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Case Report

ARCHIVES OF CLINICAL AND MEDICAL CASE REPORTS

ISSN: 2575-9655

Successful Resuscitation and Use of VA ECMO Following Cardiovascular Collapse during Ethanol Sclerotherapy - A Case Report

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Abstract

Ethanol sclerotherapy is widely used for the treatment of vascular malformations and can rarely lead to fatal cardiopulmonary collapse due to pulmonary vasospasm and right heart failure, pulmonary embolism, or cardiac arrhythmias. We present a case of successful resuscitation after sudden cardiovascular collapse with the use of venous-arterial extracorporeal membrane oxygenation (ECMO) in an eighteen year old female following ethanol sclerotherapy for a venous malformation. This report raises awareness of the most fatal and rare risk factors for ethanol sclerotherapy. More research is needed to determine the efficacy of invasive monitoring techniques such as continuous pulmonary artery pressure monitoring in preventing such complications during sclerotherapy procedures.

Keywords: Ethanol Sclerotherapy; Pulmonary Vasospasm; Venous-Arterial; ECMO; Angiography

Abbreviations list: ACLS – Advanced Cardiac Life Support; CT – Computed Tomography; ECMO - Extracorporeal Membrane Oxygenation; ICU – Intensive Care Unit; LMA – Laryngeal Mask Airway; MRI - Magnetic Resonance Imaging; PAP – Pulmonary Artery Pressure; SBP - Systolic Blood Pressure; VA – Venous Arterial

Introduction

Ethanol sclerotherapy is an effective treatment for venous malformations [1], as it causes necrosis of the vascular wall [2,3]. Although complications are mostly minor (local erythema, blistering), ethanol may lead to fatal complications such as pulmonary vasospasm with severe pulmonary hypertension and right heart failure and pulmonary embolism [4,5]. We report a case of successful resuscitation with venous-arterial (VA) extracorporeal membrane oxygenation (ECMO) after cardiovascular collapse following ethanol sclerotherapy.

Case Description

An 18-year-old healthy female (60 kg, 170 cm) with no allergies was admitted for elective sclerotherapy of a venous malformation. The malformation was diagnosed by magnetic resonance imaging (MRI) and vessels with systemic communication were ruled out. Preanesthetic evaluation was unremarkable. After standard monitors were applied, anesthesia was induced, a laryngeal mask airway (LMA) was inserted under spontaneous ventilation, and the patient was transferred to the prone position. Fluoroscopy

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Citation: Orie Sella, Amir Gal-Oz, Isaac Kori, Uri Carmi, Yael Lichter, Adi Nimrod, Elisheva Fiszer. Successful Resuscitation and Use of VA ECMO Following Cardiovascular Collapse during Ethanol Sclerotherapy - A Case Report. Archives of Clinical and Medical Case Reports. 8 (2024): 36-39.

Received: December 11, 2023
Accepted: January 03, 2024
Published: February 29, 2024

Sella O, et al., Arch Clin Med Case Rep 2024
DOI:10.26502/acmcr.96550656

confirmed a venous malformation with subcutaneous veins draining the gluteus muscle, and 35 ml 98% ethanol was injected by the interventional radiologist. The LMA was removed after recovery of airway reflexes.

Fifteen minutes after arrival to recovery, the patient became bradycardic (50 BPM), hypotensive (systolic blood pressure (SBP) 70 mm Hg), and unresponsive. The anesthesiologist administered 10 mg Ephedrine. Her heart rate decreased to 30 BPM, two doses of atropine 1 mg were administered, and the patient was intubated. The intensive care unit (ICU) and cardiology department were alerted. A central venous catheter was inserted into the right internal jugular vein, and an arterial line was inserted in the right femoral artery. An adrenaline infusion was initiated. SBP was 70 mm Hg, and saturation was 75%. Echocardiography showed an enlarged right ventricle with a D sign, suspicious for pulmonary embolism. The patient was transferred to the CT suite for a lung scan.

On the scanning table, the patient went into asystole. Chest compressions were immediately initiated, followed by 1 mg of epinephrine. Return of spontaneous circulation was achieved after two minutes of advanced cardiac life support (ACLS), and a norepinephrine infusion (0.08 mg/ml) was initiated at a rate of 10 ml/h. Analysis of the scan showed extreme vasospasm of the pulmonary veins and collapse of the left atrium, consistent with right heart failure. The patient was transferred to the ICU. Vitals were 85/50 mm Hg, HR 128 BPM, despite maximal infusion rates of vasopressors, and echocardiography demonstrated right heart enlargement and pulmonary hypertension. Arterial blood gases showed lactic acidosis. Decision to initiate VA ECMO was made. Cannulation (37F) of the right femoral artery and left femoral vein was performed and a distal perfusion catheter was inserted into the right femoral artery. ECMO was initiated (flow 2.5 L), as well as a dobutamine infusion and inhaled nitric oxide.

Twenty-four hours later, the patient was weaned from vasopressors and ECMO. An urgent thrombectomy was needed to restore blood flow to the left leg as no pulse was felt distal to the popliteal artery. Four days after thrombectomy, the patient was extubated. One day later, she was discharged from the ICU for rehabilitation.

Discussion

Sclerotherapy has replaced surgery in the treatment of venous malformations, as it provides less recurrence and better aesthetic results [6,7]. Ethanol is the preferred agent due to its low cost and high efficiency [6,8] but may rarely lead to cardiovascular collapse [9]. A possible mechanism is pulmonary artery vasospasm, with increased pulmonary artery pressure and strain on the right ventricle. Left ventricular cardiac output is affected leading to cardiovascular collapse

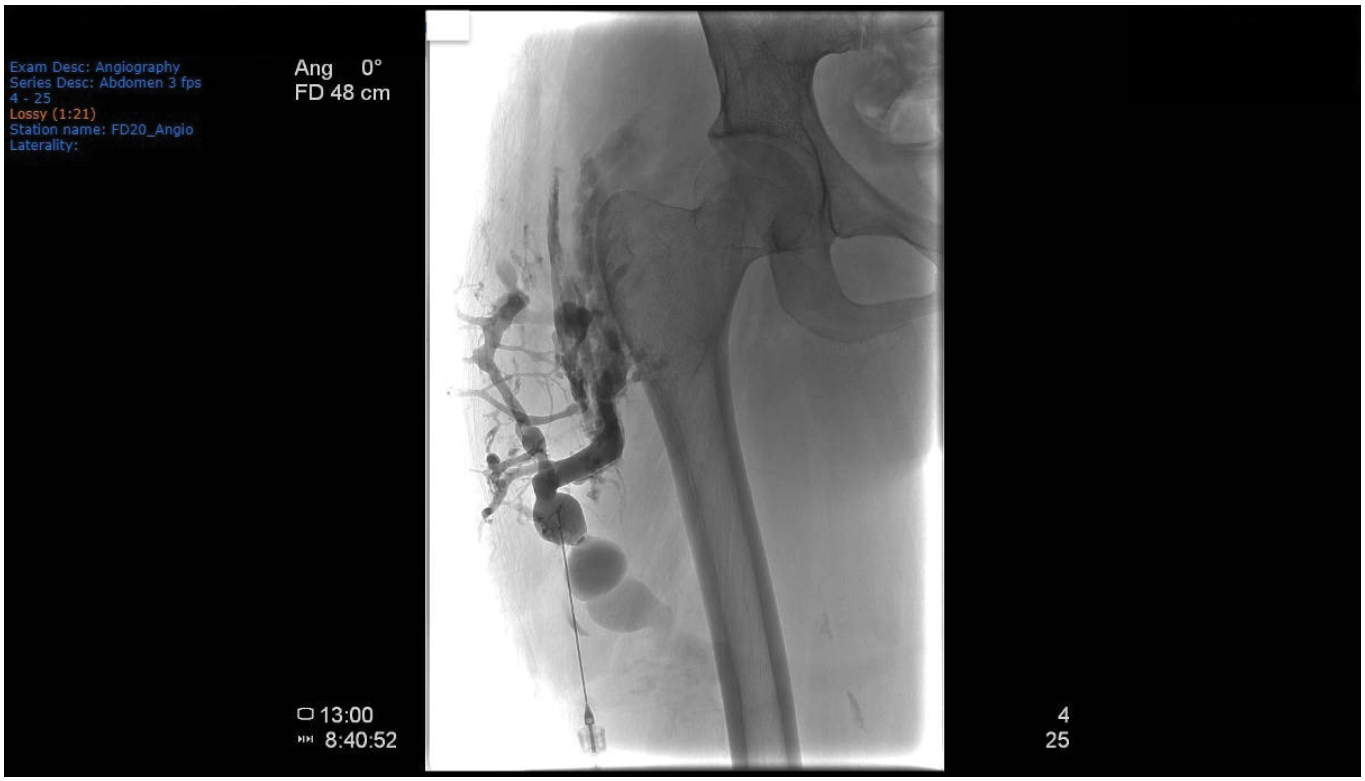

Figure 1: Angiography during sclerotherapy of venous malformation in gluteal area.

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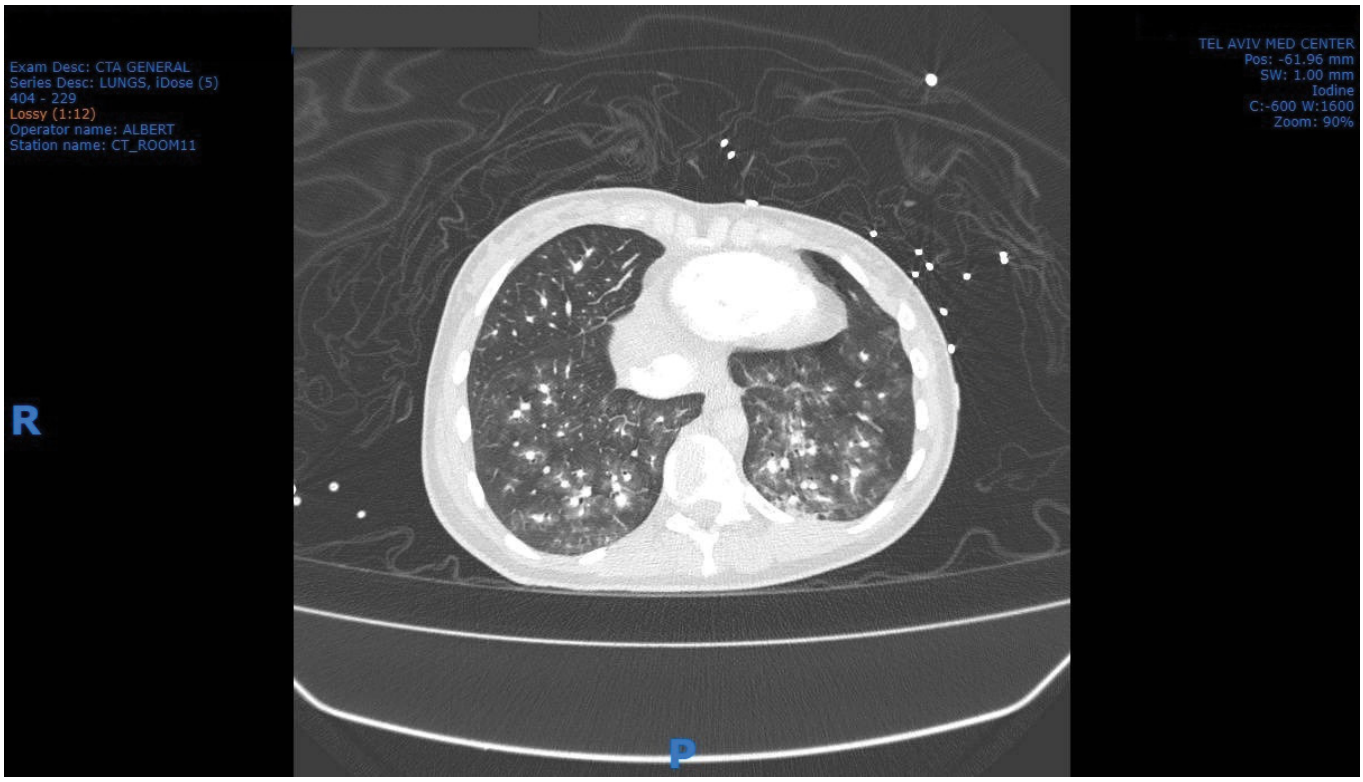

Figure 2: Computed Tomography angiography of chest exhibiting extreme pulmonary vasospasm.

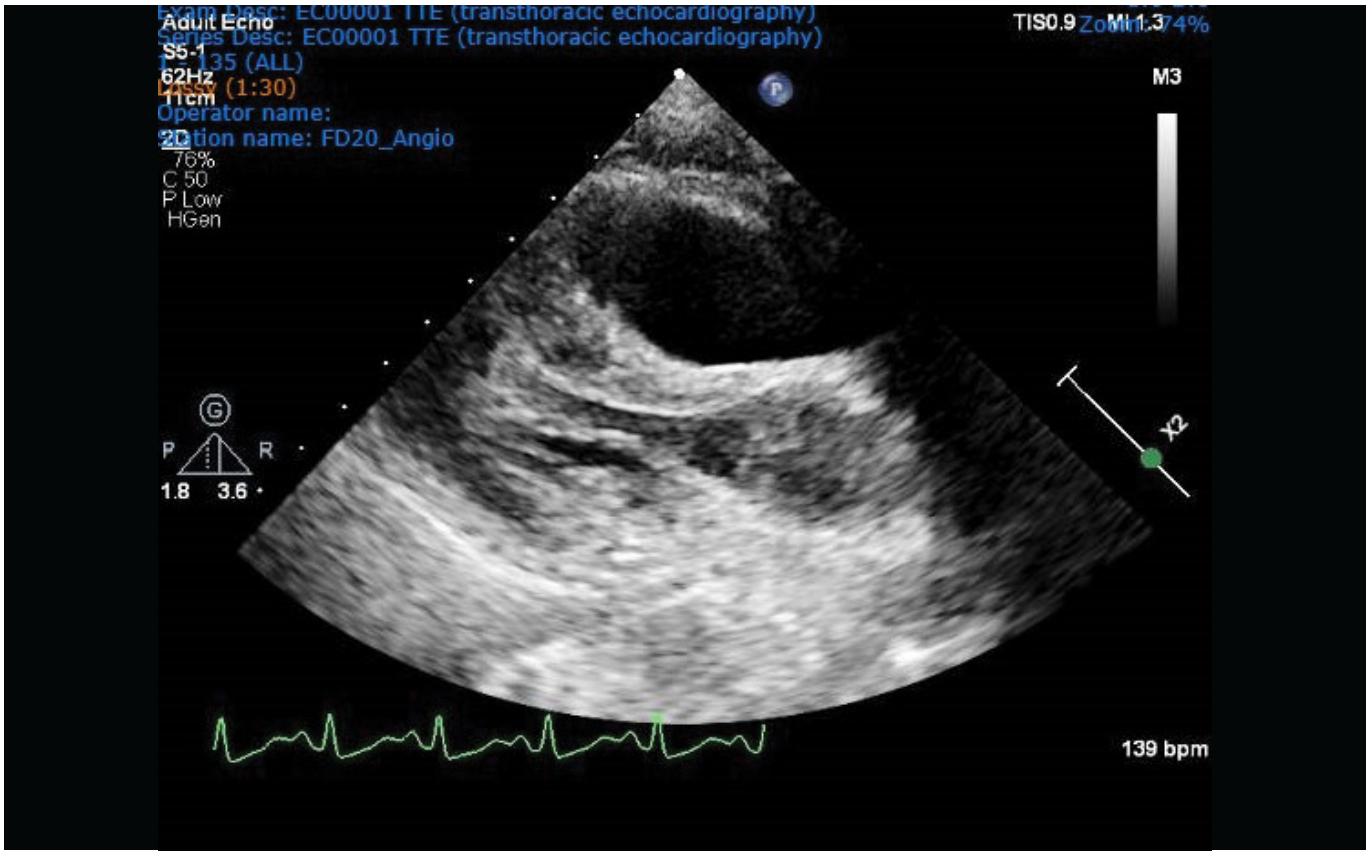

Figure 3: Transthoracic echocardiography showing right heart enlargement consistent with right heart failure.

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[10]. Ethanol may contaminate the systemic circulation (in correlation with volume injected)2 and cause direct damage to the endothelium and cardiac myocytes [11]. The upper volume limit is 1 ml/kg.1.

Few of the cases reported of cardiovascular collapse following sclerotherapy had positive outcomes. Our case is the first to describe VA ECMO for the management of this complication. We can assume that cardiovascular collapse was due to pulmonary vasospasm, as suggested by the echocardiographic findings and the dose of ethanol being within the accepted range. Early detection and management of the patient's hemodynamic instability lead to a successful outcome. The decision to initiate ECMO was fast (within two hours), leading to an early reduction in right ventricular strain.

Monitoring of pulmonary artery pressure (PAP) during ethanol sclerotherapy has been proposed to anticipate hemodynamic instability [9,12]. If PAP increases, nitroglycerin infusion is recommended [12]. We do not routinely measure PAP during sclerotherapy in our institution, but further research should determine whether PAP monitoring can improve morbidity and mortality.

Conclusion

In conclusion, awareness of this rare but catastrophic complication during sclerotherapy should be raised to ensure fast response and diagnosis. Furthermore, we believe that ECMO should be investigated as a potential tool to treat cardiovascular collapse following ethanol sclerotherapy.

Conflicts of interest

There are no conflicts of interest.

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