A case report of hemorrhagic cardiac tamponade with rapid blood clot formation
A serious complication of acute type a aortic dissection

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Abstract

Rationale: Acute type A aortic dissection (AAAD) remains a life-threatening disease. We previously reported a case with ultrasound findings of a homogeneous hemopericardium and evidence highly indicative of hemorrhagic cardiac tamponade complicated by AAAD. Here, we report a similar case who presented with a more serious situation and for whom critical care ultrasound revealed fast blood clot formation within the hemopericardium.

Presenting concerns: A 63-year-old man was admitted to our emergency department with a complaint of a tearing chest pain for 10 minutes. Asymmetric blood pressure was detected in the upper limbs and AAAD was highly suspected. An electrocardiogram (ECG) monitor was placed in a timely manner. However, during this procedure, he went into cardiac arrest and cardiopulmonary resuscitation (CPR) was initiated.

Diagnoses: Critical care ultrasound revealed hemorrhagic cardiac tamponade with blood clot formation surrounding the epicardium, strongly indicating the rupture of an ascending aortic root dissection.

Interventions: Standard CPR continued for 30 minutes.

Outcomes: Spontaneous cardiac rhythm was not restored and the patient died.

Lessons: Critical care ultrasound is a useful tool for assessing emergency cardiac arrest. Ultrasound findings of fast clot formation within the hemopericardium may indicate faster bleeding due to the rupture of an AAAD and may predict poor clinical outcomes.

Abbreviations: AAAD = acute type A aortic dissection, CPR = cardiopulmonary resuscitation, ECG = electrocardiogram, ECMO = extracorporeal membrane oxygenation, IRAD = International Registry of Acute Aortic Dissection, LV = left ventricle, RV = right ventricle.

Keywords: acute type a aortic dissection, blood clot formation, critical care ultrasound, hemorrhagic cardiac tamponade

1. Introduction

Acute aortic dissection (AAD) is the leading cause of death in patients with cardiovascular disease.[1] Furthermore, of all AAD cases documented in the International Registry of Acute Aortic Dissection (IRAD), 67% presented with acute type A aortic dissection (AAAD).[2] When left untreated, or surgery is delayed, 18% to 20% of AAAD cases may develop into hemorrhagic cardiac tamponade,[3] at which point it is associated with a two-fold higher rate of mortality.[4] Due to the rapid progression of the disease, a large amount of blood from the aortic roots rushing into the pericardium causes acute pericardial tamponade, leading to sudden death. Therefore, it remains a challenge to diagnose and treat.

Recently, we reported a life-threatening case of hemorrhagic cardiac tamponade complicated by AAAD with clear evidence from critical care ultrasound.[5] In that case, the content of the hemopericardium was homogeneous. Herein, we describe another case of sudden death due to AAAD. Interestingly, in addition to a hemopericardium, ultrasound findings also indicated that a layer of annular blood clots formed rapidly around the epicardium.

This study adhered to the tenets of the Declaration of Helsinki, and the ethics committee of the First Affiliated Hospital of Chongqing Medical University approved the study. Informed consent was obtained from the patient for publication of this report and its related images.

2. Case report

A 63-year-old man was admitted to our emergency department with a complaint of a tearing chest pain for 10 minutes. He described acute-onset pain at rest, and denied any recent chest
trauma. The pain was described as persistent and not relieved by resting or treatment with nitrates. At presentation, he was conscious and sweating on the forehead. We quickly documented the patient’s medical history, which revealed that he had a >30-year history of hypertension and diabetes. His blood glucose levels and blood pressure had not been well controlled for years. At presentation, his blood glucose level was 19.9 mmol/L, and our manual sphygmomanometer failed to measure blood pressure. Considering these data and the high risk of unexplained chest pain, he was immediately transferred to the emergency room.

The electronic blood pressure monitor indicated a pressure of 181/46 mmHg for his left upper limb and 200/55 mmHg for his right upper limb. Therefore, AAAD was highly suspected. The nurse prepared to connect an electrocardiogram monitor (ECG). However, the patient was too fatigued and he lay down on the rescue bed.

At this moment, he suddenly lost consciousness and carotid artery beats were not detected. Therefore, standard cardiopulmonary resuscitation (CPR) with high-quality chest compression was initiated, followed by endotracheal intubation and deep vein catheterization. Simultaneously, critical care ultrasound was introduced for fast assessment of cardiac arrest.

Vascular ultrasound of the lower limbs did not detect a thrombus and physical examination revealed bilateral symmetrical breath sounds. Therefore, pulmonary embolism and tension pneumothorax were initially excluded.

The echocardiography provided an important clue, revealing a hemopericardium and a homogeneous layer of blood clots around the epicardium (Fig. 1). Further abdominal ultrasounds did not reveal an intimal flail in the lumen of the abdominal aorta. Hence, based on the patient’s hypertension, asymmetrical upper limb blood pressure, tearing chest pain, and hemopericardium, the cardiac arrest was highly suspected to be hemorrhagic cardiac tamponade complicated by the rupture of an AAAD. Unfortunately, after 30 minutes of resuscitation, a spontaneous cardiac rhythm was not restored and the patient died.

### 3. Discussion

Hemorrhagic cardiac tamponade is found in 8% to 10% of patients with AAAD and is associated with poor clinical outcomes. A population-based longitudinal study indicated that it is the leading cause of mortality in patients with AAAD. Hence, early recognition and urgent operative therapy are crucial to reduce the incidence of early mortality. In a previous case treated at our hospital, critical care ultrasound detected hemorrhagic cardiac tamponade secondary to AAAD in a timely manner. The ultrasound findings demonstrated a homogeneouse and echogenic hemopericardium without clot formation. However, in the present case, an annular layer of clots formed quickly around the epicardium. Because the blood coagulation rate is affected by shear forces and frictional effects, rapid blood clot formation may indicate faster bleeding or tearing due to the rupture of an AAAD. Meanwhile, the clots themselves can compress the heart and cause tamponade-like physiology. Therefore, this phenomenon may help predict poor clinical outcomes. However, large-scale retrospective studies should be conducted in the future to ascertain whether there are causal relationships between fast clot formation within hemorrhagic cardiac tamponade and poor clinical outcomes, and to investigate the clinical value of this phenomenon.

Another important lesson that can be learned from this case is that, when treating patients with highly suspected AAAD, position management is worth taking into consideration. To the best of our knowledge, the nature of initial medical therapy, including tight blood pressure control and the administration of analgesia, is to decrease wall stress to limit the extension of the dissection and reduce the risk of developing end-organ damage and rupture. In our case, we speculate that, due to extreme fatigue, when the patient rapidly changed position from standing to supine, lying down on the rescue bed, a sudden increase in blood flow to the heart may have further increased the burden of the dissected vessel; this could have contributed to the induction of the rupture of the aortic dissection.

Although this increase in blood flow was negligible for the average healthy population, for patients in the acute stage of aortic dissection with hypertensive crisis and intimal lesions, it may become the straw that breaks the camel’s back, so to speak. Thus in patients with AAAD, when blood pressure is not effectively under control, it is wise to maintain a sitting or a semi-reclined position.

AAAD is a time-critical disease, especially in terms of the timing of surgical intervention. Based on IRAD data, the acute unoperated mortality rate for AAAD is 1% to 2% per hour during the first 48 hours. However, the median time from arrival

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**Figure 1.** Critical care ultrasound findings of fast blood clot formation within a hemopericardium complicated by the rupture of an aortic dissection. (A) Apical 4-chamber view. (B) Subcostal 4-chamber view. The white arrow indicates the annular blood clot surrounding the epicardium. Hemopericardium, LV = left ventricle, RV = right ventricle.
in the emergency department to the definitive diagnosis of AAAD is 4.3 hours, and it takes an additional 4 hours before surgical treatment. This time delay may further increase the difficulty of treating patients with hemorrhagic pericardial tamponade complicated by AAAD. Although pericardiocentesis is a contraindication in this patient group, Hayashi et al reported an alternative strategy using controlled pericardial drainage for emergency treatment. Meanwhile, open-chest massage may also help.

However, not all hospitals that patients initially visit are equipped to perform these procedures. In the meantime, the long-term clinical outcomes of cardiac arrest due to hemorrhagic tamponade complicated by AAAD depend on the quality of cerebral resuscitation. In addition, based on recent experiences with the treatment of AAAD in the perioperative and postoperative periods using extracorporeal membrane oxygenation (ECMO), it is necessary to investigate the feasibility of long-distance transfer of such patients. To this end, large clinical trials should be designed to verify whether ECMO could help maintain organ perfusion during this period of transportation.

4. Conclusions

Hemorrhagic cardiac tamponade complicated by AAAD is a clinical crisis. Critical care ultrasound offers an effective tool for timely etiology screening of highly suspected patients. Rapid blood clot formation in bloody pericardial effusion may indicate a fast rupture and may predict poor clinical outcomes. Large cohort studies are warranted to investigate this imaging sign in the future.

Author contributions

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