prompting a consultation with our department.

Echocardiography revealed an ejection fraction of 60% and minimal mitral regurgitation (Fig. 1). The patient was admitted with a suspected diagnosis of myocarditis. During follow-up, she remained asymptomatic in terms of cardiac symptoms, with no ECG changes or additional echocardiographic abnormalities. To rule out coronary artery disease, myocardial perfusion scintigraphy was performed within the capabilities of our hospital, and no hypoperfused areas were observed. The scan was reported as normal. On the fourth day of hospitalization, her troponin I levels returned to negative. The patient was discharged with a prescription for a low-dose beta-blocker and an angiotensin-converting enzyme inhibitor.

Conclusion: Turkey is home to many scorpion species, making scorpion stings a common occurrence in the country. One of these species, Mesobuthus gibbosus (Anatolian yellow scorpion), is found in Western, Southern, and Central Anatolia, and literature has reported cases of severe myocarditis associated with this species (Fig. 2). Based on the patient's history, we suspect that this was the species responsible for our case.

Scorpion venom contains neurotoxins, hemolysins, agglutinins, hemorrhagins, leukocytolysins, coagulins, enzymes, lecithin, and cholesterol. It has both local and systemic effects. Local effects include edema, ecchymosis, and burning pain. Systemic effects occur through the autonomic nervous system. The venom stimulates peripheral sympathetic nerve endings, leading to catecholamine release from the adrenal medulla. In severe cases, early-stage catecholamine discharge can result in hypertension and increased left ventricular contraction. In later stages, catecholamine depletion syndrome and activation of the kinin/prostaglandin pathway may cause biventricular systolic dysfunction, hypotension, pulmonary edema, and cardiogenic shock, with more pronounced effects in the left ventricle.

Scorpion stings can lead to fatal outcomes through their toxic effects on the cardiovascular system. To avoid missing potential complications, myocarditis should be considered in patients presenting with scorpion stings, particularly those experiencing respiratory distress or general deterioration in condition.

Keywords: Scorpion string, myocarditis, venom, hemolysis

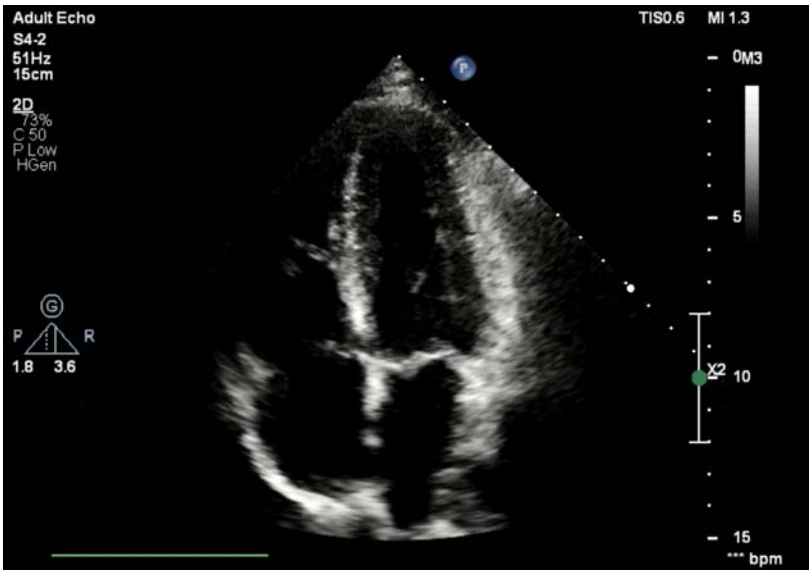

Fig. 1. **The patient's echocardiography image shows a normal ejection fraction.**

Fig. 2. **Mesobuthus gibbosus (Anatolian yellow scorpion).**

OP-02.
Myocarditis Causing Atrioventricular Complete Block

Emrah Kaya, Yakup Han Yılmaz

Department of Cardiology, Kutahya Health Sciences University, Faculty of Medicine, Kütahya, Türkiye

Introduction: Myocarditis is a myocardial disease characterized by inflammatory infiltration and necrosis of myocytes. Many agents, primarily viruses but also bacteria, fungi, autoimmune diseases, and pharmacological agents, can cause myocarditis. While myocarditis may often be asymptomatic, in some cases, it can lead to severe congestive heart failure and death. Various electrocardiographic (ECG) abnormalities have been reported in patients with myocarditis. It includes abnormalities in the ST-T wave segment, Q waves, atrioventricular block (AV block) or bundle branch blocks. It is rare to see heart block as the first-and-only presentation of infectious myocarditis. The diagnosis should be considered when physicians encounter a young patient with high-degree heart block associated to atypical symptoms of infection.
Case Report: A 47-year-old male patient presented to the Emergency Department with complaints of chest pain and dyspnea. His past medical history was unremarkable. Three-days prior the chest pain, he developed an influenza-like illness consisting of fevers, rhinorrhea, and sore throat. His ECG showed normal sinus rhythm, and troponin levels were elevated at 25,000 ng/L, prompting initial diagnoses of NSTEMI or myocarditis. Laboratory studies were remarkable for a white blood cell count of 12870/mm³ and elevated C-reactive protein at 66mg/L. Serum electrolytes, kidney and liver functions were within normal limits. Cardiac examination revealed normal heart sounds and respiratory system examination was unremarkable. The patient underwent coronary angiography in the catheterization laboratory, revealing normal LMCA, a 30-40% stenosis in LAD, normal CX, and plaque in RCA. Myocarditis was suspected as the primary diagnosis. Post-procedure, he was transferred to the intensive care unit where a transthoracic echocardiogram showed an ejection fraction of 60% with normal valve movements. During intensive care monitoring, an ECG revealed complete AV block (Fig. 1), prompting consideration for permanent pacemaker implantation. After one week of observation confirming persistent AV block, the patient underwent planned permanent pacemaker implantation following necessary preparations in the catheterization laboratory. Post-implantation, an electrocardiogram in the ward showed pacing rhythm. The patient's medical management was adjusted, and he was discharged.
Conclusion: The true incidence of infectious myocarditis in young healthy patients is unknown because of the frequently asymptomatic nature of infection. Several viruses are believed to account for the majority of cases, but a causative pathogen is identified in fewer than 10% of cases. More serious arrhythmias or conductive disturbances such as AV conduction block, as seen in our patient, are estimated to occur in fewer than 20% of cases and are correlated to worse prognosis. Myocarditis, although often asymptomatic, should not be forgotten as it can rarely lead to congestive heart failure, complete AV block, and even death in patients.
Keywords: Atrioventricular block, myocarditis, inflammatory infiltration

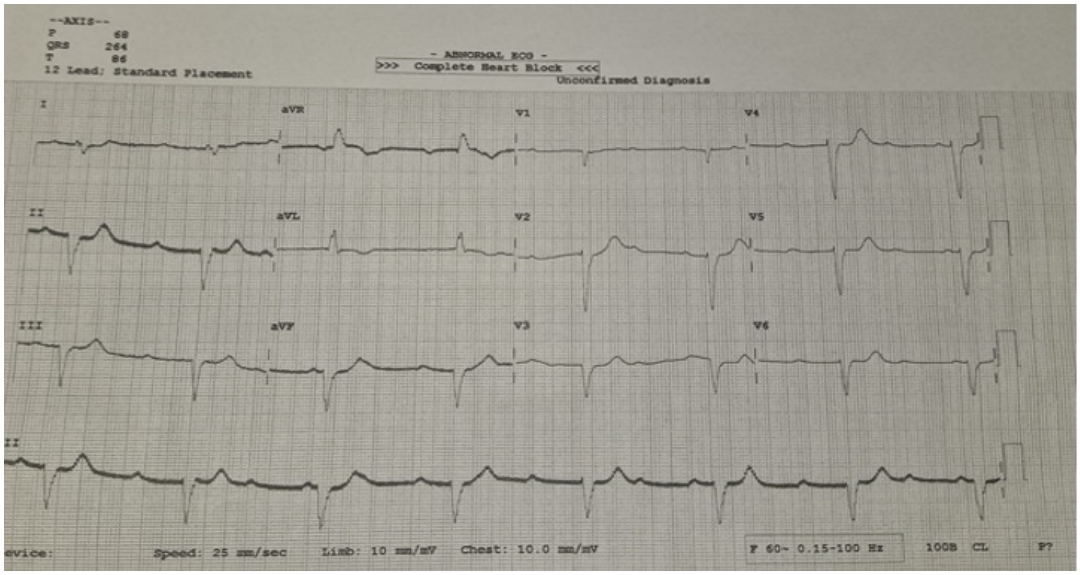

Fig. 1. The ECG showing the development of complete AV block during the patient's follow-up.

OP-03.
Intrapericardial Bleomycin Administration in Malignant Pericardial Effusion

Canan Aydoğan[1], Hüseyin Tezcan[2]

[1]Department of Cardiology, Konya Numune Hospital, Konya, Türkiye; [2]Department of Cardiology, Konya City Hospital, Konya, Türkiye

Introduction: Malignant pericardial effusions are often asymptomatic and are usually detected incidentally via echocardiography or computed tomography. Treatment options include pericardiocentesis and pericardial sclerosing agents. In this case, we aim to highlight the use of a sclerosing agent in recurrent malignant pericardial effusion.

Case Report: A patient who had been under follow-up for three years due to lung cancer and receiving regular chemotherapy was consulted to our department after experiencing worsening dyspnea while being monitored in the pulmonology clinic. Physical examination revealed jugular venous distension, muffled heart sounds, and a blood pressure of 90/60 mmHg. Electrocardiography (ECG) showed sinus tachycardia and decreased amplitude. Transthoracic echocardiography (TTE) was performed, revealing a left ventricular ejection fraction (LVEF) of 55% and a pericardial effusion exerting pressure on the right ventricle during diastole. The patient was diagnosed with pericardial tamponade, and pericardiocentesis was planned. A total of 1200 cc of pericardial fluid was drained. No further pericardial fluid accumulation was observed in the follow-up echocardiography after 24 hours of free drainage, and the patient was discharged.

Fifteen days after discharge, the patient was admitted to the emergency department with dyspnea. Echocardiography revealed recurrent pericardial effusion compressing the right ventricle during diastole. The patient was hospitalized, and pericardiocentesis was performed. Although the control echocardiography showed no pericardial effusion, the recurrence of pericardial tamponade within 15 days necessitated intrapericardial administration of bleomycin. A total of 15 mg of bleomycin was diluted in 35 cc of normal saline and administered intrapericardially. The patient was discharged without any recurrence of pericardial effusion and was scheduled for follow-up in one month. At the one-month follow-up, echocardiography confirmed the absence of pericardial effusion. The patient continued routine follow-ups without any active complaints.

Discussion: Malignant pericardial effusions are frequently asymptomatic and are often detected incidentally by echocardiography or computed tomography. Symptomatic cases can lead to cardiac tamponade, cardiovascular collapse, and even death. Lung cancer is the most common cause of malignant pericardial effusion, but it can also be seen in esophageal cancer, leukemia, lymphoma, and breast cancer. Various pericardial sclerosing agents are available for malignant pericardial effusion, including tetracycline, thiotepa, cisplatin, carboplatin, vinblastine, and bleomycin. These agents induce an inflammatory adhesion process within the pericardial cavity. Although positive outcomes have been reported with these agents in the control and prevention of malignant pericardial effusion, sufficient clinical studies are lacking. In the literature, a study by Kunitoh et al. compared intrapericardial bleomycin administration with pericardial effusion drainage in lung cancer patients with malignant pericardial effusion, finding no significant difference in mortality and morbidity between the two approaches. Randomized trials to date have demonstrated that, compared to other chemotherapeutic agents, bleomycin is associated with lower morbidity and is an effective and safe agent for pericardial sclerosis following drainage.

Conclusion: In this case, we successfully prevented the recurrence of malignant pericardial effusion by inducing pericardial fibrosis through intrapericardial administration of bleomycin. This approach reduced the patient's hospitalization frequency. Bleomycin is an effective agent for the management of recurrent malignant pericardial effusion.
Keywords: Bleomycin, lung cancer, pericardial tamponade

OP-04.
Left Atrial Thrombus in a Patient With Fibrotic Mitral Stenosis

Canan Aydoğan

Department of Cardiology, Konya Numune Hospital, Konya, Türkiye

Introduction: Malignant pericardial effusions are often asymptomatic and are usually detected incidentally via echocardiography or computed tomography. Treatment options include pericardiocentesis and pericardial sclerosing agents. In this case, we aim to highlight the use of a sclerosing agent in recurrent malignant pericardial effusion.
Case Report: A patient who had been under follow-up for three years due to lung cancer and receiving regular chemotherapy was consulted to our department after experiencing worsening dyspnea while being monitored in the pulmonology clinic. Physical examination revealed jugular venous distension, muffled heart sounds, and a blood pressure of 90/60 mmHg. Electrocardiography (ECG) showed sinus tachycardia and decreased amplitude. Transthoracic echocardiography (TTE) was performed, revealing a left ventricular ejection fraction (LVEF) of 55% and a pericardial effusion exerting pressure on the right ventricle during diastole. The patient was diagnosed with pericardial tamponade, and pericardiocentesis was planned. A total of 1200 cc of pericardial fluid was drained. No further pericardial fluid accumulation was observed in the follow-up echocardiography after 24 hours of free drainage, and the patient was discharged.
Fifteen days after discharge, the patient was admitted to the emergency department with dyspnea. Echocardiography revealed recurrent pericardial effusion compressing the right ventricle during diastole. The patient was hospitalized, and pericardiocentesis was performed. Although the control echocardiography showed no pericardial effusion, the recurrence of pericardial tamponade within 15 days necessitated intrapericardial administration of bleomycin. A total of 15 mg of bleomycin was diluted in 35 cc of normal saline and administered intrapericardially. The patient was discharged without any recurrence of pericardial effusion and was scheduled for follow-up in one month. At the one-month follow-up, echocardiography confirmed the absence of pericardial effusion. The patient continued routine follow-ups without any active complaints.
Discussion: Malignant pericardial effusions are frequently asymptomatic and are often detected incidentally by echocardiography or computed tomography. Symptomatic cases can lead to cardiac tamponade, cardiovascular collapse, and even death. Lung cancer is the most common cause of malignant pericardial effusion, but it can also be seen in esophageal cancer, leukemia, lymphoma, and breast cancer. Various pericardial sclerosing agents are available for malignant pericardial effusion, including tetracycline, thiotepa, cisplatin, carboplatin, vinblastine, and bleomycin. These agents induce an inflammatory adhesion process within the pericardial cavity. Although positive outcomes have been reported with these agents in the control and prevention of malignant pericardial effusion, sufficient clinical studies are lacking. In the literature, a study by Kunitoh et al. compared intrapericardial bleomycin administration with pericardial effusion drainage in lung cancer patients with malignant pericardial effusion, finding no significant difference in mortality and morbidity between the two approaches. Randomized trials to date have demonstrated that, compared to other chemotherapeutic agents, bleomycin is associated with lower morbidity and is an effective and safe agent for pericardial sclerosis following drainage.
Conclusion: In this case, we successfully prevented the recurrence of malignant pericardial effusion by

inducing pericardial fibrosis through intrapericardial administration of bleomycin. This approach reduced the patient's hospitalization frequency. Bleomycin is an effective agent for the management of recurrent malignant pericardial effusion.

Keywords: Bleomycin, lung cancer, pericardial tamponade

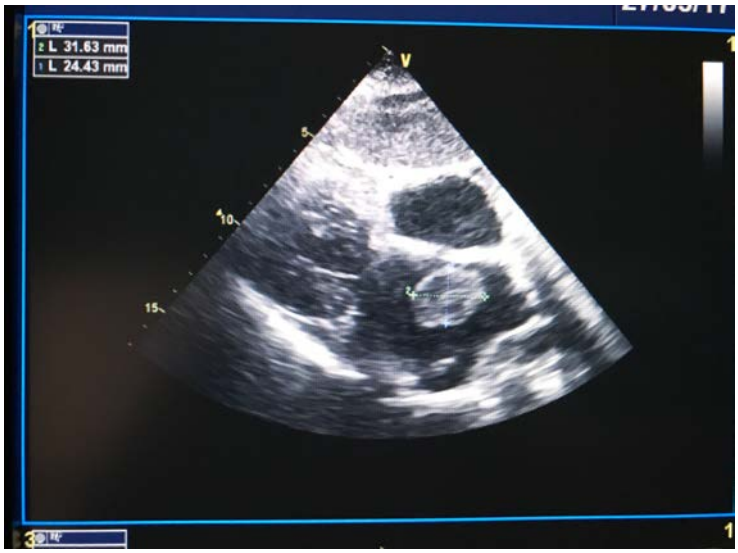

Fig. 1. **Trombus in the left atrium.**

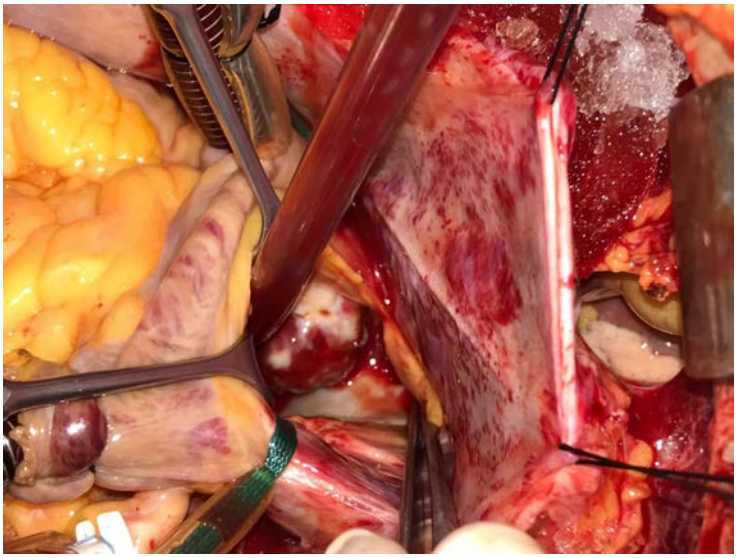

Fig. 2. **Trombus in the left atrium during surgery.**

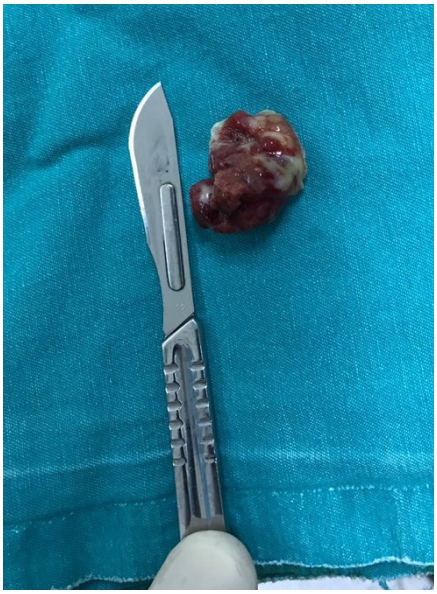

Fig. 3. **Trombus removed by surgery.**

OP-05.
Prognostic Relevance of TAPSE/PASP Ratio in Patients Undergoing Transcatheter Aortic Valve Implantation

Duygu İnan[1], Ayşe İrem Demirtola Mammadli[2], Anar Mammadli[3]

[1]Department of Cardiology, Başakşehir Çam ve Sakura City Hospital, İstanbul, Türkiye; [2]Department of Cardiology, Ankara Bilken City Hospital, Ankara, Türkiye; [3]Department of Cardiology, Bayındır Söğütözü Hospital, Ankara, Türkiye

Background: The ratio of tricuspid annular plane systolic excursion (TAPSE) and pulmonary arterial systolic pressure (PASP) obtained by echocardiography, as a more sensitive indicator of right ventricular dysfunction, has recently been reported as an independent prognostic parameter in many different diseases such as heart failure and pulmonary hypertension.
Aims: This study aims to evaluate the association between TAPSE/SPAP ratio and all-cause mortality in patients undergoing transcatheter aortic valve implantation (TAVI).
Methods: A retrospective analysis was conducted on a cohort of patients who underwent TAVI. Patients were categorized into tertiles based on their TAPSE/SPAP ratio: low (<0.42), middle (0.42-0.75), and high (>0.75). The demographic and clinical characteristics of these groups were compared. The main outcome was all-cause mortality. Kaplan-Meier curves were generated to depict mortality incidence, and differences between groups were assessed using the log-rank test. Multivariable Cox proportional hazards regression models were used to calculate adjusted hazard ratios (HR) and 95% confidence intervals (CI) for mortality, adjusting for clinically and statistically significant variables identified in univariable analysis, including age, sex, diabetes, hypertension, coronary artery disease, chronic kidney disease, mean aortic gradient, aortic valve area, and the type of implanted valve.
Results: The study included 109 patients, with 35 in the low TAPSE/SPAP ratio group, 37 in the middle group, and 37 in the high group. Total all-cause mortality was 38 (35%) during a median follow-up of 17 (9-27) months. Significant differences were observed in the prevalence of atrial fibrillation (P=0.003) and all-cause mortality rates (P<0.001) across the groups. Other demographic and baseline characteristics are presented in Table 1. The Kaplan-Meier analysis revealed a notably lower cumulative mortality rate in the high TAPSE/SPAP group compared to the low and middle groups (P<0.001) (Fig. 1). The unadjusted Cox proportional hazards analysis showed that the low TAPSE/SPAP group had a significantly higher risk of mortality (HR=1.087; 95% CI: 0.558-2.115; P=0.806) compared to the high TAPSE/SPAP group (HR=0.141; 95% CI: 0.041-0.486; P=0.002). After adjusting for confounding factors, the high TAPSE/SPAP group remained significantly associated with lower mortality risk (adjusted HR=0.116; 95% CI: 0.025-0.529; P=0.005) (Table 2).
Conclusion: The TAPSE/SPAP ratio is a significant predictor of all-cause mortality in patients undergoing TAVI. Patients with a higher TAPSE/SPAP ratio demonstrated a markedly lower risk of mortality. These findings underscore the potential utility of the TAPSE/SPAP ratio as a prognostic marker in this patient population. Further prospective studies are needed to validate these results and elucidate the underlying mechanisms.
Keywords: TAPSE/SPAP ratio, transcatheter aortic valve implantation, TAVI, all-cause mortality.

Table 1. The demographic and clinical properties of the study population according to tertiles of the TAPSE/PASP ratio.

	Total	TAPSE/SPAP ratio, mmHg			P value
		Low<0.42	Middle (0.42-0.75)	High>0.75	
		n=35	n=37	n=37	
Age, years	78.5 (73-82.2)	78 (73-81)	80 (74.7-80)	77 (71-82.5)	0.376
Sex (female), n (%)	61 (55%)	9 (26%)	8 (21%)	6 (16%)	0.056
Hypertension, n (%)	78 (71%)	20 (57%)	30 (79%)	28 (76%)	0.090
Diabetes mellitus, n (%)	56 (51%)	19 (54%)	20 (53%)	17 (46%)	0.752
CVA, n (%)	7 (6%)	3 (9%)	2 (5%)	2 (5%)	0.810
CAD, n (%)	54 (49%)	19 (54%)	17 (45%)	18 (49%)	0.716
CKD, n (%)	34 (31%)	10 (29%)	14 (37%)	10 (27%)	0.646
PAD, n (%)	8 (7%)	4 (11%)	3 (8%)	1 (3%)	0.356
AF, n (%)	28 (25%)	9 (26%)	16 (42%)	3 (8%)	0.003
Smoking, n (%)	23 (21%)	9 (26%)	8 (21%)	6 (%16)	0.612
BMI, kg/m^2	27.8 (24.5-33)	27.3 (22-35)	28 (24.7-31.3)	27.8 (25.1-33)	0.811
SBP (mmHg)	124 (114-135)	120 (110-130)	127 (111-135)	130 (10-140)	0.142
DBP (mmHg)	70 (63-75)	70 (61-74)	70 (61-75)	73 (67-77)	0.181
Type of implated valve					0.996
SE+annuler	42 (38%)	13 (37%)	15 (39%)	14 (38%)	
SE+supra-annuler	52 (47%)	17 (49%)	17 (45%)	18 (48%)	
BE	16 (15%)	5 (14%)	6 (16%)	5 (14%)	
LVEF, %	55 (50-60)	55 (35-60)	57 (50-60)	60 (50-60)	0.074
Peak aortic velocity, m/s	4.3 (4.1-4.5)	4.1 (3.9-4.4)	4.3 (4.1-4.6)	4.3 (4.1-4.7)	0.045
Mean aortic velocity, mmHg	44 (41-50)	42 (40-46)	43 (41-54)	45 (42-54)	0.024
Aortic valve area, cm^2	0.7 (0.56-0.8)	0.74 (0.6-0.83)	0.65 (0.53-0.77)	0.71 (0.54-0.79)	0.254
TAPSE, mm	20 (17-24)	19 (16-21)	19 (17-22)	24 (20-25)	<0.001
SPAP, mmHg	36 (25-50)	56 (47-70)	37 (32-40)	22 (20-26)	<0.001
eGFR, ml/min/1.73 m^2	65 (48-73)	60 (47-71)	65 (47-73)	70 (50-79)	0.369
CRP, mg/dL	5 (2-8)	5 (2-10)	5 (2-8)	3 (1-8)	0.448
LDL-C, mg/dL	97 (71-127)	92 (70-126)	102 (76-125)	97 (66-131)	0.870
HDL-C, mg/dL	42 (34-50)	38 (33-50)	42 (33-54)	44 (36-50)	0.532
Triglyceride, mg/dL	104 (82-160)	96 (72-148)	115 (84-164)	107 (82-172)	0.351
Diuretics, n (%)	16 (15%)	5 (14%)	6 (16%)	5 (14%)	0.960
ACE-i/ ARB, n (%)	68 (62%)	18 (51%)	28 (74%)	22 (59%)	0.138
B-blocker, n (%)	32 (29%)	11 (31%)	11 (29%)	10 (27%)	0.919
All cause mortality, n (%)	38 (%35)	19 (%54)	16 (%42)	3 (%8)	<0.001
Follow up time, months	17 (9-24)	17 (6-18)	15.5 (6-23)	20 (16-29)	0.020

ACE-I=angiotensin converting enzyme inhibitor, AF=atrial fibrillation, ARB=angiotensin II receptor blocker, BE=ballon- expandable, BMI=body mass index, CAD=coronary artery disease, CKD=chronic kidney disease, CRP=C- reactive protein, CVE=cerebrovascular events, DBP=diastolic blood pressure, DM=diabetes mellitus, eGFR=estimated glomerular filtration rate, HDL-C=high density lipoprotein cholesterol, HT=hypertension, LVEF=left ventricular ejection fraction, LDL-C=low density lipoprotein cholesterol, PAD=perıpheral arterıal dısease, SBP=systolic blood pressure, SE=self- expandable, SPAP=systolic pulmonary artery pressure, TAPSE=tricuspid annular plan systolic excursion.

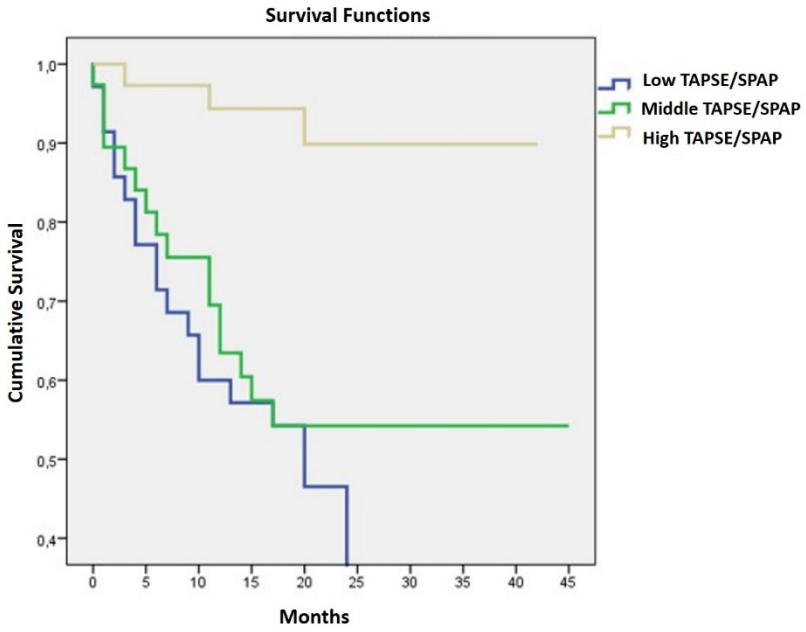

Fig. 1. Kaplan-Meier curves depicting mortality incidence. High TAPSE/SPAP group exhibited a notably lower cumulative mortality rate compared to the other two groups.

Table 2. Cox proportional hazards analysis for all cause mortality

Group	Unadjusted				Adjusted *			
	P value	HR	95.0% CI for HR		P value	HR	95.0% CI for HR	
			Lower	Upper			Lower	Upper
I	Reference				Reference			
II	0.806	1.087	0.558	2.115	0.349	1.455	0.664	3.188
III	0.002	0.141	0.041	0.486	0.005	0.116	0.025	0.529

*Models were adjusted for age, sex, diabetes, hypertension, coronary artery disease, chronic kidney disease, mean aortic gradient, aortic valve area, and the type of implated valve. CI=confidence interval, HR=hazard ratio.

OP-06.
Single Center Experience in Percutaneous Patent Foramen Ovale Closure: Short Term Clinical Outcomes

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Background: Patent foramen ovale (PFO) is associated with cryptogenic stroke, migraine, platypnea-orthodeoxia syndrome, and decompression sickness in divers. With the positive results of randomized controlled trials conducted in the last few decades, percutaneous PFO closure has been approved for appropriate indications. We aimed to investigate the short-term results of PFO closure procedures performed in our hospital.
Methods: Eleven consecutive patients who were planned to undergo percutaneous PFO closure were included in our study. These patients were indicated for PFO closure by a multidisciplinary team including cardiology and neurology clinics. Procedure indications, devices used, procedure complications, and major adverse events (death, stroke, transient ischemic attack, migraine, myocardial infarction, intracranial hemorrhage) during the follow-up period were recorded and analyzed.
Results: The procedure was performed successfully and without complications, except for one of the 11 patients included in the study (procedural success rate 90.9%). Age distribution of the patients was: 40-65 years. AF did not develop in any patient after the procedure. No major adverse events were observed in the patients during the follow-up period (median: 5.5 months).
Conclusion: If percutaneous PFO closure can be performed with the right indication, with the right device and by experienced hands, it is very successful and safe in the short term. Detailed evaluation by a multidisciplinary team in patients with cryptogenic stroke is very important for the clinical outcome of these patients.
Keywords: Patent foramen ovale, percutaneous closure, outcomes

Table 1. Baseline characteristics of patients who underwent percutaneous PFO closure.

Variable	All Patients (n=10)
Age, mean±SD	48.9±8.5
Female ratio, n (%)	7(70)
Diabetes, n (%)	0(0)
Hypertension, n (%)	4(40)
Active smoking, n (%)	5(50)
Migraine, n (%)	4(40)
Prior stroke	8(80)
Stroke counts in patients with prior CVE, median (24-75th)	1(1-2), n=8
Resistant migraine, n (%)	1(10)
Recurrent TIA, n (%)	1(10)
Creatinine clearance, median (24-75th)	106.1(89.3-109.5)
LDL, mg/dL, mean±SD	105.7±31
Amplatzer PFO occluder, n (%)	7(70)
MemoPart PFO occluder, n (%)	
Dual antiplatelet for 6 months, n (%)	10(100)
Follow up time(month), median (24-75th)	5.5(2-16.2)
Post procedural atrial fibrillation, n (%)	0(0)
Post procedural stroke	0(0)
Post procedural TIA	0(0)

CVE=Cardiovascular event, OLDL=Low density lipoprotein, SD=Standart deviation, TIA=Transient ichemic attack.

OP-07.
Shock Index as a Prognostic Indicator in Acute Coronary Syndrome

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Introduction: The Shock Index (SI) has been established as an early prognostic marker in various critical conditions, including Acute Coronary Syndrome (ACS). Its simplicity makes it especially valuable in emergency settings, where timely clinical decisions are crucial. The aim of this study was to evaluate the characteristics of patients with low and high SI in ACS, and to assess whether the SI can effectively identify high-risk patients and predict adverse intrahospital outcomes.
Methods: This retrospective study included 117 ACS patients (87 with NSTEMI, 30 with STEMI), excluding those with advanced renal insufficiency. The Shock Index (SI) was calculated as the ratio of heart rate (HR) to systolic blood pressure (SBP), with a cut-off value of 0.7 to categorize patients into two groups: SI<0.7 and SI>0.7. Clinical characteristics, comorbidities, laboratory values, treatment strategies, and clinical outcomes were compared between the two groups.
Results: There were no significant differences in terms of age, gender, or comorbidities between the groups. NSTEMI was more prevalent in the SI<0.7 group (60.9% vs. 23.3%, P<0.001). Among STEMI patients, anterior infarctions were more common in the SI>0.7 group (P<0.001). Vital parameters (blood pressure, heart rate, oxygen saturation) were significantly impaired in the SI>0.7 group (P<0.001), along with elevated troponin levels (P=0.02), while no significant differences were observed in other laboratory parameters. Treatment strategies (PCI, conservative management, CABG) did not differ significantly. Overall complication rate was low across the entire study cohort, with a slightly higher frequency in the SI > 0.7 group, though without statistical relevance. The proportion of patients with EF<30% was higher in the SI>0.7 group (P=0.014). A significant negative correlation was found between SI and ejection fraction (EF) (r= -0.53, P<0.001). Logistic regression analysis revealed that SI was a significant predictor of EF <30% (OR=1.94, 95% CI=1.54-24.4, P=0.022), with an AUC of 0.822.
Conclusions: Our results demonstrate that the Shock Index (SI) is a reliable tool for identifying critically ill patients in both STEMI and NSTEMI populations. Patients with a higher SI were more frequently in a clinically critical condition and prone to complications. This trend is consistent with the literature. Notably, patients with a higher SI had a significantly higher risk of developing heart failure, with a strong correlation to reduced ejection fraction (EF). These findings align with studies identifying SI as a marker for cardiovascular instability, impaired left ventricular function, and an increased risk of heart failure. In conclusion, our study highlights the Shock Index as an important tool for early risk stratification in ACS patients. An elevated SI helps identify those at greater risk for developing heart failure, allowing for more timely interventions. Incorporating the SI into clinical practice could enhance risk assessment and improve management of critically ill patients.
Keywords: Shock index, acute coronary syndrome, STEMI, NSTEMI

OP-08.
Ferritin-to-CRP Ratio: A New Indicator for Mortality Risk in Heart Failure With Reduced Ejection Fraction

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Background: Heart failure (HF) is a prevalent condition with significant morbidity and mortality. Predicting outcomes in HF, particularly for patients with reduced ejection fraction (EF), is crucial for effective management. Iron metabolism and inflammation are emerging as important factors in HF prognosis. Ferritin and C-reactive protein (CRP) are commonly used biomarkers, but their combined ratio (FER/CRP) might offer additional insights into patient outcomes. This study investigates the prognostic value of the FER/CRP ratio for predicting mortality in HF patients with EF<50%, who were admitted either as outpatients or inpatients.
Methods: This retrospective study encompassed patients diagnosed with heart failure and an ejection fraction (EF) of less than 50%, who were admitted to our hospital either as outpatients or inpatients from September 2018 to September 2023. The study specifically included patients for whom serum iron parameters were measured.
Results: A total of 275 patients were included in the analysis. The median follow-up time was 33 months. The analysis categorized patients into two groups: those who experienced mortality (n=102) and those who survived (n=173). Significant differences between these groups were observed in several key variables. Patients who died had a significantly lower median ejection fraction (30) compared to survivors (38), with a P-value of <0.001. Mortality was also associated with higher rates of smoking (P=0.009) and a higher prevalence of coronary artery heart disease (P=0.007). Additionally, deceased patients had lower hemoglobin levels (11.7 g/dL vs. 12.2 g/dL, P=0.011), higher C-reactive protein levels (7.37 mg/dL vs. 5.85 mg/dL, P=0.018), and lower albumin levels (37.9 g/L vs. 40.3 g/L, P<0.001). The CONUT score, indicating nutritional status, was higher in the deceased group (2.39 vs. 1.74, P<0.001). Furthermore, the CRP/albumin ratio was elevated in those who died (0.20 vs. 0.16, P=0.026), while the ferritin/CRP ratio was lower (14.5 vs. 20.4, P=0.004). No significant differences were found in age, sex, hypertension, diabetes mellitus, or intravenous iron therapy between the two groups. Linear regression analysis was performed on the statistically significant laboratory parameters and inflammation scores. This analysis determined that albumin (P<0.001) and the ferritin-to-CRP ratio (P=0.026) are independent risk factors for mortality. Table 1
Conclusions: Existing literature highlights that inflammatory and nutritional markers, such as CONUT and the CRP-to-albumin ratio, predict mortality in heart failure patients. Although intravenous iron replacement has shown no mortality benefit, chronic inflammation in these patients elevates ferritin levels, setting a higher ferritin cutoff. The ferritin-to-CRP ratio has been suggested to effectively identify iron deficiency amid chronic inflammation. Our study indicates that, in patients with EF <50% and evaluated serum iron parameters, the ferritin-to-CRP ratio and albumin are independent mortality predictors. Future research should investigate the ferritin-to-CRP ratio's role in detecting absolute iron deficiency and its prognostic value in heart failure.
Keywords: Ferritin-to-CRP ratio, heart failure, reduced ejection fraction

Table 1. **Baseline characteristics of deceased and surviving heart failure patients**

	Deceased (n=102)	Survived (n=173)	P value
Age (years) (mean±SD)	67.5(+12.1)	65.3(+12.3)	0.074
Gender, n (%)			0.257
Female	32 (31.4)	66 (38.2)	
Male	70 (68.6)	107 (61.8)	
Hypertension, n (%)	79 (77.5)	132 (76.3)	0.414
Diabetes mellitus, n (%)	57(55.9)	87 (50.3)	0.186
Coronary artery disease, n (%)	81 (79.4)	114 (65.8)	**0.007**
Active smoking, n (%)	63 (61.8)	79 (45.7)	**0.009**
Ejection fraction (%) (median, IQR)	30 (25-38)	38 (28-44)	**<0.001**
Intravenous iron therapy (n, %)	12 (11.8)	22 (12.7)	0.409
ACE inhibitors/ARBs/ARNI, n (%)	74 (72.5)	140 (80.9)	0.120
Beta-blockers, n (%)	94 (92.2)	154 (89)	0.2
Mineralocorticoid receptor antagonists, n (%)	54 (52.9)	90 (52)	0.442
NYHA Classification, n (%)			**<0.001**
1	1 (1,0)	50 (28.9)	
2	19 (18.6)	68 (38.7)	
3	51 (50)	46 (26.6)	
4	31 (30.4)	10 (5.8)	
Hemoglobin (g/dL)	11.7	12.2	**0.011**
Ferritin (ng/dL)	83.5	74.5	0.158
Ferritin (ng/dL), n (%)			0.168
<100	74 (72.5)	135 (78)	
Transferrin saturation <20 and 100-299	16 (15.7)	21 (12.1)	
Transferrin saturation >20 and 100-299	8 (7.8)	13 (7.5)	
≥300	4 (3.9)	4 (2.3)	
CRP (mg/dL)	7.37	5.85	**0.018**
Albumin (g/L)	37.9	40.3	**<0,001**
CONUT score	2.39	1.74	**<0.001**
CRP/Albumin ratio	0.20	0.16	**0.026**
Ferritin/CRP ratio	14.5	20.4	**0.004**

OP-09.
Impact of the SYNTAX Score on Mortality in Older Patients Treated With Percutaneous Coronary Intervention

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Background: Elderly patients (≥75 years) with coronary artery disease (CAD) became an increasing patient population in real life practice. Treatment strategies are hot topics in elderly patients with CAD. Recently, randomized SENIOR-RITA trial demonstrated that routine invasive therapy did not reduce cardiovascular death or myocardial infarction compared with conservative therapy [1]. In our study, we aimed to determine the role of SYNTAX score in elderly patients undergoing percutaneous coronary intervention (PCI).
Methods: A total of 236 elderly patients undergoing PCI between January 2017 and April 2023 from our university hospital enrolled to the analysis. Eighty-two non-ST segment elevation myocardial infarction patients, 90 ST segment elevation myocardial infarction patients, 12 unstable angina patients and 52 chronic coronary syndromes patients were enrolled. Clinical, procedural and follow-up data was obtained from the medical records. The SYNTAX score (SS) was calculated due to the web site calculator and patients were divided into 2 groups: Low SS group (<22) and high SS group (≥22).
Results: Out of 236 elderly patients; 193 patients were assigned to the low SS group and 43 patients were assigned to the high SS group. There was no significant difference between 2 groups in terms of diabetes, hypertension, dyslipidaemia, CAD and clinical presentation. Mean SYNTAX score was 12 in low SS group and 28 in high SS group. While median baseline PRO-BNP level was 2213 pg/mL in low SS group, it was 3521pg/mL in the high SS group (P=0.032). Median troponin value was higher in high SS group (0.79 vs. 3). Ejection fraction was significantly decreased in high SS group (46.7 vs. 40.7, P=0.006). SYNTAX score was not associated with early (P=0.498) and late (P=0.082) mortality (median: 39 months). Age, ejection fraction, TIMI flow, postdilatation, PRO-BNP, Killip class and in hospital cardiac arrest were independent predictors on late mortality.
Conclusion: Our study showed that higher SS SYNTAX score was associated with higher PRO-BNP and troponin level and lower ejection fraction in elderly patients undergoing PCI in setting of different clinical scenarios. However, SYNTAX score was not associated with early and late mortality at follow-up.
Keywords: SYNTAX score, percutaneous coronary intervention, mortality, older patients

Reference

1. Kunadian V, Mossop H, Shields C, et al. Invasive Treatment Strategy for Older Patients with Myocardial Infarction. N Engl J Med. 2024;391(18):1673-1684. doi: 10.1056/NEJMoa2407791.

OP-10.
Unusual Complication of Prostate Cancer Case Report; Moustache Type Intracardiac Thrombus

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Introduction: Hypercoagulopathy and thrombus formation are leading cause of death in cancer. Although this risk is not so high in prostate cancer, it must be keep in mind. In this case report we presented 68-year old man with thrombus that seen both in right and left atrium passing through patent foramen ovale (PFO).
Case Report: 68-year old patient reffered to our clinic from neurology service with a history of transient ischemic attack (TIA). This neurological disease was demonstrated with sypmtoms and brain diffusion magnetic resonance (MR). From patients anamnesis we have learned that he had fatigue, weight loss and some urological problems, begining from few months earlier. When we performed transthoracic echocariography (TTE) to our patient, it demonstrates moustache type intracardiac thrombus, being both in right atrium (RA) and left atrium (LA) passing through PFO (Figs. 1 and 2). At this point we have found the ethiology of TIA. Due to its right chamber origin, we took D-dimer and also pulmonary artrey computed scan (CT) to exclude pulmonary embolism. As we know, these thromboembolic particules mostly come from deep veins so we performed lower extremity venous Doppler ultrasonography. Both pulmonary embolism and deep vein thrombosis were excluded. For this hypercoagulopathic situation, we have started low-molecular weight heparin (LMWH, enoxaparin, S.C.) after urgent cardiovascular surgery expert consultation and also reffered to hematologist and urologist. After these consultations, it's found that underlying disease of this patient was prostate cancer. He is still taking his medication from urology department and also taking LMWH. After 7 days of thrombus demonstration, we perform TTE again and see the cardiac chambers clear (Fig. 3).
Conclusion: Venous thromboembolism (VTE) and cancer are two frequently entangled pathologies. The existence of active cancer in a patient is a known risk factor for VTE. Although VTE in prostate cancer mostly seen in patients that on endocrine therapy or undergoing prostatectomy (up to 60%), active cancer have an increased risk of thromboembolic disease by itself. So it's keep in mind that, patients reffered from neurology with ischemic history and patients with cancer suspicion have to be evaluated with TTE.
Keywords: Prostate cancer, venous thromboembolism, thrombus

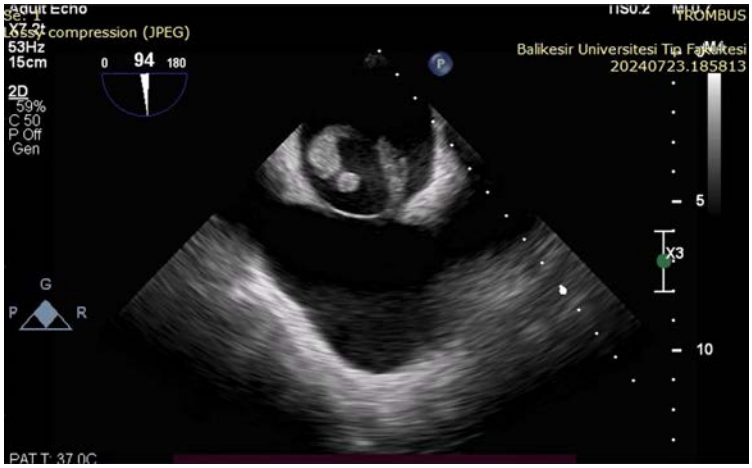

Fig. 1. **Thrombus in LA, passing through PFO.**

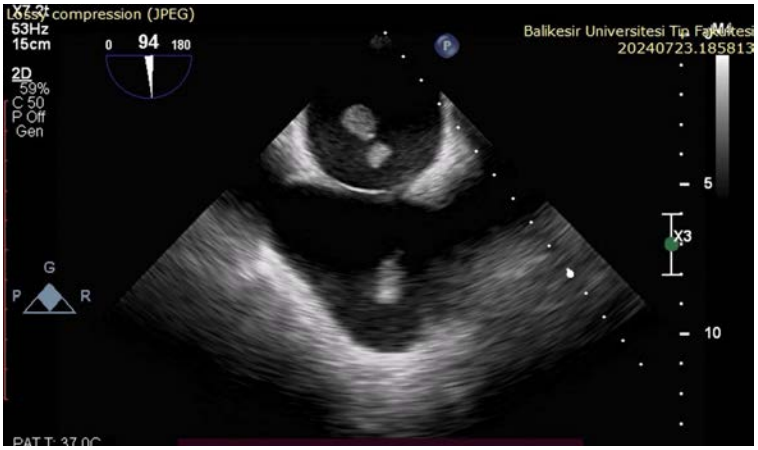

Fig. 2. **Thrombus in RA and LA.**

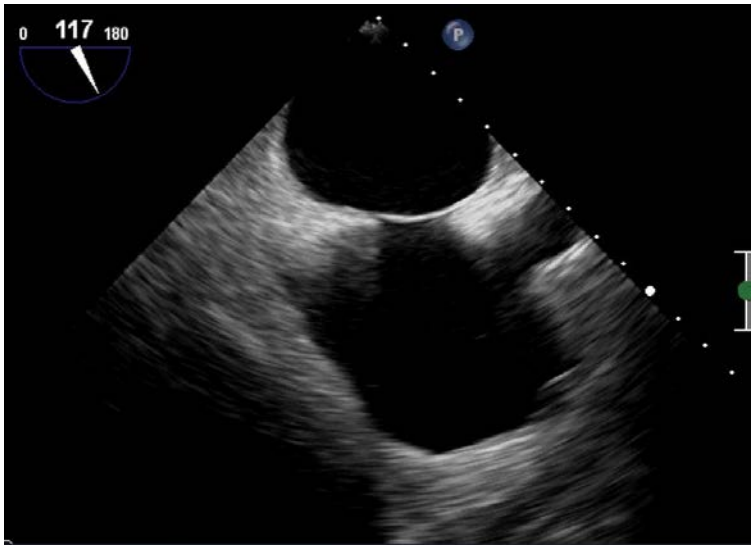

Fig. 3. **Clear cardiac chambers.**

OP-11.
Diagnostic Performance of the Visceral Adiposity Index in Predicting High Cardiovascular Risk Among Postmenopausal Women Without Atherosclerotic Cardiovascular Disease

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Background: There is increasing evidence of elevated cardiovascular risk among postmenopausal women. The menopausal transition is associated with detrimental changes in lipid profiles, a tendency toward weight gain, and heightened susceptibility to metabolic syndrome. These factors are linked to increased epicardial and paracardial fat deposition. The Visceral Adiposity Index (VAI) is a gender-specific formula that integrates such risk factors. Multiple studies have suggested that VAI may serve as a prognostic indicator of cardiovascular diseases. However, its predictive role in estimating cardiovascular risk among postmenopausal women without atherosclerotic cardiovascular disease (ASCVD) has not yet been thoroughly evaluated. This study aimed to investigate the predictive value of VAI for identifying high cardiovascular risk in postmenopausal women without ASCVD or other major comorbidities.

Methods: This retrospective study included 72 postmenopausal women, aged 45-55 years, who visited the Cardiology Clinic for routine check-ups between August 2024 and October 2024. Participants had no confirmed ASCVD or other chronic conditions. Anthropometric measurements (waist circumference, body mass index), complete blood count and biochemical parameters were obtained. VAI was calculated using the formula: [waist circumference/(36.58+(1.88×BMI))]×(triglyceride/0.81) (1.52/high-density lipoprotein cholesterol). Cardiovascular risk was assessed with the ASCVD risk score. Logistic regression models were built to identify independent predictors, and receiver operating characteristic (ROC) curve analysis determined the optimal VAI cut-off.

Results: High cardiovascular risk was observed in 20.8% (n=15) of postmenopausal women who had no ASCVD or other chronic illnesses. Women with high ASCVD risk were older (51.7±2.6 vs. 49.1±2.9 years, P=0.003), had higher BMI (29.8±7.1 vs. 26.3±4.2 kg/m², P=0.016), and significantly elevated blood pressures (P<0.001) compared to the low-risk group. The median triglycerides (134.0 vs. 95.0 mg/dL, P=0.017), and VAI (4.4 vs. 2.8, P<0.001) were higher in postmenopausal women with high ASCVD risk group compared to those with low-risk group (Table 1). In multivariable analysis, diastolic blood pressure (OR=1.12, 95% CI=1.03-1.21, P=0.009) and VAI (OR=2.02, 95% CI=1.31–3.13, P=0.002) independently predicted high ASCVD risk group (Table 2). For predicting high ASCVD risk, ROC curve analysis revealed a VAI cut-off ≥3.5 with 93.3% sensitivity and 57.9% specificity (AUC=0.80, 95% CI=0.66-0.94) (Fig. 1).

Conclusion: This study underscores the importance of incorporating VAI as part of routine cardiovascular risk assessment in postmenopausal women, particularly for those without overt ASCVD or other comorbidities. Postmenopausal women with higher VAI values exhibited a significantly elevated risk, emphasizing the role of visceral fat distribution in cardiovascular health during this stage of life. Future studies with larger, more diverse populations and longitudinal designs are needed to validate these findings and explore the potential of

VAI in guiding preventive strategies for cardiovascular health in postmenopausal women.
Keywords: Visceral adiposity index, postmenopausal women, cardiovascular risk, prognostic indicator

Table 1. Demographic and clinical findings associated with high cardiovascular risk in postmenopausal women without atherosclerotic cardiovascular disease

Variables	All population (n=72)	ASCVD risk		P value
		Low (n=57)	High (n=15)	
Age, years	49.7 ± 3.0	49.1 ± 2.9	51.7 ± 2.6	**0.003***
BMI, kg/m²	27.1 ± 5.1	26.3 ± 4.2	29.8 ± 7.1	**0.016***
Waist circumference, cm	85.4 ± 4.5	84.5 ± 4.5	88.7 ± 4.6	**<0.001***
Smoking, n (%)	46 (63.9)	31 (54.4)	11 (73.3)	**0.031***
SBP, mmHg	117.4 ± 14.1	113.4 ± 12.5	132.7 ± 8.2	**<0.001***
DBP, mmHg	75.1 ± 10.5	73.2 ± 9.7	82.7 ± 10.3	**0.001***
Total cholestrol, mg/dL	215.6 ± 35.5	210.2 ± 36.1	236.2 ± 24.9	**0.011***
HDL, mg/dL	56.0 ± 14.3	58.7 ± 14.3	45.5 ± 8.3	**0.001***
LDL, mg/dL	130.8 ± 29.2	129.3 ± 28.2	136.7 ± 32.9	0.387
Triglycerides, mg/dL	97.0 (74.0-135.2)	95.0 (61.0-131.0)	134.0 (97.0-196.0)	**0.017***
VAI	3.5 (2.1-4.5)	2.8 (1.8-4.2)	4.4 (3.7-7.4)	**<0.001***
Glucose, mg/dL	92.9 ± 8.8	93.2 ± 9.1	91.7 ± 7.6	0.548
Leukocytes, 10³/µL	6.2 ± 1.4	6.2 ± 1.4	6.5 ± 1.4	0.391
RBS, 10⁶/µL	4.6 ± 0.3	4.6 ± 0.3	4.6 ± 0.4	0.923
Hemoglobin, g/dL	13.2 ± 1.4	13.1 ± 1.4	13.5 ± 1.3	0.267
Hematocrit, %	39.9 ± 3.6	39.6 ± 3.6	40.7 ± 3.7	0.319
Platelet, 10³/µL	268.0 ± 52.3	268.7 ± 52.3	265.3 ± 54.4	0.823
Neutrophil, 10³/µL	3.6 ± 1.1	3.5 ± 1.1	3.9 ± 1.0	0.275
Lymphocyte, 10³/µL	2.1 ± 0.5	2.1 ± 0.5	2.1 ± 0.5	0.748
Monocyte, 10³/µL	0.4 ± 0.1	0.4 ± 0.1	0.4 ± 0.1	0.854
TSH, µIU/mL	2.5 (1.6-3.0)	2.5 (1.6-3.0)	2.4 (1.7-3.1)	0.972
Creatinine, mg/dL	0.7 ± 0.1	0.7 ± 0.1	0.7 ± 0.1	0.718
eGFR, mL/min/1.73 m2	98.4 ± 9.6	98.8 ± 9.7	96.7 ± 9.2	0.451
AST, U/L	17.3 ± 5.3	17.0 ± 5.5	18.2 ± 4.5	0.449
ALT, U/L	14.0 (11.0-19.0)	13.0 (11.0-19.0)	14.0 (13.0-19.5)	0.341
Albumin, g/L	4.4 ± 0.2	4.4 ± 0.2	4.3 ± 0.2	0.586
Vitamin B12, pg/mL	397.3 ± 128.8	409.9 ± 124.6	349.7 ± 137.7	0.108
Vitamin D, ng/mL	28.3 (20.6-35.4)	29.5 (23.4-35.7)	20.9 (15.7-28.2)	**0.009***
Insufficiency, n (%)	18 (25.0)	11 (19.3)	7 (46.7)	**0.029***
Folic acid, ng/mL	10.5 ± 3.9	10.6 ± 4.0	10.3 ± 3.8	0.789

Data are shown as mean±standard deviation or median (IQR), or number (%). ALT=alanine aminotransferase, AST=aspartate aminotransferase, BMI=body mass index, DBP=diastolic blood pressure, FBG=fasting blood glucose, eGFR=estimated glomerular filtration rate, HDL-C=high-density lipoprotein cholesterol, LDL=low-density lipoprotein cholesterol, SBP=systolic blood pressure, TSH=thyroid-stimulating hormone, VAI=visceral adiposity index

*P<0.05 indicates statistical significance.

Table 2. Independent predictors of high cardiovascular risk in postmenopausal women without atherosclerotic cardiovascular disease.

Variables	Univariable			Multivariable		
	OR	95% CI	P value	OR	95% CI	P value
DBP	1.11	1.03 - 1.19	**0.005**	1.12	1.03 - 1.21	**0.009**
Vitamin D	3.66	1.09 - 12.26	**0.035**	-	-	-
VAI	1.98	1.33 - 2.95	**0.001**	2.02	1.31 - 3.13	**0.002**
					Nagelkerke R^2= 0.47	

*P<0.05 indicates statistical significance. CI=confidence interval, DBP=diastolic blood pressure, OR=odds ratio, VAI=visceral adiposity index

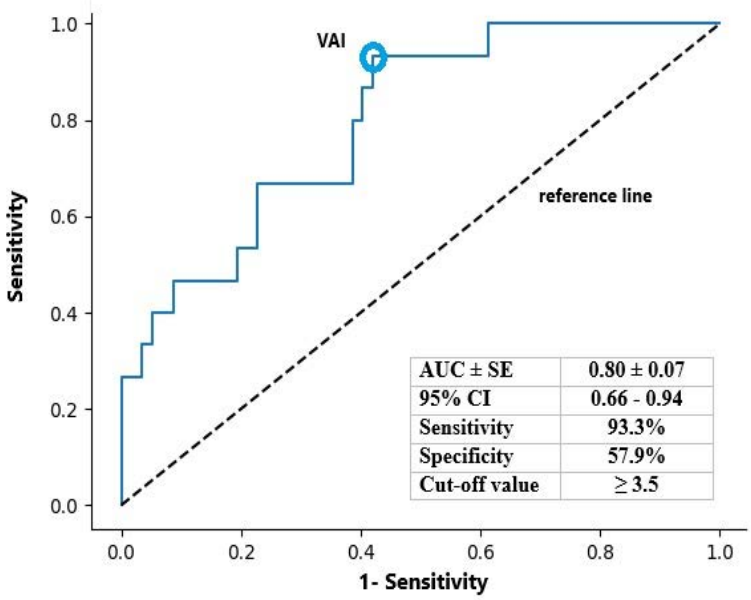

Fig. 1. Diagnostic performance of visceral adiposity index (VAI) in predicting high cardiovascular risk in postmenopausal women without atherosclerotic cardiovascular disease. AUC=area under the curve, CI=confidence interval, SE=standard error, VAI=visceral adiposity index.

OP-12.
Relationship Between Chronic Renal Failure and Cardiac Electrophysiological Balance Index

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Introduction: The Index of Cardiac Electrophysiological Balance (ICEB/ICEBc) is an index derived from the QT/QRS ratio obtained from a 12-lead ECG. Previous studies have shown that ICEB is a predictor of malignant arrhythmias. The parameters affecting ICEB have been well documented, but studies on the effect of chronic kidney disease (CKD) on ICEB remain controversial. The aim of this study was to investigate the effect of CKD on ICEBc.
Methods: Our study included 429 patients who presented to cardiology clinics. Patients were divided into two groups based on their estimated glomerular filtration rate (eGFR). Patients with eGFR<30 (stage 4 and 5 CKD patients) were included in the first group, while patients with eGFR≥30 were included in the second group. Since there is no accepted cut-off value for ICEBc in the literature, we categorized ICEBc values as high or low based on the median ICEBc value of our study population (median ICEBc=5.01).
Results: The mean age of our study population was 62.6±15 years, and the proportion of women was greater than men (53.7%). Parameters with a P-value<0.1 in univariate logistic regression analysis were included in multivariate logistic regression analyses. In univariate analyses, gender, CKD, left ventricular ejection fraction, albumin and hemoglobin levels, and coronary artery disease were included in multivariate logistic regression analyses. When all parameters predicting ICEBc were evaluated together, only gender (OR=1.770, 95% CI=1.132-2.767, P=0.012) was identified as an independent predictor.
Conclusions: Our study found an association between stage 4 and stage 5 CKD and ICEBc. Although CKD was not an independent predictor for ICEBc, it should be noted that stage 4 and stage 5 CKD may prolong ICEBc, potentially increasing the risk of malignant arrhythmias.
Keywords: Index of cardiac electrophysiological balance, chronic kidney disease, malignant arrhythmia

OP-13.
Can TCBI Predict Mortality and Length of Hospitalization in Heart Failure Patients?

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Introduction: Heart failure (HF) is a systemic disease with complex neurohormonal, biochemical and inflammatory systems. Patients with heart failure have a poor prognosis with high rates of hospitalization and mortality. Proinflammatory cytokines are chronically elevated in patients with heart failure. These inflammatory cytokines, together with other inflammatory mediators, directly or indirectly elicit many components of HF, including catabolism in protein-based tissues, altered nutrient intake and metabolism, and retention of sodium and water. Monitoring nutritional status and prevention or correction of nutritional deficiency in heart failure patients is of great importance in the prognosis of the disease. Therefore, the nutritional status of patients should be determined and monitored. However, it is very important to choose the correct method to be used in determining nutritional status in these patients. In order to assess the nutritional status of patients, many tools are used in the clinic, including anthropometric or biochemical data as well as subjective parameters. Previous studies have shown that several nutritional indicators, including serum albumin, body mass index (BMI) and cholesterol, are associated with the risk of cardiovascular events. Maruyama et al. found an association between Triglycerides, total Cholesterol, and Body weight Index (TCBI), a new and simple nutritional index, and major adverse cardiac and cerebrovascular events (MACCE) in patients with coronary artery disease. In our study, we aimed to investigate the relationship between TCBI and mortality and length of hospitalization in patients with heart failure.

Methods: The study included adult patients with systolic heart failure (EF 40% or less) due to any symptom-independent cause and the primary endpoint was all-cause mortality after 1-year follow-up. A total of 101 patients who were followed up in our clinic were included in the study. During the 1-year follow-up, the outcomes of patients who died (21) and those who survived (80) were compared. The length of hospitalization of these patients was noted. Blood parameters of the patients were obtained from the hospital automation system. TCBI was calculated by the formula triglyceride x total cholesterol x body weight (kg)/1000.

Results: When the TCBI values of both groups were compared, the TCBI value was found to be significantly lower in the group that died compared to the group that survived (P=0.033). There was also a significant correlation between the duration of hospitalization and TCBI (P<0.005).

Conclusions: Nutrition is very important in heart failure, which is the leading cause of death worldwide. Malnutrition and cachexia are seen at high rates and can lead to mortality. Nutritional index has been studied in many previous studies and compared with mortality. We believe that TCBI may help clinicians in predicting mortality and length of hospitalization since the results were significant. Another advantage is that it can be easily calculated in daily practice.

Keywords: Heart failure, hospitalization, mortality, TCBI

OP-14.
Retrograde Transmitral Paravalvular Leak Closure Through an Antegrade Transseptal Approach

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Introduction: Mitral paravalvular leak is a rare but serious complication after mitral valve replacement. Although most paravalvular leaks are small and asymptomatic, more than 10% of patients may develop large and symptomatic paravalvular leaks. Most of the small leaks that develop after surgery close spontaneously and most do not require treatment. However, when regurgitation is severe, congestive heart failure may occur and haemolysis may occur due to the high-velocity jet associated with the paravalvular leak, even if the defect is small. The most symptomatic leaks are associated with the mitral valves and less frequently with the aortic valves. Reoperation of such leaks is associated with significant morbidity and mortality, and the likelihood of recurrent leaks after reoperation is high.
Case Report: We report a case of a 65-year-old woman with moderate paravalvular mitral regurgitation successfully closed by a 3D TEE-guided retrograde transfemoral arterial approach. A 65-year-old woman presented to the cardiology outpatient clinic with complaints of fatigue and exertional dyspnoea. The patient had a history of MVR+ Triscuspid Ring operation in 2012. AF rhythm was present on ECG. On cardiac auscultation, a metallic valve sound was heard in the mitral focus. She was admitted to the cardiology department after the echo showed a moderate paravalvular MR and profound anemia was detected in blood tests. After 2 units of ES replacement, TEE was planned for further investigation. TEE revealed a 10 mm paravalvular defect in the 5-6 o'clock direction and moderate paravalvular MR associated with this defect. Percutaneous paravalvular closure was decided in a symptomatic patient with hemolysis findings and profound anemia. Firstly, septostomy was performed by right femoral vein puncture, but since the lesion could not be crossed with hydrophilic wire because of the intra-defect tortuosity, left femoral artery puncture was performed for retrograde wire delivery. The hydrophilic wire was passed into the left atrium via the ventricular route using EBU 3.5 catheter, captured with SNARE in the left atrium and removed from the right femoral vein. The defect was successfully closed with a paravalvular closure device sent through the right femoral vein. The defect was measured with 3D TEE. AVP3 8×4 device was successfully placed in the defect. No paravalvular MR was observed on post-procedure control TEE. No complications were observed and the procedure was terminated.
Conclusion: The mitral paravalvular leak is most commonly traversed antegradely by transseptal puncture of the catheter and rarely retrogradely via the femoral artery. Different techniques have been adopted for mitral PVL closures. The most frequently applied procedures are antegrade cannulation of the defect through a transseptal puncture, retrograde cannulation from the LV through the aortic valve, and retrograde cannulation via transapical access. In the antegrade transseptal and retrograde transfemoral approaches, many operators prefer to create an arteriovenous wire loop. This was the only case in which we could not pass through the antegrade route and needed to pass through the retrograde route because of the intra-defect tortuosity that prevented the passage of the hydrophilic wire.
Keywords: Mitral paravalvular leak, percutaneous paravalvular closure, antegrade transseptal approach

OP-15.
Percutaneous Closure of Challenging Ruptured Sinus Valsalva Aneurysm

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Introduction: Sinus of Valsalva aneurysm (SVA) is a rare condition in the general population, with a prevalence of approximately 0.09%. It may arise from congenital weakness of the elastic connective tissue or abnormal development of the bulbus cordis, but it can also be acquired. Although most cases remain asymptomatic until rupture, the most common site of occurrence is the right coronary sinus (70%). Congenital aneurysms are frequently associated with anomalies such as ventricular septal defect (VSD) (30-60%), aortic regurgitation (20%), and bicuspid aortic valve (10%).

In this report, we present two male patients, aged 21 and 31, who presented with ruptured sinus of Valsalva aneurysm at Etlik City Hospital over a two-year period, along with our treatment strategies.

Case Reports: Case 1. A 21-year-old male patient with a known history of VSD was under routine cardiac follow-up and presented to the cardiology outpatient clinic for a check-up. Cardiac auscultation revealed a moderate-to-severe holosystolic murmur at the aortic focus. Electrocardiography (ECG) showed sinus rhythm with incomplete right bundle branch block (RBBB). Echocardiography (Echo) revealed an aortic sinus diameter of 3.9 cm, left atrial diameter of 4.6 cm, and an ejection fraction (EF) of 65%. A 4 mm subaortic defect with left-to-right shunting was observed on color Doppler, and a pouch formation adjacent to the non-coronary cusp (NCC) of the aortic valve was noted near the superior aspect of the tricuspid septal leaflet. Transesophageal echocardiography (TEE) confirmed an aneurysmal non-coronary sinus with rupture into the right atrium (RA). Cardiac catheterization revealed a QP/QS ratio 2.3 and a pulmonary vascular resistance (PVR) of 2.5 Wood units. The patient was diagnosed with VSD and a ruptured non-coronary sinus aneurysm. Percutaneous closure of the ruptured sinus of Valsalva aneurysm was planned under 3D TEE guidance. (Figs. 1 and 2)

Following right femoral venous and left femoral arterial access, a catheter was advanced from the left femoral artery into the main pulmonary artery (MPA) and through the ruptured non-coronary sinus. A 0.035-inch guidewire was advanced into the superior vena cava (SVC) and snared from the right femoral vein to create an arteriovenous loop. (Fig. 3)

The defect diameter was measured as 1.3 cm on 3D TEE. Initially, a 12 ·10 mm CONAR VSD device was deployed but was found to be undersized and was subsequently retrieved. A 14 mm muscular VSD device was then successfully deployed, achieving complete closure. The procedure was concluded with plans for a separate session to address the VSD. (Figs. 4 and 5)

Case 2. A 31-year-old male patient presented with a 3/6 holodiastolic murmur on auscultation across all foci. TEE revealed an aneurysm of the non-coronary sinus with a 0.7 cm rupture. Cardiac catheterization demonstrated a QP/QS ratio of 2 and a PVR of 1.6 Wood units. Aortography confirmed the rupture of the non-coronary sinus aneurysm. A percutaneous closure procedure was planned following a multidisciplinary council decision. (Figs. 6 and 7)

After right femoral venous and left femoral arterial access, a catheter was advanced from the left femoral artery through the ruptured non-coronary sinus. A 7 mm fistula from the non-coronary sinus to the right atrium was identified. An arteriovenous loop was created, and a 10-8 mm ADO-1 device was successfully deployed to

close the defect without complications. (Fig. 8)

Conclusion: The ruptured sinus of Valsalva aneurysm is an exceedingly rare condition. Over the past two years, two patients presenting to our hospital underwent successful percutaneous closure. Both patients remain under follow-up without complications.

Keywords: Sinus of Valsalva aneurysm, ruptured aneurysm, percutaneous closure

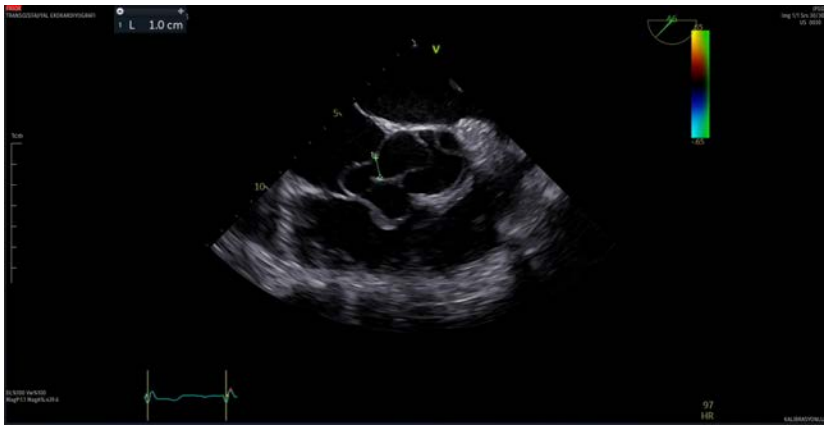

Fig. 1. **Image of ruptured noncoronary sinus aneurysm measured at 10 millimeters on TEE.**

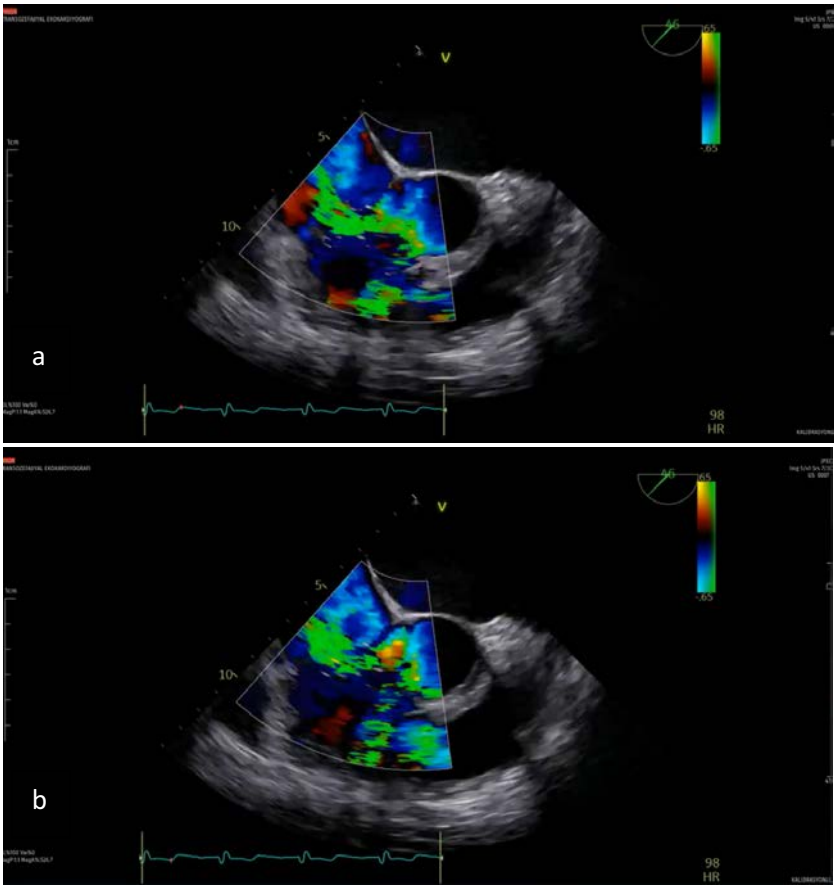

Fig. 2. **(a) Color image of ruptured noncoronary sinus aneurysm and transition to right atrium on TEE, (b) Color image of ruptured noncoronary sinus aneurysm and transition to right atrium on TEE.**

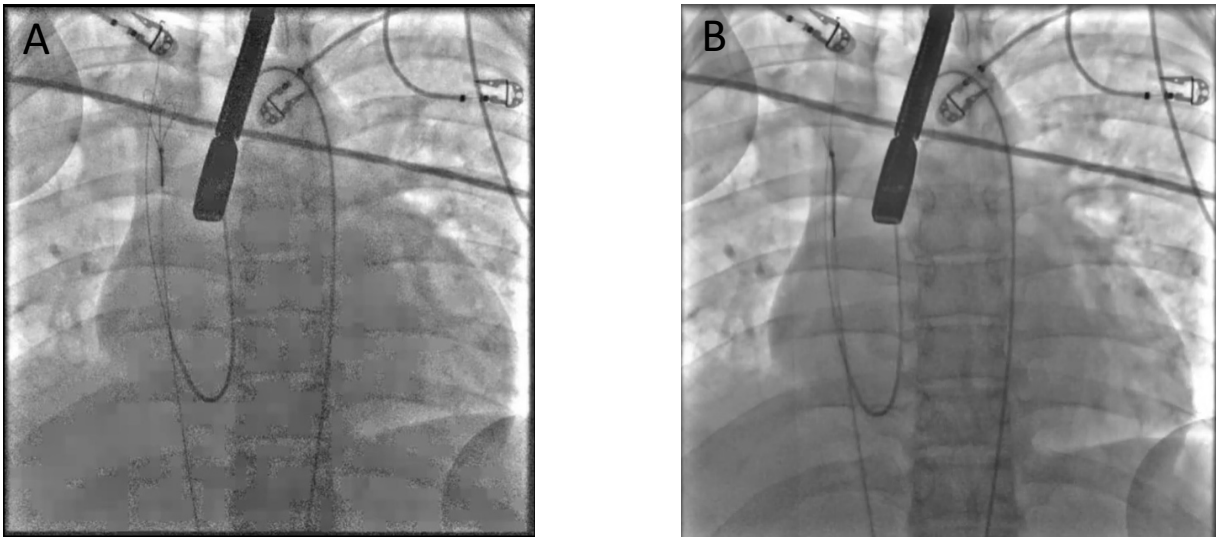

Fig. 3. (A and B) Fluoroscopic image of the arteriovenous loop formed by the wire grasped with snare in the superior vena cava.

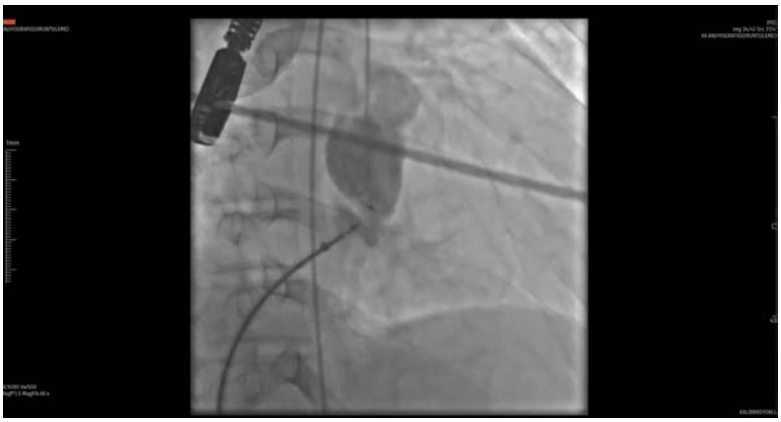

Fig. 4. Fluoroscopic image of the implanted VSD occluder device, showing the management of residual shunting through radiopacity enhancement.

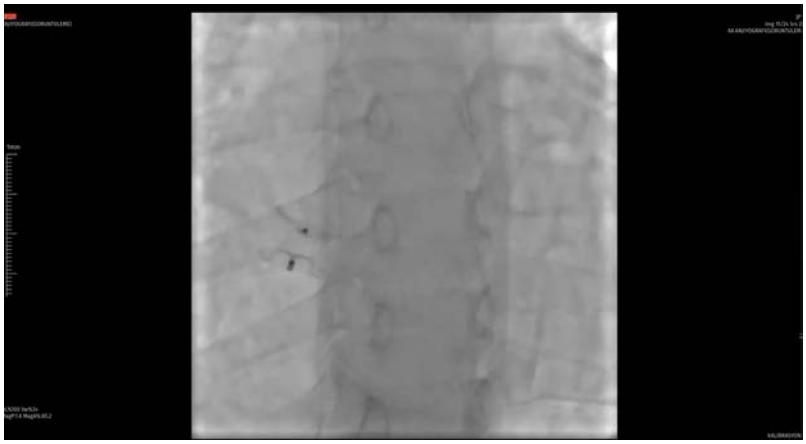

Fig. 5. Fluoroscopic image after placement of the VSD occluder device in the defect.

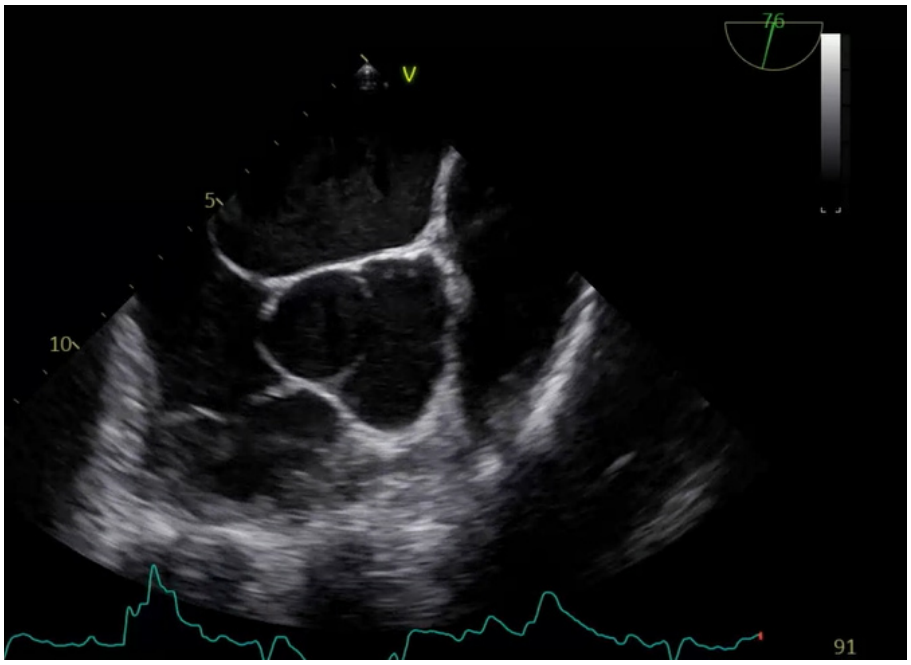

Fig. 6. **Image of ruptured noncoronary sinus aneurysm measured at 7 millimeters on TEE.**

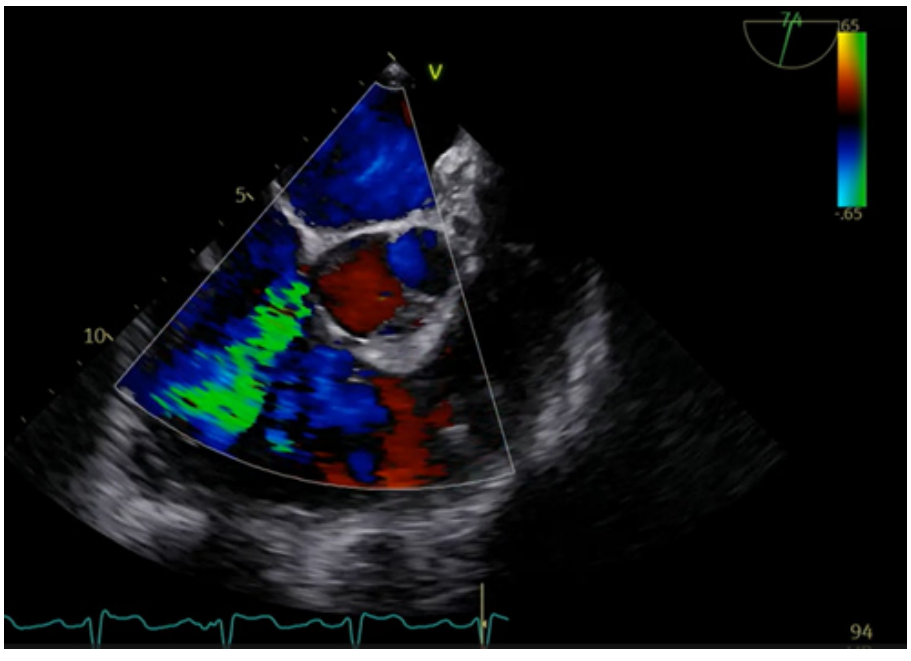

Fig. 7. **Color image of ruptured noncoronary sinus aneurysm on TEE.**

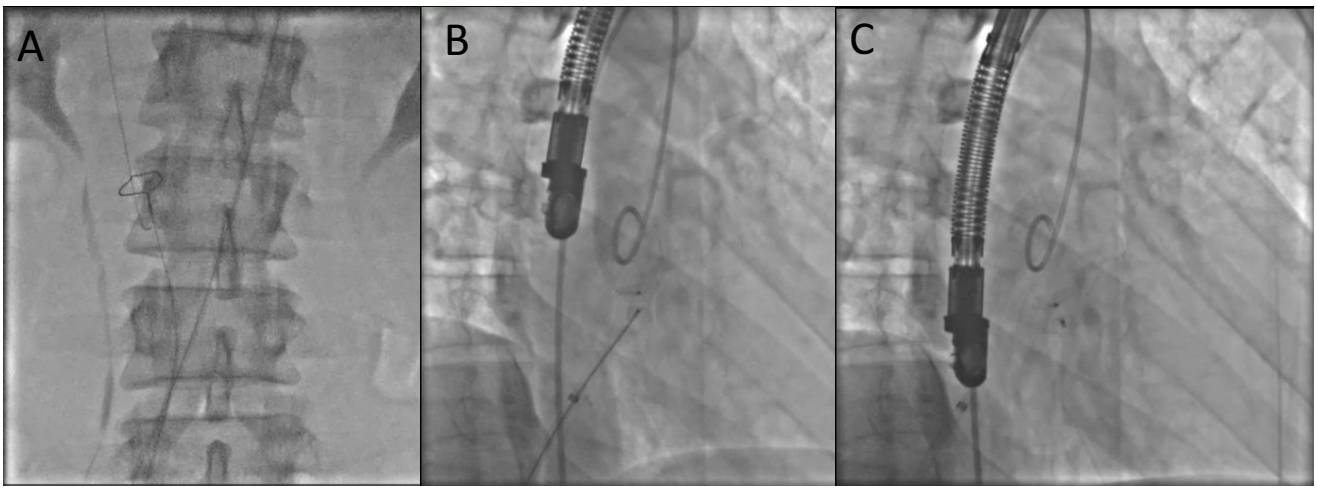

Fig. 8. (A) Fluoroscopic image of arteriovenous loop formation, (B) Image in which the VSD occluder device is placed in the existing defect, and (C) Fluoroscopic image after placement of the VSD occluder device in the defect.

References

1. Kassab K, Jolly N, Vij A, Kattoor AJ. Percutaneous closure of ruptured sinus of Valsalva: A review. Cardiovascular Revascularization Medicine: Including Molecular Interventions. 2024;60: 91-94. doi: 10.1016/j.carrev.2023.09.006
2. Chen W, Li T, Lei X, Liu H. An observational study of symmetrical VSD occluder for transcatheter closure of ruptured sinus of Valsalva aneurysm. Exp Ther Med. 2024;27(6):244. doi: 10.3892/etm.2024.12532.
3. Sivadasanpillai H, Valaparambil A, Sivasubramonian S, et al. Percutaneous closure of ruptured sinus of Valsalva aneurysms: intermediate term follow-up results. EuroIntervention. 2010;6(2):214-219.

OP-16.
Relationship Between High-Density Lipoprotein Cholesterol-Monocyte Ratio and Coronary Artery Disease

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Objective: To examine the relationship between coronary artery disease (CAD), as demonstrated by coronary angiography, and the high-density lipoprotein cholesterol (HDL-C)-to-monocyte ratio (HMR).
Methods: The retrospective analysis included 328 patients in total. Whether or not one coronary artery's stenosis surpasses 50% determined the classification. To assess the degree of coronary artery stenosis, use the Gensini Score (GS). The study employed receiver operating characteristic (ROC) curve analysis, progressive logistic regression, and Spearman correlation analysis.
Results: The CAD and non-CAD groups' HMR differences were statistically significant (P<0.001) (Table 1). Spearman correlation analysis showed that the HMR was slightly and moderately correlated with other independent risk factors of CAD (r<0.5). Progressive logistic regression analysis revealed that HMR was a new independent risk factor (odds ratio =0.782, 95% CI: 0.68-0.93). CAD patients' GS and HMR were correlated (r=-0.244, P<0.001) (Fig. 1). Additionally, when comparing AUC, the HMR outperformed HDL-C and HDL-C-to-cholesterol (AUC HDL-C-to-monocyte=0.644, AUC HDL-C=0.605, AUC HDL-C-to-cholesterol=0.586) (Fig. 2).
Conclusions: Based on our study's findings, it may be summed up as follows. Initially, there was a statistically significant difference in the HMR between patients with and without CAD, and this difference persisted even after propensity score matching (PSM). Second, HMR does not exhibit a strong association with other coronary heart disease risk factors. Third, CAD risk might be independently assessed by HMR. The HMR was linked to the GS of CAD severity and was better at reflecting CAD than HDL-C and HDL-C-to-cholesterol. HMR can be utilized as a biomarker to assess the severity of CAD and was an independent factor of CAD.
Keywords: Coronary artery disease, high-density lipoprotein cholesterol (HDL-C)-to-monocyte ratio (HMR), risk factors

Tablo 1. Baseline clinical and angiographic characteristics of the study population

	Non-CAD group（n=150）	CAD group（n=178）	P value
Male, n（%）	108（71.8）	133（75）	0.3
Age (years)	64.18±7.41	67.15±7.26	0.03
Smoking, n (%)	35（23.3）	72（40.6）	**<0.001**
HTN, n (%)	73（48.6）	90（50.6）	0.48
DM, n (%)	32（21.3）	56（31.5）	**0.007**
LVEF（%）	61（56-67）	60.00（55-65）	0.08
TC (mmol/L)	4.68±1.2	4.59±1.25	0.416
TG (mmol/L)	1.8±1.14	1.86±1.3	0.44
C-LDL (mmol/L)	2.98±1.02	2.95±1.15	0.93
HDL-C(mmol/L)	1.22±0.37	1.08±0.38	**<0.001**
NEU (10^9/L）	4.75（2.65-7.5）	5.63（3.9-7.47）	**<0.001**
LYMPH (10^9/L)	1.94±0.59	2.05±0.49	0.914
MONO (10^9/L）	0.45±0.28	0.55±0.26	**<0.001**
Target coronary artery			
LMS, n (%)	3 (2)	16 (9.1)	**<0.001**
LAD, n (%)	40（26.4）	146（82.1）	**<0.001**
LCx, n (%)	11（7.5）	158（89.0）	**<0.001**
RCA, n (%)	27（18.3）	114 (64.1)	**<0.001**
HDL-C/monocyte	2.96(1.87-3.80)	2.1(1.65-2.91)	**<0.001**

HTN=Hypertension, DM=Diabetes mellitus, TC=Cholesterol, TG=Triglycerides, C-LDL=Low density lipoprotein cholesterol, HDL-C=High density liptein cholesterol, LVEF=Left Ventricular Ejection Fractions, NEU=Neutrophils count, LYMPH=Lymphocyte count, MONO=Monocyte count, LMS=Left main coronary artery, LAD=Left main coronary artery, LCx=Left circumflex artery, RCA=Right coronary artery

Table 2. Multivariable analysis of independent factors for coronary artery disease

Variables	OR	95% CI	P value
Age	1.029	1.018- 1.048	**<0.001**
Smoking	2.35	1.56- 3.37	
Neutrophils count (10^9/L）	1.20	1.074- 1.348	
HDL-C-to-monocyte ratio	0.782	0.68- 0.93	

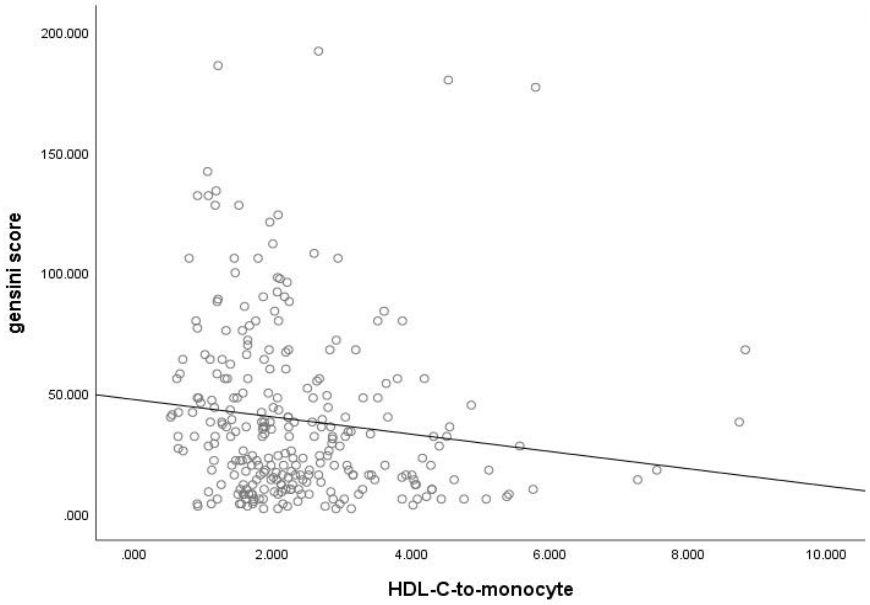

Fig. 1. **Distribution among of HDL-C-to-monocyte ratio and Gensini score.**

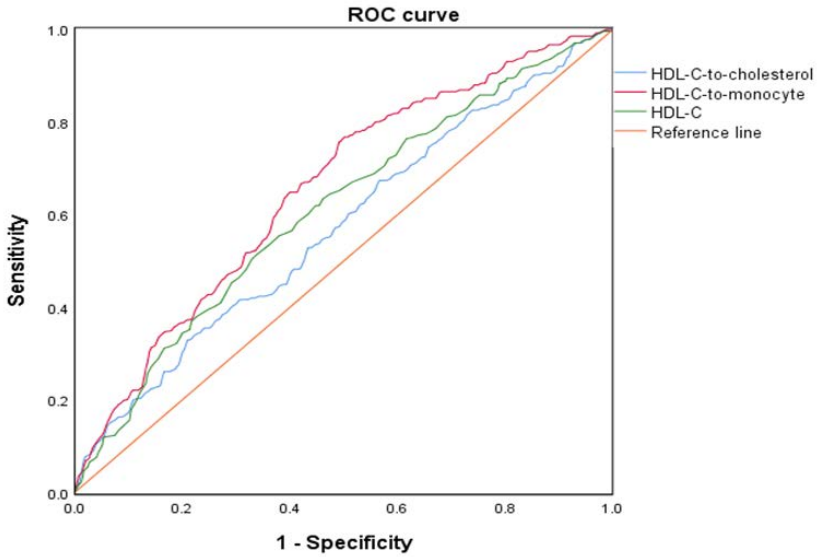

Fig. 2. **ROC curve for HDL-C-to-monocyte ratio and HDL-C and HDL-C-to-cholesterol.**

OP-017.
Can Antiaggregant Treatments Reduce Major Adverse Cardiovascular Events in Ischemia With Non-Obstructive Coronary Arteries?

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Objectives: The rate of MACE (Major Adverse Cardiovascular Events) associated with INOCA (Ischemia with non-obstructive coronary arteries) requires improvement in the treatment of affected patients. Our study aims to contribute to the role of antithrombotic therapy in patients with chronic coronary syndrome diagnosed with INOCA.
Methods: Retrospectively, patients diagnosed with chronic coronary syndrome were screened. Patients with ischemia detected in myocardial perfusion scintigraphy imaging (SPECT-MPI) and no obstructive coronary artery disease detected in invasive coronary angiography were included in the study. Patients with similar demographic characteristics were divided into two groups according to the use of antiaggregant agents. Patients who started antiaggregant treatment were included in Group 1, and those who did not were included in Group 2. The MACE rates of the two groups were compared.
Results: A total of 229 patients were included in our study. 115 patients were treated with an antiaggregant during the follow-up period (Group 1). Four patients were treated with only clopidogrel, and 111 were treated with only aspirin as an antiplatelet agent. 114 patients were not treated with an antiaggregant (Group 2). The mean follow-up period was 89±21 months. During follow-up, there was a significant difference between the two groups in terms of total deaths (5% and 2%; P=0.01), cardiac deaths (4% and 1%; P=0.01), myocardial infarctions (7% and 1%; P=0.02), and hospitalizations (13% and 4%; P=0.03).
Conclusion: Our study data suggest that adding an antiplatelet agent to treating patients diagnosed with INOCA may reduce MACE rates.
Keywords: Ischemia with non-obstructive coronary artery (INOCA), major adverse cardiovascular events (MACE), antiplatelet treatment

OP-18.
Relationship Between C-Reactive Protein to Albumin Ratio (CAR) and Mortality in Patients With Diabetes Mellitus Type 2

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Introduction: Diabetes mellitus (DM) is a metabolic disease common worldwide and associated with increased morbidity and mortality. Mortality prediction in DM patients, whose frequency is increasing and is associated with adverse cardiovascular risks and mortality, provides important contributions to both treatment planning and prognosis estimation. C-reactive protein/Albumin Ratio (CAR) is a new inflammatory marker. Therefore, we aimed to investigate the relationship between CAR, which can be easily calculated from routine biochemical tests, and mortality in DM patients.
Methods: Our study is a retrospective single-center study. Patients who applied to our hospital's outpatient clinic and were diagnosed with non-insulin-dependent type 2 diabetes (T2DM) were included in the study. Clinical data of the patients, comorbidities, medications used, laboratory data, and death information were obtained from the hospital information system and MERNIS. Patients were divided into two groups based on mortality status, and mortality-related parameters were determined. Univariate and multivariate Cox regression analyses were performed for parameters associated with mortality, and parameters independently associated with mortality were determined. To assess the ability of these parameters to predict mortality, receiver operating characteristic (ROC) curves were plotted, and the area under the curve (AUC) was determined.
Results: Eight thousand one hundred seventy-one patients who applied to the outpatient clinic diagnosed with T2DM were included in our study. Mean age was 59.2±12.8 years, and 42.2% (3450 patients) were male. The mean follow-up period was 585 days, and the mortality rate was 1% (81 patients). Mortality was significantly associated with age, gender, heart failure (HF), hemoglobin (Hb), C-reactive protein (CRP), albumin, aspartate aminotransferase, high-density lipoprotein (HDL), low-density lipoprotein (LDL), creatinine, uric acid, glucose, and CAR (Table 1). In the multivariate Cox regression analysis, mortality was independently associated with age, HF, Hb, creatinine, uric acid, and CAR (Table 2). In ROC curve analysis, CAR predicted mortality better than CRP and albumin (AUC: 0.742 %95 CI 0.682-0.803, P<0.001). At a cut-off value of 0.287, CAR predicted mortality with 70.7% sensitivity and 66.7% specificity (Fig. 1).
Conclusions: Inflammation plays a vital role in the progression of DM. High levels of inflammatory biomarkers are associated with an increased risk of adverse cardiovascular events in patients with DM. CRP, albumin, and CAR, a new biomarker derived from them, are important indicators of inflammation. CRP and albumin are independent risk factors for in-hospital and long-term prognosis in patients with acute coronary syndrome, some cancers, and diabetes mellitus. In addition, they are associated with adverse cardiovascular events and mortality. Our study observed that CAR was significantly associated with mortality in DM patients and could provide insight into their prognosis. In Conclusion, in our study CAR, which can be calculated from routine biochemical tests, was independently associated with mortality in T2DM patients. Thus, CAR can serve as an easily calculated, inexpensive, and practically helpful marker for predicting mortality.
Keywords: Diabetes mellitus, C-reactive protein/Albumin Ratio (CAR), inflammatory biomarkers

Table 1. Demographic data of groups based on mortality development status

Variables	Total Gruop (n=8171)	Mortality (+) (n=82)	Mortality (–) (n=8089)	P value
Age (year)	59.2±12.8	71.9±10.8	59.1±12.8	**<0.001**
Gender (male), n (%)	3450 (42.2)	45 (54.9)	3405 (42.1)	**0.020**
HT, n (%)	3441 (42.1)	35 (43.2)	3406 (42.1)	0.513
CAD, n (%)	2770 (33.9)	28 (34.6)	2742 (33.9)	0.194
AF, n (%)	764 (9.3)	8 (9.8)	756 (9.3)	0.493
CRF, n (%)	1168 (14.2)	12 (14.8)	1156 (14.2)	0.119
COPD, n (%)	872 (10.7)	9 (11.2)	863 (10.6)	0.594
CHF, n (%)	418 (5.1)	27 (32.9)	391 (5)	**<0.001**
Medications				
ASA, n (%)	3996 (48.9)	42 (51.8)	3954 (48.9)	0.233
Statins, n (%)	3720 (45.5)	38 (46.9)	3682 (45.5)	0.268
NOAC, n (%)	654 (8)	6 (7.4)	648 (8)	0.345
ACEI / ARB, n (%)	3055 (37.3)	29 (35.8)	3026 (37.3)	0.682
MRA, n (%)	369 (4.5)	3 (3.6)	368 (4.5)	0.146
BB, n (%)	2303 (28.1)	22 (27.1)	2281 (28.2)	0.249
Diuretics, n (%)	432 (4.1)	4 (4.8)	328 (4)	0.524
Metformin, n (%)	5131 (62.8)	46 (56.1)	5085 (62.9)	0.237
Sulfonylureas, n (%)	695 (8.5)	5 (6.1)	690 (8.5)	0.432
Glitazone, n (%)	1166 (14.3)	12 (14.6)	1154 (14.3)	0.925
SGLT-2i, n (%)	1559 (19.1)	17 (20.7)	1542 (19.1)	0.702
GLP-1i, n (%)	91 (1.1)	0 (0)	91 (1.1)	0.334
DPP-4i, n (%)	1892 (23.2)	15 (18.3)	1877 (23.2)	0.294
Laboratory				
Hb (g/dL)	14.9± 1.8	13.3± 2.8	14.9±1.8	**<0.001**
Wbc (103/µL)	8.8±2.44	9.1±2.2	8.8±2.4	0.100
Platelet count (103/µL)	302± 82	325± 141	302± 81	0.904
AST (U/L)	28.1 (7-1000)	74 (9-1000)	27 (7-1000)	**<0.001**
ALT (U/L)	36.5 (4-1000)	75 (8-1000)	36 (4-1000)	0.625
CRP (mg/L)	12.5 (1-399)	93.7 (3-399)	11.4 (1-255)	**<0.001**
Creatinine (mg/dL)	0.93± 0.42	1.63±1.18	0.93±0.4	**<0.001**
HbA1c	8±2.1	7.9±1.9	8±2.1	0.992
Glucose (mg/dL)	193±106	249±161	154±93.3	**0.001**
Albumin (g/L)	43.7±8.2	38.4±12.5	35.6±13.7	**<0.001**
TG (mg/dL)	230±134	201±106	230±134	0.051
HDL (mg/dL)	43±11	40±15	44±11	**0.008**
LDL (mg/dL)	143±41	201±106	144±41	**<0.001**
Uric Acid (mg/dL)	5.4±1.5	7.2±2.4	5.4±1.5	**<0.001**
CAR	0.28 (1-10.5)	1.05 (0.1-10.5)	0.27 (0.1-7.73)	**<0.001**

HT=hypertension, DM=diabetes mellitus, CAD=coronary artery disease, AF=atrial fibrillation, CRF=chonic renal failure, COPD=chronic obstructive pulmonary disease, CHF=congestive heart failure, ASA=acetylsalicylic acid, NOAC=New oral anticoagulant, ACEI=angiotensin converting enzyme inhibitor, ARB=angiotensin reseptor blocker, MRA=mineralocorticoid receptor antagonist, BB=beta blocker, SGLT-2i=sodium-glucose cotransporter-2 inhibitor, GLP1i=glucagon-like peptide-1 inhibitor, DPP-4i=Dipeptidyl peptidase IV inhibitör, Hb=haemoglobin, Wbc=white blood cell, AST=aspartate aminotrasferase, ALT=alanine aminotrasferase, CRP=C-reactive protein, TG=triglycerides, HDL=high density lipoprotein, LDL=low density lipoprotein, CAR=creatinine/albümine ratio

Table 2. Regression analysis according to mortality development status

Variables	Univariate OR, 95 CI%	P value	Multivariate OR, 95 CI%	P value
Age	1.097 (1.076-1.119)	**<0.001**	1.063 (1.037-1.090)	**<0.001**
Sex	1.639 (1.059-2.538)	**0.027**	1.173 (0.656-2.000)	0.590
HF	10.430 (6.570-16.556)	**<0.001**	2.956 (1.646-5.310)	**<0.001**
HB	0.667 (0.602-0.738)	**<0.001**	0.787 (0.701-0.884)	**<0.001**
AST	1.004 (1.003-1.005)	**<0.001**	1.001 (0.999-1.003)	0.234
HDL	0.973 (0.951-0.995)	**0.017**	0.985 (0.965-1.005)	0.145
LDL	0.989 (0.983-0.995)	**<0.001**	1.001 (0.993-1.006)	0.890
Creatinine	1.839 (1.663-2.034)	**<0.001**	1.499 (1.252-1.795)	**<0.001**
Uric acid	1.487 (1.386-1.594)	**<0.001**	1.195 (1.038-1.376)	**0.013**
CAR	1.913 (1.748-2.093)	**<0.001**	1.437 (1.234-1.674)	**<0.001**

Hb=haemoglobin, HF=heart failure, AST=aspartate aminotranferase, HDL=high density lipoprotein, LDL=low density lipoprotein, CAR=creatinine/albümine ratio

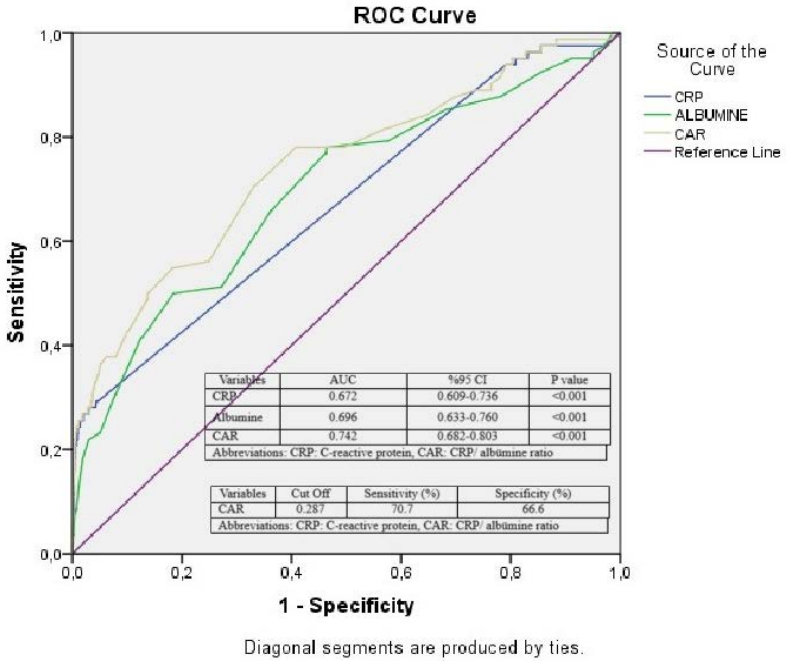

Fig. 1. Receiver operating characteristic (ROC) cure analysis and area under the curve values (ACUC) of parameters.

OP-19.
Percutaneous Closure of a Ascending Aortic Graft Pseudoaneurysm With a Konar VSD Occluder

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Introduction: Aortic pseudoaneurysms (APA) are rare aortic conditions that are generally asymptomatic but have the potential to be fatal. They most commonly occur following cardiac surgery but can also result from trauma or infection. Large APAs may compress adjacent structures, leading to serious complications such as rupture, thrombosis, embolization, and fistula formation. Therefore, routine follow-up with computed tomography (CT) is necessary. The traditional treatment method is surgical repair, which carries a high risk of morbidity and mortality, especially in patients with a history of cardiac surgery. Alternative treatment options include thoracic endovascular aortic repair (TEVAR), coil embolization, thrombin injection, vascular plugs, and occluder devices.

Case Report: A 70-year-old female patient, who had undergone coronary artery bypass grafting (CABG) and ascending aortic replacement (AAR) four years prior, was presented. Following her initial surgery, the patient developed mediastinitis, which required prolonged antibiotic therapy and an additional open surgical intervention. During routine follow-up, CT imaging revealed a collection in the substernal area, irregularity in the left anterolateral wall of the ascending aorta, and focal contrast fillings at two levels suggestive of pseudoaneurysms (Fig. 1). Transesophageal echocardiography (TEE) identified a rupture (pseudoaneurysm) measuring 14×5 mm (Fig. 2) confined by a hematoma, located 12 mm superior to the left main coronary artery (LMCA), along with a collection beneath the sternum. Blood cultures and other laboratory tests showed no signs of mediastinitis. However, to rule out infection, a sample was obtained from the collection via needle aspiration through the left second intercostal space (Fig. 3), confirming the absence of infection.

Given the high surgical risk associated with aortic rupture in a case with a hematoma located just beneath the sternum, percutaneous closure was preferred. To minimize the risk of further rupture, no guidewire or catheter was advanced directly through the defect. Instead, an 8.5F Agilis Small catheter was introduced via the right femoral artery and positioned at the aortic root. After achieving the appropriate angulation, the device was placed at the defect opening. A 14×12 mm Konar VSD Occluder device was deployed distally through the catheter (Fig. 4), and successful closure of the rupture was confirmed via 3D TEE and angiography (Figs. 4-6). No complications were observed. Follow-up transthoracic echocardiography (TTE) and contrast-enhanced CT performed a few days after the procedure confirmed the correct positioning of the device. The patient was discharged one week later.

Conclusion: APA development following AAR surgery is a known potential complication. Although conventional treatment involves open surgical repair, percutaneous closure is emerging as a viable alternative, particularly in high-risk patient groups due to the associated morbidity and mortality risks. In this case, we aimed to highlight the efficacy of percutaneous closure using a Konar VSD Occluder device as a potential alternative treatment for APA.

Keywords: Aortic pseudoaneurysms, ascending aortic replacement, Konar VSD Occluder device

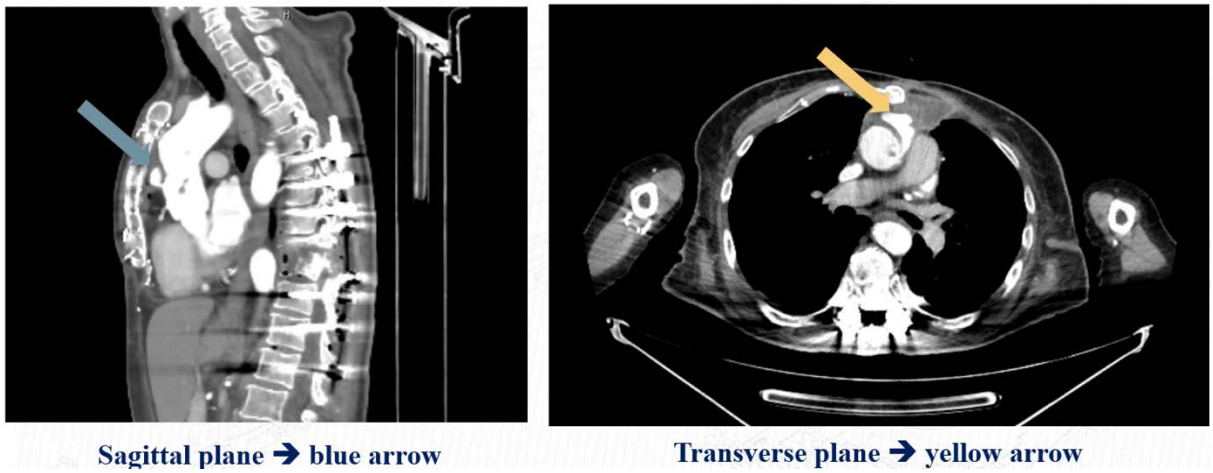

Fig. 1. CT sections of thoracic aorta.

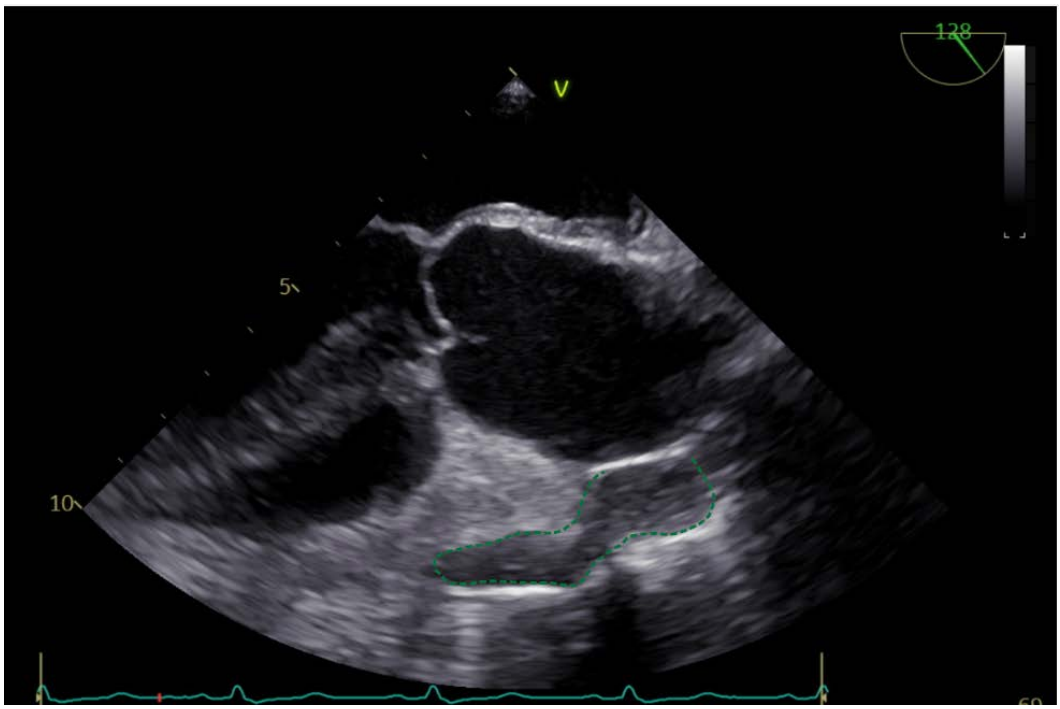

Fig. 2. Pseudoaneurysm sized 14×5 mm, green dashed area.

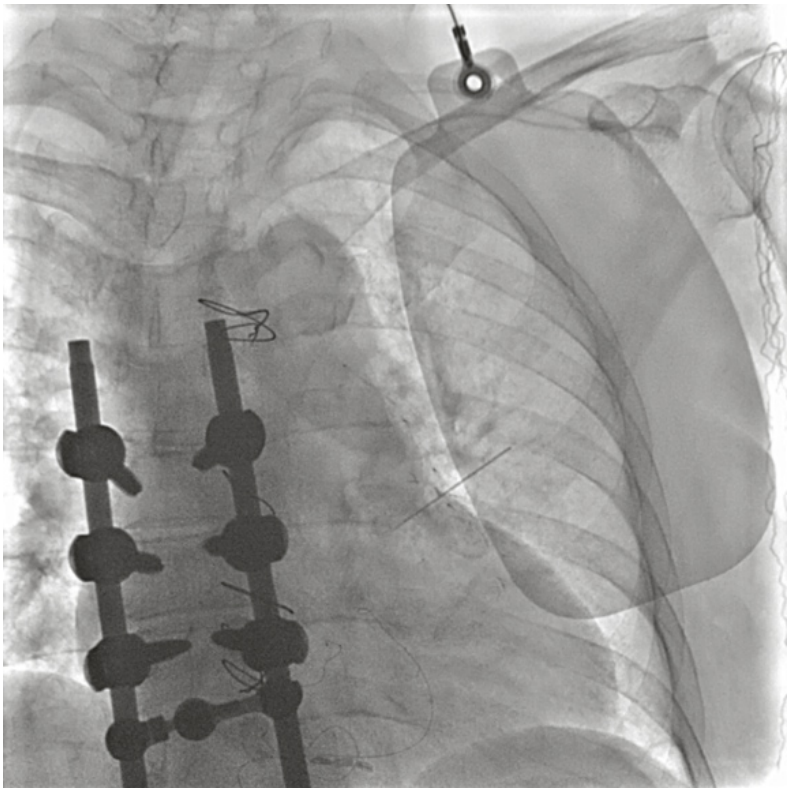

Fig. 3. **Left second intercostal space needle sampling.**

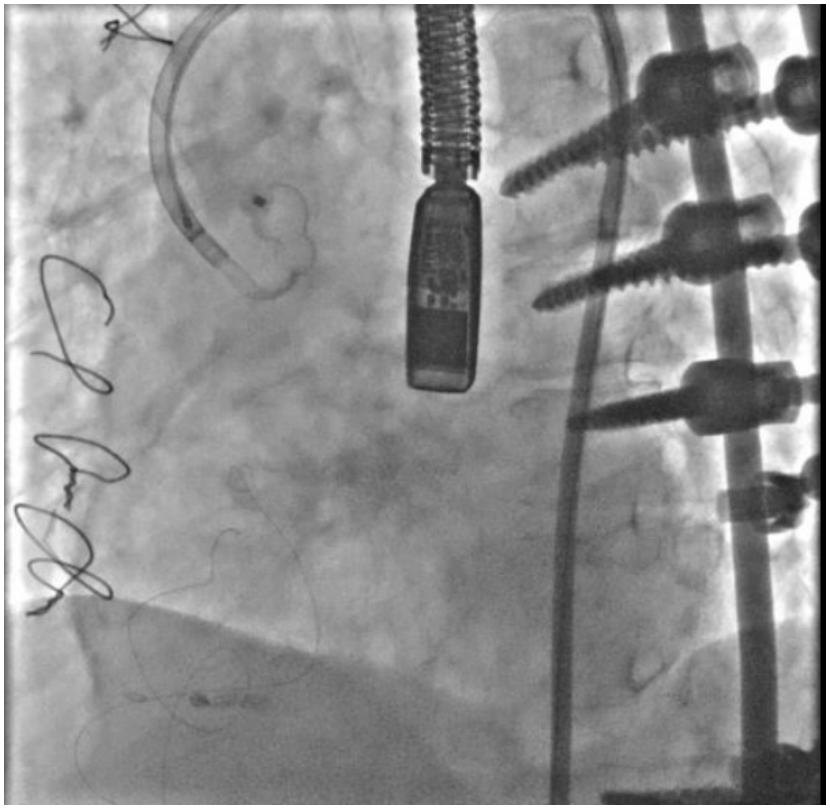

Fig. 4. **Percutaneous closure angiographic imaging.**

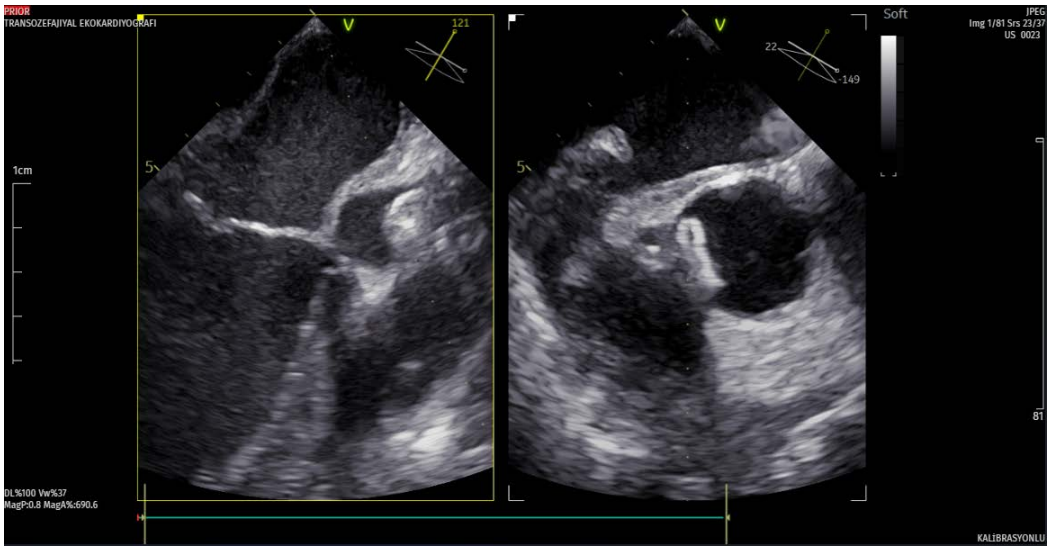

Fig. 5. **Percutaneous closure TEE imaging.**

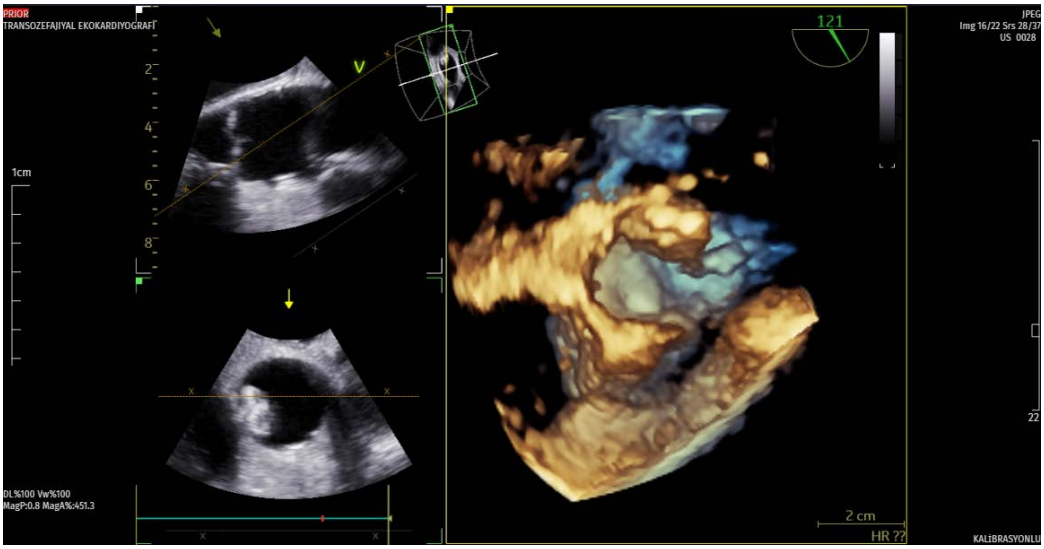

Fig. 6. **Percutaneous closure TEE imaging.**

OP-20.
Approach to a Patient With a Presumptive Diagnosis of Hypereosinophilic Syndrome Presenting With an Acute Coronary Syndrome

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Introduction: Hypereosinophilic Syndrome (HES) is diagnosed when eosinophilia of at least 1,500/µL persists for six months, secondary and clonal eosinophilia are excluded, there is evidence of organ involvement, and no phenotypically abnormal or clonal T lymphocytes are found. Its incidence is around 0.036 per 100,000, with a male/female ratio of 1.47 and peak prevalence between ages 65 and 74. Common symptoms include fatigue, cough, dyspnea, myalgia, angioedema, rhinitis, rash, and fever. Eosinophilic cardiomyopathy occurs in about 20% of patients and can progress to heart failure, thrombus formation, and restrictive cardiomyopathy.
Objective: To raise awareness of HES, manage its cardiac complications, and underscore uncertainties in its treatment.
Case Report: A 48-year-old male presented on December 23, 2024, with new-onset chest pain and left arm pain. He had a history of hypertension; physical examination was unremarkable. ECG showed sinus rhythm and biphasic T waves in the anterior leads. Echocardiography revealed an LVEF of 40%, marked apical hypokinesia, and dense spontaneous echo contrast in the left ventricular apex. Laboratory results included WBC 36,220/µL with 23,300/µL eosinophils, and troponin of 260 ng/mL (normal: 0-100).
His records showed persistent hypereosinophilia since August 2024, along with fatigue, dysuria, decreased oral intake, and visual disturbances. An earlier eye exam revealed changes in the inferior branch of the retinal artery suggesting a thrombus. At that time, his troponin levels were markedly elevated (hs-troponin: 3,615, rising to 4,578). Coronary angiography (CAG) demonstrated a 100% distal LAD occlusion, which was treated with balloon PTCA. Although his symptoms initially improved, he re-presented with persistent angina, elevated troponin, and ischemic ECG changes. Repeat CAG indicated a 95% mid-distal LAD thrombus. A 2.5×16 mm drug-eluting stent was successfully placed, after which ECG abnormalities resolved, troponin levels decreased, and no new cardiac symptoms arose. The echocardiogram remained unchanged. He was discharged on rivaroxaban 20 mg once daily and clopidogrel; aspirin therapy was stopped.
Conclusion: Although HES is rare, it should be considered in patients presenting with what appears to be coronary embolism. Because of its low prevalence, consensus on the optimal approach to such complications is lacking. In this case, placement of a drug-eluting stent and a combination therapy of a NOAC plus clopidogrel were employed, which appeared effective in preventing further cardioembolic events.
Discussion: In a patient with HES presenting with coronary embolism, DES implantation was successfully performed. We believe that with appropriate anticoagulant therapy and HES-specific treatment, the cardioembolic complications of HES can be kept under control.
Keywords: Hypereosinophilic Syndrome, cardioembolism, acute coronary syndrome

OP-21.
Artificial Intelligence in Primary Electrical Cardiac Diseases: Evaluating ChatGPT as a Clinical Decision Support Tool

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Introduction: Primary electrical heart diseases (PEHD) are generally classified as channelopathies--hereditary genetic abnormalities that affect the electrical behavior of cardiomyocytes and increase the risk of malignant arrhythmias leading to sudden cardiac death (SCD). Notably, approximately 54% of sudden and unexpected deaths among individuals under 35 years of age occur without any evidence of structural heart disease at autopsy, highlighting the potential importance of channelopathies in this demographic. Channelopathies encompass a heterogeneous group of disorders, including long QT syndrome (LQTS), short QT syndrome (SQTS), idiopathic ventricular fibrillation (IVF), Brugada syndrome (BrS), catecholaminergic polymorphic ventricular tachycardia (CPVT), and early repolarization syndromes (ERS). Although significant progress has been made in diagnostic evaluations, the relationship between these conditions and SCD remains incompletely understood. Emerging data from electrophysiological studies and genetic testing indicate that these are complex diseases involving multiple genes and a broad spectrum of SCD-associated factors. This study aims to propose a novel approach to the management of these rare and complex diseases by exploring the role of artificial intelligence (AI)-based tools in their assessment and clinical decision-making processes, with a particular focus on the potential of ChatGPT, an AI-based language model, to provide guideline-based information for the diagnosis, risk stratification, and clinical management of PEHD.

Methods: In this cross-sectional study, 30 PEHD-specific questions were posed to ChatGPT-4. These questions covered the fundamental principles of PEHD, diagnostic workflows, risk stratification, and various clinical scenarios. The responses were categorized based on guideline adherence as “completely appropriate,” “partially appropriate,” “appropriate but insufficient,” or “inappropriate.” Two cardiologists independently evaluated the responses, and inter-rater reliability was assessed using Cohen’s kappa coefficient.

Results: ChatGPT provided answers to all 30 questions. Among the 15 questions focused on general knowledge, 11 were deemed completely appropriate, while 4 were deemed appropriate but insufficient. Of the 15 clinical scenario-based questions, 12 were considered completely appropriate, and 3 were evaluated as appropriate but insufficient. ChatGPT performed strongly in domains such as diagnostic algorithms, recommendations for genetic analysis, and risk stratification. However, certain limitations were noted, particularly regarding Brugada syndrome management and risk assessment in pediatric/adolescent patients. The inter-rater reliability, as measured by Cohen’s kappa, was 92%.

Conclusion: ChatGPT demonstrates promise as a supplementary decision support tool by providing guideline-concordant information in the management of PEHD. Nonetheless, limitations related to contextual understanding and individualized risk assessment restrict its independent clinical application. Future enhancements should focus on personalized recommendations, clinical validation, and integration with healthcare professionals.

Keywords: Primary electrical heart diseases, artificial intelligence, supplementary decision support tool

OP-22.
G6PD Deficiency With Severe Mitral Stenosis and Heart Failure: Hard Case to Manage

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A 61-year-old patient was admitted with complaints of dyspnea, swelling in the legs. She had a history of mitral valvotomy due to mitral valve stenosis, known atrial fibrillation. In addition, she had glucose-6-phosphate dehydrogenase (G6PD) deficiency. The patient was taking warfarin 5mg, spironolactone 25mg, metoprolol 50mg. Cardiac auscultation revealed 2/6 systolic, 4/6 diastolic murmur and rales were heard in the bases of both lungs. Thorax CT revealed bilateral pleural effusion at the basal regions. Echocardiography revealed LVEF 30%, moderate aortic, mitral and tricuspid valve regurgitation and a gradient of 22/11 mmHg in themitral valve. Mitral valve area was 1.1 cm2 and Wilkins score was 12. Cardiac catheterization and coronary angiography showed mean gradient of 12 mmHg was measured in the mitral valve and no lesion was found in the coronary arteries. Heart team decided to operation and mitral valve replacement was performed with a 29 mm mechanical valve (St Jude Medical). The patient was transferred to intensive care unit with dobutamine (unknown risk of hemolysis in patients with G6PD deficiency) as an inotropic agent at 15 mcg/kg/min and glyceryl trinitrate (high risk) was used at 1 mcg/min to reduce systemic venodilatation and preload for 3 days. On the 5th postoperative day, the patient intubated and 12.5 mcg/kg/min dopamine (moderate risk) and 0.25 mcg/kg/min noradrenaline (unknown risk) was administered for 5 days. Intermittent hemodialysis and ultrafiltration were administered to the patient with signs of overload and pulmonary edema and decreased urine output on postop day 7 because of the moderate risk of furosemide use in G6PD deficiency and a history of hemolysis after furosemide tablet use in the past. Clinical findings and laboratory values were compatible with intravascular hemolysis (shown as tables and graphics) and were thought to be related to glyceryl trinitrate use. (Fig. 1, 2) (Table 1).

After one month, acetazolamide (intermediate risk) 250 mg was started because there was no evidence for the use of most pharmacologic agents in G6PD deficiency and diuretic agents that can be used in heart failure are risky. Due to the continuation of overload findings and failure to achieve the desired urine output, tolvaptan (unknown risk) 30 mg was started. Urine output reached the target amount with the use of tolvaptan. After one week of treatment, sodium values increased from 135 mEq/L to a maximum of 141 mEq/L. The patient's congestion symptoms regressed. After treatment, tolvaptan and acetazolamide was discontinued and the patient was discharged with lifestyle changes, medication and additional recommendations. During follow-up, perindopril 5 mg and dapagliflozin 10 mg were added to the current treatment. Although studies did not provide any recommendation for the patients with heart failure and G6PD deficiency, we could not give furosemide treatment to the patient and symptomatic improvement was achieved by adding acetazolamide and tolvaptan in acute care, dapagliflozin for maintenance treatment.

Keywords: Glucose-6-phosphate dehydrogenase (G6PD) deficiency, severe mitral stenosis, heart failure, cardiac surgery, acetazolamide

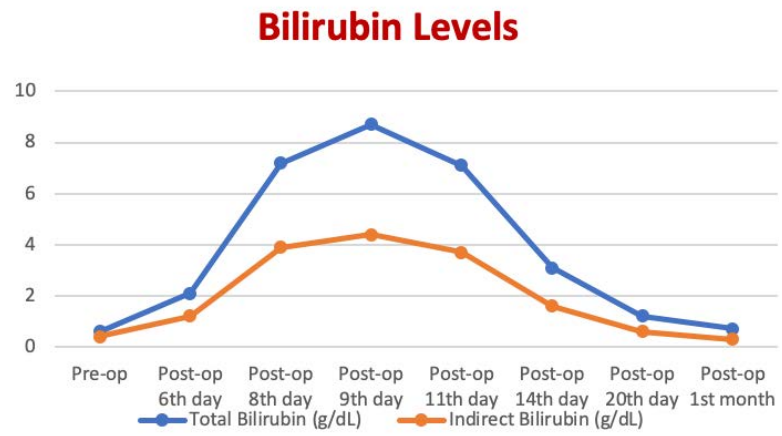

Fig. 1. Change graph of bilirubin values.

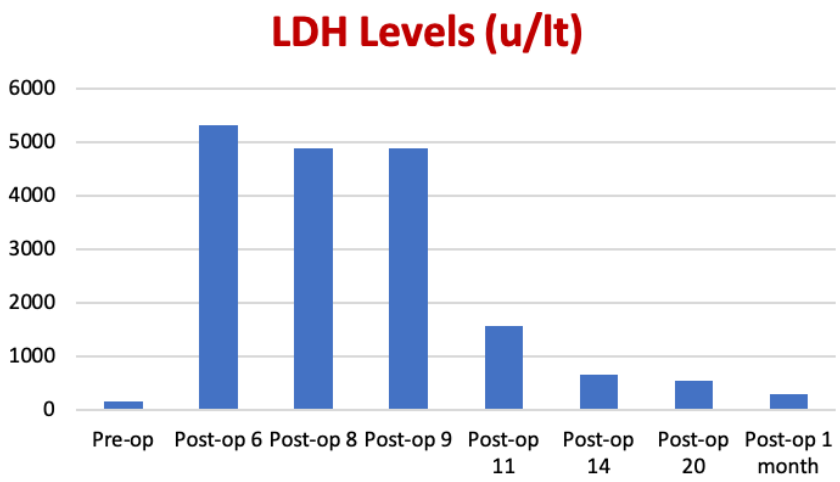

Fig. 2. Change graph of lactate dehydrogenase values.

Table 1. Hemoglobin level and hemolysis parameters

	Total Bilirubin (mg/dL)	Indirect Bilirubin (mg/dL)	Hemoglobin (g/dL)	Haptoglobulin (mg/dL)	LDH (U/L)
Pre-op	0.6	0.4	13.2		147
Post-op 6th day	2.1	1.2	7.3	<8	5335
Post-op 8th day	7.2	3.9	8.3	<8	4901
Post-op 9th day	8.7	4.4	9.9	<8	4887
Post-op 11th day	7.1	3.7	7.2	<8	1554
Post-op 14th day	3.1	1.6	8.6	<8	646
Post-op 20th day	1.2	0.6	9	<8	528
Post-op 1st month	0.7	0.3	10.1	60.3	292

OP-23.
The Triglyceride Glucose Index may Predict Significant Coronary Stenosis in Moderate Left Main Coronary Artery Lesions: An Intravascular Ultrasonography Study

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Background: There may be severe difficulties in determining the severity of LMCA (left main coronary artery) lesions. The use of intravascular ultrasound (IVUS) facilitates the decision about the lesion severity in these patients. Previous studies have shown a close association of the triglyceride glucose (TyG) index with coronary atherosclerosis. The aim of this study was to investigate the relationship between TyG index and lesion severity in patients who have undergone LMCA IVUS.
Methods: The study included 180 patients who were determined with ICS (intermediate coronary stenosis) in LMCA and underwent an IVUS procedure. The patients were separated into two groups according to the TyG index values as those <9.83 and ≥9.83. In the IVUS measurements of these patients, the plaque burden (PB) and the minimal lumen area (MLA) showing lesion severity were measured. Lesions calculated as MLA <6 mm^2 or PB ≥65% were considered critical lesions (Table 1).
Results: The patients comprised 136 (75.6%) males and 44 (24.4%) females with a median (IQR) age of 59 (53-68) years. In the group with high TyG index, MLA values were significantly lower (7.74±4.1 vs. 6.24±2, P=0.032) and plaque burden percentages were significantly higher (54.5±15.2 vs. 59.8±11.4, P=0.05). In addition, the number of patients with critical lesions detected in IVUS measurements was significantly higher in the group with high TyG index (69 vs. 19, P=0.005). SYNTAX score was significantly higher in patients with critical lesions detected on IVUS (17±9 vs. 6±8, P<0.001) (Table 2 and 3).
Conclusions: The results of this study suggested that there was a significant association between the TyG index and lesions evaluated as critical on IVUS, which may predict anatomically important lesions in patients with moderate degree of LMCA stricture. TyG index can be a surrogate marker for invasive treatment modality fort the management of patients with lessions of LMCA in the grey zone for intervention.
Keywords: Triglyceride glucose (TyG) index, left main coronary artery, intravascular ultrasound

Table 1. The baseline characteristics and laboratory investigations of all patients according to TyG index

	All patients (n=180)	TyG index <9.83 (n=155)	TyG index ≥9.83 (n=25)	P value
Demographic characteristics				
Age, y	59 (53-68)	59 (52-68)	62 (58-68)	0.051
Male sex, n (%)	136 (75.6)	115 (74.2)	21 (84)	0.451
Body mass index, kg/m^2	26.8 (24.7-29)	26.8 (24.5-29)	26.2 (25.2-28.1)	0.978
Comorbidites				
Hypertension, n (%)	112 (62.2)	97 (62.6)	15 (60)	0.827
Diabetes mellitus, n (%)	42 (23.3)	31 (20)	11 (44)	0.019
Hyperlipidemia, n (%)	46 (25.6)	41 (26.5)	5 (20)	0.624
Smoking, n (%)	114 (63.3)	97 (62.6)	17 (68)	0.661
Chronic kidney disease, n (%)	27 (15)	22 (14.2)	5 (15)	0.544
Medications				
Acetylsalicylic acid use, n (%)	179 (99.4)	154 (99.4)	25 (100)	1.000
P2Y12 Inhibitors use, n (%)	144 (80)	123 (79.3)	21 (84)	0.717
B-blocker use, n (%)	169 (93.9)	144 (92.9)	25 (100)	0.366
ACE Inhitors use, n (%)	154 (85.6)	131(84.5)	23(92)	0.539
Statin use, n (%)	176 (97.8)	152 (98.1)	24 (96)	0.453
OAD use, n (%)	46 (25.6)	32 (20.6)	14 (56)	**0.001**
Laboratory assessment				
Hemoglobin, g/dL	13.2 (12-14.5)	13.2 (12-14.6)	13.2 (11.8-13.9)	0.610
eGFR, ml/min/1.73 m^2	90.3 (70.2-101.9)	92.3 (70.1-103.3)	88.2 (74-99.2)	0.450
Total cholesterol, mg/dL	167 (144-206.5)	166 (138-204)	183 (159-232)	**0.047**
Triglycerides, mg/dL	145.5 (100-204)	135 (95-173)	302 (232-345)	**<0.001**
LDL cholesterol, mg/dL	90.7 (68-126)	89.9 (69-127.6)	95.9 (62.2-124.1)	0.675
Albumin, g/L	42 (39-44)	42.1 (39.1-44.3)	41.6 (38.8-44.9)	0.849
FBG, mg/dL	117 (98-171.5)	110 (95-159)	175 (124-229)	**0.001**
Uric asid, µmol/L	5.4 (4.7-6)	5.4 (4.7-6.1)	5.2 (4.5-5.9)	0.928
TyG index	9.2 (8.7-9.5)	9.04 (8.6-9.4)	10.1 (10-10.3)	**<0.001**

Data are shown as median (interquartile range) or number (percentage) of patients. ACE=angiotensin converting enzyme, eGFR=estimated glomerular filtration rate, FBG=fasting blood glucose, IQR=interquartile range, LDL=low-density lipoprotein, OAD=oral antidiabetic, TyG=triglyceride glucose

Table 2. Angiographic and procedural status of patients according to TyG index

Angiographic parameters		All patients (n=180)	TyG index <9.83 (n=155)	TyG index ≥9.83 (n=25)	P value
Procedural data					
SYNTAX score		11±10	11±10	13±10	0.361
Critical number of vessels, n (%)	0	40 (22.2)	37 (23.9)	3 (12)	0.651
	1	55 (30.6)	45 (29)	10 (40)	
	2	53 (29.4)	45 (29)	8 (32)	
	3	31 (17.2)	27 (17.4)	4 (16)	
	4	1 (0.6)	1 (0.6)	0	
CAG result, n (%)	Medical follow up	70 (38.9)	60 (38.7)	10 (38.9)	0.883
	PCI	65 (36.1)	57 (36.8)	8 (32)	
	CABG	45 (25)	38 (24.5)	7 (28)	
IVUS-based volume parameters in LMCA					
EEM volume, mm^3		149.2 ± 48.8	151.1 ± 47.6	137.5 ± 55.1	0.253
Lumen volume, mm^3		67.2 ± 37	69.3 ± 38.2	54.2 ± 24.2	0.011
Plaque volume, mm^3		82 ± 33.1	81.7 ± 32	83.5 ± 40	0.833
MLA, mm^2		7.56 ± 3.9	7.74 ± 4.1	6.24 ± 2	**0.032**
MLA< 6 mm^2		80 (44.4)	65 (41.9)	15 (60)	0.071
Plaque burden, %		55.2 ± 14.8	54.5 ± 15.2	59.8 ± 11.4	0.050
Plaque burden ≥ 65%		64 (35.6)	52 (33.5)	12 (48)	0.181
Critical lesion, n (%)		88 (48.9)	69 (44.5)	19 (76)	**0.005**

Data are shown as mean±standard deviation or number (percentage) of patients. CABG=coronary artery bypass surgery, CAG=coronary angiography, EEM=external elastic membrane, IVUS=intravascular ultrasound, MLA=minimal lumen area, PCI=percutaneous coronary intervention, SYNTAX=the synergy between percutaneous coronary intervention with taxus and cardiac surgery, TyG=triglyceride glucose.

Table 3. Basic characteristics and laboratory examinations of all patients according to critical lesions on IVUS

	All patients (n=180)	Critical lesion (+) (n=88)	Critical lesion (-) (n=92)	p-value
Age	59 (53-68)	59 (55-70)	59 (51-68)	0.059
Male gender	136 (75.6)	67 (76.1)	69 (75)	0.864
Hypertension, n (%)	112 (62.2)	54 (61.4)	58 (63)	0.878
Diabetes mellitus, n (%)	42 (23.3)	22 (25)	20 (21.7)	0.725
Hyperlipidemia, n (%)	46 (25.6)	26 (29.5)	20 (21.7)	0.238
Chronic kidney disease, n (%)	27 (15)	14 (15.9)	13 (14.1)	0.835
Hemoglobin, g/dL	13.2 (12-14.5)	12.9 (11.9-14.3)	13.6 (12.2-14.6)	0.218
eGFR, ml/min/1.73 m^2	90.3 (70.2-101.9)	91.4 (70.1-101.9)	89.3 (71.1-101.9)	0.941
Total cholesterol, mg/dL	167 (144-206.5)	162.5 (133.5-204)	175.5 (151-209.5)	0.073
LDL cholesterol, mg/dL	90.7 (68-126)	84.2 (61-120)	97.5 (71-135)	0.064
Triglycerides, mg/dL	145.5 (100-204)	146 (112-197.5)	144.5 (94.5-205.5)	0.439
FBG, mg/dL	117 (98-171.5)	160 (114.5-194)	105 (89.5-123)	<0.001
TyG index	9.2 (8.7-9.5)	9.39 (8.96-9.72)	8.98 (8.51-9.33)	<0.001
SYNTAX score	11±10	17±9	6±8	<0.001

Data are shown as median (interquartile range), mean ± standard deviation or number (percentage) of patients. eGFR=estimated glomerular filtration rate, FBG=fasting blood glucose, LDL=low-density lipoprotein, SYNTAX=the synergy between percutaneous coronary intervention with taxus and cardiac surgery, TyG=triglyceride glucose

OP-24.
A Case of Giant Left Atrial Myxoma Presenting With Heart Failure Symptoms

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Myxomas are the most common primary tumors of the heart. Primary cardiac myxomas are most frequently located in the left atrium. Histopathologically, myxomas are generally considered benign. The size of the myxoma primarily determines the clinical symptoms. They may present with cerebral or peripheral embolic findings, as well as with dyspnea due to mitral obstruction, peripheral and pulmonary edema, or arrhythmic symptoms. Additionally, they may rarely cause myocardial infarction due to coronary artery embolization and, when infected, may lead to endocarditis and sepsis. This case report presents a 52-year-old male patient who was admitted with symptoms of heart failure, including shortness of breath and leg swelling.

Myxomas are the most common benign tumors of the heart and are typically located in the left atrium and interatrial septum. They can, although rarely, also be found in the right atrium and ventricles. Clinically, they often present with fever, anemia, and an elevated erythrocyte sedimentation rate, along with systemic embolism and mitral stenosis symptoms.

A 52-year-old male patient with no known comorbidities presented to our clinic with complaints of shortness of breath, leg swelling, and palpitations for the past three months. On physical examination, his temperature was 36.6°C, heart rate 120 bpm, blood pressure 110/70 mmHg, and respiratory rate 24 breaths per minute. A 2/6 systolic murmur was auscultated at the mitral focus. Bilateral inspiratory crackles were heard up to the middle lung zones.

Laboratory findings: creatinine: 0.9 mg/dL, erythrocyte sedimentation rate: 67 mm/h, hemoglobin: 11.9 g/dL, WBC: 12.8×10³/µL, NT-proBNP: 1007 pg/mL, D-dimer: 4617 ng/mL, CRP: 101 mg/L. ECG was consistent with sinus tachycardia. Chest X-ray showed open costophrenic sinuses, pulmonary congestion, and Kerley B lines.

Transthoracic echocardiography revealed a 92×70 mm hyper-echogenic mass originating from the interatrial septum in the left atrium, prolapsing into the left ventricle during diastole, occupying 70% of the left ventricular volume, and obstructing blood flow through the mitral valve. Transesophageal echocardiography confirmed these findings and additionally showed that the mass had a well-defined capsule. Based on these findings, an emergency surgical procedure was planned. The histopathological examination of the excised mass confirmed the diagnosis of cardiac myxoma.

Keywords: Left atrial myxoma, heart failure, surgical procedure

OP-25.
Effect of Nutritional Status, Visseral Adiposity and Renal Function on Central Arterial Stiffness in Patients With Acute Coronary Syndrome

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Objectives: Increased aortic stiffness or arterial distensibility is a major factor in cardiovascular morbidity and mortality in patients with vascular risk factors. Decreased estimated glomerular filtration rate (eGFR), nutritional status and increased visceral adiposity are associated with fatal and non-fatal cardiovascular events. The pathophysiological mechanisms of this association are not fully defined. The aim of this study was: (1) To analyse the central aortic distensibility, comparing several markers as nutritional status and increased visceral adiposity, in subjects with acute coronary syndrome. (2) To study the possible association of aortic distensibility with GFR, prognostic nutrition index (PNI) and visceral adiposity index (VAI).

Material and methods: A total of 158 subjects with acute coronary syndromes (ACS) were included consecutively. Two groups were created according to ACS type: ST elevation myocardial infarction (STEMI) (n=75) and non-ST elevation myocardial infarction (n=83). Central arterial stiffness indices obtained by echocardiographic aortic assessment to determine the aortic distensibility. Prognostic nutritional index (PNI) and visceral adiposity index were calculated according to the previously used formulations. As a marker of renal function, eGFR was classified in 2 ways as the determined values below the cut off points of 60 and 90 ml/min/1.73m². Optimal statistical analysis were used to study the association between aortic distensibility and the parameters of interest.

Results: Patients age was 59±12 years and 65% was male. VAI and PNI values were 3.93±3.61 vs 2.92±5.19; P=0.16 and 32.4±10.4 vs 29.7±8.3; P=0.08 respectively. Nutritional status, visceral adiposity and renal dysfunction severity did not differed according to coronary syndrome type (P>0.05). Only eGFR was significantly correlated with central arterial distensibility (r=0.16, P=0.04). Aortic distensibility was more worse affected in STEMI group according to Non-STEMI group (2.18±0.85 vs 2.49±1.11 cm²/dyn/10³; P=0.03). Only the presence of previous coronary artery disease and presence of hypertension effected aortic distensibility independently (OR: 2.73 (1.27-5.87); P=0.01and 3.20 (1.58-6.53); P=0.001 (Table 1)

Conclusions: Central arterial distensibility is reduced and worse affected in STEMI patients regardless of the presence of other risk factors such as PNI, VAI, DM and CKD (chronic kidney disease). Only the presence of previous coronary artery disease and hypertension were independently associated with worse aortic distensibility.

Keywords: Acute coronary syndrome, arterial stiffness, visseral adiposity, nutritional status, renal function

Tablo 1. Patinents' medical data

	Total (n=158)	STEMI (n=75)	Non-STEMI (n=83)	P value
Men, n (%)	102 (65)	48 (64)	54 (65)	0.89
Age (year)	59±12	60±12	57±13	0.24
Body mass index (kg/m^2)	28.2±4.6	28.1±4.5	28.4±4.7	0.69
Smoke, n (%)	67 (42)	30 (40)	37 (45)	0.56
Diabetes mellitus, n (%)	89 (56)	38 (51)	51 (61)	0.17
Hypertension, n (%)	63 (40)	43 (57)	20 (24)	**0.0001**
Coronary artery diseas, n (%)	105 (67)	61 (81)	44 (53)	**0.0001**
ACEI/ARB, n (%)	131 (83)	66 (88)	65 (78)	0.11
Beta blocker, n (%)	140 (89)	68 (91)	72 (87)	0.44
CaCB, n (%)	31 (20)	13 (17)	18 (22)	0.49
Statins, n (%)	154 (97)	73 (98)	81 (97)	0.92
Total cholesterol (mg/dL)	194±44	193±48	196±40	0.68
LDL (mg/dL)	124±38	125±41	123±34	0.71
Triglyceride (mg/dL)	152±132	150±148	153±117	0.89
Glucose (mg/dL)	145±71	147±57	143±81	0.69
Urat (mg/dL)	5.6±1.6	5.8±1.6	5.4±1.5	0.11
Albumin (g/L)	41±4	40±4	43±4	**0.001**
Ejection fraction (%)	49±11	46±11	51±10	**0.001**
VAI	3.40±4.52	3.93±3.61	2.92±5.19	0.16
PNI	31.0±9.4	32.4±10.4	29.7±8.3	0.08
Aortic distensibility (cm^2/dyn/10^3)	2.34±1.01	2.18±0.85	2.49±1.11	**0.03**
eGFR< 90 (mL/dak/1, 73m^2)	70 (44)	35 (47)	35 (42)	0.57
eGFR< 60 (mL/dak/1, 73m^2)	20 (13)	12 (16)	8 (10)	0.23

Data are shown as mean±standard deviation or n (%).eGFR=estimated glomerular filtration rate, VAI= visceral adiposity index, PNI=Prognostic nutritional index, CaCB=calcium channel bloker, LDL=low-density lipoprotein, ACEI=angiotensin converting enzyme inhibitor, ARB=angiotensin receptor bloker

OP-26.
Association Between Cerebrovascular Events And Blood Markers in Non-Valvular Atrial Fibrillation Patients

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Introduction: Non-valvular atrial fibrillation (NVAF) is a significant condition that increases the risk of cerebrovascular events, such as ischemic stroke. Various clinical and laboratory parameters, including age, albumin, fibrinogen, and D-Dimer, are known to play critical roles in the prediction of stroke risk in these patients. Understanding these relationships can help guide preventative strategies and improve patient outcomes. Aim: This study aimed to investigate the association between NVAF-related stroke risk and routine blood parameters, including albumin, D-Dimer, fibrinogen, and other hematological markers.
Methods: A total of 92 NVAF patients were included in the study. Patients were divided into two groups: those with stroke (n=26) and those without stroke (n=66). Routine blood tests [red blood cell count (RBC, 10^6/μL), hemoglobin (HGB, g/dL), hematocrit (HCT, %), mean platelet volume (MPV, fL), white blood cell count (WBC, 10^3/μL), neutrophil-to-lymphocyte ratio (NLR, %), platelet count (PLT, 10^3/μL), plateletcrit (PCT, %), eosinophil count (EOS, 10^3/μL), basophil count (BASO, 10^3/μL), albumin (g/dL), fibrinogen (mg/dL), and D-Dimer (μg/L)] were analyzed retrospectively. Multiple binary logistic regression analysis was used to evaluate predictors of stroke risk.
Results: The mean age of the stroke group was 69.12±14.83 years, while the non-stroke group was younger (52.78±17.35 years, $P<0.01$). Albumin, RBC, HGB, HCT, and EOS values were significantly lower in the stroke group ($P<0.01$), while NLR, fibrinogen, D-Dimer, and WBC levels were significantly higher ($P<0.01$) (Table 1). Logistic regression analysis showed that older age, elevated fibrinogen, increased D-Dimer, and low albumin levels were independent predictors of stroke risk in NVAF patients (Table 2)
Discussion: The findings of this study highlight the importance of routine blood parameters, such as albumin, fibrinogen, and D-Dimer, in assessing stroke risk among NVAF patients. Elevated fibrinogen and D-Dimer levels reflect hypercoagulability, which is a key factor in thrombus formation and stroke. Low albumin levels may indicate systemic inflammation and malnutrition, further contributing to cerebrovascular risk. Age remains a significant, non-modifiable risk factor, reinforcing the need for vigilant monitoring and preventative strategies in elderly NVAF patients.
Keywords: Non-valvular atrial fibrillation, cerebrovascular events, albumin, fibrinogen, D-Dimer, age

Table 1. Comparison of blood parameters and demographic characteristics in hospitalized NVAF patients

Parameters	Group 1 (n=26)	Group 2 (n=66)	P value
Age (years)	69.12 ± 14.83	52.78 ± 17.35	**<0.01****
Albumin (g/dL)	3.1 ± 0.4	4.2 ± 0.5	**<0.01****
RBC (10^6/µL)	4.1 ± 0.5	4.8 ± 0.6	**<0.01****
HGB (g/dL)	12.8 ± 1.2	14.3 ± 1.4	**<0.01****
HCT (%)	38.5 ± 3.2	42.7 ± 3.5	**<0.01****
MPV (fL)	10.4 ± 0.9	10.1 ± 0.8	0.12
Fibrinogen (mg/dL)	405 ± 65	320 ± 50	**<0.01****
D-Dimer (µg/L)	1.8 ± 0.5	0.9 ± 0.3	**<0.01****
WBC (10^3/µL)	9.7 ± 2.1	7.4 ± 1.8	**<0.01****
NLR (%)	5.3 ± 1.4	2.8 ± 1.0	**<0.01****
PLT (10^3/µL)	240 ± 60	270 ± 50	0.08
PCT (%)	0.21 ± 0.05	0.22 ± 0.04	0.65
EOS (10^3/µL)	0.1 ± 0.04	0.2 ± 0.06	**<0.05***
BASO (10^3/µL)	0.02 ± 0.01	0.03 ± 0.01	0.15

RBC=Red blood cell count, HGB=Hemoglobin, HCT=Hematocrit, MPV=Mean platelet volume, WBC=White blood cell, NLR=Neutrophil to lymphocyte ratio, PLT=Platelet, PCT=Plateletcrit, EOS=Eosinophil, BASO=Basophil. *Comparison is statistically significant P<0.05, ** Comparison is statistically significant P<0.01.

Table 2. Multiple binary logistic regression analysis of NVAF-related stroke

Variable	B	Sig.	Exp(B)	95% C.I. (Lower)	95% C.I. (Upper)
Age (ref: 15–34)					
35–64	1.25	**0.04***	2.63	1.01	6.88
65 or higher	2.75	**<0.01****	15.65	5.48	45.50
Albumin (ref: 3.4–5.5 g/dL)					
3.4 or lower	-1.45	**<0.01****	0.23	0.10	0.51
D-Dimer (ref: 200–1000 µg/L)					
1000 or higher	1.85	**<0.01****	6.38	2.70	15.11
MPV (ref: 6–10 fL)					
10 or higher	0.45	0.15	1.57	0.85	2.90
Fibrinogen (ref: 200–300 mg/dL)					
300 or higher	2.10	**<0.01****	8.15	3.42	19.41
Constant	-3.20	**<0.01****	0.04		

OP-27.
Nutritional Indices as Predictors of No-Reflow Phenomenon After Saphenous Vein Graft Percutaneous Coronary Intervention in Patients Presenting With Non-St Elevation Myocardial Infarction

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Background: Percutaneous coronary intervention (PCI) of saphenous vein grafts (SVGs) is associated with an increased risk of complications, particularly the no reflow phenomenon and distal embolization. The association between nutritional status and adverse outcomes in individuals with cardiovascular disease has been documented. We aimed to explore the correlation between the Prognostic Nutritional Index (PNI) and Controlling Nutritional Status (CONUT) scores as malnutrition indices and the occurrence of the no reflow phenomenon following PCI of SVGs.
Methods: The study included 199 patients who underwent PCI for SVG disease at our tertiary cardiovascular center between January 2020 and January 2023, presenting with non-ST elevation myocardial infarction (NSTEMI). The study included 199 patients who underwent PCI for SVG disease in our tertiary cardiovascular center January 2020 to January 2023. Patients were divided into Group 1 without no-reflow phenomenon (n=169) and Group 2 with no-reflow phenomenon (n=30) following PCI and were then compared based on PNI and CONUT scores.
Results: The mean age was 66 years. The incidence of the no reflow phenomenon in the study group was 15%. The NR group had lower lymphocyte count, hemoglobin, glomerular filtration rate, albumin, PNI score, and left ventricular ejection fraction (EF), and higher creatinine, CRP, and CONUT scores (Table 1). Coronary angiography and procedural characteristics revealed that intraluminal thrombus, thrombectomy, use of glycoprotein IIb/IIIa receptor antagonist, degenerated SVGs, stent length, and stent diameter were more common in the NR group (Table 2). Univariate regression analysis showed several significant variables (EF, hemoglobin, glomerular filtration rate, CRP, intraluminal thrombus, thrombectomy, degenerated SVGs, stent diameter, and length) (Table 3). Multivariate analysis confirmed that PNI and CONUT scores were independent predictors of the NR phenomenon (PNI: OR: 0.835, P=0.002; CONUT: OR: 1.397, P=0.039). PNI demonstrated superior predictive capability. ROC analysis identified the optimal cut-off values: PNI 45 (AUC: 0.794) with 87% sensitivity and 66% specificity, and CONUT (AUC: 0.702) with 60% sensitivity and 75% specificity. EF, degenerated SVG count, and stent diameter were also independent predictors of NR (Table 4).
Conclusion: Our findings suggest that PNI and CONUT scores are independent predictors of the no-reflow phenomenon in patients undergoing SVG interventions. The simplicity and efficiency of calculating the PNI and CONUT scores can make them valuable risk assessment tools for stratifying patients at risk of experiencing the no-reflow phenomenon before SVG interventions.
Keywords: Saphenous vein graft failure, percutaneous intervention, nutritional indices

Table 1. Demographic and clinical characteristics of the analyzed patients

Variables	Overall	No-reflow (+) (n=30)	No-reflow (-) (n=169)	P value
Age, years, median (IQR)	66 (59-72)	67 (56-71)	65 (60-72)	0.731
Gender, male, n (%)	167 (83.9%)	23 (76.7%)	144 (85.2%)	0.241
Hypertension, n (%)	160 (80.8 %)	25 (83.3%)	135 (80.4%)	0.703
Diabetes mellitus, n (%)	91 (45.7%)	13 (43.3%)	78 (46.2%)	0.775
CAD, n (%)	19 (9.5%)	3 (10%)	16 (9.5%)	0.927
CKD, n (%)	38 (19.1%)	6 (20%)	32 (18.9%)	0.891
Time interval since CABG, years	6 (3-8)	6 (3-9.75)	6 (3-8)	0.714
AF, n (%)	22 (11.1%)	3 (10%)	19 (11.2%)	0.841
Medication, n (%)				
Acetyl salicylic acid	186 (93.5%)	26 (86.7%)	160 (94.7%)	0.102
Beta-blockers	189 (95.5%)	28 (96.6%)	161 (95.3%)	0.759
RAAS inhibitors	144 (72.7%)	22 (73.3%)	122 (72.6%)	0.936
Statin	150 (75.4%)	23 (76.7%)	127 (75.1%)	0.859
Oral antidiabetic	46 (23.4%)	5 (16.7%)	41 (24.6%)	0.347
Insulin	44 (22.2%)	7 (23.3%)	37 (22%)	0.874
Oral anticoagulant	15 (7.5%)	4 (13.3%)	11 (6.5%)	0.167
GIIb-IIIa inhibitors	34 (17.1%)	12 (40%)	22 (13%)	**0.004**
Clopidogrel	124 (62.3%)	18 (60%)	106 (62.7%)	0.657
Ticagrelor	58 (29.1%)	9 (30%)	49 (29%)	0.809
Prasugrel	15 (7.5%)	3 (10%)	12 (7.1%)	0.536
Laboratory data, median (IQR)				
WBC, $\times 10^9$/L	8.4 (6.15-10.30)	8.2 (5.88-10.4)	8.4 (6.2-10.3)	0.926
Neutrophils, $\times 10^9$/L	5.1 (3.60-6.60)	5.5 (3.83-7.2)	5.1 (3.6-6.5)	0.486
Lymphocytes, $\times 10^9$/L	1.60 (1.15-2.20)	1.3 (0.83-2)	1.7 (1.2-2.2)	**0.044**
Monocytes, $\times 10^9$/L	0.6 (0.50-0.90)	0.55 (0.5-0.9)	0.6 (0.5-0.9)	0.772
Hemoglobin, g/dL	13.2 (12.00-14.10)	12.25 (11.35-14.08)	13.2 (12-14.1)	**0.045**
Platelets, $\times 10^9$/L	239 (198-267)	241.5 (195.25-268.5)	238 (198-266)	0.757
Serum creatinine, mg/dL	0.96 (0.87-1.35)	1.11 (0.93-2.07)	0.96 (0.87-1.2)	**0.025**
GFR	74 (52.80-92.00)	62.1 (31.45-84.85)	75.5 (53-94.2)	**0.030**
Uric acid, mg/dL	5.7 (5.40-6.40)	6.4 (5.9-6.9)	5.65 (5.45-6.33)	0.113
Total cholesterol, mg/dL	191 (153.5-215)	195 (159.25-204)	191 (153-216)	0.587
HDL cholesterol, mg/dL	42 (38-50)	45 (38-55)	42 (38-50)	0.257
Triglycerides, mg/dL	137 (96-217)	114 (89.25-195.75)	143 (97-222)	0.133
LDL cholesterol, mg/dL	113 (85-138)	112 (89.25-136.75)	113 (85-138)	0.706
Albumin, g/dL	3.6 (3.20-4.00)	3.3 (2.9-3.75)	3.6 (3.2-4)	**0.020**
CRP	10 (3.00-42.00)	20 (5.75-52.75)	9 (3-40)	**0.045**
CONUT score	1 (0-3)	3 (1-6)	1 (0-3)	**<0.001**
PNI score	48 (39.50-52.00)	38.75 (33.13-43.88)	49 (42-52)	**<0.001**
EF	60 (50-65)	50 (41.3-60)	65 (50-65)	**<0.001**

AF=Atrial fibrillation, CABG=coronary artery bypass grafting, CAD=coronary artery disease, CKD=chronic kidney disease, CONUT=Controlling Nutritional Status, CRP=C-reactive protein, GFR= glomerular filtration rate, HDL=high-density lipoprotein, LDL=low-density lipoprotein, PNI=Prognostic Nutritional Index, RAAS=renin-angiotensin-aldosterone system, WBC=white blood cells

Table 2. Coronary angiographic findings and procedural characteristics of the analyzed patients

Variables	Overall	No-reflow (+)	No-reflow (-)	P value
Narrowed saphenous vein graft to, n (%)				
Left anterior descending artery	14 (7%)	2 (6.7%)	12 (7.1%)	0.932
Diagonal artery	22 (11.1%)	4 (13.3%)	18 (10.7%)	0.666
Circumflex artery	71 (35.7%)	12 (40%)	59 (34.9%)	0.592
Right coronary artery	89 (44.7%)	12 (40%)	77 (45.6%)	0.572
Lesion site, n (%)				
Osteal	26 (13.1%)	5 (16.7%)	21 (12.4%)	0.525
Proximal	66 (33.2%)	10 (33.3%)	56 (33.1%)	0.983
Middle	62 (31.2%)	10 (33.3%)	52 (30.8%)	0.676
Distal	43 (21.6%)	5 (16.7%)	38 (22.5%)	0.536
Intraluminal thrombus, n (%)	41 (20.6%)	14 (46.7%)	27 (16%)	**0.004**
Drug-eluting stent, n (%)	141 (70.9%)	20 (66.7%)	121 (71.6%)	0.494
Predilation, n (%)	101 (50.8%)	17 (56.7%)	84 (49.7%)	0.607
Postdilation, n (%)	47 (23.6%)	8 (26.7%)	39 (23.1%)	0.943
Stent diameter, mm	3 (3.00-3.50)	3.5 (3-4)	3 (3-3.5)	**0.010**
Stent length, mm	24 (21-29)	28 (23-33)	24 (21-29)	**0.010**
Distal protection device usage, n (%)	8 (4%)	2 (6.7%)	6 (3.6%)	0.394
Thrombectomy, n (%)	10 (5%)	4 (13.3%)	6 (3.6%)	**0.019**
Degenerated SVG count	1 (0-1)	1 (1-2)	0 (0-1)	**<0.001**

SVG=Saphenous vein graft

Table 3. Univariate predictors of no-reflow phenomenon after saphenous vein graft percutaneous coronary intervention

	Univariate			
Variables	**OR**	**95% CI lower**	**95% CI upper**	**P**
EF	0.936	0.904	0.968	**<0.001**
Acetyl salicylic acid	0.366	0.105	1.274	0.114
Intraluminal thrombus	4.602	2.013	10.521	**<0.001**
GIIb-IIIa	4.455	1.891	10.494	**<0.001**
Thrombectomy	4.179	1.104	15.821	**0.035**
Degenerated SVG count	2.280	1.487	3.498	**<0.001**
Lymphocytes	0.683	0.374	1.248	0.215
Hemoglobin	0.785	0.611	1.010	**0.059**
Serum creatinine, mg/dL	1.277	1.007	1.621	**0.044**
GFR	0.985	0.973	0.997	**0.018**
Albumin	0.917	0.856	0.982	**0.014**
CRP	1.007	1.001	1.014	**0.035**
CONUT score	1.468	1.233	1.750	**<0.001**
PNI score	0.875	0.828	0.924	**<0.001**
Stent diameter, mm	3.481	1.442	8.399	**0.006**
Stent length, mm	1.087	1.031	1.146	**0.002**

CONUT=Controlling Nutritional Status, CRP=C-reactive protein, EF=ejection fraction, GFR=glomerular filtration rate, PNI=Prognostic Nutritional Index, SVG=saphenous vein graft

Table 4. Multivariate Model 1 (PNI) and Model 2 (CONUT) for prediction of no-reflow phenomenon

	Multivariate for PNI		Multivariate for CONUT	
Variables	**OR, 95% CI (lower-upper)**	**P**	**OR, 95% CI (lower-upper)**	**P**
Age	1.007, 0.954-1.063	0.799	1.004, 0.951-1.060	0.888
EF	0.951, 0.911-0.997	**0.044**	0.958, 0.918-0.998	**0.048**
Diabetes mellitus	0.844, 0.304-2.337	0.743	0.761, 0.281-2.061	0.592
Hemoglobin	0.982, 0.658-1.464	0.928	1.021, 0.691-1.508	0.918
GFR	0.999, 0.982-1.017	0.943	0.996, 0.980-1.013	0.676
CRP	0.993, 0.982-1.004	0.231	0.995, 0.983-1.007	0.381
Intraluminal thrombus	1.088, 0.177-10.143	0.941	1.230, 0.129-11.703	0.857
GIIb-IIIa inhibitor use	2.119, 0.202-22.232	0.531	1.692, 0.162-17.653	0.660
Thrombectomy	1.610, 0.222-11.674	0.638	1.899, 0.291-12.395	0.503
Degenerated SVG count	0.111, 0.018-0.666	**0.016**	0.145, 0.027-0.734	**0.028**
Stent diameter (mm)	3.239, 1.049-10.003	**0.041**	3.525,1.179-10.534	**0.024**
Stent length (mm)	1.048, 0.978-1.123	0.183	1.054, 0.985-1.129	0.128
CONUT			1.397, 1.018-1.918	**0.039**
PNI	0.835, 0.747-0.934	**0.002**		

CONUT=Controlling Nutritional Status, CRP=C-reactive protein, EF=ejection fraction, GFR=glomerular filtration rate, PNI=Prognostic Nutritional Index, SVG=saphenous vein graft

OP-28.
The Role of White Blood Cell Levels in Predicting Ejection Fraction in Young ACS Patients

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Background: Elevated white blood cell (WBC) levels have been associated with clinical outcomes in patients with acute coronary syndrome (ACS). The inflammatory response triggered by myocardial necrosis during ACS is a well-established contributor to adverse outcomes. However, there is limited evidence on the prognostic value of WBC as an inflammatory marker in younger ACS patients. This retrospective study aimed to assess the ability of WBC levels, measured at the time of emergency department admission before coronary angiography, to predict the risk of reduced left ventricular ejection fraction (LVEF) at discharge in patients under 50 years of age.

Method: This retrospective study included 546 consecutive patients under the age of 50 who were diagnosed with ACS between 2021 and 2024. Patients with ACS were included if they had a culprit lesion in at least one coronary artery and underwent stent implantation or balloon angioplasty. Demographic data, clinical characteristics, and laboratory parameters were obtained from patient medical records. WBC levels measured in the emergency department prior to coronary angiography were recorded. Patients with WBC levels >10,000/mm³ were classified as having leukocytosis. Patients were categorized into two groups based on their diagnosis: ST-elevation myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI). LVEF was classified into two categories: preserved EF (≥50%) and reduced EF (<50%). WBC levels and other clinical characteristics were compared between the preserved EF and reduced EF groups within both STEMI and NSTEMI subgroups. The relationship between WBC levels and LVEF was analyzed using Pearson correlation. Logistic regression analysis was performed to identify predictors of reduced EF. Additionally, ROC curve analysis was conducted to determine the optimal WBC cutoff value for predicting reduced EF, and the sensitivity and specificity of this cutoff value were calculated.

Results: A total of 546 patients were included in the study, comprising 335 diagnosed with STEMI and 211 with NSTEMI. Among STEMI patients, 41.5% were discharged with reduced EF, compared to 16.9% of NSTEMI patients. STEMI patients discharged with reduced EF had significantly higher WBC levels and a higher prevalence of leukocytosis compared to those with preserved EF, whereas no such differences were observed between the reduced and preserved EF groups in NSTEMI patients (Table 1). A significant negative correlation between WBC levels and EF values was identified in STEMI patients (P<0.001) (Fig. 1), whereas no such association was observed in NSTEMI patients (Fig. 2). Logistic regression analysis revealed that elevated WBC levels were a strong predictor of reduced EF at discharge in STEMI patients (OR: 1.131; 95% CI: 1.082–1.183; P<0.001) (Table 2). In contrast, WBC levels did not predict reduced EF in NSTEMI patients. ROC curve analysis determined that the optimal WBC cutoff value for predicting reduced EF in STEMI patients was ≥12.15 (AUC: 0.627, P<0.001), with a sensitivity of 60.7% and a specificity of 60.6% (Fig. 3).

Conclusion: This study highlights that elevated WBC levels, as an inflammatory marker, are a strong predictor of reduced EF, especially in young STEMI patients. WBC can be considered a simple, cost-effective, and practical parameter for predicting reduced EF in STEMI patients.

Keywords: Acute coronary syndrome, ejection fraction, white blood cell level

Table 1. Clinical characteristics of STEMI and NSTEMI patients according to preserved and reduced ejection fraction

Parameter	STEMI EF≥50% (n=196)	STEMI EF<50% (n=139)	P-value (STEMI)	NSTEMI EF≥50% (n=177)	NSTEMI EF<50% (n=34)	P-value (NSTEMI)
Age (years)	45.25 ± 4.37	39.71 ± 4.48	0.111	45.31 ± 4.34	45.85 ± 4.41	0.507
Male gender, n (%)	171 (87.2%)	123 (88.5%)	0.732	150 (84.7%)	31 (91.2%)	0.325
History of MI/Stent/CABG, n (%)	20 (10.2%)	19 (13.7%)	0.330	19 (10.7%)	7 (20.6%)	0.109
DM, n (%)	27 (13.8%)	29 (20.9%)	0.223	29 (16.4%)	7 (20.6%)	0.551
HT, n (%)	30 (15.3%)	21 (15.1%)	0.960	44 (24.9%)	14 (41.2%)	0.051
Smoking, n (%)	63 (32.1%)	54 (38.8%)	0.478	49 (27.7%)	10 (29.4%)	0.697
HbA1c (%)	6.16±1.52	6.23±1.45	0.754	6.46±1.84	6.23±1.53	0.655
Creatinine (mg/dL)	0.88±0.21	0.92±0.37	0.135	0.89±0.41	1.04±0.51	0.434
Hemoglobin (g/dL)	14.15±1.55	14.25±1.28	0.540	14.15±1.55	14.25±1.28	0.547
WBC count (×10³/µL)	12.66±4.28	14.66±4.68	**<0.001**	10.90±3.08	11.56±3.59	0.266
Leukocytosis, n (%)	137 (69.8%)	122 (87.7%)	**<0.001**	99 (55.9%)	21 (61.7%)	0.529
Platelet count (×10³/µL)	279.56±76.58	285.43±70.98	0.477	271.98±68.92	250.08±66.21	0.089
Total cholesterol (mg/dL)	194.40±42.08	187.43±52.89	0.289	200.88±55.29	190.55±68.67	0.467
LDL cholesterol (mg/dL)	119.51±34.48	118.56±45.14	0.865	122.24±45.03	127.04±58.86	0.679

Data are shown as mean± standard deviation or n (%). CABG=Coronary Artery Bypass Grafting, DM=Diabetes Mellitus, HbA1c=Hemoglobin A1c, HDL=High-Density Lipoprotein, HT=Hypertension, LDL=Low-Density Lipoprotein, LVEF=Left Ventricular Ejection Fraction, MI=Myocardial Infarction, NSTEMI=Non-ST-Elevation Myocardial Infarction, STEMI=ST-Elevation Myocardial Infarction, WBC=White Blood Cell

Table 2. Predictors of Reduced Ejection Fraction in STEMI and NSTEMI Patients Based on Logistic Regression Analysis Results

	STEMI		NSTEMI	
Parameter	**OR (95% CI)**	**P value**	**OR (95% CI)**	**P value**
Age	0.961 (0.915–1.009)	0.113	1.032 (0.941–1.132)	0.506
Male gender	1.124 (0.576–2.194)	0.732	1.860 (0.531–6.517)	0.332
WBC count	1.131 (1.082–1.183)	**<0.001**	1.066 (0.953–1.192)	0.266
DM	1.440 (0.799–2.595)	0.225	1.323 (0.526–3.326)	0.552
HT	0.985 (0.537–1.804)	0.960	2.116 (0.986–4.539)	0.054
Smoking	1.369 (0.858–2.186)	0.188	0.974 (0.429–2.213)	0.950
History of MI/stent/CABG	1.393 (0.713–2.721)	0.331	2.156 (0.827–5.619)	0.116
Total cholesterol (mg/dL)	0.997 (0.991–1.003)	0.272	0.997 (0.998–1.006)	0.463
LDL cholesterol (mg/dL)	0.999 (0.993–1.006)	0.860	1.002 (0.992–1.012)	0.676

CABG=Coronary Artery Bypass Grafting, CI=Confidence Interval, DM=Diabetes Mellitus, HT=Hypertension, LDL=Low-Density Lipoprotein, MI=Myocardial Infarction, OR=Odds Ratio, STEMI=ST-Elevation Myocardial Infarction, NSTEMI=Non-ST-Elevation Myocardial Infarction, WBC=White Blood Cell

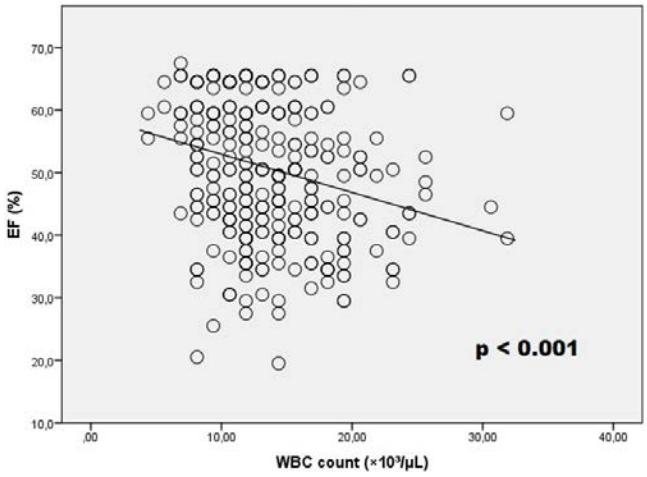

Fig. 1. The correlation between ejection fraction and white blood cell count in STEMI patients.

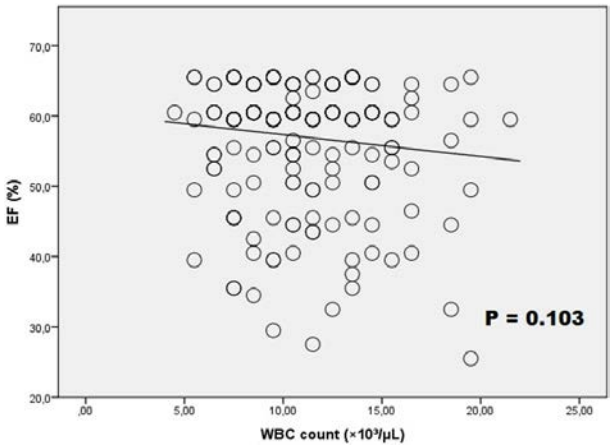

Fig. 2. The correlation between ejection fraction and white blood cell count in STEMI patients.

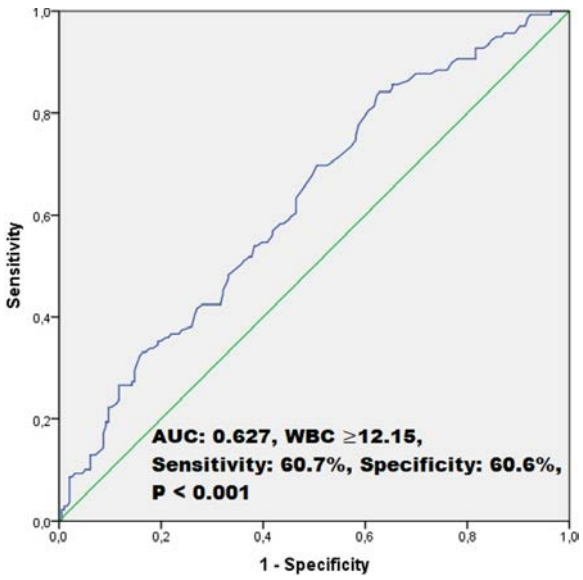

Fig. 3. ROC analysis identified a WBC threshold of ≥12.15 for predicting reduced ejection fraction (EF) in patients with STEMI (AUC: 0.627, P<0.001). This threshold demonstrated a sensitivity of 60.7% and a specificity of 60.6%.

OP-29.
Myocarditis Associated With Trabectedin

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Summary: Cancer treatment-related cardiovascular toxicity encompasses cardiac dysfunction and cardiovascular complications. The ideal timing for protection against cardiovascular toxicity should occur after diagnosis but before the start of treatment. Screening for ideal protection involves medical history, clinical examination, ECG, cardiac imaging, and laboratory tests. During cancer treatment, many cardiovascular complications may arise due to medications, including coronary artery disease, heart valve diseases, arrhythmias, pericarditis-myocarditis, and thromboembolic events. In our case, a 42-year-old woman developed myocarditis due to trabectedin.

Introduction: A 42-year-old female patient underwent surgery for endometrial cancer two years ago and achieved remission. One year after remission, she was diagnosed with ovarian cancer, and chemotherapy was initiated. Initially, she received gemcitabine + docetaxel treatment every 3 weeks. Later, based on imaging results, she began pazopanib therapy for 9 months. In December 2024, after a PET-CT scan, her treatment regimen was updated, and 3 cycles of trabectedin were planned instead of pazopanib. Before changing chemotherapy agents, an echocardiogram (ECHO) revealed an ejection fraction (EF) of 65%, mild mitral insufficiency, and an E/A ratio <0.8. Five days after receiving the first dose of trabectedin, the patient presented to our clinic with severe abdominal pain and shortness of breath. The ECG taken in the emergency department (Fig. 1) showed ST elevation in the inferior leads. Laboratory results showed: HGB: 14, WBC: 10,000, CRP: 136, CRE: 1.2, TROP: 3700, CK-MB: 2, Na: 123, K: 3.5. The patient underwent angiography (Figs. 2, 3), which showed a plaque in the RCA coronary artery, but no lesions were found in other arteries. Given her ongoing dyspnea and signs of heart failure, medication was initiated. Upon further review of her medical history, there was no history of infection. Follow-up ECGs showed increasing ST elevation, which became evident in all leads (Fig. 4). Subsequent echocardiograms showed EF: 20%, global hypokinesia, moderate left ventricular hypertrophy (LVH), PAB: 35 mmHg, mild mitral insufficiency, and a decrease in tissue Doppler velocities. Pancytopenia, a known side effect of trabectedin, was observed. Despite inotropic support, the patient entered cardiogenic shock, and vital stabilization could not be achieved. Unfortunately, the patient was clinically declared dead.

Discussion: Trabectedin is currently used in the treatment of ovarian cancer and liposarcoma. Although it's exact mechanism of action remains unclear, it has been shown to block the DNA binding of the oncofetal transcription factor FUS-CHOP, reversing the transcriptional program in myxoid liposarcoma. Trabectedin promotes differentiation by reversing the oncogenic phenotype in cells. Another proposed mechanism involves direct covalent DNA damage, leading to alterations in DNA base pairs that are not fully understood. Known side effects of trabectedin include pancytopenia, jaundice, bruising, nausea, and increased susceptibility to infections. However, there is limited research on its cardiac toxicity.

Conclusion: Chemotherapeutic agents can lead to cardiac dysfunction and cardiovascular disease. Therefore, before starting chemotherapy, patients should undergo an ECG, cardiac biomarkers, and, most importantly, transthoracic echocardiography. Throughout chemotherapy, patients should be periodically evaluated from a cardiac perspective. Even after achieving remission, the risk of cardiac complications should not be overlooked.

Keywords: Myocarditis, trabectedin, endometrial cancer

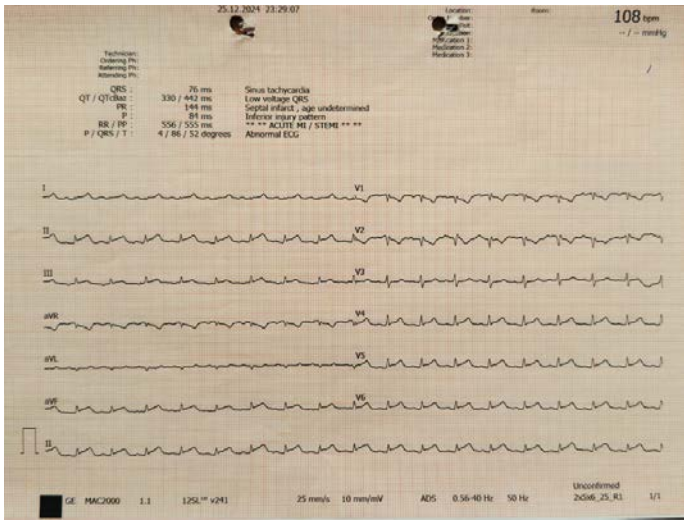

Fig. 1. **Inferior elevation ECG of the patient at the time of admission to the emergency department.**

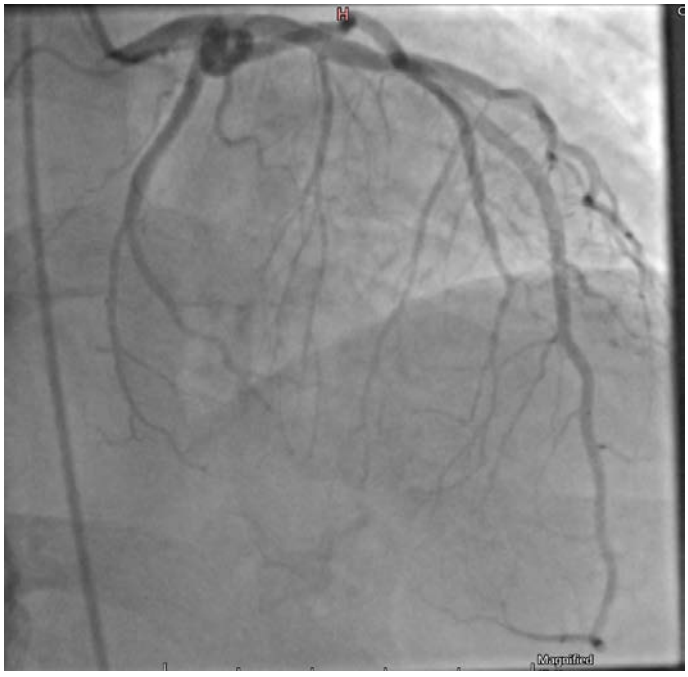

Fig. 2. **CAG left shot; LAD+Lcx image.**

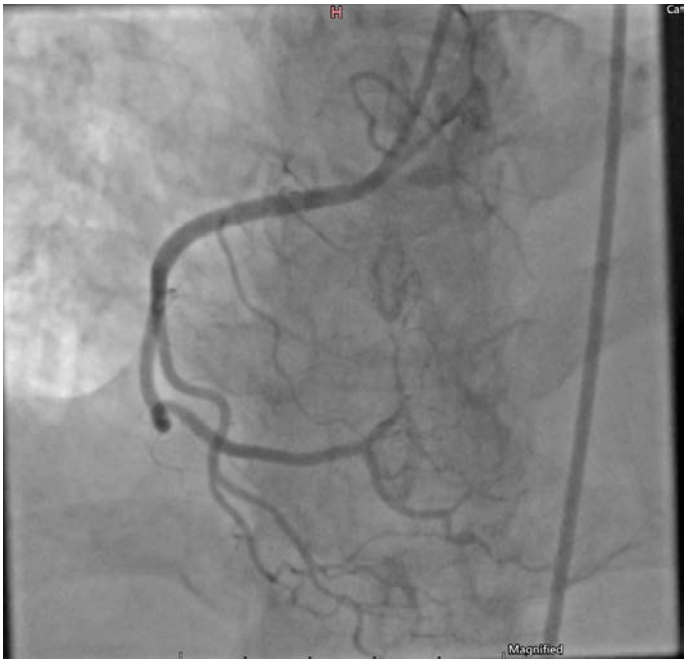

Fig. 3. **CAG right shot RCA image.**

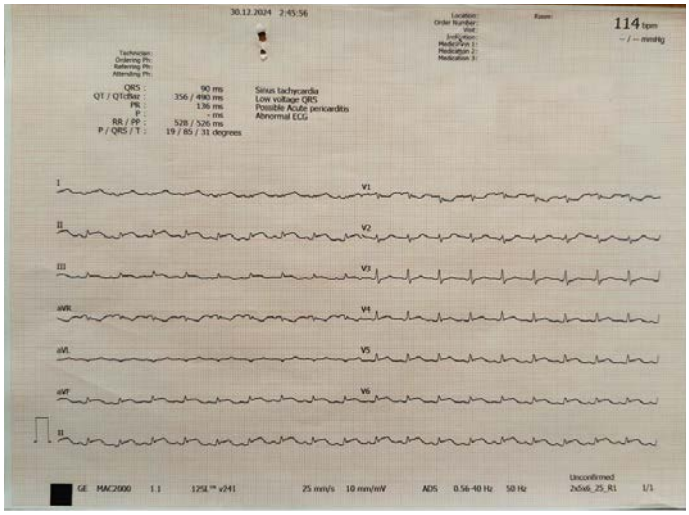

Fig. 4. **CAG widespread ST elevation ECG that developed during the patient's follow-up.**

OP-30.
Are Incidental Non-Cardiac Radiological Findings Detected in Preoperative Thoracic CT a Barrier for Cardiac Surgeons?

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Objectives: Thoracic computed tomography (CT) is a modality utilized by some surgeons to detect aortic calcifications prior to primary cardiac surgery. This study aimed to identify incidental radiological abnormalities detected on thoracic CT scans performed to evaluate calcified aortas and assess their impact on the clinical course of patients.
Methods: Demographic characteristics and surgical data of patients undergoing cardiac surgery were recorded. Patients with a prior diagnosis of malignancy were excluded. Thoracic CT scans were retrospectively analyzed for non-cardiac pathologies. The time from CT findings to surgery, hospital and intensive care unit stay, and postoperative complications were compared.
Results: A total of 616 patients, including 184 women (29.9%) and 432 men (70.1%), were included in this study. Isolated CABG was performed in 61% of the patients, isolated valve surgery in 20%, and combined CABG and valve surgery in 19%. Among the 93 patients (15.1%) with normal chest X-rays, incidental pathological findings were identified on CT scans. Noncardiac pathology was detected in 310 (50.3%) thoracic CT scans, with the most common findings being emphysema/chronic bronchitis (18.8%) and pulmonary edema (10.7%). Thoracic CT findings were classified into five categories based on severity: normal (49.7%), clinically insignificant findings (32.5%), findings requiring follow-up (9.7%), infection (5.5%), and severe findings such as malignancy (2.6%). Postoperative complications, including stroke (P=0.013), acute kidney injury (P=0.012), postoperative pneumonia (P=0.001), and in-hospital mortality (P=0.001), were significantly higher in the infection group. In the two groups with severe findings (infection and malignancy), the time from the CT findings to surgery was significantly longer than that in the other groups (P=0.002).
Conclusions: Even in asymptomatic patients undergoing cardiac surgery, radiological findings suggestive of infection are a risk factor for in-hospital mortality and complications. Our study demonstrated the importance of confirming these risks with thoracic CT as they may not be distinguishable from chest X-rays alone.
Keywords: cardiac surgery, incidental findings, thorax CT

OP-31.
From Colon to Heart: Enterococcus Faecalis Infective Endocarditis and Colorectal Neoplasia

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Introduction: Streptococcus gallolyticus (formerly Streptococcus bovis) is known as an infectious agent strongly associated with colorectal malignancies. In contrast, whether Enterococcus faecalis is associated with colorectal lesions through a similar mechanism is less known? Although Enterococcus faecalis is one of the important causes of infective endocarditis, it is also considered a microorganism found in the intestinal flora and its connection with colonic pathologies has not been sufficiently investigated.
Case Report: A 72-year-old male patient was admitted to our clinic with complaints of fever, fatigue, and shortness of breath. The patient's medical history included a TAVI procedure performed in 2019. Physical examination revealed fever (38.2°C) and tachycardia (110 beats/min). Because the patient had a history of bioprosthetic valve implantation and fever, four sets of blood cultures were taken with a preliminary diagnosis of infective endocarditis and evaluated with transthoracic echocardiography (TTE). TTE revealed tachycardia, normal left ventricular diameters, but global hypokinesia and eccentric hypertrophy of the left ventricle. Ejection fraction (EF) was measured as 22% by the modified Simpson method. 2nd degree mitral insufficiency and minimal aortic insufficiency were detected. When the aortic valve was evaluated after TAVI, a maximum systolic gradient of 20 mmHg and an average of 12 mmHg was observed. Although no obvious vegetation was observed on TTE, TEE was planned due to the growth of Enterococcus faecalis in the patient's blood cultures. In the TEE evaluation, a mass appearance compatible with vegetation, measuring 7 ·12 mm, was detected on the bioprosthetic aortic valve (Fig. 1). First degree aortic insufficiency and second degree mitral insufficiency were observed.
On the 4th day of follow-up, the patient developed complaints of loss of orientation, difficulty in finding words, and numbness in the right arm. Cranial MRI showed a few millimeter diffusion restrictions in the right frontal and left parietooccipital regions, consistent with cardioembolic infarction. PET-CT imaging was performed due to the possibility of septic embolism. PET-CT revealed a polypoid lesion with increased FDG uptake at the sigmoid colon level. (Fig. 2). Colonoscopy revealed a 20 mm pedunculated polyp in the sigmoid colon and polypectomy was performed. Pathological examination revealed low-grade mucosal neoplasia (low-grade adenoma).
Discussion: TTE is often used as a first-line imaging modality in the diagnosis of infective endocarditis, but its sensitivity is limited in bioprosthetic valve endocarditis. Advanced imaging methods such as TEE and PET-CT are pivotal to confirm the diagnosis and determine complications. PET-CT may be a useful tool, especially in patients with suspected septic embolism and malignancy.
Current guidelines recommend early surgery in cases of heart failure, uncontrolled infection, and septic embolism. However, there is no definitive recommendation regarding the timing of surgery in patients with suspected malignancy.
In this case, early surgery could have been considered due to ischemic embolism, but since suspicion of colon malignancy may increase the risk of postoperative infective endocarditis, colonoscopy and pathological evaluation were performed first to clarify the etiology. After the definitive diagnosis, the patient was evaluated

by the Heart Team and a decision for surgery was made. However, the patient preferred to continue the treatment process in an external center.

The diagnosis and treatment process of infective endocarditis is quite complex and requires a multidisciplinary approach. Multimodality imaging modalities, especially PET-CT, play an important role in detecting malignancies and determining surgical timing. In patients with Enterococcus faecalis growth, colorectal neoplasms should be kept in mind and evaluated with further examinations. In cases where colon neoplasm is detected, it is of great importance that the timing of surgery is evaluated on an individual patient basis and that this process is managed together with the Heart Team.

Conclusion: This case is important as it demonstrates the association of Enterococcus faecalis infective endocarditis and colorectal neoplasia developing after TAVI. Screening for colorectal malignancy is recommended in patients diagnosed with infective endocarditis, especially when Enterococcus faecalis is isolated. In this direction, further studies are needed.

Keywords: Enterococcus faecalis, infective endocarditis, colorectal neoplasia

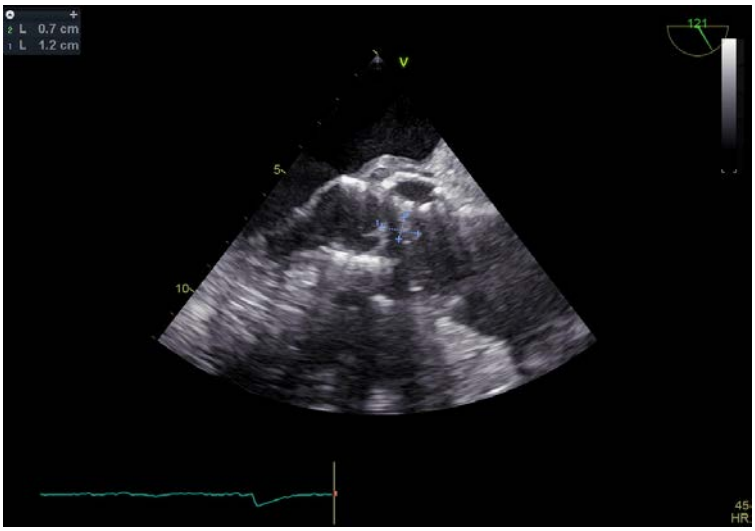

Fig. 1. Transesophageal echocardiography, A vegetation measuring 7×12 mm is observed on the bioprosthetic valve.

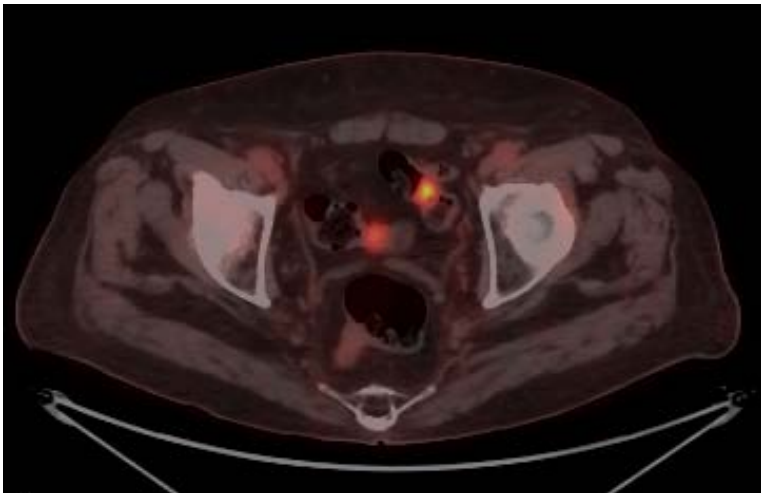

Fig. 1. Oncological PET-CT, Increased FDG uptake with polypoid appearance was observed at the sigmoid colon level.

OP-32.
Management of Spontaneous Coronary Artery Dissection

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Introduction: Spontaneous coronary artery dissection (SCAD) is characterized by the disruption of the intimal layer of the coronary artery wall, leading to impaired coronary flow due to intramural hematoma caused by trauma, atherosclerosis, or iatrogenic factors. It commonly occurs in young women, during pregnancy, and in individuals without typical atherosclerotic changes in the coronary arteries. SCAD accounts for 1% to 4% of all acute coronary syndrome (ACS) cases and 25% of ACS cases in women under 50 years of age.
Case Report: A 45-year-old male patient presented to the outpatient clinic with retrosternal, compressive chest pain that had been present for 5 months and worsened with exertion. Laboratory values did not reveal any pathological findings.The echocardiogram showed an ejection fraction (EF) of 65%, mild mitral insufficiency, left ventricular hypertrophy, and stage-1 diastolic dysfunction.The patient was scheduled for elective coronary angiography. The admission ECG showed sinus rhythm with ventricular extrasystoles (Fig. 1). The patient had a known history of hypertension and central obesity, and 8 years ago had undergone primary PCI due to acute inferior MI (Fig. 2). Coronary angiography revealed a 90-99% distal dissection thrombus in the left anterior descending (LAD) artery (Fig. 3), a total dissection thrombus in the right coronary artery (RCA) (Fig. 4), and a normal circumflex (CX) artery. Given the inferior MI on the ECG, the RCA was intervened first, followed by balloon angioplasty (2.0·20 mm) and stent implantation distal 2.75·16 mm, proximal 3.5·20 mm, and 4.0·16 mm bare-metal stents (Fig. 5). Elective intervention on the LAD was planned. One week later, during the LAD procedure, a 2.75·20 mm drug-eluting stent (DES) was implanted in the distal lesion, and a 3.5·20 mm bare-metal stent was placed in the proximal lesion, followed by post-dilatation with a 4.0·12 mm balloon, and medical follow-up was advised (Fig. 6). On the current admission, coronary angiography showed that the proximal LAD stent was open, with a dissection at the distal end of the stent, a total dissection in the distal instent, and a double-lumen appearance post-stent (Fig. 7). The CX artery was dissected, with a total occlusion of CXOM1 (Fig. 8). The RCA had open proximal and distal stents, with dissection and a double-lumen appearance between the stents (Fig. 9). After angiography, the patient was evaluated by a cardiology and cardiovascular surgery team, and a follow-up coronary angiography and intravascular ultrasound (IVUS) were planned. The control angiography showed that the proximal LAD stent remained open, the distal instent had 30-50% stenosis, and there was a dissection between the stents (Fig. 10). The D1 and CX arteries were dissected, but CXOM1 was open (Fig. 11). The RCA stents were open, but there was dissection between the stents (Fig. 12). IVUS imaging of the left main coronary artery (LMCA) and LAD confirmed that the LMCA was normal, while SCAD starting from the distal part of the LAD stent was confirmed. Since coronary flow was still present, the patient was recommended for medical follow-up, and the procedure was concluded. Upon discharge, the patient was prescribed DAPT, beta-blockers, ACE inhibitors, high-dose statins, and colchicine and referred to the rheumatology clinic with a suspected vasculitis diagnosis. The patient tested positive for HLA B52, and treatment with methotrexate and methylprednisolone was started. The patient was discharged without additional pathology during follow-up.
Keywords: Spontaneous coronary artery dissection, intravascular ultrasound, treatment

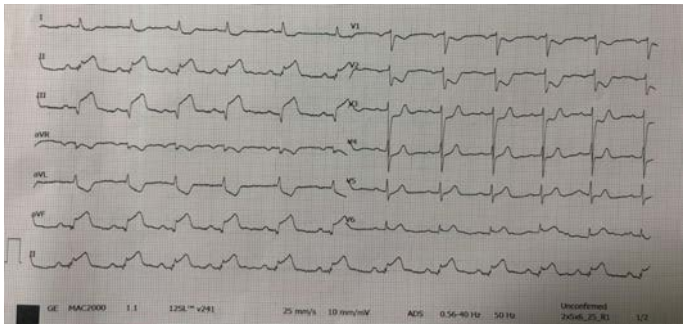

Fig. 1. Inferior miyocardial infaction.

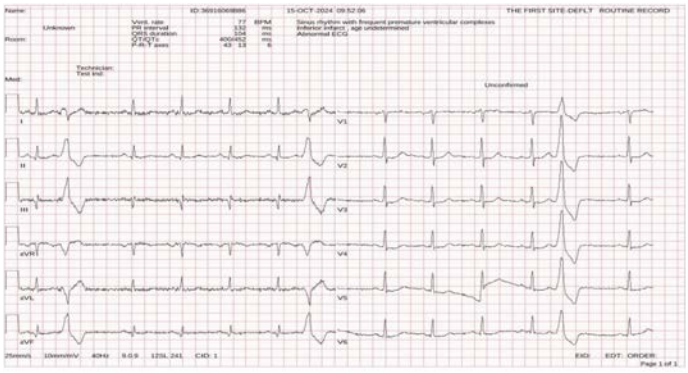

Fig. 2. ECG of Non-STEMI patient.

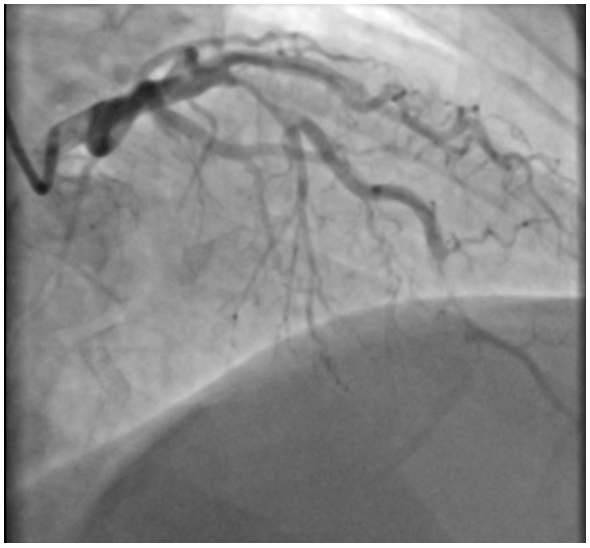

Fig. 3. LAD spontaneous coronary dissection.

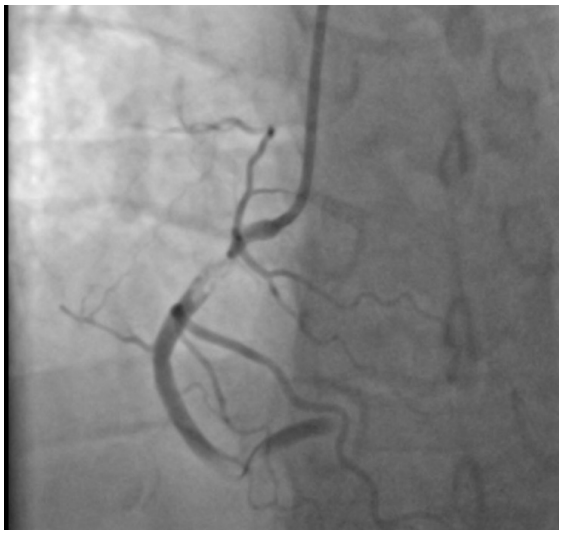

Fig. 4. RCA spontaneous coronary dissection.

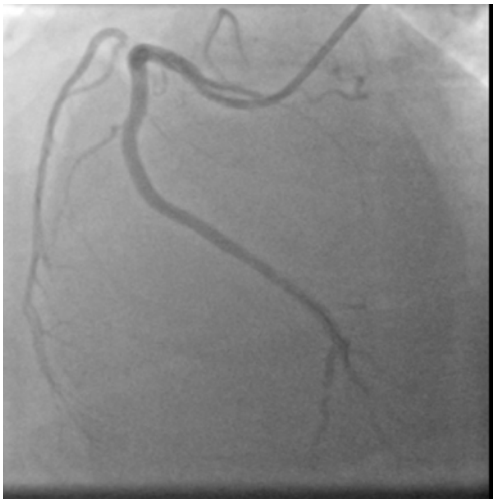

Fig. 5. RCA after stenting.

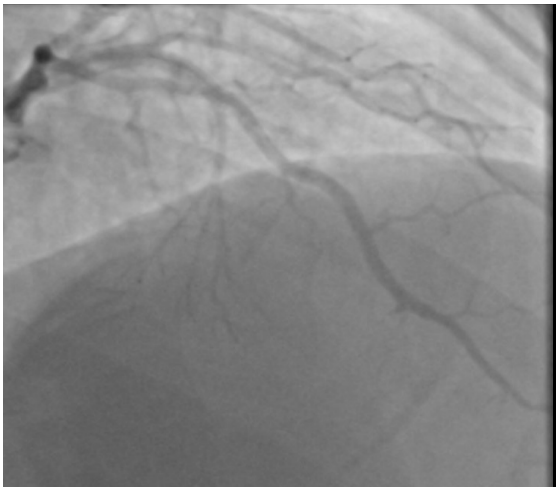

Fig. 6. LAD after stenting.

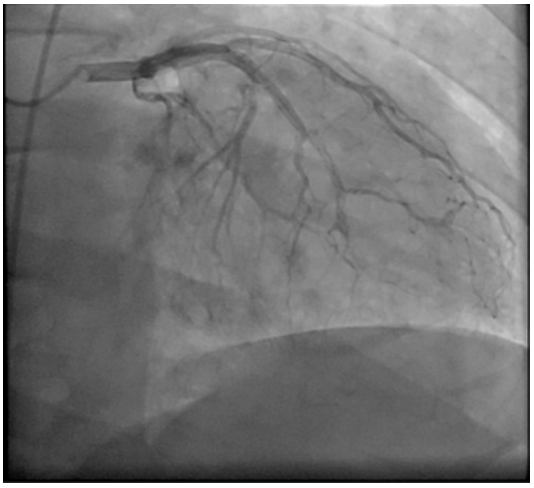

Fig. 7. Coronary angiographic image of dissected LAD.

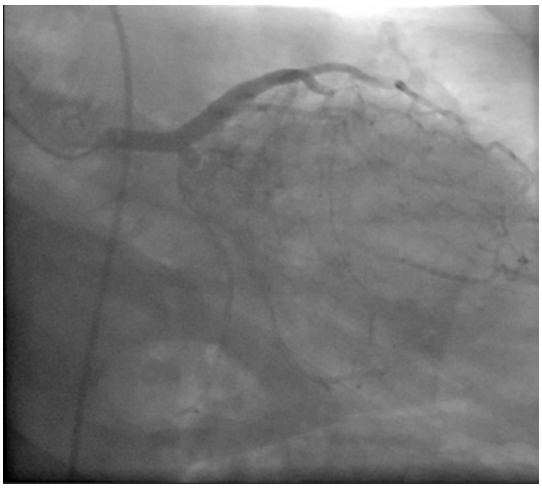

Fig. 8. Coronary angiographic image of occluded CX.

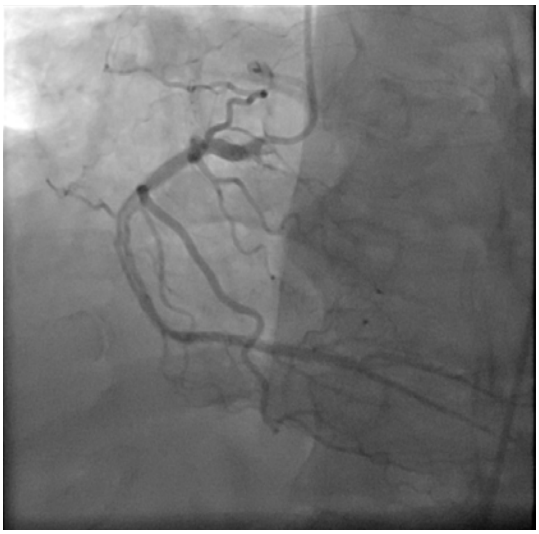

Fig. 9. Coronary angiographic image of dissected RCA.

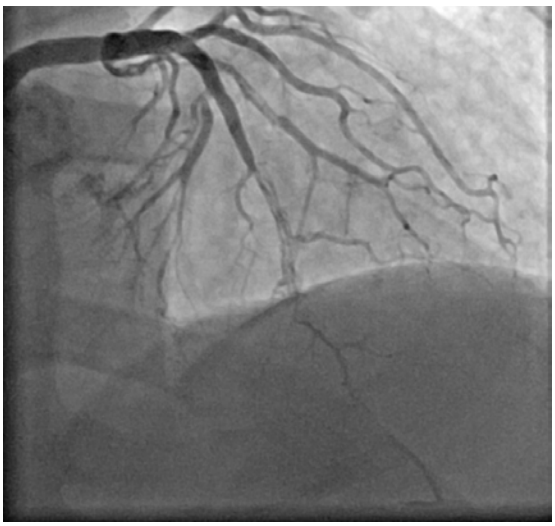

Fig. 10. Control coronary image of LAD.

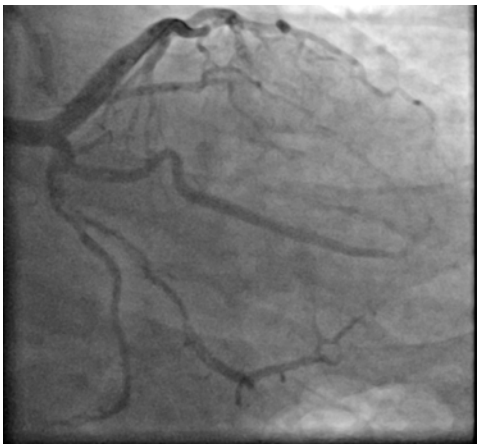

Fig. 11. Control coronary image of CX.

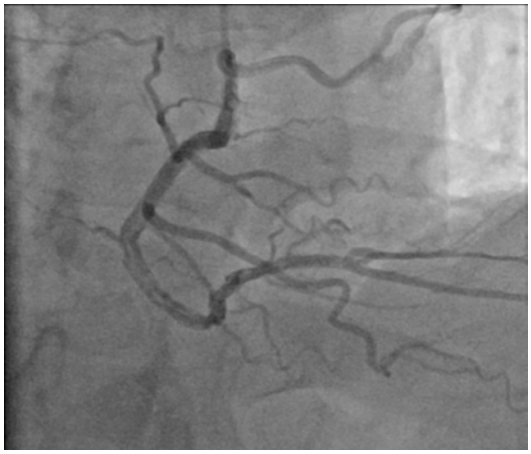

Fig. 12. Control image of RCA.

OP-33.
Relationship Between Persistent Left Superior Vena Cava and Horseshoe Kidney

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We use echocardiography very frequently in our daily cardiology practice. The small clues we obtain as a result of the imaging we perform lead us to undiagnosed diseases or conditions. Left persistent superior vena cava, which is a condition that we can suspect and diagnose as a result of echocardiographic imaging, is a relatively common congenital anomaly (0.3-0.5%). This anomaly, which is often detected incidentally and does not cause any problems, needs to be investigated. A 34-year-old male patient had no complaints when he applied to the cardiology clinic. He had severe shortness of breath when he was young and this shortness of breath completely disappeared after an angiographic procedure was performed on him when he was 7 years old. He had no complaints until this age. Since they did not have any medical records from that period, he applied to the cardiology clinic for information about what could have been done and for a general cardiological examination. It was learned that he had a horseshoe kidney and an accessory spleen in his anamnesis. The patient's ECG was in sinus rhythm, normal axis, and heart rate was 69/min. Kidney and liver functions and hemogram were observed at normal levels in the blood samples taken. Heart diameter measurements were observed at normal levels in echocardiography. Minimal-1 Degree TR was observed. PaP (systolic) was calculated as 25 mmHg. Pulmonary blood flow velocity was observed to be increased by 1.5 m/sec. The most striking finding was that the coronary sinus was significantly widened on the parasternal long-axis imaging and its diameter was measured as 1.2·1.2 cm. Thoracic angiography CT was performed considering that the patient may have a persistent left superior vena cava. The imaging results showed that there was a persistent left superior vena cava anomaly, the right superior vena cava was in its normal position and there was no connection between the two venous structures. It was determined that the persistent left superior vena cava drained into the right atrium via the coronary sinus. Diagnosing persistent left superior vena cava anomaly is important for possible central venous catheter interventions and possible pacemaker implantations. Knowing the presence of this anatomy in patients who will undergo the procedure can increase the success of the procedure and reduce possible complications. Although the presence of this anomaly is mostly innocent on its own, its association with congenital heart diseases and rotation anomalies of embryological origin is important. Another anomaly that is relatively common in the population, horseshoe kidney (0.1-0.3%) is usually seen with inferior vena cava anomalies. It is also rare to see it with persistent left superior vena cava. Although the association of these two conditions seems to be independent events, it may be a situation that should be considered in terms of the presence of other congenital anomalies. In a study conducted by Ichikawa et al., it was determined that superior vena cava anomalies were seen more frequently in individuals with horseshoe kidney anomaly. It is thought that more studies are needed.

Keywords: Persistent left superior vena cava, horseshoe kidney anomaly, congenital anomalies

OP-34.
On the Trail of Cholesterol Crystals: A Journey Through a Diagnostic Maze

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Introduction: Cholesterol Embolization Syndrome (CES) is a complication that results from an advanced degree of atherosclerosis affecting multiple organs. CES can occur after invasive cardiovascular procedures, but it can also occur spontaneously. The clinical manifestations and symptoms are heterogeneous in nature. The definitive diagnosis is made through a positive biopsy of the affected organ. This case discusses a patient with CES that developed following renal angiography.
Case Report: A 65-year-old female patient with a history of hypertension, diabetes, and heart failure presented to the emergency department with complaints of dyspnea, leg swelling, and pain in the epigastric region and lower extremities. Her heart rate was 80 bpm, and blood pressure was 144/110 mmHg. Physical examination revealed moderate pretibial edema and cyanotic appearance of the feet (Fig. 1).
Upon taking a detailed history, the patient revealed that she had undergone renal angiography to investigate the cause of her hypertension. After the procedure, she began experiencing pain in her feet, and they developed a cyanotic appearance. An echocardiogram showed an ejection fraction of 35%, with moderate regurgitation of the mitral and tricuspid valves, and a pulmonary artery pressure of 28 mmHg.
Laboratory tests revealed a hemoglobin level of 8.3 g/dL, platelets of 102,000/mm³, and CRP of 77.2 mg/L. Lipid profile, C3, C4, ANA, ANA profile, ANCA profile, and cryoglobulin tests were requested for cholesterol embolization syndrome, but no abnormalities were detected.
During follow-up, the patient developed severe abdominal pain and nausea. Contrast-enhanced CT angiography was performed to exclude mesenteric ischemia. Although mesenteric ischemia was not detected, aneurysmal changes in the splenic and renal arteries, widespread atherosclerotic plaques in the abdominal and thoracic aorta, and atherosclerotic ulcers were observed (Fig. 2).
The patient was then evaluated with fundus optical coherence tomography, given the preliminary diagnosis of CES, due to the appearance of livedo reticularis in the extremities and progressive renal function loss. Cholesterol plaques in the retinal arterioles and retinal hemorrhage in the temporal region were detected (Fig. 3). A skin biopsy was performed on the cyanotic areas of the toes, which confirmed the diagnosis of CES, with typical cholesterol crystals detected in the arterial lumen.
Discussion: Cholesterol Embolization Syndrome occurs as a result of the embolization of atherosclerotic plaque contents (especially cholesterol crystals) from proximal large arteries to distal small and medium-sized arteries. This embolization causes end-organ damage through both mechanical obstruction and inflammatory response. CES often develops after interventional procedures such as cardiac catheterization, aortography, and vascular surgery. It commonly presents with renal failure, gastrointestinal ischemia, skin lesions, and neurological symptoms.
Although no significant inflammatory response was observed in our patient, CES was suspected based on her clinical history, laboratory findings, and skin and eye findings. A skin biopsy was performed to confirm the diagnosis.
There is no specific treatment for CES; the main goal is supportive care and the prevention of new embolization episodes. Some evidence suggests that statin use may prevent recurrence of CES. However, there is no clear

consensus on the use of antiplatelet agents, ACE inhibitors/ARBs, corticosteroids, or immunosuppressive treatments. Anticoagulant or thrombolytic therapy should only be continued if there is another indication. Despite intensive supportive treatment, the clinical outcome in our patient was fatal due to renal failure and cardiovascular complications.

Conclusion: This case emphasizes the diagnostic and management challenges associated with cholesterol embolization syndrome following invasive vascular procedures. High clinical suspicion and biopsy are crucial for diagnosis. Treatment is primarily supportive, with the goal of controlling atherosclerosis. It is important to remember that, despite correct diagnosis and intensive supportive treatment, the atherosclerotic burden in this patient group is high, and the clinical outcome can be fatal.

Keywords: Cholesterol embolization syndrome, renal angiography, invasive vascular procedures

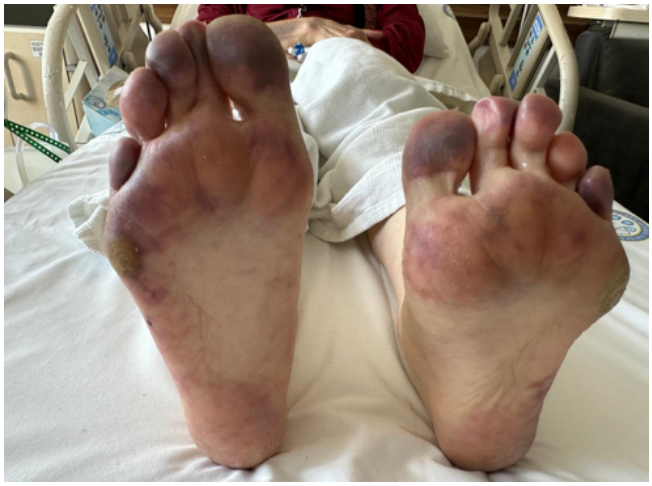

Fig. 1. Rivedo Retikülaris in the toes.

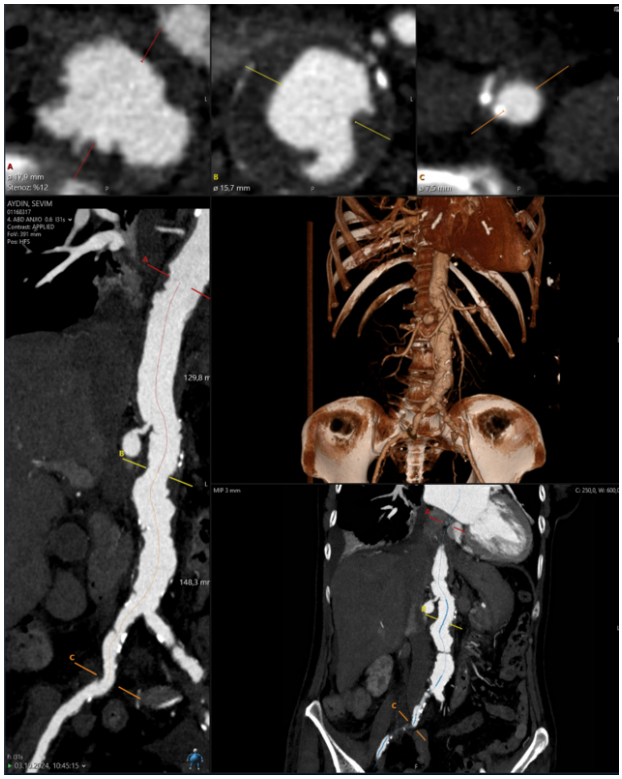

Fig. 2. Coronary CT angiography showing progressive atherosclerotic plaques.

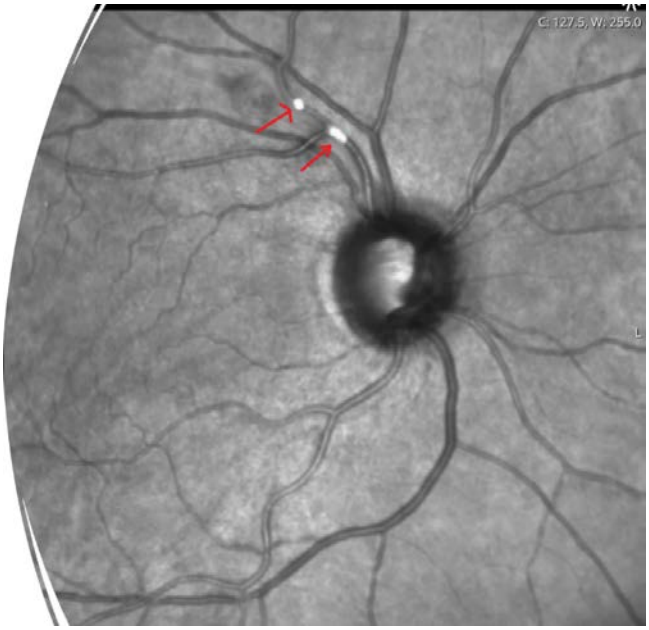

Fig. 3. Retinal OCT showing cholesterol crystals (Hollenhorst plaques).

OP-35.
Effects of Anatomical Variations on Radial Artery Spasm in Radial Artery Interventions

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Objectives: The radial artery is the first preferred intervention site in angiographic procedures. Due to reasons such as fewer bleeding-related complications, shorter hospital stays, and patient comfort, the radial artery is the recommended and commonly used primary access route in angiographic interventions. However, radial artery spasm is a complication that occurs in radial artery interventions, which is not present when using the femoral artery. Three main factors play a role in the development of this complication. The first is the materials and medications used, the second is patient-related factors, and the third is operator-related factors. In our study, we aimed to investigate the frequency and causes of radial artery spasm and to demonstrate the effect of radial artery anatomy on this.

Methods: Patients who underwent radial angiography at our hospital in the last 7 months were retrospectively analyzed. Radial artery spasm was defined as more than 75% narrowing in the vessel lumen and pain that prevented further procedure. The patients' age, gender, referring clinic, sheath diameter, catheter diameter, use of single or multiple catheters, whether angioplasty was performed, heart rate during the procedure, and smoking habits were recorded. In addition, anatomical variations, abnormal origin of the radial artery, presence of a radioulnar loop, and tortuous course of the artery were documented. The effects of these factors, which are known from previous studies to potentially influence radial artery spasm, were analyzed. Patients who developed hematomas in the arm or required crossover to the femoral artery or the other arm due to radial artery spasm were also recorded.

Results: The total number of patients included in the study was 672. The mean age of the patients was 58±13 years. 409 (60.6%) patients underwent angiography due to acute coronary syndrome. Radial artery spasm occurred in 31 (4.6%) patients. Crossover to the femoral or other radial artery was required in 9 (1.3%) patients; in other patients, the procedure was successfully completed with catheter maneuvers or medications. A radioulnar loop was detected in 21 (3.1%) patients. An abnormally originated radial artery was identified in 7 (1%) patients (Table 1). In the group with radial artery spasm, female gender, radioulnar loop, and abnormal radial artery origin were significantly more frequent, while other data were similar (Table 2). The rate of abnormal origin of the radial artery was significantly higher in patients who required crossover (5 [55.6%] vs. 2 [0.3%]; P<0.001).

Conclusions: According to the results of our study, the main risk factors for radial artery spasm were female gender and anatomical variations of the radial artery (Table-3). However, in most patients, the spasm was resolved through catheter maneuvers or medications, allowing the procedure to continue. The biggest obstacle to continuing the procedure was found to be an abnormally originating radial artery. Factors such as sheath and catheter size, age, and heart rate, which have been considered risk factors in many studies, did not yield significant results in our study.

Keywords: Radial artery spasm, anatomical variation, radial artery intervention

Table 1. **Patient Charecteristics**

Parameter	Data
Number of patients	672
Age	58±13
Gender (female)	259 (38.5%)
ACS clinic	409 (60.6%)
Sheat size (>5f)	491 (73.1%)
Catheter size (>5f)	479 (71.3%)
Policatheter use	488 (72.6%)
Angioplasty	264 (39.3%)
Heart rate (>70 bpm)	292 (43.5%)
Smoking	253 (37.6%)
Radial artery spasm	31 (4.6%)
Crossover	9 (1.3%)
Radioulnar loop	21 (3.1%)
Anormal origin	7 (1%)
Tortuosity	128 (19%)
Hematoma	8 (1.2%)

Table 2. **Comparison of groups**

Parameter	Radial artery spasm+	Non-spasm	P value
Age (year), median (IQR)	65 (55-79	56 (48-68)	**0.02**
Gender (female)	19 (61.3%)	240 (37.4%)	**0.01**
ACS clinic	15 (48.4%)	394 (61.5%)	0.1
Sheat size (>5f)	19 (61.3%)	472 (73.6%)	0.1
Catheter size (>5f)	19 (61.3%)	460 (71.8%)	0.2
Policatheter use	19 (61.3%)	469 (73.2%)	0.1
Angioplasty	13 (41.9%)	251 (39.2%)	0.8
Heart rate >70 bpm	17 (54.8%)	275 (42.9%)	0.1
Smoking	12 (38.7%)	241 (37.6%)	0.5

Table 3. **Regression Analysis**

	Univariate analysis			Multivariate analysis		
Parameter	**Odds ratio**	**95 % CI**	**P**	**Odds ratio**	**95 % CI**	**P**
Age	1	1,003-1,059	0.02			
Gender (female)	2,64	1.26- 5.54	0.01	3.2	1.31-8.61	0.01
ACS clinic	1,7	0.82-3.5	0.1			
Radioulnar loop	21.44	8.18-56.18	<000.1	38.75	13.17-113.97	<0.001
Anormal origin	153	17.81-1324	<0.001	209	22-1935	<0.001
Tortuosity	1,61	0.5-4.7	0.3			
Sheat (>5f)	1,7	0.8-3.7	0.1			
Catheter(>5f)	1,6	0.7-3.3	0.2			
Policatheter use	1,7	0.8-3.6	0.1			
Angioplasty	0,8	0.4-1.8	0.7			
Heart rate >70 bpm	0,6	0.3-1.2	0.1			
Smoking	0,9	0.4-2	0.9			

OP-36.
STEMI or an Illusion? An Unexpected Cardiac Reflection of Post-CPR Pneumomediastinum

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Introduction: ST-segment elevation is one of the most important electrocardiographic findings of acute myocardial infarction (MI). However, rare clinical conditions such as pneumomediastinum can also cause similar ST-segment changes, complicating the diagnosis.In this case report, we present a patient initially evaluated with a preliminary diagnosis of STEMI but later diagnosed with pneumomediastinum.
Case Report: A 63-year-old male patient presented to the emergency department with a sensation of chest pressure.The patient was transferred to a district hospital by the emergency team, where the initial electrocardiography (ECG) showed no ST-segment elevation,and patient's general condition was evaluated as stable.However, after the patient developed cardiac arrest, cardiopulmonary resuscitation (CPR) was performed, he was intubated, and mechanical ventilation was initiated.Post-CPR and post-intubation ECG showed ST-segment elevation in leads V1-V6 and DII-aVF, and the patient was transferred to our center with a preliminary diagnosis of STEMI.
Emergency laboratory tests revealed a troponin-I level of 2072.6 ng/L, WBC of $21.53 \cdot 10^3/\mu L$, and an eGFR of 60 mL/min. Transthoracic echocardiography (TTE) showed an ejection fraction of 50%, minimal mitral and tricuspid regurgitation, and a systolic pulmonary artery pressure of 30 mmHg. Urgent coronary angiography (CAG) was performed, revealing no critical stenosis. During follow-up, swelling on both sides of the patient's neck and subcutaneous emphysema detected by palpation led to an urgent thoracic computed tomography (CT) scan. CT revealed bilateral pneumothorax, extensive pneumomediastinum, and subcutaneous emphysema. The patient underwent tube thoracostomy performed by the thoracic surgery department. Follow-up showed regression of the pneumomediastinum and ECG findings.
Discussion: Pneumomediastinum is a rare condition characterized by the accumulation of air within mediastinal tissues, which can cause various ECG abnormalities, including ST-segment elevation. Cases of pneumomediastinum mimicking STEMI have been reported in the literature. The possible mechanisms underlying these electrocardiographic abnormalities include positional changes of the heart and altered conduction due to the presence of mediastinal air. In our patient, pneumomediastinum was attributed to mechanical ventilation and barotrauma following CPR. The most significant indicator of this was the absence of ST elevation upon admission to the emergency department, followed by the emergence of ST-segment changes after CPR and intubation. Additionally, the absence of significant coronary artery stenosis on angiography and the preserved ejection fraction of 50% in echocardiography further support pneumomediastinum as the primary cause. From a differential diagnosis perspective, other potential causes such as MINOCA (myocardial infarction with non-obstructive coronary arteries), myocarditis, Takotsubo cardiomyopathy, coronary artery spasm, pulmonary embolism, and acute pericarditis should be considered. However, given the clinical and echocardiographic findings, along with the timeline of events following mechanical ventilation, pneumomediastinum was determined to be the most likely diagnosis.
Conclusion: Pneumomediastinum is a rare condition that can present as STEMI, particularly in patients who undergo traumatic CPR and are exposed to barotrauma during mechanical ventilation. Careful interpretation

of clinical assessment, laboratory findings, and imaging studies is crucial to prevent misdiagnosis and unnecessary invasive procedures. This case highlights that pneumomediastinum can mimic STEMI through ECG changes and highlights the importance of clinical awareness.

Keywords: Cardiopulmonary resuscitation, pneumomediastinum, acute myocardial infarction

OP-37.
Intervention of Calcific Left Main Coronary Artery Lesion With Rotational Atherectomy

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Rotational atherectomy is a technique used to treat coronary artery lesions with a high degree of calcification. This technique is particularly used in severely calcified lesions that cannot be treated with standard balloon angioplasty or stent placement. Rotating diamond-tipped catheter is used to reduce calcified plaques (Fig. 1). This device rotates at high speed to break up plaque into microparticles, which are then removed by the reticuloendothelial system. Indications are preferred in lesions that are densely calcified, circular on IVUS imaging, >270°, have a thickness of >0.5 mm, or have calcification in a segment longer than 5 mm, and where the stent or balloon cannot be advanced or full expansion cannot be achieved. Its use is not recommended in soft, non-calcified plaques, and in vessels at risk of perforation or dissection. 77-year-old male with a diagnosis of hypertension, who applied to an external center with chest pain and underwent coronary angiography, and was referred to us after severe calcific LMCA, D1, IM, and RCA lesions were detected (Fig. 2). The patient was considered high risk for CABG surgery due to his comorbidities. In the evaluation, LMCA intervention was planned after RCA percutaneous coronary intervention. Echocardiographic findings are 65% ejection fraction, mild mr and tr. In the IVUS examination performed during the procedure, 360° calcification was observed in the LMCA (Fig. 3) and a plaque modification was planned with rotablator.

After rotaablation with 1.75 mm Burr (Fig. 4), dilatation was provided with balloon PTCA. Intervention with DEB to IM was performed after LAD-D1 bifurcation PCI with crush technique and a drug-eluting stent was implanted from the LAD to the LMCA and postdilatation was applied with NC balloons (Fig. 5). In the control IVUS examination, it was seen that the stents had full expansion and the procedure was terminated.

73-year-old male with hypertension and type 2 diabetes, was admitted to the emergency room with angina complaints and was hospitalized with a diagnosis of acute coronary syndrome due to increased troponin values. Echocardiographic findings are 45% ejection fraction, mild ar and mr, moderate tr. CABG was recommended after coronary angiography showed LMCA 90%, LAD and CX non-critical, RCA 70-90% lesions (Fig. 6), but later the heart team made a council and decided to perform PCI due to the high surgical risk.After RCA intervention, LMCA was evaluated with IVUS and MLA was measured as 2.16 mm² with 270-360° calcification (Fig. 7) and LMCA rotaablation was planned. After successful atherectomy with 2 mm burr (Fig. 8), PTCA was performed with NC balloons and a drug-eluting stent was implanted in the LMCA. Full stent expansion was achieved after postdilatation (Fig. 9).

Conclusion: Although surgical treatment is primarily recommended due to the difficulty of percutaneous procedures in calcific LMCA lesions, patients with high surgical risk can also be treated percutaneously in line with the developments in plaque modification methods. Rotational atherectomy technique mainly aims to reduce calcification in calcific plaques and thus provide optimal stent patency, and in selected cases, it increases the success of the procedure and can prevent the risk of restenosis.

Keywords: Rotational atherectomy, calcified left mail coronary stenosis, optimal stent patency

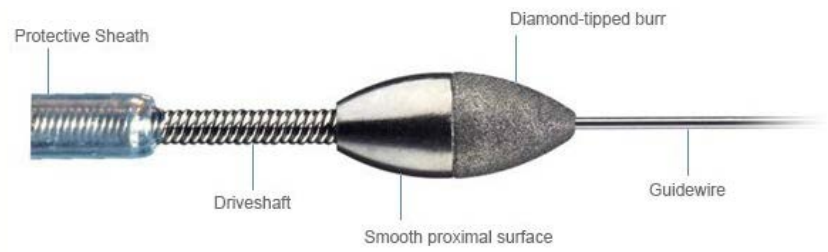

Fig. 1. **Rotational atherectomy catheter.**

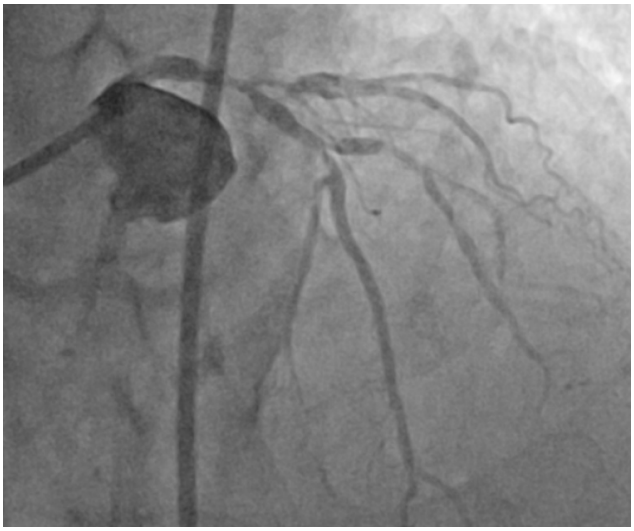

Fig. 2. **Angiographic image shows severe calcified lesions on LMCA and LAD-D1 bifurcation (First patient).**

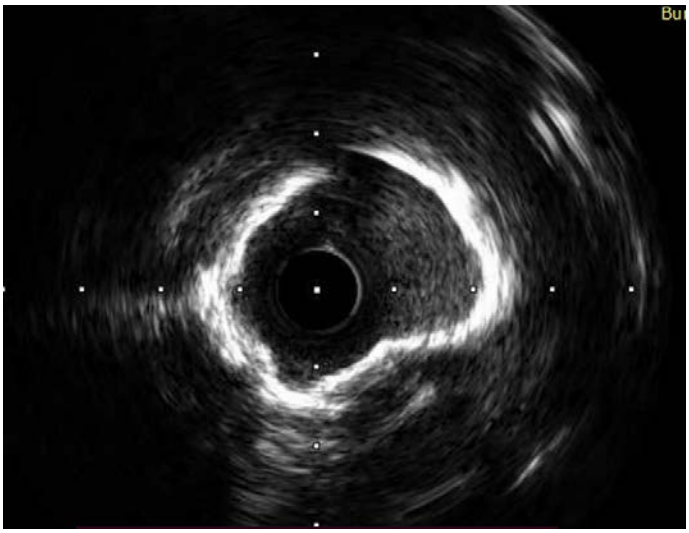

Fig. 3. **IVUS image shows 360° calcification.**

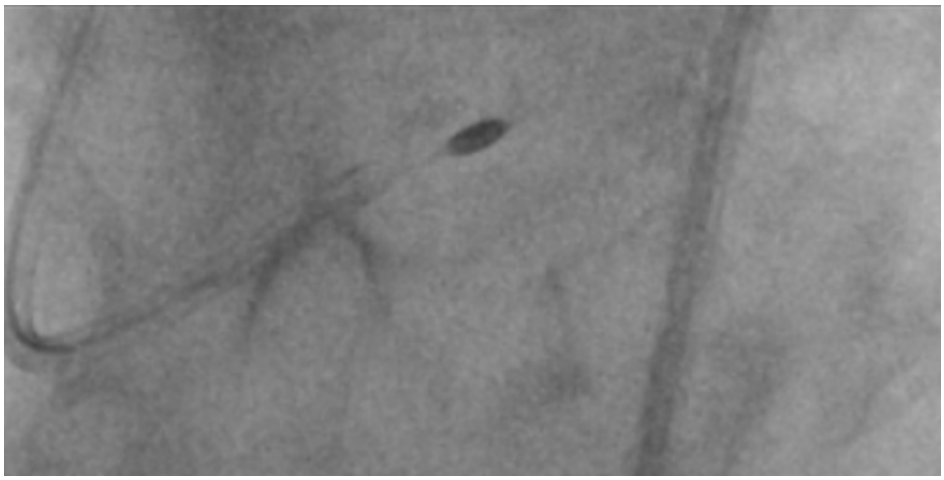

Fig. 1. **Rotaablation of LMCA lesion.**

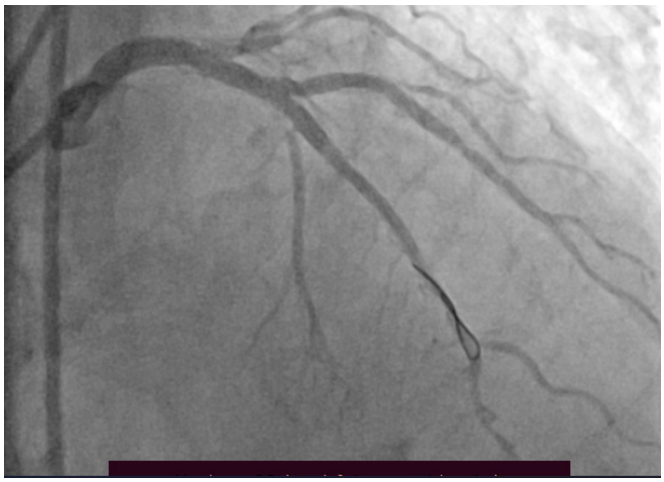

Fig. 5. Final image (first patient)

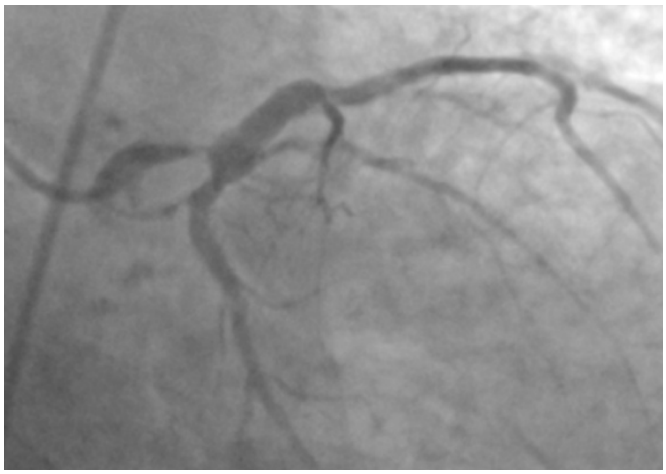

Fig. 6. Angiographic image of severe calcified LMCA lesion (Second patient)

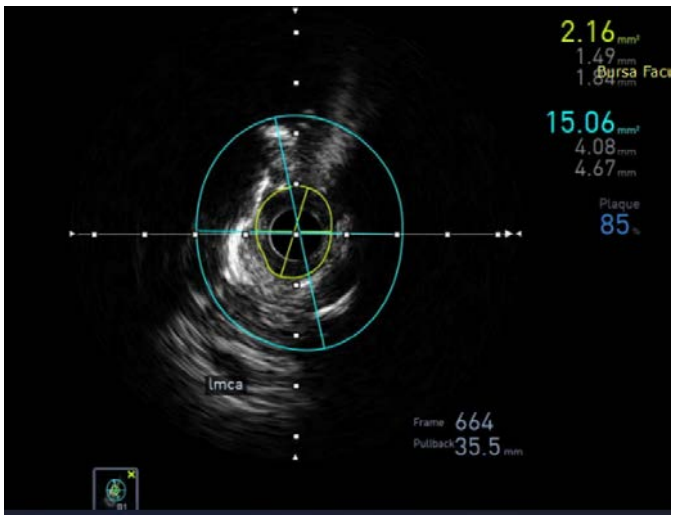

Fig. 7. IVUS image of narrowed LMCA.

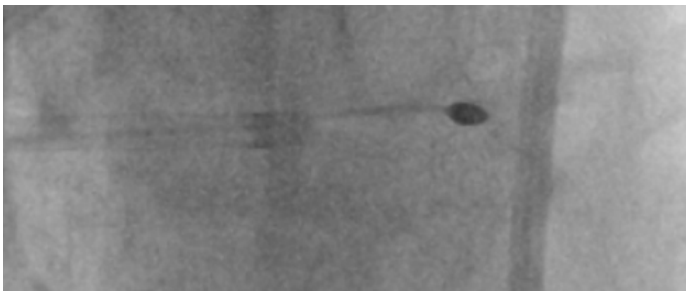

Fig. 8. Rotaablation of lesion.

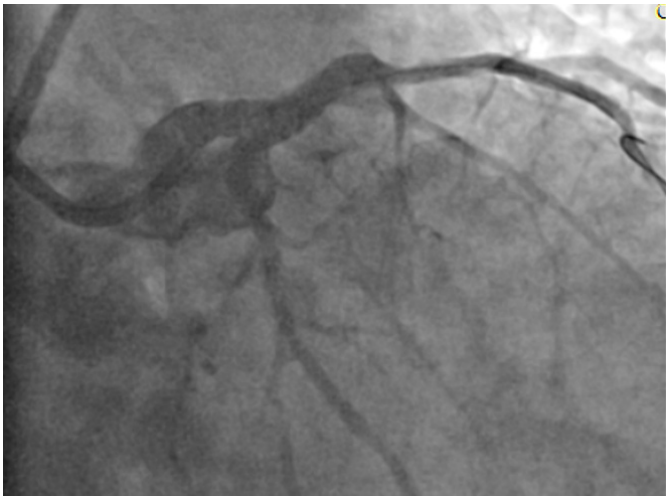

Fig. 9. Final image (second patient).

OP-38.
Impact of Intravenous Nitroglycerin Treatment on Antiplatelet Effects of Clopidogrel in Acute Coronary Syndrome Patients: A Pilot Study

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Background: Clopidogrel is a pro-drug that requires metabolic activation by cytochrome P450 enzymes in 2 steps. Previous study showed that, P450-dependent drug metabolism may be strongly affected after continuous organic nitrate administration. One previous study found that, sustained-release nitrate using was increased PRU values in patients receiving clopidogrel treatment, compared to those not receiving sustained-release nitrate. In this pilot study, we evaluate the effect of intravenous nitroglycerin treatment on antiplatelet platelet effect of clopidogrel in Non-STEMI patients.
Methods: We randomingly include 20 Non-STEMI patients, 15 in active group and 5 in control group. Inclusion criteria; patients with blood pressure higher than 120/80 mmHg, ECG changes and Troponin T elevation. Exclusion criteria; patients with Hb<10 g/dL, Platelet count <100000 uL, renal insufficiency, hepatic insufficiency, used morphine, on chronic clopidogrel, nitrate treatment and patients with ST elevation in ECG. Study protocol; After the diagnosis (Non-STEMI) baseline blood sampling was get, then clopidogrel 600 mg was load and 10 ugr/min nitroglycerin infusion was started for 48 hours. The other blood sampling was get 30 min, 60 min, 120 min, 240 min, 360 min, 48 hours after nitroglycerin infusion started, according to our protocol (Fig. 1). The antiplatelet effect of clopidogrel was evaluated with PRU (platelet reactivity unit) value with Verify-Now P2Y12 assay, from the blood samples.
Results: Baseline charecteristics of the nitroglycerin group and control group were similar (Table 1). Hematologic and Biochemical characteristics, Tn T (maximal value) and LV EF were similar in both groups (Table 1). Baseline (P=0.29), 30th min (P=0.63), 60th min (P=0.96), 120th min (P=0.22) and 48 th hour (P=0.10) PRU values were similar in both groups (Fig. 2). The PRU values were lower in control group than nitroglycerin group at 240 th min (274.13±53.90 vs 162.50±46.14; P=0.002) and 360 th min (270.61±72.27 vs 185.75±20.90; P=0.03) (Fig. 2).
Conclusions: Intravenous nitroglycerin treatment was delayed the antiplatelet effect of clopidogrel in acute coronary syndrome patients in early phase. Direct acting P2Y12 inhibitor treatment may be reliable options for patients under nitroglycerin treatment for preventing thrombotic complications.
Keywords: Intravenous nitrate, clopidogrel, acute coronary syndrome

Table 1. Baseline characteristics of nitrate and control groups.

Variable	Nitrate Group (n=15)	Control Group (n=5)	P value
Age (year)	65.6 ± 9.6	56.2 ± 18.5	0.50
Gender, n (%)			
Male	8 (53.3 %)	3 (60 %)	0.79
Female	7 (46.7 %)	2 (40 %)	
Systolic blood pressure (mmHg)	142.06 ± 16.5	139.0 ± 15.1	0.71
Diastolic blood pressure (mmHg)	83.5 ± 9.2	90.6 ± 6.9	0.13
Heart rate (beats/min)	82.8 ± 11.4	88.6 ± 5.7	0.41
Diabetes mellitus, n (%)	7 (46.7 %)	1 (20 %)	0.60
Smoking, n (%)	6 (40 %)	3 (60 %)	0.61
White blood cells (10^3/µL)	9.85 ± 3.92	8.36 ± 2.88	0.55
Hemoglobin (g/dL)	12.7 ± 1.2	13.2 ± 1.6	0.48
Platelet (10^3/µL)	232.9 ± 60.6	205.3 ± 47.3	0.47
Glukoz (mg/dL)	144.08 ± 57.9	112.6 ± 27.1	0.38
Creatinine (mg/dL)	0.84 ± 0.18	0.83 ± 0.05	0.90
LDL-cholesterol (mg/dL)	137.4 ± 57.4	136.0 ± 16.5	0.98
Troponin T (max) (ng/mL)	4.2 ± 5.1	4.7 ± 5.6	0.86
Left ventricular EF (%)	54.2 ± 9.7	60.0 ± 2.7	0.33

Data are shown as mean±standard deviation or n (%). EF=ejection fraction.

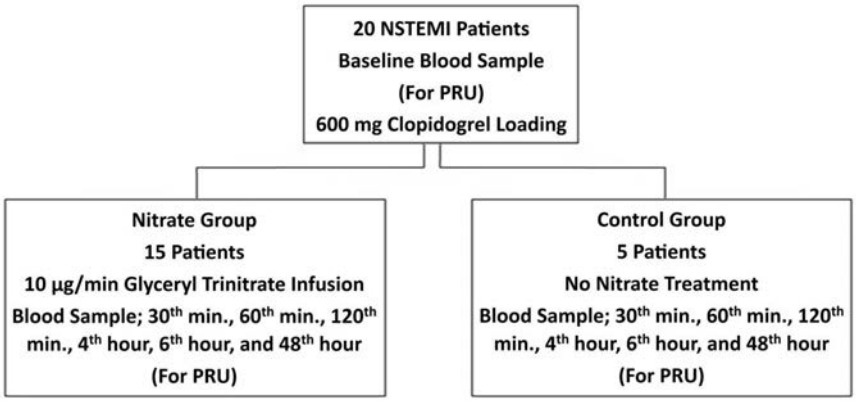

Fig. 1. Study design.

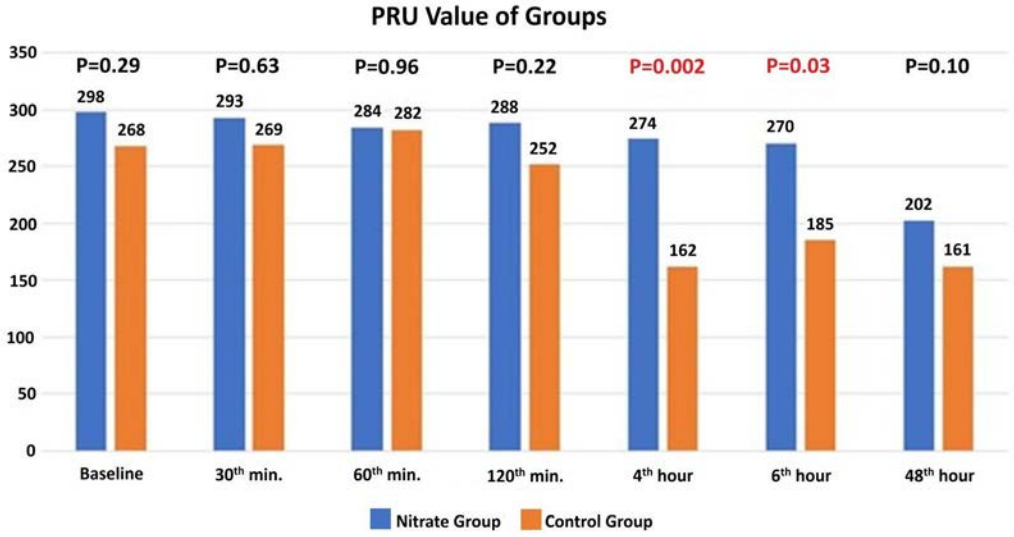

Fig. 2. **PRU values of the nitrate and control groups.**

OP-39.
From the Perspective of Sports Cardiology; Heart Rate Variability Analysis in Predicting Overuse Syndrome in Elite Athletes

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Objectives: There is a balance between training and disruption of this balance may cause overuse syndrome (OS) as a result of chronic low-grade trauma, strain, inflammation, and alterations in the autonomic nervous system. Elite athletes often overlook overuse injury, despite it being a common cause of injuries. Detecting OS presents a challenge. Because there is no clear history of trauma and the pain is usually ignored by the athlete. Early detection and prevention of OS can prevent many non-traumatic injuries in professional athletes. In our study, we examined the relationship between heart rate variability (HRV) and prediction OS.
Methods: 15 athletes diagnosed with OS and 27 healthy athletes from various sports were compared. A Polar H10 heart rate device (Polar Electro Oy in Kempele, Finland) was used to collect the R-R series. The HRV analyses were conducted using commercially available Kubios HRV software (Kempele, Finland; Kubios HRV 3.3, Kuopio, Finland). Additionally, athletes were required to complete the OS questionnaire.
Results: HRV analyses show statistically significant RMSSD, PNS and Poincaré SD1 (P=0.003, P=0.021 and P=0.018, respectively). Considering the entire study group, specificity for RMSSD ≤31 cutoff in OS prediction was observed as 89%, sensitivity as 54% and AUC as 0,738 (0,580-0,862). In isolated upper extremity OS, while the p value was 0.001, the specificity was 89% and the sensitivity was 71% for the RMSSD ≤31 cutoff value (AUC 0,829 (0,660-0,935)).
Conclusion: HRV can be utilized with validated questionnaires for predicting OS. Various methodological follow-up studies may contribute to increasing the sensitivity of this prediction.
Keywords: Heart rate variability, overuse syndrome, non-traumatic injuries, athletes

OP-40.
Dynamics of Smoking Behavior in Turkey: An Analysis of Initiation Reasons, Maintenance Motivations, and Nicotine Dependence Levels

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Objectives: The prevalence of smoking in Turkey poses a significant public health concern. This study aims to examine individuals' reasons for initiating smoking, their motivations for continuing, and their levels of nicotine dependence.
Methods: This study was conducted using an electronic survey among voluntary participants who visited a cardiology outpatient clinic for any reason. Participants were asked questions regarding their age, gender, educational level, income status, and history of chronic diseases. Additionally, the Fagerström Nicotine Dependence Test (FNDT) was applied to assess nicotine dependence levels. Furthermore, participants' reasons for starting smoking, their thoughts on quitting, and their perceptions of the health effects of smoking were explored.
Results: A total of 310 participants (168 males and 142 females) were included in the study. The average age of smoking initiation was 19.5 years. A statistically significant relationship was found between nicotine dependence levels and age, educational level, income status, and history of chronic diseases ($P=0.014$, $P<0.001$, $P=0.039$, and $P<0.001$, respectively). Among smokers, 42% had low, 52% had moderate, and 6% had high nicotine dependence levels. The average daily cigarette consumption was 14 cigarettes. While 79.4% of the participants agreed that smoking is highly harmful, only 34.8% expressed an intention to quit in the near future.
Conclusions: Smoking remains a significant public health issue in Turkey. Our study highlights individuals' reasons for initiating smoking, their motivations for maintaining the habit, and their levels of nicotine dependence. The findings suggest that smoking addiction is influenced not only by nicotine dependence but also by the pleasure derived from smoking and various sociocultural factors. These insights can serve as a **guide for developing more effective smoking cessation programs.**
Keywords: Smoking, nicotine dependence, cardiology outpatient clinic

OP-41.
Effect of Colonoscopy on Atrial Parameters

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Introduction: Colonoscopy is an endoscopic procedure performed for screening, diagnosis, treatment and follow-up of many diseases, especially colon cancer, inflammatory bowel diseases and gastrointestinal bleeding. More than 15 million colonoscopy procedures are performed in the USA each year [1]. The diagnostic accuracy of colonoscopy depends on good colon preparation and adequate visibility of the colon mucosa. Various types of bowel preparation agents are available to improve the quality of bowel cleansing. In addition to side effects such as electrolyte imbalances and acute renal failure [2, 3], bowel cleansing agents can cause acute heart failure, arrhythmias and even, rarely, cardiac arrest [4-6]. Although some patients with a history of cardiac disease may be more prone to developing cardiac arrhythmia after bowel preparation for colonoscopy, new-onset atrial fibrillation, atrial tachycardia, and atrial flutter have been reported after bowel preparation solution intake, even in patients without a history of cardiac arrhythmia or heart failure, and with normal serum electrolyte and thyroid stimulating hormone levels [7]. In addition to possible electrolyte imbalances, the colonoscopy process may induce atrial arrhythmias by causing abnormal activation in the autonomic nervous system. There is limited information on the development of cardiac arrhythmia as a result of bowel preparation before colonoscopy. Electrocardiography (ECG) findings that may predict the risk of arrhythmia have not been examined in this patient group. There are previous studies in the literature evaluating the risk of AF with the MVP ECG risk score [8]. This study aimed to investigate the ECG findings of patients who underwent colonoscopy and the effect of bowel cleansing and colonoscopy on atrial parameters in the ECG.
Methods: This prospective single-center study included patients between the ages of 18 and 75 who needed colonoscopy. The baseline ECGs taken before colonoscopy preparation, the ECGs taken after the colonoscopy procedure, and the patients' files were reviewed, and ECG parameters, comorbidities, and laboratory values were noted. ECG traces were enlarged and atrial arrhythmia markers (P wave duration, P wave voltage, P wave morphology, P wave axis, PR interval) were measured and MVP ECG score was calculated using the formula previously found in the literature and the baseline values and the values obtained after colonoscopy were compared.
Results: Thirty-six patients with a mean age of 55.08±11.23 years were included in this study. 55.6% of the patients were female. The basic clinical characteristics and ECG parameters of the patients are summarized in Tables 1 and Table 2.
Discussion: Patients with structurally normal hearts tend to show a vagal atrial fibrillation pattern, while patients with structural heart disease tend to show a sympathetic pattern [9]. Increased luminal distension in the colon after ingestion of the bowel preparation solution, rapid gastrointestinal motility and increased colonic secretions may increase parasympathetic tone. The atrial refractory period can affect automaticity and atrioventricular conduction, causing significant changes in cardiac electrical activity and thus facilitating the induction of AF. In addition, >50% of patients experience moderate anxiety before colonoscopy [10]. Anxiety also increases this automaticity. Although electrolyte imbalance that may develop after bowel cleansing is mostly asymptomatic, since there is a tendency for myocardial irritability at the cellular level, it may be necessary to monitor electrolytes before colonoscopy, especially in patients with heart or kidney disease and

in the elderly. However, most studies in the literature did not include patients with kidney or heart disease or elderly patients who may be at greater risk for electrolyte disturbances.

Conclusion: No significant changes were detected in atrial parameters between ECGs taken before and after colonoscopy. Further large-scale studies including ambulatory rhythm monitoring are needed in high-risk patient groups.

Keywords: Colonoscopy, atrial parameters, ambulatory rhythm monitoring

Table 1. Baseline characteristics of patients

Study Variables		Data
Age (years)		55.08±11.23
Sex**	**male, n (%)**	20 (55.56)
	female, n (%)	16 (44.44)
Diabetes mellitus		6 (16.67)
Hypertension		9 (25.00)
Hyperlipidemia		1 (2.78)
Coronary aretry disease		3 (8.33)
Heart failure		0 (0.00)
Malignancy		1 (77.78)
Urea, mg/dL		29.68±8.04
Creatinin,mg/dL		0.80 (0.50-1.20)
Glomerular filtration rate, ml/min/1.73m^2		94.62±16.04
Sodium, mmol/L		139.50 (137-145)
Potassium, mmol/L		4.49±0.36
Calcium, mg/dL		9.31±0.48
Chloride, mmol/L		105.68±2.10
Glucoseoz, mg/dL		100.78±15.74
White blood cell, 10^9/L		6.85±2.09
Haemoglobin, g/dL		13.37±1.83
Platelets, 10^9/L		227.35±69.49

Data are shown as mean±standard deviation or n (%) or median (minimum–maximum) where appropriate

Table 2. **ECG parameters**

ECG parameters		Pre-colonoscopy	Post-colonoscopy	P value
Heart rate, bpm		74.5 (60-108)	68.5 (53-108)	**0.021**
P wave axis, degree		57.2±14.6	55.8±14.1	0.598
PR interval, ms		153.1±18.1	153.3±18.3	0.919
P wave duration, ms		110 (80-132)	111 (66-129)	0.919
MVP score		2 (0-4)	2 (0-49)	0.506
P wave morphology (inferior), n (%)	non biphasic <120 ms	29 (80.6)	31 (%86.1)	0.727
	non biphasic >120 ms	7 (19.4)	5 (13.9)	
	biphasic	0 (0	0 (0	
P wave voltage (lead 1), mV, n (%)	>0.2	1(2,8)	1(2.8)	1.000
	0.1-0.2	13(36.1)	13(36.1)	
	<0.1	22(61.2)	22(61.2)	

Data are shown as mean±standard deviation or n (%) or median (minimum–maximum) where appropriate bpm: beat per minute, ms: milisecond

References

1. Wang P, Xu T, Ngamruengphong S, Makary MA, Kalloo A, Hutfless S. Rates of infection after colonoscopy and osophagogastroduodenoscopy in ambulatory surgery centres in the USA. Gut. 2018;67(9):1626-1636. doi: 10.1136/gutjnl-2017-315308.
2. Shaukat A, Kahi CJ, Burke CA, Rabeneck L, Sauer BG, Rex DK. ACG Clinical Guidelines: Colorectal Cancer Screening 2021. Am J Gastroenterol. 2021;116(3):458-479. doi: 10.14309/ajg.0000000000001122.
3. Kim SY, Kim HS, Park HJ. Adverse events related to colonoscopy: Global trends and future challenges. World J Gastroenterol. 2019;25(2):190-204. doi: 10.3748/wjg.v25.i2.190.
4. Ashraf S, Singh M, Singh M, Afonso L. Polyethylene Glycol Preparation for Colonoscopy Associated With Heart Failure Exacerbation. Am J Ther. 2018;25(4):e495-e496. doi: 10.1097/MJT.0000000000000582.
5. Marschall HU, Bartels F. Life-threatening complications of nasogastric administration of polyethylene glycol-electrolyte solutions (Golytely) for bowel cleansing. Gastrointest Endosc. 1998;47(5):408-410. doi: 10.1016/s0016-5107(98)70229-9.
6. Mo Y, Gandhi S, Orsini J. Possible GoLytely-Associated Cardiac Arrest: A Case Report and Literature Review. J Pharm Pract. 2020;33(3):364-367. doi: 10.1177/0897190019825965.
7. Kajy M, Ramappa P. A Galvanizing Solution: Colonoscopy Bowel Preparation as a Trigger for Supraventricular Tachycardia. Ann Pharmacother. 2022;56(3):297-302. doi: 10.1177/10600280211023808.
8. Alexander B, Milden J, Hazim B, et al. New electrocardiographic score for the prediction of atrial fibrillation: The MVP ECG risk score (morphology-voltage-P-wave duration). Ann Noninvasive Electrocardiol. 2019;24(6):e12669. doi: 10.1111/anec.12669.
9. Coumel P. Paroxysmal atrial fibrillation: a disorder of autonomic tone? Eur Heart J. 1994;15 Suppl A:9-16. doi: 10.1093/eurheartj/15.suppl_a.9.
10. Yang C, Sriranjan V, Abou-Setta AM, Poluha W, Walker JR, Singh H. Anxiety Associated with Colonoscopy and Flexible Sigmoidoscopy: A Systematic Review. Am J Gastroenterol. 2018;113(12):1810-1818. doi: 10.1038/s41395-018-0398-8.

OP-42.
Evaluation of the Risk of Developıng Atrial Fibrillation Using Echocardiography and Surface Electrocardiography in Patients With Asymptomatic Carotid Artery Stenosis

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Background: Stroke is one of the top three causes of death in developed societies. Ischemic strokes are linked to carotid artery stenosis (CAS). Atrial fibrillation (AF) is a commonly encountered clinical arrhythmia. It has been shown that the prolongation of intra- and interatrial conduction times, known as atrial electromechanical delay (EMD), is associated with a higher risk of AF. We aimed to determine the correlation of atrial conduction abnormalities between surface electrocardiographic and TDI measurements in the CAS patient group.
Methods: The study included 76 patients diagnosed with extracranial internal carotid artery (ICA) stenosis. Asymptomatic severe CAS was defined as patients with 70-99% stenosis detected by carotid digital subtraction angiography (DSA). The longest P wave and the longest atrial conduction time ACT were considered as the maximal P wave duration. The difference between the longest P wave (Pmax) and the shortest P wave (Pmin) was accepted as PD. (PD=Pmax-Pmin). Atrial EMD was defined as the time interval from the onset of atrial electrical activity to the beginning of mechanical atrial contraction.
Results: The CAS group had significantly longer Pmax and PD values compared to the control group (Pmax 104.72±6.03 and 93.06±7.26 ms, P<0.001; PD 48.55±6.72 and 38.50±8.12 ms, P<0.001) (Table 1). In the TDI examination, the atrial EMD parameters (PA lateral, PA septum) were significantly longer in the CAS group compared to the control group. (77.88 ±5.13 vs 65.53 ±9.11 ms; P <0.0001; 63.77±3.95 vs 54.56±7.13 ms; P<0.001, respectively) Both interatrial and intra-atrial EMD times were found to be longer in the CAS group compared to the control group (31.72±7.39 vs 22.13±8.67 ms; P<0.001; 17.61±7.76 vs 11.16±7.76 vs 11.16±7.04 ms; P<0.001, respectively) (Fig. 1). In the correlation analysis, a positive relationship was found between interatrial and interatrial EMD and Pmax and PD. (P<0.001, both) (Fig. 2).
Conclusion: We found that both intra-atrial and inter-atrial electromechanical conduction times were longer in CAS patients. This suggests that CAS patients are at risk for AF in their follow-up.
Keywords: Carotid artery stenosis, atrial fibrillation, atrial conduction time

Table 1. Electrocardiographic characteristics of the study population

Variables	Control group (n=54)	Carotis Artery Disease (n=76)	P value
Heart rate (min)	77.33±10.90	80.05±2.31	0.149
P Max(ms)	93.06±7.26	104.72±6.03	**<0.01**
P Min (ms)	54.56±3.53	56.16±3.67	0.077
PD (ms)	38.50±8.12	48.55±6.72	**<0.01**

Pmax=maximum P-wave duration, Pmin=minimum P-wave duration, PD=P-wave dispersion, min=minute, ms=millisecond

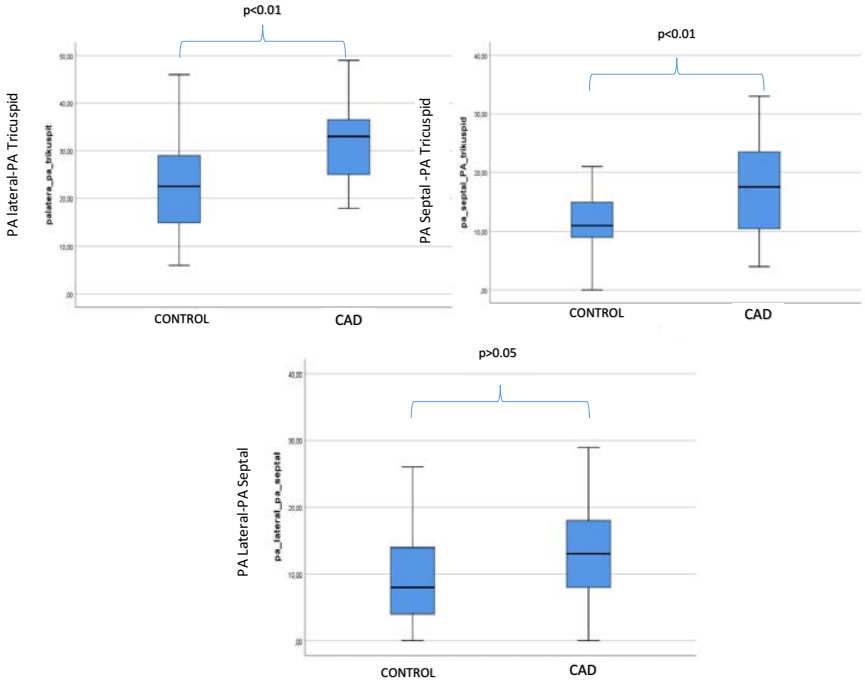

Fig. 1. **Change PA Lateral-PA Tricuspid, PA Septal-PA Tricuspid, PA Lateral-PA Septal between study groups.**

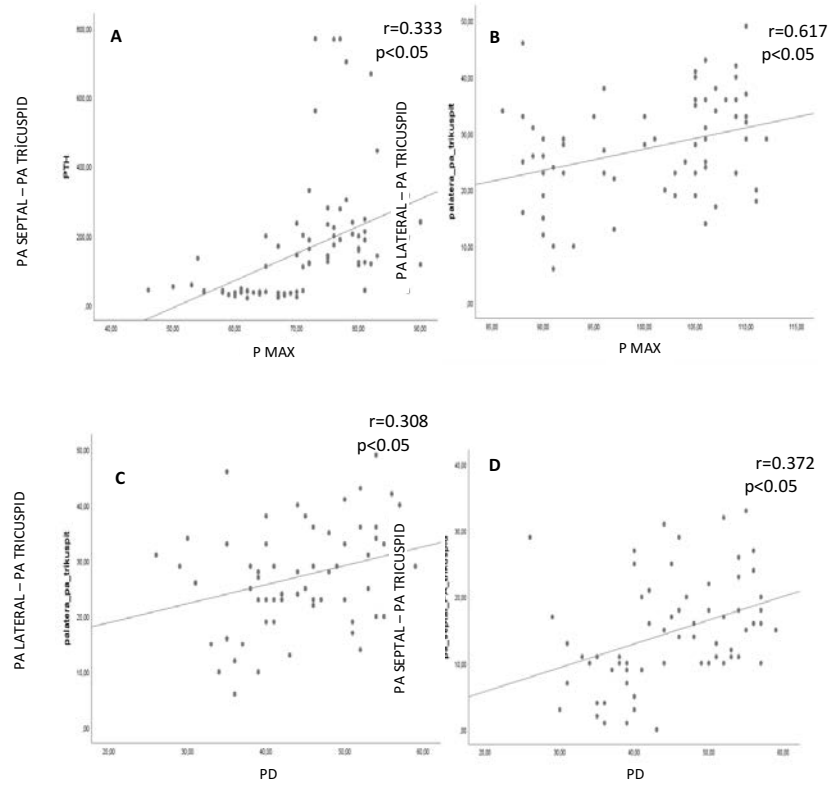

Fig. 2. **Correlation between PA Septal – PA Tricuspid and P MAX duration. (A) Correlation between PA Lateral – PA Tricuspid and P MAX duration. (B) Correlation between PA Lateral – PA Tricuspid and PD duration. (C) Correlation between PA Septal– PA Tricuspid and PD duration (D). Pmax = maximum P-wave duration: PD = P wave dispersion**

OP-43.
New Inflammatory Markers for Predicting In-Hospital Major Adverse Cardiac Events in Patients With Non-ST-Segment Elevation Myocardial Infarction: A New Perspective

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Background: Acute coronary syndromes (ACS), including non-ST-segment elevation myocardial infarction (NSTEMI), are still the leading cause of mortality and morbidity worldwide. This study aimed to evaluate whether pan-immune-inflammation value (PIV), a novel marker of inflammation, is associated with the risk of in-hospital major adverse cardiac events (MACE) assessed by the Global Registry of Acute Coronary Events (GRACE) risk score (GRS) in patients with NSTEMI.
Methods: A total of 426 patients were admitted to the ED of our hospital with chest pain and hospitalized in the coronary intensive care unit and diagnosed with NSTEMI. PIV was calculated as: neutrophil count times platelet count times monocyte count divided by lymphocyte count.
Results: Of the patients included in this study, 80 (18.7%) had low GRS, 158 (37%) had medium GRS, and 188 (44%) had high GRS. Monocyte count, platelet count, neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SII) and PIV were higher in the high GRS group compared to the other two groups with a statistically significant difference ($P<0.001$). (Table 1) There was a positive, strong and statistically significant correlation between PIV and GRS. ($r=0.71$, $r=0.68$, $r=0.61$, $P<0.001$). (Table 2) Inflammatory markers such as PIV, SII and NLR were found to be statistically significantly higher in the MACE (+) groups. ($P<0.001$) (Table 3) The receiver-operator characteristic (ROC) analysis with inflammatory markers in Mace (+) (group-2) patients: PIV in cut-off value 756.03, 82 % sensitivity, 84% specificity (area under ROC curve 0.86 [95% CI: 0.82-0.89], $P<0.001$) (Fig. 1)
Conclusion: Inflammatory markers such as PIV were found to be statistically significantly higher in the MACE (+) groups. Moreover, there was a statistically significant correlation between PIV value and GRS.
Keywords: Non-ST-segment elevation myocardial infarction, Global Registry of Acute Coronary Events risk score, pan-immune-inflammation value

Table 1. Demographic features and laboratory findings of patients in Grace risk score groups

Variables	GRACE<108 (n=80)	GRACE 108-140 (n=158)	GRACE >140 (n=188)	P value
Neutrophil count (10^3 /µL)	2.9±1	4.57±1.8	11±9.1	0.28
Lymphocyte count (10^3 /µL)	1.51±0.1	1.7±0.5	2±0.79	0.62
Monocyte (10^3 /µL)	0.5±0.13[a]	0.62±0.1[a]	0.76±0.01[b]	**<0.001**
Platelet count (10^3 /µL)	212±63.4[a]	221.8±59.4[a]	255.1±69.1[b]	**<0.001**
NLR	1.64 (1.3-2)[a]	1.98 (1.5-2.8)[a]	3.8 (2.7-6.2)[b]	**<0.001**
SII	352.3 (267.6-395.6)[a]	454.9 (322.3-610.7)[a]	963.2 (660.9-1480)[b]	**<0.001**
PIV	177.6 (129-233.7)[a]	284.6 (195.9-355.2)[a]	709.6 (499.4-1195.3)[b]	**<0.001**

PIV=pan-immune-inflammation value, SII=systemic immune-inflammation index, NLR=neutrophil /lymphocyte ratio, Kruskal–Wallis test and one-way ANOVA test were used when comparing three groups. Groups with statistically significant differences are labeled with different letters (a, b, c).

Table 2. Correlation relationship between inflammatory markers and GRACE risk score

	r	P
PIV	0.71	<0.001
SII	0.56	<0.001
NLR	0.5	<0.001

PIV=pan-immune-inflammation value, SII=systemic immune-inflammation index, NLR=neutrophil /lymphocyte ratio

Table 3. Comparison of groups according to the demographic features and laboratory findings

	MACE- (n=398)	MACE+ (n=28)	P value
Neutrophil count (10^3 /µL)	5.12 (3.43-7.55)	6.17(3.2-8.3)	0.76
Lymphocyte count (10^3 /µL)	2.1 (1.5-2.7)	1.6 (0.5-2.55)	0.65
Monocyte count (10^3 /µL)	0.55 (0.3-0.68)	0.97 (0.78-1.23)	**<0.001**
Platelet count (10^3 /µL)	227.7 (190-269)	259.6 (204.5-312.6)	**0.04**
NLR	2.37 (1.6-3.5)	4.45 (3.1-6.4)	**<0.001**
SII	537.7 (368.2-853.7)	1277.8(882.03-1571.3)	**<0.001**
PIV	350.7 (219-583.7)	1120.7 (915.5-1334.4)	**<0.001**

PIV=pan-immune-inflammation value, SII=systemic immune-inflammation index, NLR=neutrophil /lymphocyte ratio

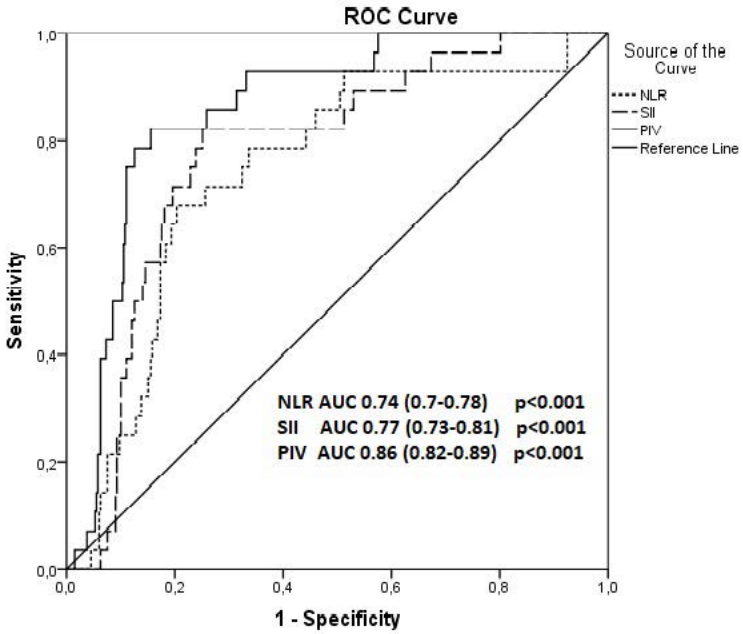

Fig. 1. ROC analysis with inflammatory markers in major adverse cardiac events (+) patients.

OP-44.
Mitral Valve-in-Valve Procedure in a High-Risk Patient With Bioprosthetic Valve Dysfunction

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Bioprosthetic valve dysfunction presents a significant therapeutic challenge in patients with high surgical risk due to advanced age and comorbidities. The valve-in-valve procedure offers a minimally invasive alternative to surgical intervention in such cases. This case report discusses the successful application of the valve-in-valve procedure in a high-risk patient with bioprosthetic mitral valve dysfunction.

A 69-year-old female patient with a history of bioprosthetic mitral valve replacement and tricuspid ring annuloplasty seven years ago, but without additional comorbidities, presented to the cardiology outpatient clinic with progressive dyspnea and exertional shortness of breath.

Transthoracic echocardiography (TTE) revealed preserved left ventricular systolic function and elevated transvalvular gradients across the bioprosthetic mitral valve (mean: 25 mmHg, peak: 31 mmHg). Pulmonary artery pressure was measured at 75 mmHg. Subsequent transesophageal echocardiography (TEE) showed mean and peak gradients of 16 mmHg and 25 mmHg, respectively. The patient was diagnosed with bioprosthetic valve dysfunction and referred to the cardiovascular surgery department for further evaluation. Coronary angiography was performed, revealing no significant coronary stenosis.

Surgical consultation deemed the patient to be at extremely high risk for open-heart surgery, and surgical intervention was not recommended. Following a multidisciplinary discussion between cardiology and cardiovascular surgery teams, the valve-in-valve procedure was determined to be the most appropriate treatment option.

The procedure was performed in a catheterization laboratory under general anesthesia with TEE guidance. A 7F sheath was introduced via the right femoral vein. After obtaining adequate imaging via TEE, interatrial septostomy was performed using a Mullins sheath, septostomy needle, 0.032-inch, and 0.014-inch guidewires. A hydrophilic wire and AL1 catheter were used to cross the degenerated bioprosthetic mitral valve. A catheter was advanced into the left ventricle, and a guidewire was positioned within the ventricle. The septum was dilated using a 14 mm × 4 cm balloon. A 30.5 mm transcatheter heart valve was then deployed within the bioprosthetic valve scaffold in an optimal position. Post-procedural assessment with a pigtail catheter confirmed the absence of mitral regurgitation.

On the first post-procedure day, TTE demonstrated a reduction in mitral valve gradients (mean: 6 mmHg, peak: 9 mmHg), with mild central mitral regurgitation and a decrease in pulmonary artery pressure to 50 mmHg.

This case highlights the valve-in-valve procedure as a safe and effective treatment option for high-risk patients with bioprosthetic mitral valve dysfunction. Given its minimally invasive nature and shorter recovery time, this approach should be considered a primary alternative in patients who are not candidates for surgery.

Keywords: Bioprosthetic valve dysfunction, valve-in-valve procedure, high risk patient

OP-45.
Iatrogenic Pneumopericardium Following Pericardiocentesis

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Introduction: Pneumopericardium, the presence of air within the pericardial space, is a rare occurrence which usually follows positive pressure ventilation in infants, or blunt and penetrating thoracoabdominal injuries in adults. It is even more infrequent for it to occur after pericardiocentesis or for it to cause tamponade.
Case Report: A 67 years old male with history of small cell lung cancer treated with chemotherapy consulted to our department with complaint of progressively worsening dyspnea on exertion and pericardial effusion. On physical examination he had a respiratory rate of 25/min, heart rate (HR) of 124/min, with vital signs otherwise within normal parameters. Cardiopulmonary examination was remarkable for muffled heart sounds with jugular venous distention, additional decreased breath sounds. Electrocardiogram (ECG) obtained on presentation was remarkable for low QRS voltage and 1st degree AV block. PR interval was measured as approximately 320 msec. In the following hours, upon observation of clinical deterioration in the patient and signs of compression on the right ventricle, emergent pericardiocentesis was performed. Initially, 700 ml of hemorrhagic pericardial effusion was drained, and in the following days, a total of 1000 ml of hemorrhagic pericardial effusion was drained. In the control TTE after drainage: 22 mm adjacent to the right ventricle and 12 mm adjacent to the right atrium. 13 mm posterior wall adjacent to the left ventricle Pericardial effusion without any signs of compression was observed in the adjacent area. 4 days after the procedure, as there was no significant improvement in the patient's clinical condition, TTE was observed, which showed a pericardial effusion of 26 mm at the apex adjacent to the right ventricle, 16 mm at the apex and 10 mm at the apex adjacent to the left ventricle, without any signs of compression. (Fig. 1) The patient, who had increased pericardial effusion, was consulted with cardiovascular surgery regarding the pericardial window. Thorax CT was planned for the patient, whose control chest radiography showed an air image at the edge of the right heart. (Fig. 2) In the thorax CT performed on the patient, the appearance of free air in the pericardial cavity was detected, consistent with pneumopericardium. Additionally, an effusion reaching 2.5 cm was observed in the pericardial cavity. (Fig. 3) The patient underwent an emergent pericardial window operation by cardiovascular surgery. It was withdrawn 3 days after the procedure due to the lack of a drain. In the control TTE, 11 mm pericardial effusion was observed near the right ventricle, 5 mm in the apex, and 4 mm in the lateral wall, with no signs of compression, and no pneumopericardium was observed in the control thorax CT. The patient was then referred to the oncology center where he was followed up.
Conclusion: Iatrogenic pneumopericardium as a consequence of pericardiocentesis is rare and mostly results from a leak in the drainage system or formation of communication between the pleura and pericardium. While it has been reported before, it is known to be usually self limited.
Keywords: Pneumopericardium, pericardiocentesis, iatrogenic complication

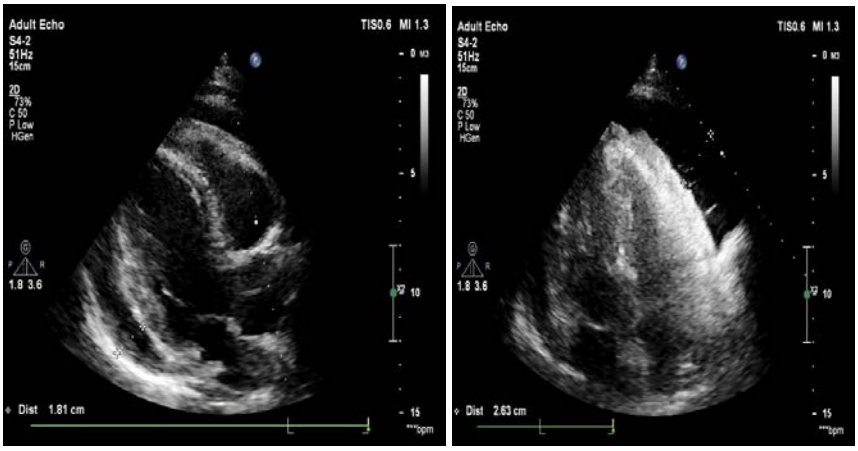

Fig. 1. Transthoracic echocardiography (TTE).

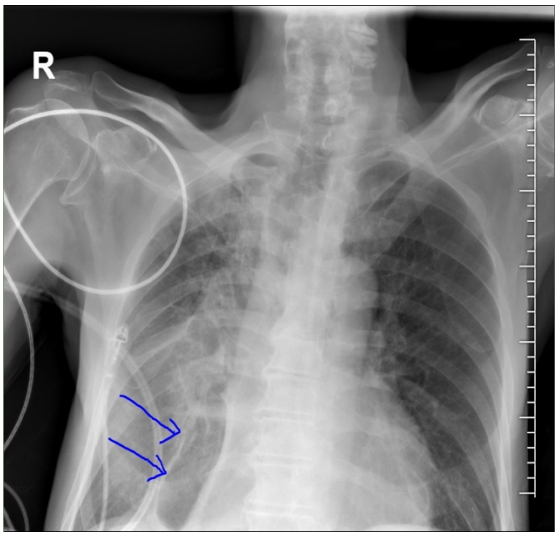

Fig. 2. Chest X-ray (CXR).

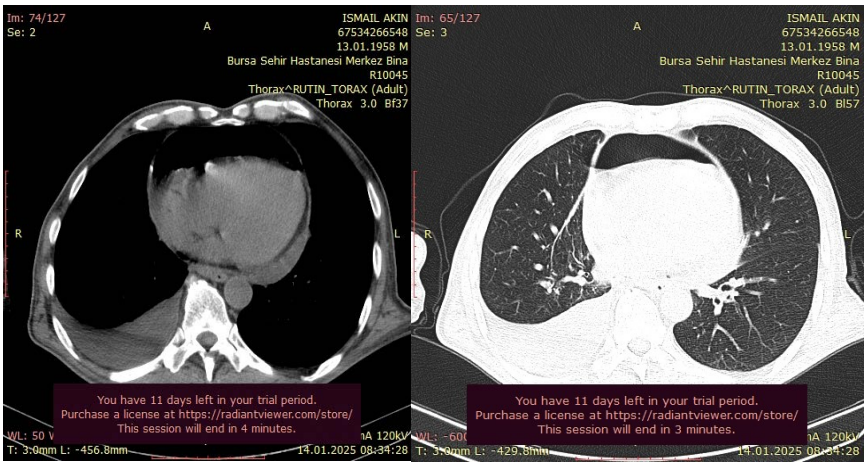

Fig. 3. Thorax CT.

OP-46.
Effect of Heart Rate and Recovery Time on Serve Success in Volleyball Sultans League

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Introduction: In recent years, performance medicine—a subfield of sports cardiology—has gained prominence as one of the scientific fields that supports industrial sports. One of the simple but effective methods used here is heart rate recovery time analysis. While heart rate recovery time is an indirect measure of performance in endurance sports, it is known to be related to muscle biomechanics in skill sports. In our study, we examined the relationship between serve success rates in the Volleyball Sultans League and the heart rate at the time of shooting and the heart rate recovery time.
Methods: Cardiac follow-ups of Bahçelievler Belediyespor players were performed in league matches for 6 consecutive matches in the 2024-2025 season. During the follow-up, the players were asked to wear a Polar H10 (Polar Electro Oy, Kempele, Finland) heart rate tracking device during the match and instant follow-ups were made from the team module of the application. The players' pulses, heart rate reserves and service quality at the time of service were recorded. Service success rates were categorized as effective service, successful service and bad service and grouped in accordance with current sports statistics definitions.
Results: When the poor serve group was examined, it was observed that the average heart rate at the time of the shot was 163 beats/min, the average of the successful serve was 149 beats/min, and the effective serve was 131 beats/min. When we look at the heart rate reserves at the time of the shot, these averages were found to be 81%, 75% and 65%, respectively. In addition, the positive effect of heart rate recovery time on service quality is being examined in the second half of the season, after the zone 5 exercise training between seasons in the first half of the season, and the data collection process continues as the second step of the study.
Conclusion: In professional sports, cardiac-based methods are components that should be taken into account in order to improve sports performance. In our study, it was clearly observed that high pulse rate at the time of the shot was associated with low service quality. The main factors in this connection are deterioration of muscle biomechanics in high pulse rate, increased rate of tremor in the hand and poor coordination. More comprehensive observation studies and zone 5-targeted training methodologies to be conducted in the future can contribute to increased success in various sports branches, both athletically and industrially.
Keywords: Heart rate recovery time, professional sports, performance medicine

OP-47.
Q Fever Endocarditis: A Rare Case

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Abstract: Q fever is caused by the Gram-negative bacillus Coxiella burnetii. It can presents as either acute or chronic illness. Acute disease is characterized by a wide spectrum of symptoms; however, infective endocarditis rarely reported in acute Q fever cases. It is generally considered a consequence of subacute or chronic Coxiella infection. This case report describes a diabetic person who was diagnosed with acute Q fever causing endocarditis with large typical vegetations and severe aortic insufficiency.

C**ase Report:** A 42-year-old man presented to the emergency department with chest pain, fatigue, fever, and poor appetite. Initially, he was diagnosed with pneumonia and admitted to an external medical center for treatment. During follow-up at that facility, the development of clinical heart failure was observed, and the patient was referred to our clinic for further evaluation. Laboratory tests revealed no significant abnormalities other than mild leukocytosis and elevated BNP levels. Transesophageal echocardiography identified a bicuspid aortic valve with a mass measuring 14×7 mm. Valvular rupture was considered the likely cause of severe aortic regurgitation. (Fig. 1) .The patient needed immediate surgery because of their clinical cardiac failure. We obtained blood cultures according to current protocols and the patient was started on vancomycin and gentamicin medications. With no postoperative complications, the patient had a 25mm St. Jude bileaflet valve implanted successfully. No bacterial growth was detected in all blood culture sets. The histopathology of the cardiac valve samples did not reveal any pathogenic microorganisms, however the tissue specimens displayed persistent inflammation, myxoid and hyaline degeneration. A blood polymerase chain reaction (PCR) assay for Coxiella burnetti was positive. Coxiella serology assays, IgG Phase I titers of 1/1024 and IgG Phase II titers of 1/8192, suggested acute Q fever, while IgM was negative. Doxycycline 2×200 mg/day and moxifloxacin 1×400 mg/day intravenously were added to antimicrobial therapy. During hospitalisation, the patient remained hemodynamically stable. After ten weeks, follow up serology showed IgG Phase I was 1/1024, and Phase II IgG was 1/4096 while Ig M Phase II remained negative. The anticardiolipin antibodies and autoimmune markers were negative. The patient has since been monitered without complications.

Conclusion: Chronic Q fever diagnosis can be challenging due to atypical vegetations and negative cultures, often requiring detailed history-taking and clinical suspicion. In contrast, our patient was identified in the acute phase, with typical vegetations and acute advanced aortic valve rupture secondary to infective endocarditis. While existing guidelines provide cut-off values for chronic Q fever, they lack specificity for acute cases. Patients with prolonged fever and new valve dysfunction, especially with negative blood cultures, should be carefully evaluated. Clinical and laboratory assessments should include routine serological testing for C. burnetii and PCR-based detection of bacterial DNA. Although Q fever is endemic in our country, it remains underdiagnosed, likely due to difficulties in culturing the bacterium and detecting vegatation. Increased clinical awareness and improved diagnostic strategies are essential for early detection and effective management.

Keywords: Q fever, endocarditis, early diagnosis, management

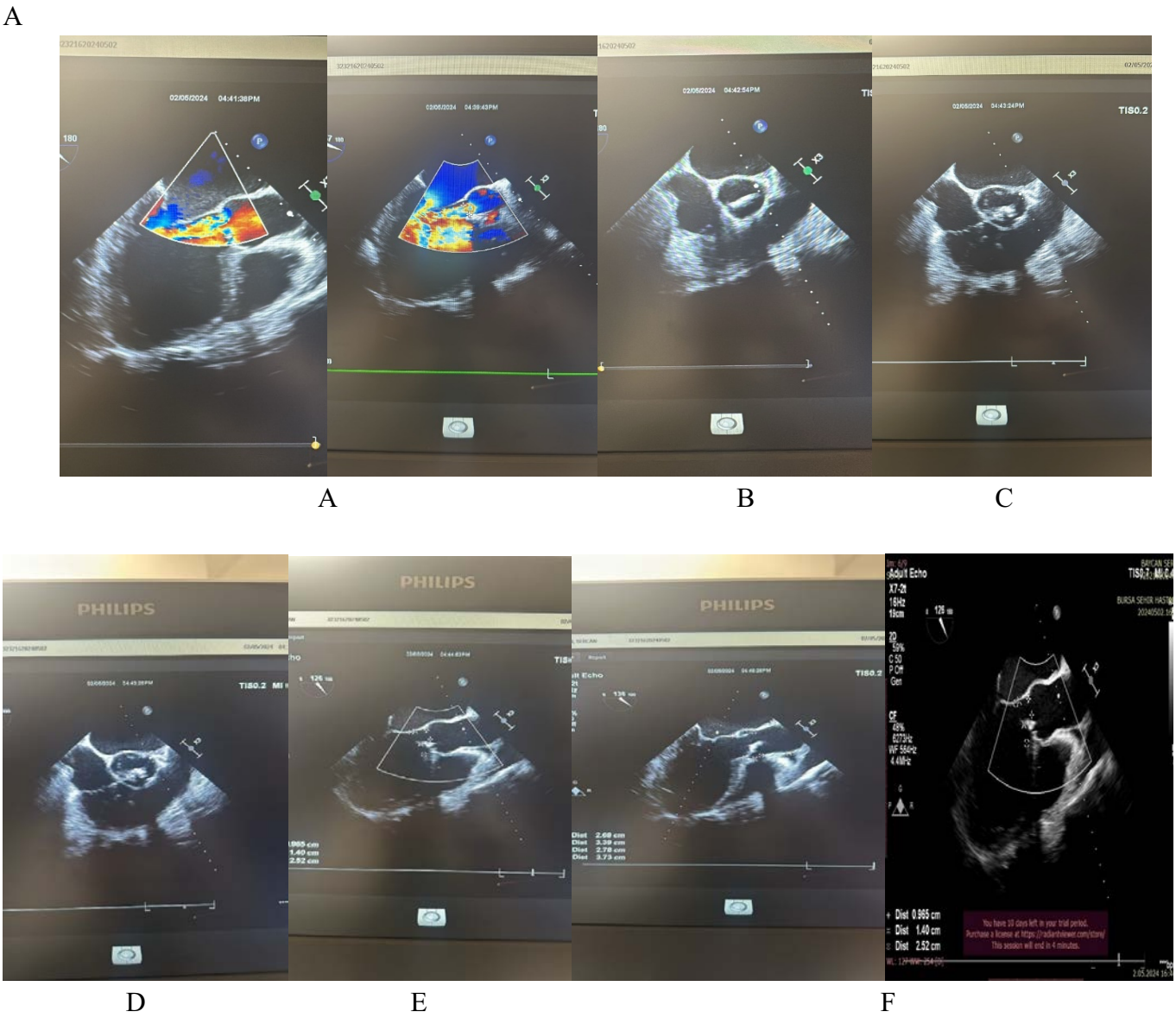

Fig. 1. **(A) Severe aortic regurgitation in the parasternal long axis, (B) bicuspid aortic valve thickening, (C) aortic bicuspid a nodular hyperechogenic additional vibratile lesion was observed on the cusp, (D) aortic bicuspid a nodular hyperechogenic additional vibratile lesion was observed on the cusp, (E) Transesophageal echocardiography (126°) zoomed in on the aortic valve and revealed a 1.4×2.5 mm nodular lesion on the valve in the parasternal long axis, and (F) Transesophageal echocardiography (126°) zoomed in on the aortic valve and revealed a 1.4×2.5 mm nodular lesion on the valve in the parasternal long axis.**

OP-48.
What Strategy Should We Follow Incoronary Bypass Graft - Native Vessel Anastomotic Stenosis?

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A 52-year-old male patient with a history of coronary artery bypass grafting (CABG) was admitted to the coronary intensive care unit with a diagnosis of unstable angina pectoris. His medical history was negative for smoking, hypertension, and diabetes. After premedication, he underwent coronary angiography (CAG).
CAG Report: LMCA: Normal, LAD: Ostial 100% occlusion, distal 60-70% stenosis, Intermediate Artery: Patent (high-origin diagonal), Cx: Proximal stent patent with intrastent plaque; mid stent patent with intrastent plaque, RCA: Proximal 100% occlusion (CTO), LIMA: Patent, LIMA-LAD anastomosis site 80-90% stenosis, Ao-RCA Saphenous Graft: Proximal stent patent, mid 50-60% stenosis, sequential 60% stenosis, distal segment with plaque
Decision: PCI planned for the LIMA-LAD anastomosis. Elective myocardial perfusion scintigraphy planned for RCA saphenous graft assessment. (Figs. 1-5).
PCI Report: The 80-90% stenosis at the LIMA-LAD anastomosis site was crossed using a 0.014 floppy wire. The LAD lesion was predilated using a 2.0×12 mm balloon (Figs. 6 and 7). Two overlapping DES stents (2.5×13 mm and 2.5×29 mm) were implanted at nominal atmospheric pressure (Fig. 8-9). Post-dilation was performed with a 2.75×10 mm NC balloon at nominal atmospheric pressure. Full stent expansion and TIMI 3 flow were achieved without complications (Fig. 10).
Outcome: Successful LIMA-LAD PCI.
Six months later, the patient experienced recurrent typical chest pain. Follow-up angiography revealed that the LIMA-LAD stent remained patent. A decision was made to proceed with full medical therapy. The purpose of sharing this case is to highlight the occurrence of stenosis at the anastomosis site between a bypass graft and a native coronary artery in patients who have undergone CABG. In such cases, percutaneous coronary intervention (PCI) with stent placement is an effective treatment option to alleviate the stenosis. There are limited studies in the literature on the successful treatment of anastomotic stenosis with stenting. For example, a case report published in 2021 detailed a successful PCI procedure in a 75-year-old male patient who experienced acute myocardial infarction and cardiogenic shock less than 24 hours after two-vessel CABG. Angiography revealed acute closure at the left internal mammary artery (LIMA) to left anterior descending artery (LAD) anastomosis. The intervention involved deploying a PK Papyrus covered stent across the anastomosis site, resulting in restored blood flow and stabilization of the patient [1]. However, it should be noted that in our case, as well as in all other patients with this condition, the characteristics of the lesions may vary. Therefore, the treatment strategy should be determined on an individual basis.
Key words: Coronary artery bypass grafting, anastomotic stenosis, percutaneous intervention

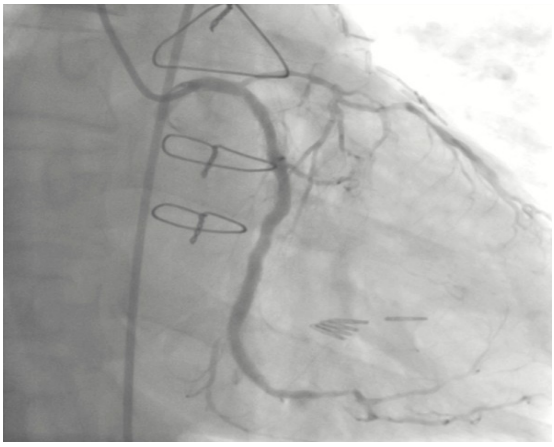

Fig. 1. Left Coronary system image.

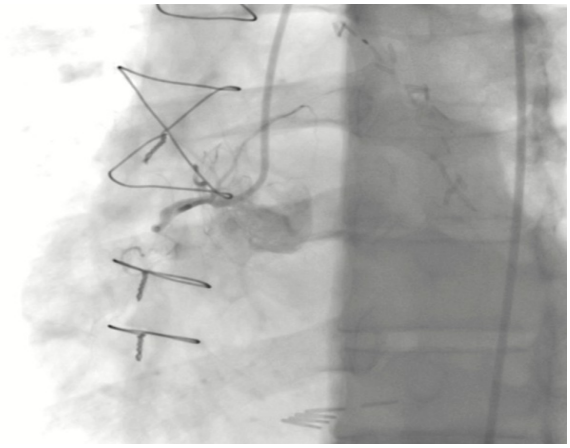

Fig. 2. RCA image.

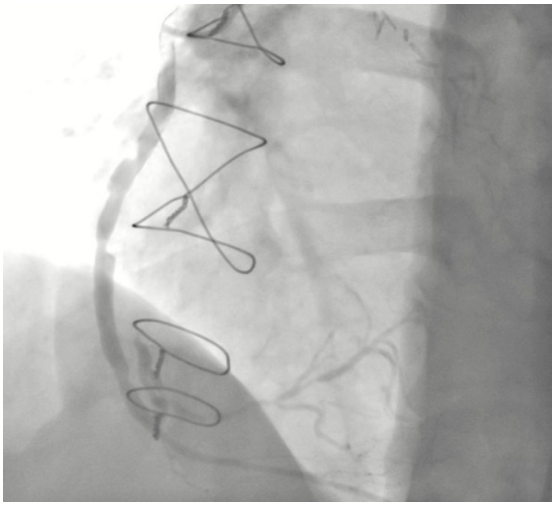

Fig. 3. Ao-RCA saphenous graft image.

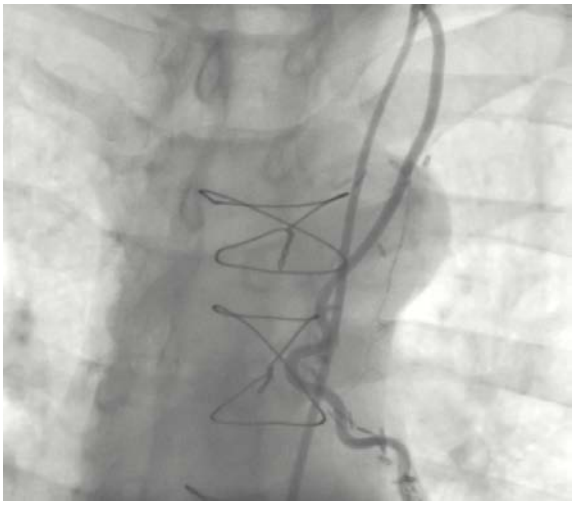

Fig. 4. LIMA-LAD graft proximal image.

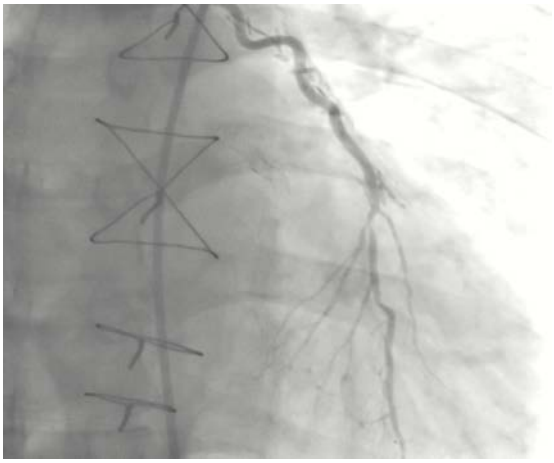

Fig. 5. LIMA-LAD graft distal image.

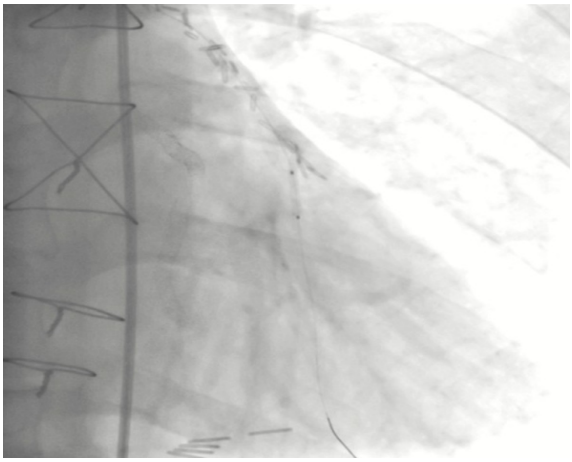

Fig. 5. LAD PTCA.

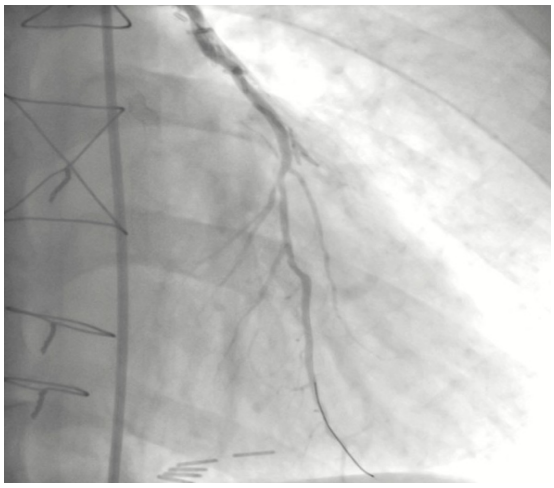

Fig. 7. LAD image after PTCA.

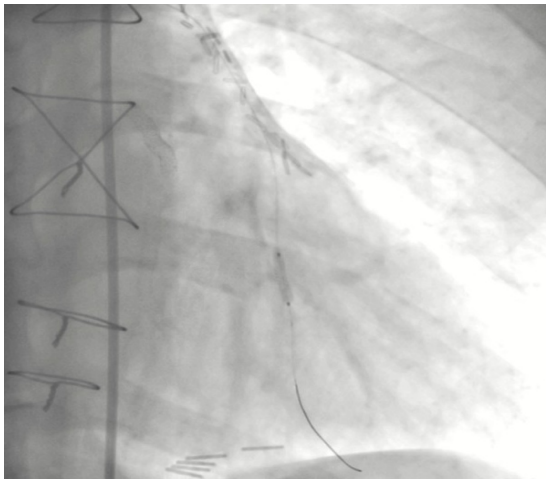

Fig. 8. LAD stenting 1.

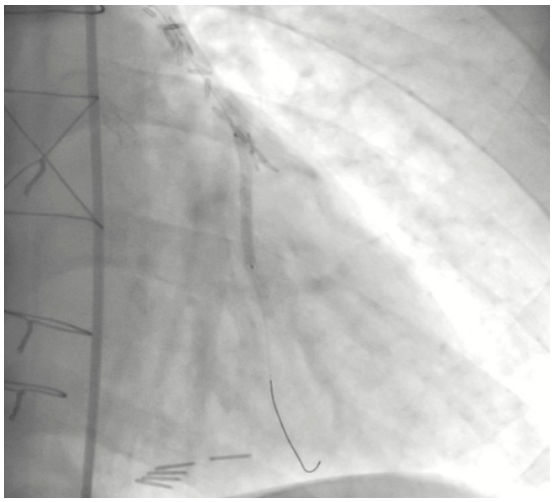

Fig. 9. LAD stenting 2.

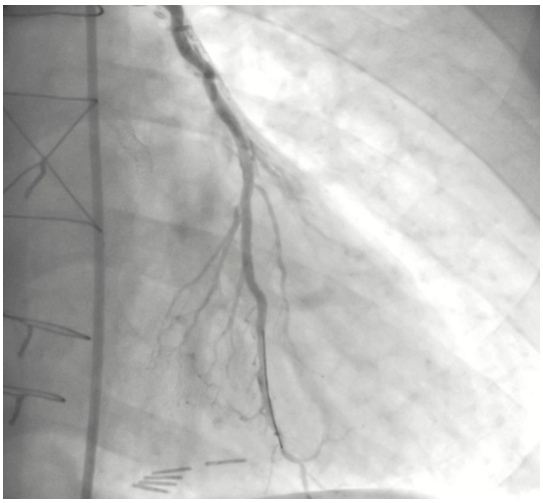

Fig. 10. 10. LAD final image.