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Evaluating Adverse Events and Management Strategies in Foam Sclerotherapy: A Case Report of Sinus Venosus Atrial Septal Defect

Köpük Skleroterapi ile Ortaya Çıkan Sinus Venosus Atrial Septal Defekt: Advers Olaylar ve Yönetim Üzerine Bir Vaka Sunumu

Ufuk Türkmen[1,*] | **Kudret Atakan Tekin**[2]

[1]Hitit University, Faculty of Medicine, Department of Cardiovascular Surgery, Çorum, Türkiye
[2]Çorum Erol Olçok Training and Research Hospital, Department of Cardiovascular Surgery, Çorum, Türkiye

Sorumlu Yazar | Correspondence Author
Ufuk Türkmen
ufukturkmen@hitit.edu.tr
Address for Correspondence: Hitit University Çorum Erol Olçok Training and Research Hospital, Çepni Dist. İnönü Str. No:176, 19040 Çorum, Türkiye

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Evaluating Adverse Events and Management Strategies in Foam Sclerotherapy: A Case Report of Sinus Venosus Atrial Septal Defect

ABSTRACT

Foam sclerotherapy (FS) is widely employed for minimally invasive varicose vein treatment. Despite contraindications in symptomatic right-to-left shunt cases like patent foramen ovale, routine pre-procedure transthoracic echocardiogram (TTE) screening isn't standard. This article discusses managing chest symptoms during FS in a young woman later diagnosed with an unknown sinus venosus atrial septal defect (SV-ASD). FS was administered to a 38-year-old female with symptomatic CEAP-1, who developed sudden shortness of breath and chest tightness. Subsequent diagnosis revealed SV-ASD. Minimally invasive repair followed four weeks later. While FS is generally safe, it poses systemic adverse event risks, emphasizing vigilance in managing SV-ASD patients undergoing FS

Keywords: Adverse events, foam sclerotherapy, sclerotherapy, minimally invasive treatment, sinus venosus atrial septal defect.

ÖZET

Köpük skleroterapi (KS), minimal invaziv varis tedavisi için yaygın olarak kullanılmaktadır. Patent foramen ovale gibi semptomatik sağdan sola şant vakalarında kontrendikasyonlara rağmen, işlem öncesi rutin transtorasik ekokardiyogram (TTE) taraması standart değildir. Bu makale, daha sonra bilinmeyen bir sinus venosus atrial septal defekt (SV-ASD) teşhisi konulan genç bir kadında KS sırasında gelişen göğüs semptomlarının yönetimini ele almaktadır. Semptomatik CEAP-1 tanısı olan 38 yaşındaki bir kadına KS uygulanmış ve hastada göğüs semptomları gelişmiştir. Sonrasında yapılan teşhis SV-ASD'yi ortaya koymuştur. Dört hafta sonra minimal invaziv onarım gerçekleştirilmiştir. KS genellikle güvenli olmakla birlikte, sistemik advers olay riskleri taşır, bu da KS uygulanan SV-ASD hastalarının yönetiminde dikkatli olunması gerektiğini vurgular.

Anahtar Sözcükler: Advers olaylar, köpük skleroterapi, minimal invaziv tedavi, sinus venosus atrial septal defekt, skleroterapi.

Introduction

Sclerotherapy, a procedure widely employed for several years in the minimally invasive treatment of telangiectasias, spider veins, reticular veins, and varicose veins in the lower extremities, is generally regarded as safe (1). Despite its safety profile, sclerotherapy encompasses a spectrum of adverse events (AEs) ranging from simple local reactions to potentially life-threatening complications such as cerebrovascular events, cardiac toxicity, pulmonary embolism, deep vein thrombosis, and severe anaphylactic reactions. Some studies have reported the detection of patent foramen ovale (PFO) in patients who experienced systemic AEs during sclerotherapy. Although systemic AEs are less frequent than local AEs, they can lead to serious outcomes, including fatalities, when they occur (1,2).

This article presents the case of a patient with an undiagnosed sinus venosus atrial septal defect (SV-ASD) who developed chest tightness and shortness of breath likely attributable to air embolism or chemical-induced vasospasm during foam sclerotherapy (FS).

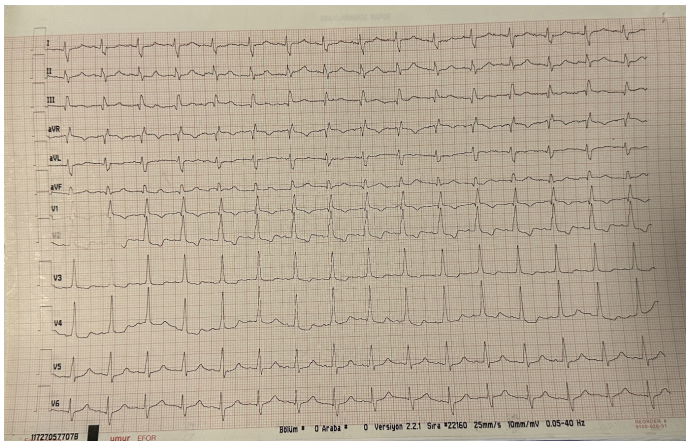
Case Presentation

Informed consent was obtained from the patient for the publication of this case report. A 38-year-old female patient with symptomatic CEAP-1 presented with sudden onset chest tightness and shortness of breath during foam injection sclerotherapy of the reticular vein in her right leg. Pre-procedure Doppler ultrasonography revealed no insufficiency in the great and small saphenous veins, deep venous system, or saphenofemoral junction, with standard diameters. The patient had no known medical history or medications.

Polidocanol foam (1%) (AethoxysklerolVR, Keussler Pharma, Wiesbaden, Germany) was prepared using the Tessari method with a double syringe and a three-way tap (3). After cleansing the target localization with ethyl alcohol, 1 ml of foam was injected into the target vein using a 30G sclerotherapy needle guided by Portable Veinlite (Veinlite LEDXVR, TransLite, LLC, Sugar Land, TX, USA). Compression was applied to the target vein.

While under compression, the patient experienced sudden shortness of breath and chest tightness. She was placed in the Trendelenburg position, and nasal oxygen at 5 L/min was administered. Peripheral vascular access was established, and the patient received 2 ml of pheniramine maleate and 40 mg of methylprednisolone. Enoxaparin 0.6 ml was injected subcutaneously, and 100 mg of acetylsalicylic acid was administered sublingually. Upon examination, her temperature was 37°C, respiratory rate 22 breaths per minute, oxygen saturation 99%, and blood pressure 138/86 mmHg. Electrocardiogram (ECG) showed no evidence of ischemia, with a heart rate of 100 beats per minute and sinus rhythm. Right bundle branch block and 150 degrees right QRS axis deviation were observed on ECG (Figure I). Neurological examination was unremarkable. The patient's symptoms improved after approximately 20 minutes, and further evaluations were conducted. Chest X-ray revealed enlargement of the right side of the mediastinum. Initial high-sensitivity troponin T levels were elevated at 760 ng/L, which decreased to 544 ng/L after 4 hours and 236 ng/L after 24 hours. D-dimer level was 0.48 µg/mL, and all other blood tests were within normal limits. Anaphylaxis due to the sclerosing agent was considered unlikely due to the absence of clinical manifestations. Myocardial infarction was ruled out based on troponin levels and the absence of ischemic findings on ECG. CT pulmonary angiogram for pulmonary embolism yielded negative results.

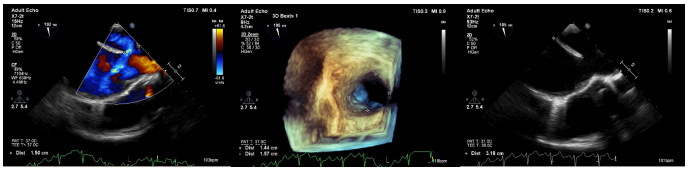
Figure I. ECG recording obtained during onset of patient symptoms.



Transthoracic echocardiogram (TTE) was performed to assess cardiac function and rule out pericardial pathologies. Left ventricular function and heart valves appeared normal, while the right atrium

and ventricle were dilated, and pulmonary artery pressure was within normal range. A left-to-right shunt was identified. After 24 hours of observation, the patient was discharged with enoxaparin 0.6 ml and 81 mg of acetylsalicylic acid.

Figure II. Clinical application of TEE images depicting sinus venosus-atrial septal defect.



Two weeks later, she was readmitted for further evaluation and preparation for atrial septal defect (ASD) surgery. Transesophageal echocardiography (TEE) revealed a left-to-right shunt with a Qp: Qs ratio of 2.1, along with SV-ASD and anomalous right superior pulmonary venous return (Figure II). Coronary artery angiography was not performed due to the absence of risk factors.

Given the patient's cosmetic concerns and young age, a minimally invasive approach was chosen. An incision was made through the fourth right intercostal space, and cardiopulmonary bypass with aortic cross-clamping was utilized with bicaval and femoral artery cannulation. Del Nido cardioplegia induced cardiac arrest under moderate systemic hypothermia.

The interatrial defect was accessed through a right atriotomy extending into the superior vena cava (SVC). Examination within the right atrial cavity revealed SV-ASD near the SVC and the right upper pulmonary vein draining proximally to the SVC. A fresh autologous pericardial patch was sutured with a 5/0 monofilament suture to close the SV-ASD and redirect pulmonary venous flow to the left atrium. SVC expansion plasty was not performed due to the absence of narrowing.

The patient was extubated 4 hours post-surgery and discharged in good condition on the fifth postoperative day with 100 mg of acetylsalicylic acid.

Discussion

Sclerotherapy is designed to induce a controlled thrombophlebitis reaction by inflicting endothelial damage with a sclerosing agent within the vein wall. This process triggers fibrosis and eventual obliteration of the vessel (4). Typically, the liquid sclerosing agent is combined with a gas (such as air or CO2) in a 1:4 ratio using the Tessari method to generate foam, which is then administered to the target vessels through various catheter techniques (3). However, this technique presents drawbacks such as variations in gas/liquid compositions, bubble size, foam behavior, and safety concerns. Systemic AEs may arise due to the pharmacological properties of the sclerosing agent, air embolism, or chemical-induced vasospasm (2).

ASDs represent the third most prevalent congenital heart anomaly in adults, with SV-ASDs accounting for approximately 4-11% of all ASDs. SV-ASD denotes a communication defect between one or more right pulmonary veins and either the superior vena cava (superior sinus venosus) or the inferior vena cava (inferior sinus venosus) (5). While the majority of SV-ASD patients remain asymptomatic throughout childhood, even those with left-to-right shunts may not exhibit discernible symptoms until adulthood. Most SV-ASDs are incidentally detected during evaluations for nonspecific clinical signs such as exertional dyspnea, arrhythmias, and paradoxical embolism.

Paradoxical embolism through ASDs can be triggered by thrombus, fat, or air (6). Several case reports have documented ischemic stroke resulting from paradoxical embolism following foam injection sclerotherapy for varicose veins in patients with PFO. The resulting ischemia may stem from air embolism or chemical-induced vasospasm (7). Moreover, rare yet potentially fatal cardiac complications, including post-sclerotherapy myocardial infarction and sudden cardiac death, have been reported, indicating a similar mechanism of myocardial injury as observed in cerebral events following foam sclerotherapy (8-9). In our case of SV-ASD, the immediate onset of symptoms following foam sclerotherapy suggests myocardial injury due to air embolism or chemically induced vasospasm.

Stroke or cardiac symptoms arising during or after foam sclerotherapy should raise suspicion for ischemia due to paradoxical embolism of the foam through ASDs. It is crucial to acknowledge that such

ischemic events may be attributed to air embolism or chemical spasm (10). While specific screening for the presence of a right-to-left shunt before foam sclerotherapy is not deemed necessary, it is imperative to recognize that foam sclerotherapy represents an absolute contraindication in patients with symptomatic right-to-left shunts (e.g., symptomatic SV-ASD).

As a result, physicians must remain cognizant of the potential serious and even life-threatening AEs associated with sclerotherapy, adequately inform their patients, and be prepared to manage such events effectively. The incidence or prevalence of complications, particularly cardiac AE, is challenging to assess but can be potentially fatal. There is ongoing debate regarding whether all patients should undergo transthoracic echocardiography screening before undergoing sclerotherapy. However, routine TTE screening is not currently feasible.

Future research, leveraging advancing technology, should prioritize the development of sclerosing agents with improved safety profiles. Furthermore, further investigation is warranted to determine the optimal foam volume for safe administration.

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