

Hemorrhagic pericardial effusion after removal of pericardial drain in penetrating atherosclerotic ulcer of the ascending aorta with pseudoaneurysm

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Introduction

A penetrating atherosclerotic ulcer (PAU) is a type of ulcerative lesion that breaches the internal elastic lamina, allowing for the formation of a hematoma within the medial wall of the aorta. PAUs typically occur in advanced atherosclerotic lesions and are more commonly observed in elderly individuals with a higher cardiovascular risk. The clinical course of PAUs can vary and may involve complications such as intramural hematomas, dissection, and pseudoaneurysms (PSA). If the ulcerative lesion extends through the adventitia, it can lead to a life-threatening bleeding, necessitating the careful management of PAUs. Here, we report the case of a 66-year-old patient with a known history of non-Hodgkin's lymphoma who presented to the hospital with shortness of breath, peripheral edema, vertigo, and low blood pressure persistent for the previous three weeks. During hospitalization, investigations revealed the presence of pericardial effusion. On the same day, the patient experienced hemodynamic instability with cardiac tamponade, requiring pericardiocentesis. Four

days after drain removal, there was a recurrence of cardiac tamponade, which was successfully managed with surgical intervention. Unfortunately, the patient passed away a few days later due to cardiac arrest. During autopsy, a then a clinically asymptomatic PSA was discovered in the ascending aorta, resulting from a PAU. The rupture of PSA was responsible for the fatal bleeding into the pericardial space during the drain removal procedure.

Patient presentation

A 66-year-old man (180 cm, 85 kg) with a history of Non-Hodgkin's lymphoma (25 years ago) and arterial hypertension was referred to our emergency department following three weeks of dyspnea, peripheral edema, vertigo, and hypotension (day 1). On initial examination, he was conscious (GCS 15), with bilateral basal inspiratory crackles on auscultation and pitting edema of the lower legs. Blood pressure was 95/55 mmHg, body temperature was 37.8C, heart rate was 84 beats per minute,

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respiratory rate was 18 breaths per minute, and oxygen saturation was 96% with oxygen therapy via a mask at 3 liters per minute.

Initial work up

Laboratory testing revealed leukocytosis of $22.1 \times 10^9/l$ (normal range: $4.5 - 11 \times 10^9$), severe grade anemia (hemoglobin 78 g/l, normal range: 130 – 160 g/l), and mild thrombocytosis of $450 \times 10^9/l$ (normal range: $150-400 \times 10^9/l$). Iron levels indicated anemia of chronic inflammatory nature. Other laboratory abnormalities included elevated levels of brain natriuretic peptide type B (NT-proBNP) at 14328 ng/l (normal range: 0-125), C-reactive protein at 199.3 mg/l (normal range: 0-10), and procalcitonin at 1.9 ug/l (normal range: below 0.046). Two-dimensional transthoracic echocardiography (TTE) (Siemens SC 2000 Prime, Siemens Medical Solutions, Inc., Mountain View, California, USA) performed on admission day showed partially organized circumferential pericardial effusion with maximum separation of pericardial layers up to 3 cm, diastolic collapse of the right ventricle, and a transmitral respiratory variation of Doppler flow of 25-30%, indicative of cardiac tamponade (Figure 1). The patient was immediately

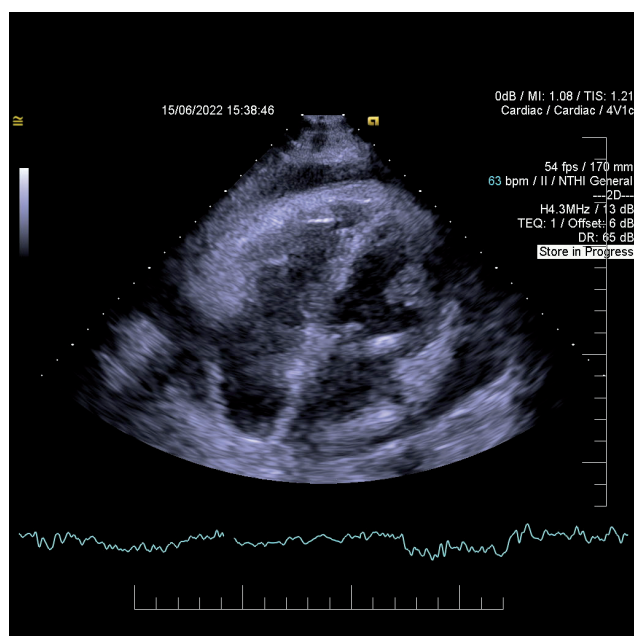


Figure 1 Circular pericardial effusion

transferred to our catheterization laboratory, where an emergency needle pericardiocentesis was performed, evacuating 650 ml of sanguineous fluid, followed by insertion of a chest drain and transfer to the intensive care unit. Due to gradual improvement, drain extraction was indicated after four days (day 5).

Diagnosis and management

However, approximately 30 minutes later, there was a sudden onset of hypotension, tachycardia, cold extremities, and elevated jugular venous pressure. Bedside TTE revealed recurrence of tamponade. Considering the overall condition of the patient, a decision was made to perform pericardial fenestration. Early in the procedure, the hemodynamic status deteriorated to the extent that brief cardiac massage was required. Medial fenestration and opening of the pericardial sac above the right ventricle proceeded, during which accumulated clots and freshly suctioned bright red blood were removed. The source of bleeding was identified to the right of the ascending aorta, within a suspicious tumorlike lesion.

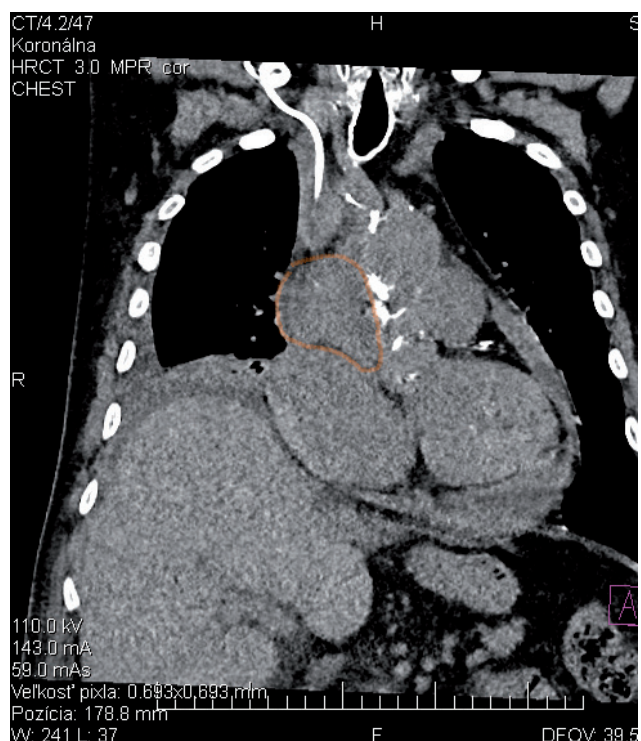


Figure 2 CT scan – suspected pericardial tumorous mass



Figure 3 Anterior surface of the heart with organizing hemorrhagic effusion and large mediastinal vessels

A total of 2000 ml of hemorrhagic fluid was evacuated, and two sutures were applied for bleeding control. Attempts to resect the tumor were unsuccessful due to evident infiltration of both the ascending aorta and the hilum of the right lung.

Additionally, further tumorlike changes were observed on the anterior and diaphragmatic surfaces of the pericardium. In total, four histological samples were taken. Additional CT examination confirmed the previous finding, at that time already a residual circumferential pericardial effusion, and particularly a suspicious heterogeneous pericardial tumor mass above the right atrium with cranial extension towards the aortic arch (**Figure 2**). During the postoperative period, the patient's condition rapidly improved, including laboratory results and clinical presentation. Three days later (day 9), he was transferred to a standard ward. However, the following day (day 10), he suddenly developed altered

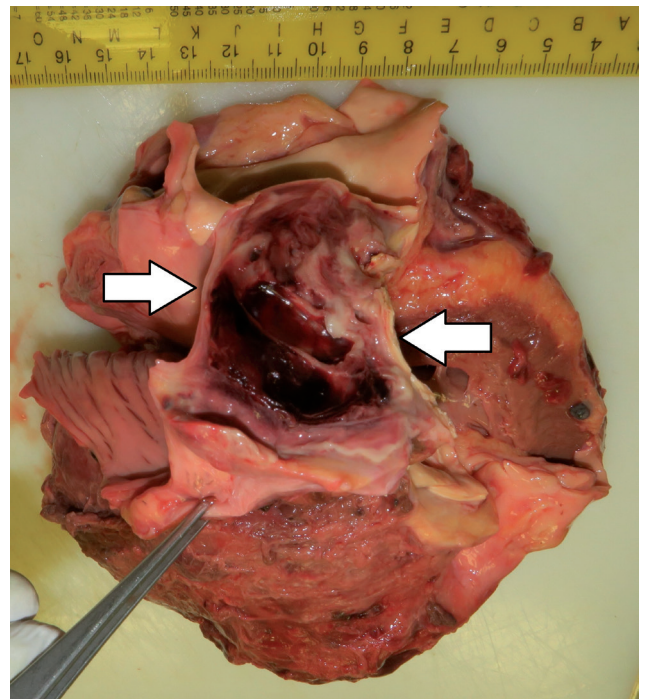


Figure 4 Base of the heart with a pseudoaneurysm between the superior vena cava (arrow pointing to the right) and the aorta (arrow pointing to the left)

consciousness accompanied by hypotension, ultimately leading to cardiac arrest. Despite immediate initiation of cardiopulmonary resuscitation lasting a total of 45 minutes, spontaneous circulation could not be restored. Death was pronounced, and a post-mortem examination was recommended by the attending physician.

Follow-up

The post-mortem examination five hours after death revealed significant findings related to cardiac surgery. Notably, 855 ml of clear fluid accumulated in the right pleural cavity, alongside 300 ml in the left. Bilateral lung edema was observed, with the left lung weighing 1199 g and the right lung weighing 1000 g, exceeding the reference range. Within the thickened pericardial sac, 80 ml of liquid blood and 100 grams of blood clots were firmly adhered to the pericardial layers (**Figure 3**). During the dissection of mediastinal structures, an irregularly shaped pseudoaneurysm (measuring 4.5 x 7 x 4-8 cm) was found, compressing the adjacent

superior vena cava. This pseudoaneurysm, delineated by fibrous bands, communicated through a 0.4-0.9 cm fissure, originating from a penetrating aortic ulcer on the right lateral wall of the ascending aorta, 2.5 cm above the aortic valve. Multiple pseudolumens were observed within the pseudoaneurysm, extending into the pericardial cavity where two stitches were present (Figure 4). Additionally, the heart exhibited severe atherosclerosis, while the liver, spleen, and kidneys were enlarged. Histological examination revealed organizing hemorrhage with fibrin reaction and significant fibrosis of the epicardium. Despite severe sclerotic changes in the ascending aorta, there were no inflammatory or degenerative alterations. In the lungs, intra-alveolar edema was noted, while the liver showed acute congestion with hepatocyte necrosis and a leukocytic reaction. The immediate cause of death was determined to be intra-alveolar lung edema due to cardiac failure secondary to hypertension and ischemic damage. Massive bleeding into the pericardial sac, following the removal of the drain for hydropericardium evacuation, originated from a ruptured penetrating aortic ulcer of the ascending aorta with a pseudoaneurysm, which leaked into the pericardial sac. Fortunately, the residual hemopericardium in the organization stage did not significantly impact cardiac function.

Conclusions

The presented case highlights the interplay between aortic conditions and pericardiocentesis complications.

The 66-year-old man initially underwent pericardiocentesis because of hydropericardium, where, even after successful procedure, removal of the pericardial drain resulted in cardiac tamponade. Although urgent surgical intervention averted immediate fatality from bleeding or tamponade, the patient passed away 5 days later. The post-mortem examination revealed that cardiac tamponade originated from a ruptured penetrating aortic ulcer of the ascending aorta with a pseudoaneurysm, which leaked into the pericardial sac. Therefore, it is necessary to always consider that even seemingly successful procedures may bring unexpected complications, including decease.

However, we would like to present that some pathologies cannot be predicted in advance, as was the case in this instance.

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