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CRITICAL CASE AND RESUSCITATION

CASE REPORT: CLINICAL CASE

An Unusual Cause of Shock

Bursting a Bubble

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ABSTRACT

This study presents the case of a previously healthy 68-year-old woman who presented with shock from tamponade due to hemopericardium. Initial noninvasive imaging did not provide a clear etiology for the hemopericardium. Given the ongoing clinical deterioration and need for diagnosis and treatment, an exploratory sternotomy was performed with successful outcome. (JACC Case Rep 2024;29:102394) © 2024 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 68-year-old woman presented to the emergency department with near-syncope and noncardiac chest pain. At the initial time of assessment, she denied any dyspnea, palpitations, or drug use. The medical history could not be completed because the patient became obtunded. She was afebrile with a blood pressure of 100/60 mm Hg, heart rate of 74 beats/min, and oxygen saturation of 99% on room air. After initial assessment, the patient had worsening hemodynamics requiring intubation and initiation of vasopressors. An echocardiogram was performed which revealed pericardial effusion with tamponade physiology.

LEARNING OBJECTIVES

- To recognize the indication for surgical exploration in patients with tamponade from hemopericardium who are otherwise healthy to avoid further hemodynamic compromise.
- To understand the role of further diagnostic imaging in otherwise healthy patients with hemopericardium to elucidate the underlying etiology.

PAST MEDICAL HISTORY

The patient's past medical history was only notable for asthma. She denied smoking and consumption of alcohol or drugs.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for tamponade included pericarditis of all etiologies, myocardial infarction with wall rupture, aortic dissection, trauma, and malignant pericardial invasion.

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ABBREVIATIONS AND ACRONYMS

CAA = coronary artery aneurysm

CI = cardiac index

CTA = computed tomography angiogram

INVESTIGATIONS

Her laboratory investigations revealed neutrophilic leukocytosis of 17.4 × 10^9/L with otherwise normal blood counts. She had evidence of metabolic acidosis from elevated lactate (bicarbonate of 18 mmol/L, pH 7.18 with lactate of 9 mmol/L). She had end-organ hypoperfusion with creatinine of 92 μmol/L (baseline, 57 μmol/L) and alanine transaminase of 206 U/L. High-sensitivity troponin-T was 37 ng/L (reference range, 0-9) with a repeat 3 hours later at 58 ng/L. Her D-dimer was elevated at more than 4,000 mg/L fibrinogen equivalent units. Coagulation function, thyroid stimulating hormone, electrolytes, and albumin levels were within normal range.

An electrocardiogram revealed sinus rhythm with nonsignificant Q waves in the inferolateral leads (**Figure 1**). She underwent routine computed tomography angiogram (CTA) with dissection protocol, which revealed pericardial fluid with attenuation values of more than 40 Hounsfield units, highly suggestive of hemopericardium of visually moderate size (**Figure 2**). There was no conclusive evidence of aortic dissection or aortic intramural hematoma.

Due to the uncertainty of the etiology of tamponade, ongoing hemodynamic instability, and hemopericardium, the patient was taken to the operating room for exploration. Transesophageal echocardiogram revealed diastolic right atrial collapse (**Figure 3**, Video 1). Epiaortic ultrasound of the ascending aorta did not reveal evidence of dissection or intramural hematoma. Further assessment of the inferior vena cava revealed that it was dilated with no respiratory variation, consistent with high right-sided pressure (Video 2). Her initial hemodynamic assessment while on intravenous infusion of norepinephrine at 20 μg/min and epinephrine 10 μg/min revealed a mean arterial pressure of 54 mm Hg, cardiac index of 0.94 L/min/m^2, and central venous pressure of 26 mm Hg. On sternotomy and removal of clots within the pericardium (**Figure 4**, Video 3), her blood pressure increased to a systolic of 180 mm Hg, with a cardiac index of 2.6 L/min/m^2 and central venous pressure of 7 mm Hg. The heart was lifted to visualize the posterolateral aspects of the left ventricle. On assessment of first obtuse marginal coronary artery, an aneurysm measuring 15 mm in diameter with a small rupture was found (**Figure 5**). The remainder of the coronary arteries did not reveal any abnormalities.

FIGURE 1 Electrocardiogram Highlighting Nonspecific Q Waves in Inferolateral Leads

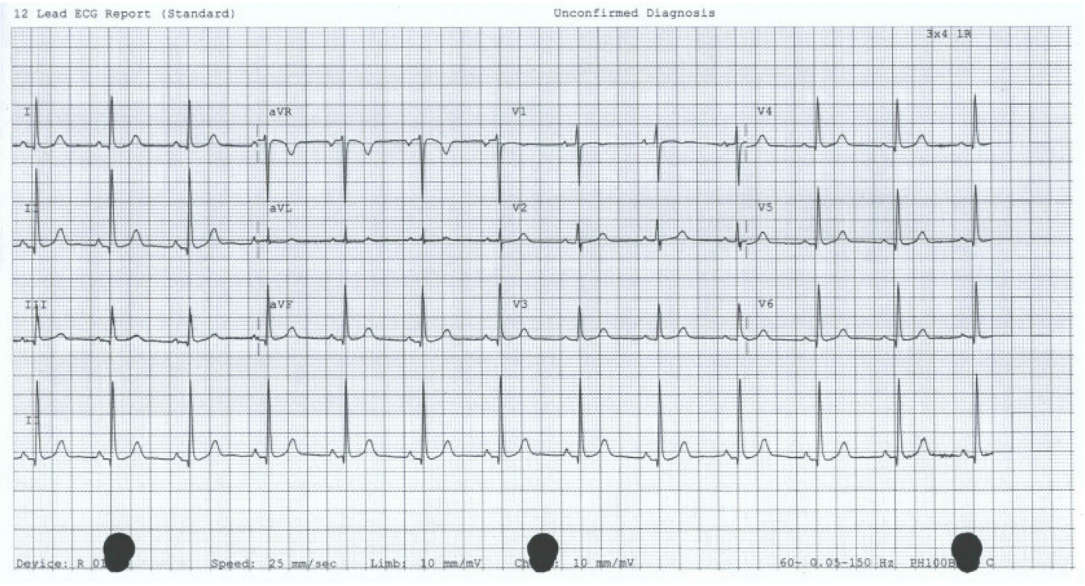
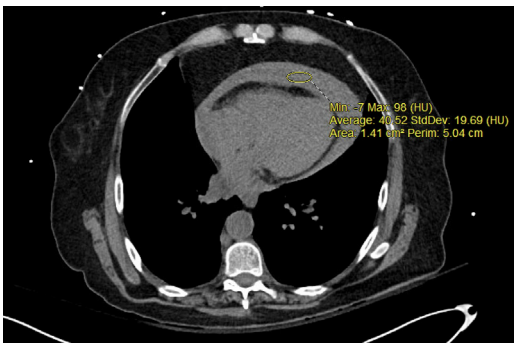
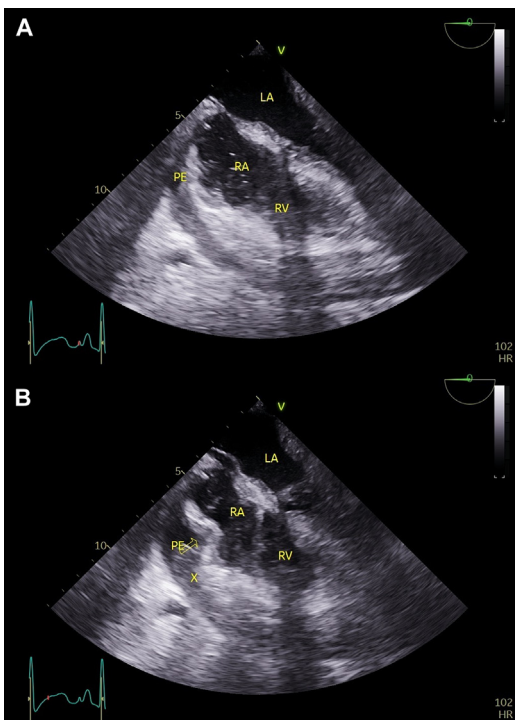


FIGURE 2 Axial Computed Tomography Angiogram



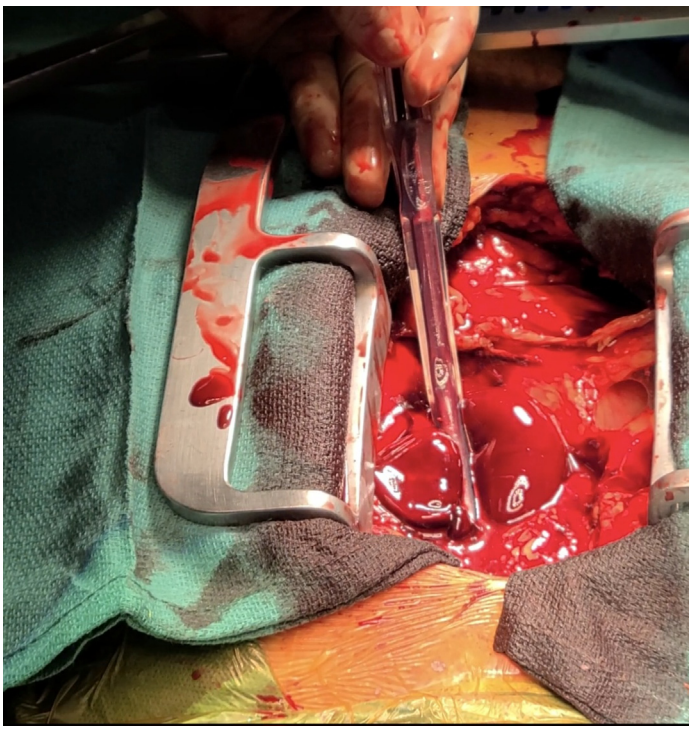
Axial computed tomography angiogram with moderate pericardial effusion of more than 40 HU, suggestive of hemopericardium.

FIGURE 3 Transthoracic Echocardiogram Revealing Tamponade Physiology



(A) Visualization of ventricular diastole with no evidence of impairment. (B) Right atrial collapse is noted during ventricular systole. LA = left atrium; PE = pericardial effusion; RA = right atrium; RV = right ventricle.

FIGURE 4 Pericardiectomy Revealing Hemopericardium With Blood Clots



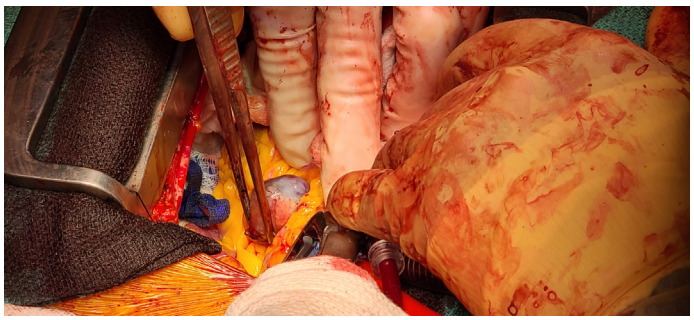
MANAGEMENT

The patient underwent marsupialization of the coronary artery aneurysm (CAA) and reconstruction with vein patch (Video 4). She was transferred to the intensive care unit for further monitoring. The remainder of her clinical course was unremarkable. She was discharged 10 days later from the hospital. At her 1-month follow-up, the patient reported no chest pain or dyspnea. The patient's condition was deemed idiopathic, and she underwent no further investigation given that there were no other aneurysms identified and given the absence of risk factors for alternative etiologies.

DISCUSSION

CAAs have been described as a rare pathology found to be present in about 1% of postmortem examinations.[1] The pathogenesis of this entity is thought to be predominantly inflammatory in children secondary to vasculitides. In adults, CAA is often due to atherosclerosis. Occasionally, other processes may be involved in their development (eg, drug-eluting stents, use of amphetamines or cocaine, vasculitides, infections, connective tissue diseases).[2,3] The

FIGURE 5 Evaluation of the First Obtuse Marginal Artery Revealing an Aneurysm

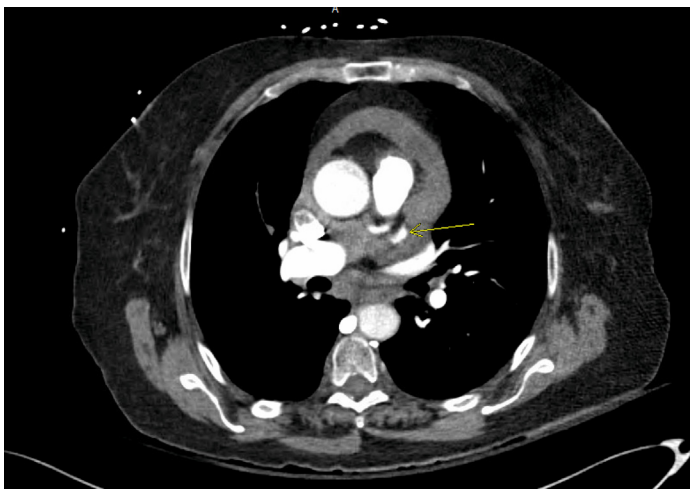


The edge of the forceps delineates a small rupture.

presentation of CAAs is variable depending on the resulting complication. CAAs are typically asymptomatic but may present as sudden cardiac death.

The gold standard diagnostic modality for detecting CAAs remains conventional angiography. With increasing use of coronary CTA, incidental CAAs have been more frequently identified. Electrocardiographic gating allows elimination of cardiac-induced motion artifacts and is commonly used in patients with already known CAA, either for characterization or follow-up. The advantage of coronary CTA over conventional angiography is the ability of CTA to assess lumen size, aneurysmal wall, and presence of thrombus. Magnetic resonance angiography also allows detection of CAA, although less often used in urgent scenarios. In our case, retrospective assessment of the CTA obtained to rule out aortic dissection revealed a 14-mm contrast-filled outpouching arising from the first obtuse marginal coronary artery (Figure 6). This case highlights that CAA may be sometimes visible on computed tomography, even when cardiac gating is not used.

FIGURE 6 Computed Tomography Angiogram



Computed tomography angiogram reveals a 14-mm contrast-filled outpouching (highlighted at the tip of the arrow) arising from the first obtuse marginal coronary artery, compatible with an aneurysm. Active extravasation of the intravenous contrast was not present. The study was performed without cardiac gating.

The patient's presenting symptom of shock precluded the possibility of investigating rarer etiologies of hemopericardium with further imaging. Although diagnosis of CAAs via echocardiography has been described in the pediatric population, its role in adults is limited.[4,5] Given the deteriorating clinical status of the patient from hemopericardium with no clear etiology on preliminary imaging, the decision was made to proceed with exploratory surgery.

Studies evaluating treatment approaches to CAA are limited. A recent literature review with proposed treatment algorithm recommends an individualized strategy.[2] In the case of symptomatic CAA or hemodynamically unstable disease, an early interventional approach is recommended. Although percutaneous coronary intervention for stable CAA has been described, its role appears to be limited. Surgical interventions such as ligation or marsupialization with interposition graft have been performed effectively.[6]

CONCLUSIONS

Understanding the role of noninvasive imaging in deteriorating patients from undifferentiated hemopericardium is necessary to help guide management. This case highlights variables that prompted surgical exploration (eg, undetermined etiology of hemopericardium). Nevertheless, retrospective analysis of the imaging suggests that CAA can be seen on noncardiac-gated CTA. Finally, guideline recommendations for the management of undifferentiated hemopericardium should focus on factors that prompt surgical exploration and include cardiac-gated CTA to assess for coronary anomalies.

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KEY WORDS coronary artery aneurysm, hemopericardium, tamponade

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