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EDITORIAL

PREFACE

With the philosophy that success is dependent on continuity and stability, we take pride in sharing another issue of our magazine with you on this journey. Each new year opens new doors for the medical community. With every scientific advancement, we aim to make our mark through our magazine. Your valuable contributions of scientific studies and writings will elevate our magazine to even greater heights in the academic arena. Hoping that this issue of our magazine, with its rich content penned with different perspectives, interesting topics, and current approaches, will contribute to all our readers, I wish you enjoyable readings.

Dr. Hatice Hanci

Editor-in-Chief

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OLGU SUNUMU / CASE REPORT **DOI:** 10.56941/odutip.1600891

A case of coronary perforation with graft-covered stent placement

Emrah Kaya[1], Taner Sen[1]

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Abstract

Coronary artery perforation is a rare but potentially fatal complication during percutaneous coronary intervention. In this case, we report a patient with myocardial infarction who experienced coronary perforation during percutaneous coronary intervention and successful application of the graft stent. In our case, coronary rupture occurred as a result of balloon inflation in a small vessel without making sure that the guidewire was in the lumen. Prolonged balloon inflation at the site of coronary perforation may provide a solution in some cases. In our case, the balloon was inflated proximal to the rupture, the bleeding was stopped by waiting, and the coronary rupture was successfully closed with a graft stent. The balloon or stent balloon should not be inflated without making sure that the coronary lumen is in place. If inflated without being sure, coronary rupture may occur. Steps to manage this complication include rapid closure of the perforated area, administration of protamine sulfate, graft covered stenting and emergency surgery if necessary.

Key Words: Coronary artery perforation, Complication, Graft-covered stent

Greft Kaplı Stent Yerleştirilen Bir Koroner Perforasyon Olgusu

Özet

Koroner arter perforasyonu perkütan koroner girişim sırasında nadir görülen fakat mortal olabilen bir komplikasyondur. Bu olguda, miyokard infaktüsü ile gelen hastada girişim sırasında koroner perforasyon ile karşılaşılması ve greft stentin başarılı bir şekilde uygulanması sunuldu. Olgumuzda küçük çaplı bir damarda kılavuz telin lümende olduğuna emin olunmadan balon şişirilmesi sonucu koroner rüptür olmuştur. Koroner perforasyon gelişen bölgede uzun süreli balon şişirilmesi vakaların bir kısmında çözüm sağlayabilir. Olgumuzda da rüptürün proksimalinde balon şişirilip beklenerek kanamanın durması sağlandı, sonrasında da greft stent ile koroner rüptür başarılı şekilde kapatıldı. Koroner lümende olunduğundan emin olunmadan balon veya stent balonu şişirilmemelidir. Eğer emin olunmadan şişirilirse, koroner rüptüre neden olunabilir. Bu komplikasyonu yönetme adımları; perfore bölgenin hızlı bir şekilde kapatılması, protamin sülfat verilmesi, greft kaplı stent uygulanması ve gerekli durumda acil cerrahidir.

Anahtar kelimeler: Koroner arter perforasyonu, Komplikasyon, Greft kaplı stent

Suggested Citation: Kaya E and Sen T. A case of coronary perforation with graft-covered stent placement. ODU Med J, 2025;12(1): 1-6.

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INTRODUCTION

Coronary artery perforation is a rare but fatal complication during percutaneous coronary intervention. Coronary perforation is observed in 0.3-0.6% of these interventions (1). The

prevalence of this complication has increased with the development of new interventional techniques such as laser coronary angioplasty, rotablator, high-pressure balloon dilation, and interference with chronic total occlusions. However, perforation can sometimes be related to a standard balloon or stent. Coronary perforation may cause pericardial tamponade, myocardial infarction, or death. Surgical intervention is required in a considerable proportion of patients (2,3). This case report presents a patient with non-ST elevation myocardial infarction, who presented with coronary perforation during intervention, and the successful application of a polyurethane-covered graft stent.

CASE REPORT

An 84-year-old male patient with a diagnosis of hypertension and no other cardiac event history was admitted to the emergency department of our hospital after having chest pain. In the emergency department, 300 mg of acetyl salicylic acid was administered. Electrocardiogram performed in the emergency department showed atrial fibrillation, D1-AVL, and V5-V6 ST depression (Figure 1). The patient's blood pressure was 130/80 mmHg, heart rate was 70 bpm, and physical examination findings were normal. The echocardiography of the patient revealed an ejection fraction of 55%, no wall motion abnormality, and mild mitral, aortic, and tricuspid regurgitation. In biochemistry tests, troponin 6125 ng/ml (normal range: 0-19.8 ng/ml) was detected, and the patient was admitted to the catheter laboratory with the diagnosis of non-ST elevation myocardial infarction.

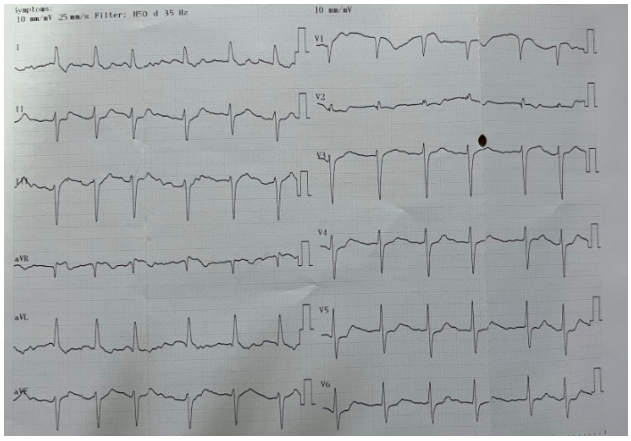

Figure 1. Electrocardiogram at admission showing atrial fibrillation and ST-segment depression in D1-AVL, V5-V6 leads.

A 7 French (Fr) sheath was inserted through the right femoral artery of the patient. The left main coronary artery was cannulated with a left Judkins 4 diagnostic catheter, and no significant stenosis was observed in the circumflex artery on imaging. In the left anterior descending artery, 60-70% stenosis was observed in the mid region, which later led to the decision to test the patient for ischemia. The right coronary artery (RCA) was cannulated with a right Judkins 4 catheter, and the obtained image showed critical stenosis with thrombus in the proximal region (Figure 2).

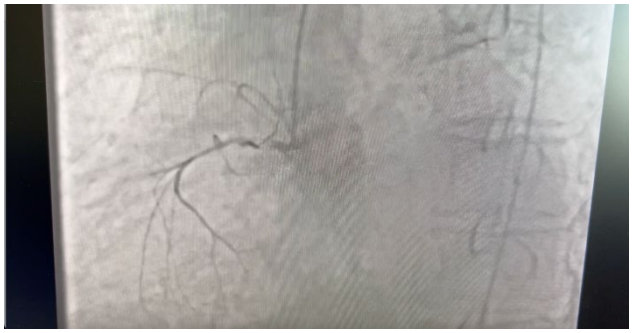

Figure 2. RCA, critical lesion.

An intervention for RCA was planned as the source of the lesion responsible for the myocardial infarction. The patient was administered 600 mg of clopidogrel and 7500 units of unfractionated heparin, adjusted according to his weight. The right Judkins guiding catheter was cannulated into RCA. The guide wire (0.014 Asahi) was sent to the distal of RCA. Pre-dilation was applied to the lesion with a 2.0x15 mm balloon. After pre-dilation, the flow was lost, and no-reflow developed. The balloon could not be advanced again over the guide wire to the area where the flow was lost. It was observed that the right Judkins 4 catheter did not provide sufficient support, and the catheter was removed. For support, RCA was cannulated with an Amplatz Left 1 catheter. The guide wire was again advanced from the lesion, but this time, it could not be sent distally as before. As far as it progressed, a 2.0x15 mm balloon was brought over the guide wire, and dilation was applied to the lesion again. Then, imaging showed a coronary rupture to the distal of the lesion. Contrast staining was observed in myocardial and epicardial adipose tissue (Figure 3). The balloon was inflated in the proximal region of the rupture, and blood flow to the rupture area was stopped as expected. The on-call cardiovascular surgery physician was informed that an emergency operation could be required. After waiting for the balloon to inflate for about 10 minutes, it was observed that the bleeding stopped in the rupture when we deflated and checked the balloon. During this period, the patient was hemodynamically stable. The guide wire was then sent to the distal again. An over-the-wire (OTW) balloon was sent so that it would be positioned in the lumen. The OTW made sure that the contrast agent given using the balloon was in the lumen. Pre-dilation was performed with the OTW balloon, and TIMI-1 flow was achieved.

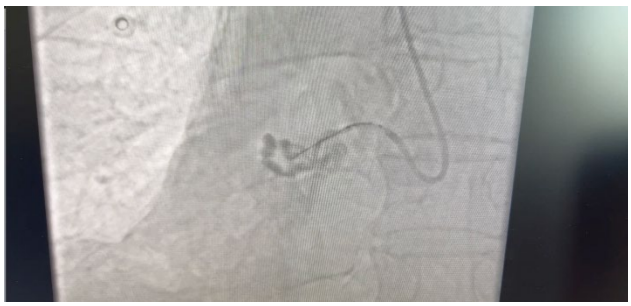

Figure 3. Coronary artery perforation.

A 2.5x20 mm polyurethane-covered graft stent (PK Papyrus; Biotronik) was placed with a pressure of 10 atm starting from the lesion to cover the rupture site as well. There was also a

lesion to the distal of the stent, and a 2.25x16 mm drug-eluting stent (Evermine50; Meril) was implanted distally from the graft stent by overlapping. Post-dilation was applied to the overlap area with a stent balloon. TIMI-2 flow was achieved, and the process was completed (Figure 4). After the intervention, serial echocardiography follow-ups were performed on the patient, and no pericardial effusion was detected. The patient was hemodynamically stable, and there were no complaints. The patient was prescribed medical treatment and discharged. There were no complaints in his follow-ups, and no pericardial effusion was detected.

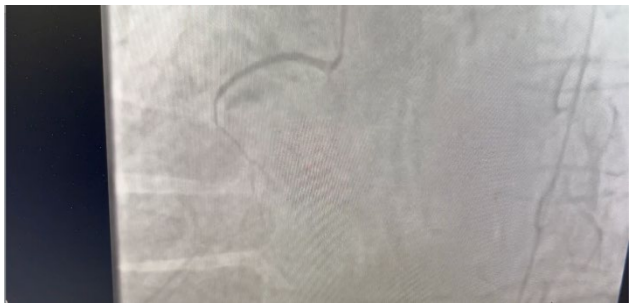

Figure 4. RCA, latest state.

DISCUSSION

The main causes of coronary perforation in the presence of a calcific, small-diameter, chronic total lesion of the intervening coronary artery, the use of rigid hydrophilic wires, and a large-diameter balloon (4). In our case, there was a coronary rupture in a small-diameter artery as a result of the inflation of a balloon without making sure that the guide wire was in the lumen. The size of the coronary perforation is very important in the prognosis of the patient. The most accepted classification related to coronary perforation was made by Ellis et al. in a multicenter review study conducted with 12900 patients. Ellis type 1 refers to the presence of crater extending out of the lumen without contrast extravasation, Ellis type 2 refers to the absence of contrast extravasation and the presence of contrast staining observed in epicardial adipose tissue or myocardium, and Ellis type 3 refers to noticeable contrast extravasation with pronounced perforation (1). In our case, contrast extravasation was not seen as a jet, but it corresponded to the Ellis type 2 class due to contrast staining in myocardial and epicardial adipose tissue. An Ellis type 3 perforation has a worse prognosis, while types 1 and 2 have a relatively better prognosis than type 3 (5). Long-term balloon inflation in the area of coronary perforation may provide a solution in some cases. In our case, the balloon was inflated proximally to the rupture, and the bleeding was stopped by waiting. Close echocardiography follow-up should be performed in patients with type 2 and 3 perforations, and pericardiocentesis should be performed if pericardial tamponade is detected. In our case, pericardial effusion was not detected during echocardiography follow-ups.

Additionally, in case of perforation, the effect of heparin could be neutralized with protamine sulfate.

Graft-covered stents are much more rigid than other standard stents and are difficult to place without adequate guiding catheter support (2). The risk of stent thrombosis is higher in graft-covered stents than in non-covered stents. The rate of restenosis in the stent placement area is 32%, which is high (6). The reason for this situation may be the fact that endothelization occurs later when these stents are used. There is no agreed-upon view about the duration of antiaggregant drug use for graft-covered stents (7). In our case, the coronary rupture was successfully closed with a graft stent. If perforation cannot be controlled with a graft stent, emergency surgery should be considered.

CONCLUSION

Coronary perforation is a fatal complication. The balloon or stent balloon used in interventions should not be inflated without making sure that it is positioned in the coronary lumen. If it is inflated without being sure of this positioning, a coronary rupture may develop as a result. The steps to manage this complication are the rapid closure of the perforated area, the administration of protamine sulfate, graft-coated stent placement, and if necessary, emergency surgery. It should be kept in mind that coronary rupture is a potential complication in balloon and stent interventions, treatments needed for coronary perforation should be known well, and the necessary equipment should be available in the catheter laboratory during the entire procedure.

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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The Intersection of Psychiatry and Neurology in Young Adults: The Hidden Link Between Patent Foramen Ovale and Stroke

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Abstract

In young patients presenting with acute neurological symptoms, distinguishing between psychogenic and organic causes is crucial. Patent foramen ovale (PFO) is a significant factor that can lead to cardioembolic stroke in young individuals. This case report presents a patient who was initially misdiagnosed with a psychogenic disorder but was later found to have suffered a cardioembolic stroke.

A 17-year-old female presented with speech disturbances (stuttering, difficulty in articulation) and became emotional during history taking. Her past medical history revealed recurrent similar episodes. Neurological examination was normal; thus, a psychogenic etiology was suspected, and she was discharged after benzodiazepine infusion. However, she returned in the evening with right-sided numbness in her arm and leg, was again diagnosed with a psychogenic disorder, and received another benzodiazepine infusion. Later that night, she fell while attempting to use the restroom and was brought to the emergency department with speech impairment and right-sided weakness. This time, a neurological evaluation revealed left central facial paralysis, right hemiparesis, dysarthria, and cerebellar dysfunction. Brain MRI showed bilateral lacunar ischemic infarcts. A cardiology evaluation suspected PFO, which was confirmed via transesophageal echocardiography. The patient was diagnosed with acute ischemic stroke, started on fractionated heparin therapy, and underwent PFO closure five days later.

Although psychogenic disorders are common in young patients, organic causes must be ruled out in cases of acute neurological deficits. PFO is a leading cause of cryptogenic strokes in young adults, often presenting with recurrent transient ischemic attacks (TIAs). Overlooking a cardioembolic etiology can lead to diagnostic and treatment delays, potentially worsening outcomes. Unexplained neurological symptoms in young patients should be thoroughly investigated before attributing them to psychogenic causes. Cardioembolic conditions such as PFO can be successfully managed with early diagnosis and appropriate intervention.

Key Words: Patent foramen ovale (PFO), Cardioembolic stroke, Stroke in young adults, Psychogenic speech disorder, Fractionated heparin.

Genç Yetişkinlerde Psikiyatri ve Nörolojinin Kesişimi: Patent Foramen Ovale'nin İnme ile Gizli Bağlantısı

Özet

Genç yaş grubunda akut nörolojik semptomlarla başvuran hastalarda psikojenik ve organik nedenlerin dikkatli şekilde ayırt edilmesi gerekmektedir. Patent foramen ovale (PFO), genç hastalarda kardiyoembolik inmeye neden olabilen önemli bir faktördür. Bu olguda, başlangıçta psikojenik olarak değerlendirilen ancak sonrasında kardiyoembolik inmeye bağlı olduğu anlaşılan bir hasta sunulmaktadır.

17 yaşındaki kız hasta, konuşma bozukluğu (kekeleme, hecelemede zorluk) ile başvurmuş, öykü alınırken ağlamaya başlamıştır. Öyküden, benzer atakların daha önce de olduğu anlaşılmıştır. Nörolojik muayene normal bulunduğu için psikojenik değerlendirilmiş, benzodiazepin infüzyonu sonrası taburcu edilmiştir. Aynı günün akşamı sağ kol ve bacakta uyuşukluk şikayetiyle tekrar başvurmuş, yine psikojenik değerlendirilerek benzodiazepin infüzyonu uygulanmıştır. Gece tuvalete kalkarken düşen hasta, konuşmada güçlük ve sağ tarafında güçsüzlük ile üçüncü kez acile getirilmiştir. Bu kez nöroloji tarafından değerlendirilen hastada sol santral fasiyal paralizi, sağ hemiparezi, dizartri ve serebellar test bozukluğu saptanmıştır. Beyin MRG'de bilateral laküner iskemik infarktlar tespit edilmiştir. Kardiyoloji değerlendirmesinde PFO şüphesi konulmuş ve transözofageal ekokardiyografi ile doğrulanmıştır. Hasta akut iskemik inme tanısı ile yatırılmış, fraksiyone heparin tedavisi başlanmış ve beş gün sonra PFO kapatılmıştır.

Genç hastalarda psikojenik bozukluklar sık görülse de, akut nörolojik defisitlerde altta yatan organik nedenler dışlanmalıdır. PFO, genç erişkinlerde kriptogenik inmelerin önemli bir nedenidir ve özellikle tekrarlayan geçici iskemik ataklar (GİA) ile kendini gösterebilir. Kardiyoembolik etiyolojinin gözden kaçırılması, tanı ve tedavi gecikmesine neden olabilir. Genç yaşta açıklanamayan nörolojik semptomlar, psikojenik olarak değerlendirilmeden önce dikkatlice incelenmelidir. PFO gibi kardiyoembolik nedenler, erken tanı ve uygun yönetimle başarılı şekilde tedavi edilebilir.

Anahtar kelimeler: Patent foramen ovale (PFO), Kardiyoembolik inme, Genç yaşta inme, Psikojenik konuşma bozukluğu, Fraksiyone heparin.

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INTRODUCTION

Stroke is commonly associated with older individuals; however, it is also a significant cause of morbidity and mortality in young adults. The etiology of ischemic strokes in younger populations is often cryptogenic, necessitating an investigation into potential cardioembolic sources (1). PFO is among the most critical contributors to cardioembolic stroke in young patients, as it facilitates paradoxical embolism, leading to recurrent TIA and ischemic strokes (2).

In evaluating young patients with acute neurological symptoms, it is essential to differentiate between psychogenic and organic neurological disorders (3). Misattributing neurological symptoms to stress, anxiety, or psychogenic factors can result in missed diagnoses of serious underlying conditions. Therefore, comprehensive neurological assessments and appropriate diagnostic testing are crucial in young stroke patients.

In this case, attributing the previous symptoms solely to recurrent TIAs caused by PFO may be inaccurate, as they appeared as symptoms unnoticed by the patient and were frequently ascribed to psychogenic factors. There may be a concurrent psychogenic comorbidity that could be influencing the presentation of these symptoms.

This case report highlights the importance of a multidisciplinary approach in diagnosing a young patient initially misdiagnosed with a psychogenic disorder who was later found to have a PFO-related cardioembolic stroke. Early symptoms were overlooked, leading to a delay in diagnosis and treatment. This underscores the need for meticulous differential diagnosis and early intervention to improve long-term neurological outcomes.

CASE REPORT

A 17-year-old female presented to the emergency department (ED) in the morning with speech disturbances characterized by stuttering and difficulty in syllabification. The patient, who had an introverted personality, became emotional and started crying when asked questions during history taking. Due to communication difficulties, her mother provided the history. The mother reported: "She is behaving this way because we did not allow her to accompany her uncle, who arrived from abroad, to the airport and instead asked her to stay home and take care of her sibling. She has had similar speech

disturbances twice before when she was upset about something, but they resolved within a few days."

Based on this history and the normal findings on neurological examination performed by the ED physician, the condition was considered psychogenic. The patient received an intravenous infusion of 10 mg benzodiazepine. By the fourth hour after admission, her speech had improved; however, due to medication-induced drowsiness, she was discharged with a recommendation for psychiatric outpatient follow-up.

The same evening, the patient returned to the ED, now also complaining of numbness in her right arm and leg in addition to speech disturbances. The same physician reassessed the patient and again considered her symptoms to be psychogenic, attributing the inconsistent responses during a detailed examination to a conversion disorder. Another 10 mg intravenous infusion of benzodiazepine was administered over one hour. The patient, in a profoundly drowsy state, was taken home at the request of her family.

Approximately five hours later, around midnight, the patient attempted to use the restroom but collapsed near the sink. Her brother, awakened by the sound of the fall, found her lying on the floor, struggling to speak and unable to stand due to right-sided weakness. She was brought back to the ED for the third time. Upon arrival, her right arm and leg strength had returned to normal; however, her speech disturbance persisted. This time, the ED specialist requested a neurology consultation, and the patient was evaluated accordingly.

Neurological Examination:

- *Inspection:* The patient was petite, had a very fair complexion compared to her family members, and exhibited an introverted demeanor.
- *Speech*: Dysarthria characterized by difficulty in word articulation and sentence formation.
- *Cranial Nerves:* Left central facial palsy.
- *Sensory System*: Hemihypoesthesia in the right upper and lower extremities, sparing the face.
- *Motor System:* Right-sided hemiparesis with a muscle strength of 4+/5 on the Mingazzini test.
- *Reflexes*: Deep tendon reflexes were normoactive in all four limbs, with bilaterally normal plantar responses. There were no pathological reflexes
- *Coordination:* Impaired cerebellar tests on the left side and a tendency to fall to the left when attempting to stand.

Imaging Studies: Diffusion-weighted magnetic resonance imaging (MRI) revealed multiple small diffusion-restricted areas suggestive of ischemic infarcts: Left centrum semiovale and internal capsule, right sylvian fissure adjacent

subcortical region and two millimetric diffusion-restricted areas in the left cerebellum.

Given the bilateral and multifocal involvement, including infratentorial lesions, the findings were suggestive of acute lacunar ischemic infarcts of cardioembolic origin. A transthoracic echocardiography (TTE) performed in the ED by the cardiology team indicated a suspected patent foramen ovale (PFO). The patient was admitted to the neurology service with a diagnosis of acute ischemic stroke.

Since the exact onset of symptoms was unclear and the patient had been asleep prior to the worsening of her condition, she did not meet the time criteria for thrombolytic therapy. Instead, the following treatment regimen was initiated intravenous hydration and fractionated Heparin (5,000 units administered over one hour in IV infusion, 25,000 units in 1,000 cc normal saline, infused over 24 hours).

The following day, transesophageal echocardiography (TEE) definitively confirmed the presence of a PFO. A percutaneous closure procedure was scheduled for five days later. During this period, daily activated partial thromboplastin time (aPTT) levels were monitored to guide heparin continuation.

On day five, with an aPTT level still below 70, the patient underwent the PFO closure procedure. Post-procedure, intravenous heparin infusion was continued. By the third postoperative day, the aPTT level had risen to 110, prompting discontinuation of fractionated heparin. The anticoagulation regimen was switched to Enoxaparin: 0.6 IU twice Daily and acetil salicilic acid: 150 mg once Daily.

At the time of discharge, the patient's neurological symptoms had improved but had not completely resolved. At the one-month follow-up, all pathological neurological findings except for hypoesthesia had resolved. Follow-up brain and diffusion-weighted MRI showed a subacute lacunar infarct persisting in the left internal capsule, while all other previously noted infarct areas had completely resolved.

DISCUSSION

Neurological disorders often present with symptoms that overlap with psychiatric conditions. Particularly in young patients with acute neurological symptoms, it is essential to consider not only psychogenic causes but also organic neurological diseases (3). Misdiagnosis and inadequate clinical evaluation can lead to delayed treatment, increasing the risk of long-term disability or mortality. Therefore, during differential diagnosis, a thorough assessment of symptoms and appropriate diagnostic testing should be conducted.

In this case, the patient's young age initially led to a diagnosis of psychogenic speech disorder. However, the subsequent development of motor symptoms suggested an underlying

cardiovascular and neurological etiology. Although stroke is traditionally considered rare in young individuals, PFO is a significant risk factor for cardioembolic stroke in this population (1). PFO is found in approximately 40% of young stroke patients and can frequently cause cerebral ischemia through a paradoxical embolism mechanism (2). Consequently, PFO should be actively investigated in young patients with unexplained stroke or TIA.

In this case, the recurrent TIAs caused by PFO manifested as symptoms that the patient did not recognize and were often attributed to psychogenic factors. However, many neurological disorders can be exacerbated by triggers such as stress or physical and mental exhaustion (4). Thus, while stress should be considered a triggering factor in patients with neurological and cardiovascular conditions, underlying organic pathology should not be overlooked.

Clinical clues used to differentiate between symptoms and findings of cerebrovascular disease and organic versus psychogenic pathologies play a critical role in the diagnostic process and shape therapeutic approaches. Organic pathologies are generally supported by distinct laboratory tests and imaging findings, whereas psychogenic pathologies are more often based on the patient's clinical history and the relationship of symptoms to psychological factors, typically lacking accompanying radiological or laboratory findings. Furthermore, psychogenic cases usually extend over a longer period.

For instance, in cerebrovascular diseases, the sudden onset of specific symptoms, such as losses in particular neurological functions like motor skills or speech, indicates an organic cause. Conversely, symptoms usually beginning or worsening with stress and patients excessively dramatizing their symptoms can point to psychogenic factors.

Additionally, while symptoms in organic pathologies are expected to present a stable and consistent pattern, those in psychogenic conditions can be more variable and inconsistent. The reproducibility of findings obtained during clinical examinations supports the diagnosis of organic disorders, whereas variable findings may indicate psychological influences. Considering our case, the clinical scenario preceding the event can very likely be attributed to a thromboembolic process.

In the management of acute ischemic stroke, thrombolytic therapy (such as tissue plasminogen activator - tPA) is the gold standard for recanalization. However, it cannot be administered to all patients due to time constraints (5). In such cases, alternative anticoagulant strategies, such as fractionated heparin, may be considered. Fractionated heparin

is used as a recanalization-supporting agent, particularly in ischemic strokes of cardioembolic origin (6). While low-molecular-weight heparins (LMWH) have gained popularity in recent years, clinical experience suggests that fractionated heparin may provide more effective recanalization in cardioembolic strokes and help preserve the penumbra area. However, LMWH has been reported to be less effective in achieving similar outcomes (7).

This case demonstrates that PFO-related cardioembolic stroke can be successfully managed with early diagnosis and a multidisciplinary approach. PFO closure is an effective method for reducing the risk of recurrent strokes and should be considered, particularly in patients who are resistant to medical therapy (8). In our patient, significant neurological improvement was achieved following PFO closure.

CONCLUSION

In conclusion, diagnosing young patients with acute neurological symptoms as psychogenic without ruling out organic causes can lead to delays in treatment and management. A collaborative assessment by neurology, cardiology, and psychiatry specialists plays a crucial role in managing such cases. A comprehensive assessment of both organic and psychogenic factors is vital in managing cerebrovascular and similar neurological conditions. However, interactions and potential overlaps between these two types of pathologies can complicate diagnostic and treatment strategies. This necessitates a multidisciplinary approach and individualized treatment plans. Additionally, investigating underlying cardiovascular risk factors such as PFO and implementing early intervention in appropriate patients is essential for minimizing the risk of recurrent strokes.

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report.

Author Contributions: Conception – Sukran Kaygisiz, Tuba Gul; Design - Sukran Kaygisiz, Tuba Gul; Data Collection and/or Processing - Sukran Kaygisiz, Tuba Gul; Analysis and/or Interpretation - Sukran Kaygisiz, Tuba Gul; Literature Search - Sukran Kaygisiz, Tuba Gul; Writing - Sukran Kaygisiz, Tuba Gul; Critical Review - Sukran Kaygisiz, Tuba Gul

Conflict of Interest: The authors declare that they have no known competing financial interests or personal relationships that could affect the work reported in this article.

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Sleep-Related Headache Following Metformin Use: Hypnic or Toxic?

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Abstract

Headache is among the most frequently encountered symptoms in clinical practice. Occipital neuralgia is characterized by sudden, sharp, electric shock-like pains in the distribution area of the occipital nerve. In this report, we present a 54-year-old female patient who developed occipital neuralgia-like symptoms shortly after the initiation of metformin treatment for type 2 diabetes mellitus. The symptoms resolved following the discontinuation of the drug. This case is discussed in light of the literature to highlight a potentially rare headache pattern triggered by metformin.

This case describes an unusual form of headache possibly associated with metformin therapy. It underscores the importance of considering uncommon side effects of commonly prescribed medications such as metformin in differential diagnoses.

Key Words: Occipital neuralgia, Metformin, Headache, Trigeminal neuralgia, Neuropathic pain

Metformin Sonrası Gelişen Uykuya Özgü Baş Ağrısı: Hipnik mi, Toksik mi?

Özet

Baş ağrısı, klinik pratikte en sık karşılaşılan semptomlardan biridir. Oksipital nevralji, özellikle oksipital sinirin innervasyon alanında ani, keskin, elektrik çarpması tarzında ağrılarla karakterizedir. Bu çalışmada, tip 2 diabetes mellitus tanısıyla başlanan metformin tedavisinden kısa süre sonra gelişen ve ilacın kesilmesiyle gerileyen, oksipital nevralji benzeri semptomlar gösteren 54 yaşındaki kadın hasta sunulmuştur. Bu olgu, metformin ile tetiklenmiş olabilecek nadir bir baş ağrısı paternini vurgulamak amacıyla literatür ışığında tartışılmıştır.

Bu olgu, metformin tedavisi ile ilişkili nadir bir baş ağrısı formunu tanımlamaktadır. Metformin gibi sık kullanılan ilaçların, alışılmadık yan etkilerle ilişkili olabileceği akılda tutulmalı ve ayırıcı tanıda bu tür ilişkiler göz önünde bulundurulmalıdır.

Anahtar kelimeler: Occipital neuralgia, Metformin, Headache, Trigeminal neuralgia, Neuropathic pain

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INTRODUCTION

Occipital neuralgia arises from irritation of the greater or lesser occipital nerves and is characterized by sudden, stabbing, electric shock-like pains, usually unilateral and radiating from the neck to the posterior head. Secondary

causes include trauma, cervical disc pathologies, and vascular compressions (1). In some cases, no specific etiology can be identified.

Metformin is a biguanide widely used in the treatment of type 2 diabetes mellitus. Although generally well tolerated, it may occasionally lead to neurological side effects such as vitamin B12 deficiency and peripheral neuropathy (2).

CASE REPORT

A 54-year-old female patient presented with a two-month history of posterior head pain described as a sensation of pressure or brief electric shock-like episodes, particularly triggered by minor stimuli. There was no history of trauma or febrile illness. Notably, 3–4 years ago, she experienced a transient loss of consciousness following a playful slap to the nape.

The patient had been taking sertraline 100 mg/day for 23 years due to panic disorder. She reported experiencing chest tightness, a pressing pain in the heart, and emotional tension when she missed a dose. Despite regular use of the medication, episodes of intense stabbing chest pain, psychological distress, and even insomnia for several days could still occur in the context of emotional upset. Otherwise, her sleep was reported as regular.

A neurosurgeon had previously evaluated the patient and found no abnormalities on brain MRI. Cervical MRI revealed signs suggestive of cervical disc herniation, for which a regimen including Geralgine-K and Dexfort was initiated, to be taken as needed. The patient reported partial symptom relief with these medications.

She experienced a baseline persistent headache accompanied by paroxysmal, electric shock-like episodes predominantly occurring at night, often waking her up. These attacks tended to recur at similar times during the night.

Two months earlier, the patient was evaluated by her primary care physician, and laboratory tests revealed an HbA1c level of 7.0%. She was diagnosed with diabetes mellitus by an internal medicine specialist and started on metformin 850 mg twice daily. Approximately one week after starting metformin, she developed severe headaches. Ambulatory blood pressure monitoring revealed elevated readings, and the patient was diagnosed with hypertension. Candesartan 16 mg/day was initiated, resulting in blood pressure normalization from 190/100 mmHg to 110/70 mmHg. She continued to experience nocturnal headaches despite normotensive readings during these episodes.

Past medical history: No prior history of trauma, febrile illness, or smoking. The patient did not have asthma. She was undergoing regular follow-up for bilateral fibrocystic breast adenomas.

Family history: Positive for neuralgic disorders. Her father had required long-term pharmacological treatment for neuralgia, and her sister had undergone surgical intervention for trigeminal neuralgia after inadequate response to medical therapy.

Neurological examination: Revealed a left peripheral facial palsy, noted as a long-standing sequela, and a minimal essential tremor in the right hand. Other neurological findings were unremarkable.

Neuroimaging findings: Brain MRI was within normal limits. Cervical MRI demonstrated mild anterior bulging at the C4–C5 level and right-sided bulging at C5–C6 with borderline nerve root contact.

Initial treatment with propranolol 2x1 and triptans for acute episodes was ineffective. One month later, due to persistent symptoms, topiramate was initiated but did not result in improvement. Occipital nerve block (GON block) was then administered, and partial benefit was achieved after the second session.

At this stage, a possible association between the clinical picture and metformin therapy was considered. The patient was referred back to internal medicine, and metformin was discontinued. A three-month period of dietary and exercise-based glucose control was advised, without initiating an alternative pharmacological agent.

Within one week of discontinuing metformin, the patient reported a rapid reduction in headache intensity. Complete resolution of symptoms was observed by the third week post-discontinuation. As symptoms resolved, there was no further need for occipital nerve block therapy.

At the three-month follow-up, the patient's HbA1c had increased to 7.2%, prompting the initiation of a sulfonylurea by the internal medicine specialist. At both the 6- and 12-month follow-up visits in the neurology outpatient clinic, the patient remained asymptomatic with regard to headaches. Neurological follow-up was therefore concluded.

DISCUSSION

Metformin, a biguanide derivative, is widely recommended as a first-line agent in the treatment of type 2 diabetes. Although generally safe, rare but significant adverse effects may occur. The most common side effects are gastrointestinal; however, serious complications such as B12 deficiency, lactic acidosis, and peripheral neuropathy are also documented (1,2).

This case highlights the development of occipital neuralgia-like headaches shortly after metformin initiation, which resolved completely following drug withdrawal. Reports linking metformin and headache are scarce. A study by Wile and Toth

suggested an increased risk of peripheral neuropathy in patients using metformin, possibly related to B12 deficiency (2).

However, B12 levels were normal in this case. One possible mechanism for the headache is the increase in nitric oxide (NO) production induced by metformin. NO's effect on vascular structures is known to trigger headaches (3). Moreover, metformin may reduce asymmetric dimethylarginine (ADMA) levels, thus increasing endothelial NO synthesis, which may lead to cerebral vasodilation and headache (4).

Occipital neuralgia is typically paroxysmal, electric shock-like pain resulting from irritation of the occipital nerves (5).

Secondary causes include cervical disc disorders, trauma, tumors, or vascular anomalies. Although cervical MRI in this case revealed only minimal pathology, the temporal relationship between symptom onset and metformin use, along with complete resolution upon discontinuation, points to a pharmacological rather than structural cause.

There are few documented cases of metformin-induced headaches in the literature. One report described a patient developing cluster-like headache following metformin initiation, with resolution upon discontinuation (3). In our case, the partial response to occipital nerve block and rapid, sustained improvement after stopping metformin further supports a causal relationship. Additionally, the family history of neuralgic disorders suggests a possible genetic predisposition that may enhance susceptibility to drug-related neurological effects (6,7).

Interestingly, the nocturnal pattern of headaches in this patient raises the differential diagnosis of hypnic headache. Hypnic headache typically affects individuals over 50, awakens them from sleep, and presents with pulsating or dull pain in brief episodes (8,9). According to ICHD-3 criteria, hypnic headache is often treated successfully with caffeine or indomethacin. However, in this case, the clear temporal correlation with metformin use and the complete resolution upon its cessation suggest a drug-induced neurological side effect rather than primary hypnic headache. Nonetheless, typical features, accompanying symptoms, and medication history should be carefully reviewed when considering this diagnosis.

CONCLUSION

While metformin is generally regarded as a safe antidiabetic agent, clinicians should remain vigilant for rare neurological side effects. A thorough drug history should be obtained in patients presenting with headache or neuralgia-like symptoms, and the potential neurotoxicity of widely used medications like metformin should be considered. This case underscores the importance of including metformin-induced

occipital neuralgia-like symptoms in the differential diagnosis.

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Little-Known Cause of Chest Pain: Tietze Syndrome Case Report

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Abstract

Tietze syndrome is a rare inflammatory disease that can be seen in all age groups and has characteristic features such as swelling, chest pain, tenderness and pain localized in the anterior chest wall, usually in the second or third costal region. The differential diagnosis includes many diseases that cause chest pain. Although the diagnosis is made by excluding other inflammatory pathologies and causes of chest pain, it usually does not require the use of additional diagnostic methods. Its etiology is not fully known, but heavy exercise and minor traumas are considered. Anti-inflammatory drugs and lifestyle changes are recommended in the treatment of Tietze syndrome. Surgical treatment can be applied for refractory cases, but it is generally not necessary. In this case, we report a 38-year-old female patient who presented with chest pain for 1 month, had normal laboratory values and the patient's pathologies were excluded. This case underscores the importance of clinical examination in diagnosing Tietze syndrome, avoiding unnecessary invasive tests.

Key Words: Tietze Syndrome, Chest pain, Costosternal

Göğüs Ağrısının Az Bilinen Nedeni: Tietze Sendromu Olgu Sunumu

Özet

Tietze sendromu, her yaş gurubunda görülebilen genelde ikinci veya üçüncü kostasternal bölgede, şişlik, göğüs ağrısı, hassasiyet ve göğüs ön duvarında lokalize ağrı ile karakteristik özelliklere sahip nadir bir inflamatuar hastalıktır. Ayırıcı tanısında birçok göğüs ağrısına neden olan hastalığı kapsamaktadır. Tanı, diğer enflamatuar patolojilerin ve göğüs ağrısı nedenlerinin dışlanması ile konmakla beraber genellikle ek tanı yöntemlerinin kullanılmasını zorunlu kılmaz. Etiyolojisi tam olarak bilinmemekte fakat ağır egzersiz ve minör travmalar düşünülmektedir. Tietze sendromunun tedavisinde anti-inflamatuar ilaçların kullanılması ve yaşam tarzı değişikliklerin uygulanması önerilir. Cerrahi tedavi refrakter olgular için uygulanabilmekle birlikte genellikle gerekli değildir. Bu olgumuzda 1 aydır göğüs ağrısı şikayeti ile başvuran, laboratuvar değerleri normal olan ve kardiyak patolojilerin ekarte edildiği 38 yaşında bayan hastayı bildiriyoruz. Bu vaka gereksiz invaziv testlerden kaçınarak Tietze sendromunun teşhisinde klinik muayenenin önemini vurgulamaktadır.

Anahtar kelimeler: Tietze sendromu, Göğüs ağrısı, Kostosternal

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INTRODUCTION

The disease known as Tietze syndrome is a rare, non-suppurative inflammatory process characterised by chest pain and swelling at the costochondral junction (1). It is a painful but

benign condition, mostly unilateral in the second or third costosternal or costochondral joints. Its etiology is not fully known, but heavy exercise and minor traumas are considered. It is usually seen in the 2nd-3rd decade, but it can occur at any age. The symptoms of Tietze syndrome may begin suddenly or develop gradually. The patient's discomfort is usually positional. The severity of the pain may increase with movement of the arm and trunk, coughing, sneezing and deep breathing (2).

CASE REPORT

A 38-year-old female patient who applied to the internal medicine clinic had been complaining of chest pain for the last 1 month. The patient had complaints of swelling and redness in the anterior chest wall. She described that it was aggravated by straining movements on the rib cage. The patient said that she had been doing heavy exercise at home approximately 1 week before the onset of her complaints and that she felt a strain on her rib cage during this time. She had no additional complaints such as fever, shortness of breath, palpitations or night sweats. No cardiac pathology was detected in the patient who applied to the cardiology department 15 days ago. There was no history of disease or medication use in the patient's medical history. Neck examination revealed redness, tenderness and a 1.5x1.5 cm moderately hard swelling in the left costosternal joint. There was no crepitation or deformity on palpation. Cardiovascular, respiratory, and musculoskeletal examinations were unremarkable. No pain, tenderness or mass was found in the neck, chest, arm, leg, hand or foot joints.

In laboratory tests, routine biochemistry and hemogram were normal. The eritrocye sedimentation rate (ESR) was 28 mm/hour and the c-reactive protein (CRP) was slightly elevated at 12 mg/dl. Rheumatoid factor and anti-nuclear antibodies were tested for the differential diagnosis of rheumatoid arthritis and other seropositive inflammatory arthritis, and both were found to be negative.The electro cardiography performed on the patient, which showed natural and sinus rhythm. The superficial USG performed on the patient, thickening of the cartilage tissue in the left 2nd costosternal joint, signs of inflammation, and edema were detected. Based on clinical findings and tests, the patient was diagnosed with Tietze syndrome. The patient was started on oral nonsteroidal antiinflamatuar drugs, and topical gel treatment. It was recommended to stay away from activities could cause trigger pain. When the patient came for a follow-up visit after 2 weeks of treatment, he stated that the pain and swelling had decreased significantly. On physical examinationin tenderness and swelling had regressed. One month later, it was determined that his

complaints had completely disappeared and his physical examination was completely normal.

DISCUSSION

Tietze Syndrome was first described in 1921 by the German surgeon Alexander Tietze (3). The exact aetiology is unknown, it is rarely encountered. Although its exact etiology is not known, it is thought that recurrent microtraumas may cause small tears in the sternocostal ligaments and paving the way for the disease. It is more frequently detected in women and in people under the age of 40, but it can be seen in all age groups (4). In a retrospective study of 24 patients from our country, the mean age was reported as 21.2 (5). Our patient was 38 years old, which isin consistency with the literature.

Patients usually present with complaints of acute chest pain without a history of trauma. Individuals usually feel the pain more acutely during movement and position changes, coughing, sneezing, and deep breathing. The pain may radiate to the neck, arms, and shoulders.

It is usually unilateral (70%) and involves the 2nd-3rd costosternal joints. Sternoclavicular joint involvement has been detected rarely (6). In our case, there was only left costosternal joint was involved. During the periods when the patient's complaints increase, high fever and CRP and ESH values can be detected (2). At the time of our patient's admission, CRP and ESH values were slightly elevated, but there was no fever.

USG imaging is one of the most common evaluation methods showing soft tissue swelling in the area of the ongoing inflammatory process. In a study, a case of Tietze Syndrome, which was diagnosed by USG imaging and treated with USG-guided corticosteroid injection, was presented, emphasising the importance of USG imaging in diagnosis and treatment (7). In our case, we performed USG imaging because it is reliable in soft tissue diagnosis and does not involve radiation, and we obtained results by identifying inflammatory findings.

Tietze syndrome can resemble cardiac, pulmonary, neurological and intra-abdominal pathologies due to the variety of its symptoms and should therefore be carefully evaluated in the differential diagnosis of patients with abdominal and chest pain (8). In addition, rheumatoid arthritis (RA), gout, pyogenic infections, neoplastic processes and chest wall pain syndromes such as costochondritis and sternal syndrome, which may involve the costosternal joints, are important clinical conditions that should be considered in the differential diagnosis.

Treatment is usually with oral or local nonsteroidal anti-inflammatory and analgesic drugs. However, in rare cases, additional

treatment protocols should be applied to the patients. Surgery may be considered in cases that do not respond to conventional treatment. In chronic cases, intercostal nerve blocks can be used successfully, but the possibility of recurrence has been reported frequently (9). In a study conducted by Şentürk E. et al., nonsteroidal anti-inflammatory drugs (NSAIDs) were compared with prolotherapy. The findings revealed that patients who received prolotherapy showed a faster recovery process compared to patients who used NSAIDs. The researchers emphasised that prolotherapy can be an effective treatment alternative in cases where other treatment approaches are not suitable (10). In our patient, one month of oral non-steroidal anti-inflammatory and local analgesic treatment was sufficient for thepatient's symptoms and physical examination to improve completely.

CONCLUSION

Although rare, Tietze syndrome should be considered in localized chest pain to reduce diagnostic delays and unnecessary interventions. However, a detailed history and physical examination are important in patients presenting with chest pain to exclude life-threatening diseases, Tietze syndrome is a disease that should be kept in mind in differential diagnosis. It is important for doctors to recognise this benign syndrome in order to minimise the patient's physical distress, the psychological effects of the disease, time loss and costs.

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