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CASE REPORT

INTERMEDIATE

CLINICAL CASE

Sudden Cardiac Arrest Due to Ascending Aortic Thrombus Originating From Penetrating Aortic Ulcer

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ABSTRACT

Penetrating aortic ulcers typically occur in severely diseased vessels. We present the case of a 46-year-old woman, without extensive atherosclerosis, who had sudden cardiac arrest related to ischemia from a mobile intraluminal aortic thrombus adherent to a penetrating ulcer in the ascending aorta. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2021;3:1617–1621) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 46-year-old woman was transported to the emergency department (ED) after sustaining a cardiac arrest. The patient's daughter had heard a fall, found the patient unresponsive, and immediately alerted emergency medical services, who arrived within 5 minutes. The initial rhythm was ventricular fibrillation (VF), and cardiopulmonary resuscitation was initiated. Two external defibrillations achieved return of spontaneous circulation.

LEARNING OBJECTIVES

- Generate a differential diagnosis for a patient with SCA to refine the initial diagnostic work-up and management after presentation to the hospital.
- Recognize the typical clinical presentation and epidemiology of PAUs to determine the need for additional investigation of uncommon causes in a patient presenting with atypical features.
- Apply the concepts of Virchow's triad for a patient presenting with an intraluminal aortic thrombus to improve diagnostic accuracy.

On arrival to the ED, the patient's blood pressure was 184/112 mm Hg, her heart rate was 132 beats/min, the Glasgow Coma Scale Score was 3, and oxygen saturation was 98% on 100% fraction of inspired oxygen through a supraglottic airway. An endotracheal tube was placed, and temperature management was initiated to target 33 °C. The cardiopulmonary examination was unremarkable, and the electrocardiogram demonstrated sinus tachycardia without ST-segment elevation and no features of pre-excitation, Brugada syndrome, or long QT syndrome. Laboratory studies revealed an undetectable troponin level on admission that peaked at 0.13 ng/mL at 2 hours and then trended to 0.08 ng/mL at 12 hours (normal, <0.04 ng/mL). The B-type natriuretic peptide level was <10 pg/mL. Chemistry studies revealed the following: potassium,

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ABBREVIATIONS AND ACRONYMS

CMR = cardiac magnetic resonance

CTA = computed tomographic angiography

ED = emergency department

PAU = penetrating aortic ulcer

RCA = right coronary artery

SCA = sudden cardiac arrest

TEE = transesophageal echocardiogram

VF = ventricular fibrillation

3.6 mmol/L; magnesium, 1.6 mg/dL; creatinine, 0.6 mg/dL; and glucose, 371 mg/dL.

PAST MEDICAL HISTORY

The patient's history included type 2 diabetes mellitus, hypertension, and hyperlipidemia. Being out of regular medical care, the patient was taking no medications or supplements. She never used tobacco or illicit substances and rarely used alcohol.

DIFFERENTIAL DIAGNOSIS

The patient presented with sudden cardiac arrest (SCA), defined as cessation of cardiac activity that causes unresponsiveness without a pulse (1). The categorical differential diagnosis of SCA with VF includes the following: acute ischemic events; structural heart disease; arrhythmogenic syndromes without structural heart disease; and noncardiac causes such as drugs, toxins, trauma, and metabolic derangements.

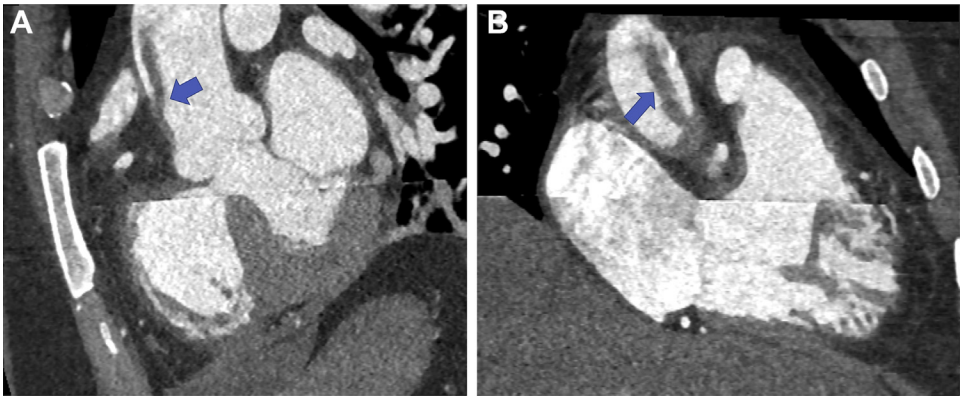
INVESTIGATIONS

The transthoracic echocardiogram revealed normal biventricular function and no structural abnormalities (Videos 1 and 2). After rewarming, full neurologic recovery, and extubation on hospital day 2, the patient reported no symptoms and could not recall the events leading up to her SCA. Coronary computed tomographic angiography (CTA) demonstrated normal coronary arteries without atherosclerosis and a coronary artery calcium score of zero. However, a 1.5-cm hypodense linear structure was seen adherent to the aorta, superior to the right coronary artery (RCA) cusp with distal mobility and extension into the coronary ostium (**Figures 1A and 1B**, Videos 3 and 4). Cardiac magnetic resonance (CMR) with thoracic angiography confirmed the intra-aortic findings and further revealed a focal area of subendocardial late gadolinium enhancement in the basal inferior wall (**Figures 2A and 2B**). Additionally, deep venous thrombi were identified along the course of the previously in situ femoral cooling catheter. Transesophageal echocardiogram (TEE) was performed and confirmed the diagnosis of mobile ascending aortic thrombus (Video 5). CTA, CMR, and TEE did not reveal any other areas of atherosclerosis or other disease in the aortic arch or descending aorta. Biomarker diagnostic values for hypercoagulable, autoimmune, infectious, and malignant states were unremarkable.

MANAGEMENT

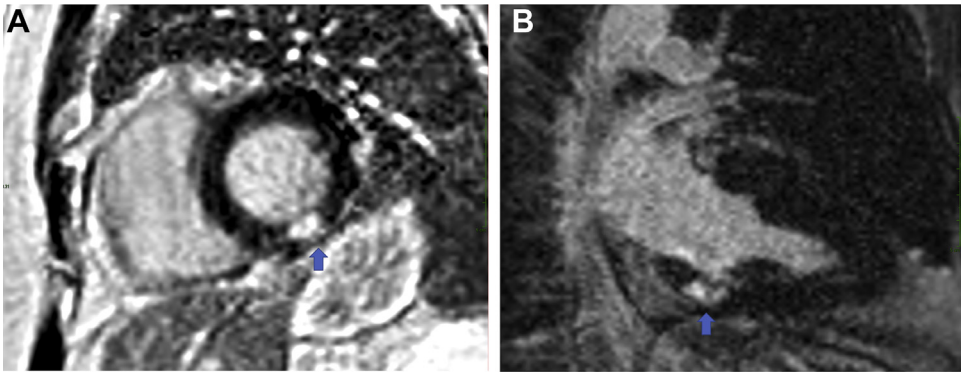
Initial therapy included unfractionated heparin administered by continuous infusion. Following extensive discussion with the patient and multidisciplinary team, she underwent surgical removal of the aortic thrombus given the otherwise ongoing risk for catastrophic embolization. Intraoperative examination revealed an aorta that was normal in size and appearance with the exception of a large thrombus, located above the RCA ostium and measuring 3.5 cm in length, adherent to a small penetrating aortic ulcer (PAU) (**Figure 3**). The PAU and the thrombus (**Figures 4A and 4B**) were resected, and the aorta was repaired using a bovine pericardial patch.

FIGURE 1 Coronary CTA With Ascending Aortic Thrombus



(A and B) Orthogonal long-axis views of the aortic root demonstrate thrombus **(arrows)** with proximal attachment above the right coronary ostium and unattached distal segment. CTA = computed tomography angiography.

FIGURE 2 Basal Inferior LGE on CMR



(A) Basal short-axis and **(B)** 2-chamber phase-sensitive inversion recovery late gadolinium enhancement (LGE) images with focal basal inferior subendocardial LGE **(arrows)**. CMR = cardiac magnetic resonance.

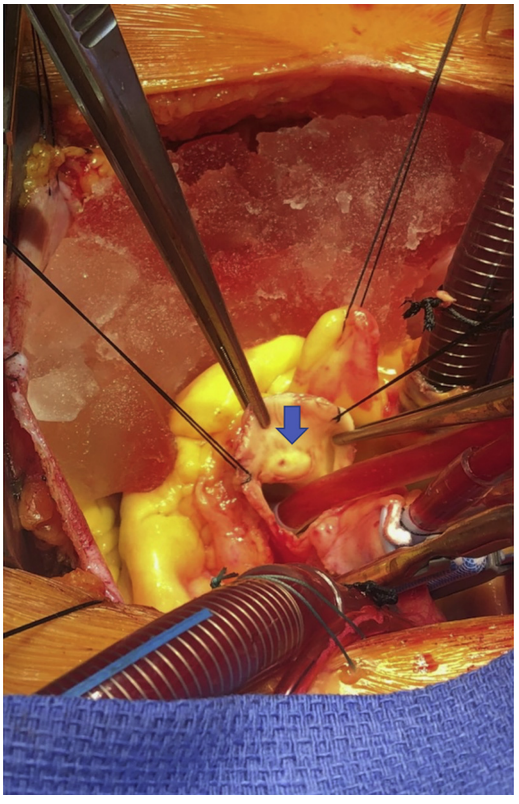
Histopathologic examination revealed extensive mucoid medial degeneration with atherosclerotic plaque surrounding the aortic ulcer and confirmation of the associated thrombus.

DISCUSSION

Defined as a deep atherosclerotic lesion wherein focal ulceration of elastic lamina extends to the medial layer of the aortic wall, PAU is often associated with hematoma formation (2,3). PAU is most frequently found in the descending thoracic aorta (60%-95%), corresponding to common locations of atherosclerotic deposition, and it is rarely described in the ascending aorta (4,5). Less commonly, PAU can also have infective, inflammatory, traumatic, or iatrogenic causes (2). Compared with our patient, individuals found to have PAU are typically older (>65 years old) with extensive atherosclerosis. Notably, PAU accounts for up to 8% of acute aortic syndromes and may manifest with a spectrum of symptoms ranging from an incidental finding on cardiothoracic imaging to severe chest or back pain (2). Although it is considered a distinct subtype of aortic disease, PAU can progress to aortic dissection and rupture (6).

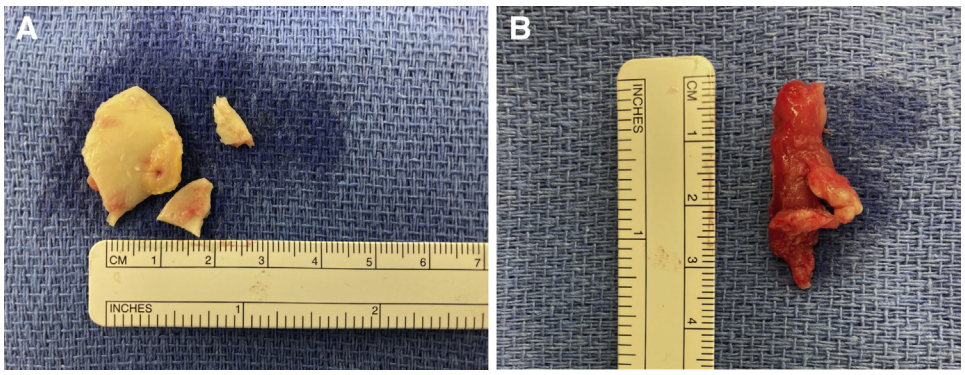
To our knowledge, intraluminal thrombus as a complication of PAU has not been well described. Thrombus formation in the aortic lumen is rare because of the high-flow, high-pressure arterial location (7). Intraluminal thrombi typically develop in the setting of venous stasis, a hypercoagulable state from autoimmune or malignant disease, and endothelial

FIGURE 3 Intraoperative Findings



Proximal ascending aorta with a small penetrating aortic ulcer **(arrow)** located 2 to 3 mm above the right coronary orifice with surrounding plaque after thrombus removal.

FIGURE 4 Gross Specimens



(A) Penetrating aortic ulcer, 1 mm × 1 mm, and **(B)** intra-aortic thrombus, 3.5 cm in length.

damage, known as Virchow's triad. In our patient's case, an underlying yet unspecified hypercoagulable state that was manifest by the finding of additional venous thromboses may have contributed to the atypical presentation. There was no evidence to suggest other potentially contributing disorders such as systemic diseases affecting the aorta (eg, autoimmune or infectious). In an alternate scenario involving multiple thrombi secondary to systemic disease, long-term anticoagulation would be appropriate.

Our patient's presentation of SCA may be attributed to ventricular tachycardia with degeneration into VF as a result of subendocardial scar, as seen on CMR, from a previous subclinical type II myocardial infarction caused by embolization from the aortic thrombus down the RCA to a small vessel. The acute arrhythmia may have been triggered by mechanical obstruction of the RCA ostium by the thrombus contributing to only transient ischemia, which may explain the minimal troponin elevation.

FOLLOW-UP

The patient recovered from surgery uneventfully. After a shared decision-making discussion, the patient underwent implantable cardioverter-defibrillator placement before discharge for secondary prevention given the scar on CMR and the uncertain associated risk. The patient was discharged on indefinite warfarin anticoagulation while awaiting completion of further work-up for malignant disease.

CONCLUSIONS

We present an atypical case of SCA attributed to ischemia from arterial thrombus formed in association with a PAU. This case highlights the importance of a broad differential diagnosis of SCA along with comprehensive evaluation and offers the opportunity to review the definition of PAU in addition to its cause, associated complications, and management.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS aorta, cardiovascular disease, imaging, myocardial ischemia, secondary prevention, thrombus, vascular disease, ventricular fibrillation

APPENDIX For supplemental videos, please see the online version of this paper.