International Journal of Emergency Medicine

CASE REPORT Open Access

Acute aortic dissection on CT: is D-dimer determination useful for a timely and correct diagnosis? A case report

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Abstract

Background Chest pain is a common reason patients are admitted to the hospital. The most clinically significant cases are those in which the pain is due to an immediate life-threatening condition, such as acute aortic dissection (AAD). A prompt and correct diagnosis is crucial to patient survival. This case report of a patient who presented with chest pain confirms the appropriateness of urgent imaging tests e.g. POCUS when AAD is suspected in high-risk patients, regardless of the results of additional laboratory tests such as the D-dimer (DD) assay.

Case report A 72-year-old female patient was brought by the emergency medical team to the emergency room due to fainting without loss of consciousness and severe chest pain. Owing to worsening hypotonia and recurrent chest pain, a thoracic computed tomography (CT scan) was performed and subsequently revealed aortic dissection within the ascending segment with bleeding into the pericardial sac. The results of previously ordered laboratory tests, including the DD assay, were unremarkable and were obtained only after the thoracic CT scan had been acquired. Despite prompt medical intervention, the patient died.

Conclusion Vigilance is required when diagnosing chest pain in high-risk patients who are suspected of having AAD. The case presented in this report confirms the importance of a thorough history and physical examination as well as prompt diagnostic imaging e.g. POCUS or CT scan. Dedicated laboratory tests such as the DD assay, while often helpful, may fail to reveal remarkable abnormalities in time for medical intervention.

Keywords D-dimer, Thoracic Aorta Dissection, Chest Pains

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Background

Chest pains of various eaetiologies are common in patients admitted to the hospital. The most clinically significant cases are those in which the pain is due to an immediate life-threatening condition [1]. It is therefore important to rule out acute conditions requiring urgent targeted treatment, such as AAD, as soon as possible. In AAD, the aortic wall is dissected to form the true and false vascular lumina, the latter of which leads to greater risk of perforation and rapid patient death [2]. In epidemiological studies, the incidence of AAD has been estimated at 6 cases per 100,000 people per year [3], while it

is higher in men than in women and increases with age [4]. According to the ESC guidelines, when AAD is suspected, the clinical likelihood of the condition should be assessed on the basis of characteristic comorbidities and history (family history of aortic diseases, Marfan syndrome, diagnosis of valvular or aortic disease, previous aortic surgeries), pain characteristics (sudden onset, very high intensity, and severe pain) and symptoms (hypotension/shock, *de novo* aortic diastolic murmur, signs of disturbed perfusion, pulse deficit, neurological deficits, and differences in systolic pressure measured in both upper extremities). One point is assigned for every condition that is observed in the patient. The clinical risk is considered high for scores greater than 2, with DD levels used to exclude AAD in low-risk patients [5]. In the available literature, elevated DD levels have been reported in most AAD patients [6–11]. Caroline et al. reported a case where the clinical presentation of AAD was atypical and the correct diagnosis and CT of the thoracic vasculature were guided by a high DD level [12]. Owing to the very high mortality rate (a 2% increase every hour during the first 48 h), prompt and accurate diagnosis is crucial to increase the chances for the implementation of targeted therapy and patients' survival [13]. For quick diagnosis, bedside cardiac ultrasound can be performed as a noninvasive and radiation-free method [14].

The objective of this case report is to highlight the possibility of patients with AAD presenting with unremarkable DD levels, which should be considered when treating patients with chest pain.

Case Report

A 72-year-old female patient was brought by the emergency medical team to the emergency room due to fainting without loss of consciousness and severe chest pain. The patient had previously been treated for anxiety-depressive disorder, hypertension, and hypothyroidism. Upon examination, the patient's general condition was assessed as moderate. The patient was lethargic and presented with psychomotor retardation, a circulatory blood pressure of 90/55 mmHg (as measured in the left upper limb), a heart rate of 53 bpm, no pathological murmurs, saturation of 96%, and a body temperature of 37.0 °C. Upon auscultation of the lung fields, the vesicular murmur was physiological and symmetrical; the abdomen was painless on palpation, the peritoneal signs were negative; slight swelling of the lower extremities was notable.

The patient was subjected to an ECG examination, and serum was collected for laboratory tests immediately after admission. The ECG (Fig. 1) revealed sinus bradycardia of 53 bpm, isolated supraventricular premature beats, and features of left ventricular hypertrophy (LVH). After approximately 15 min owing to the worsening of hypotension despite fluid resuscitation and nonremittance of chest pain, a decision was made to perform an urgent (without waiting for laboratory test results) chest CT scan with arterial evaluation (the patient was classified as being at high risk for ADD). The CT scan revealed a short segment of ascending aortic dissection (starting just above the origin of the coronary arteries), with a periaortic haematoma that was 12 mm thick and bleeding into the pericardial sac (with a fluid thickness of

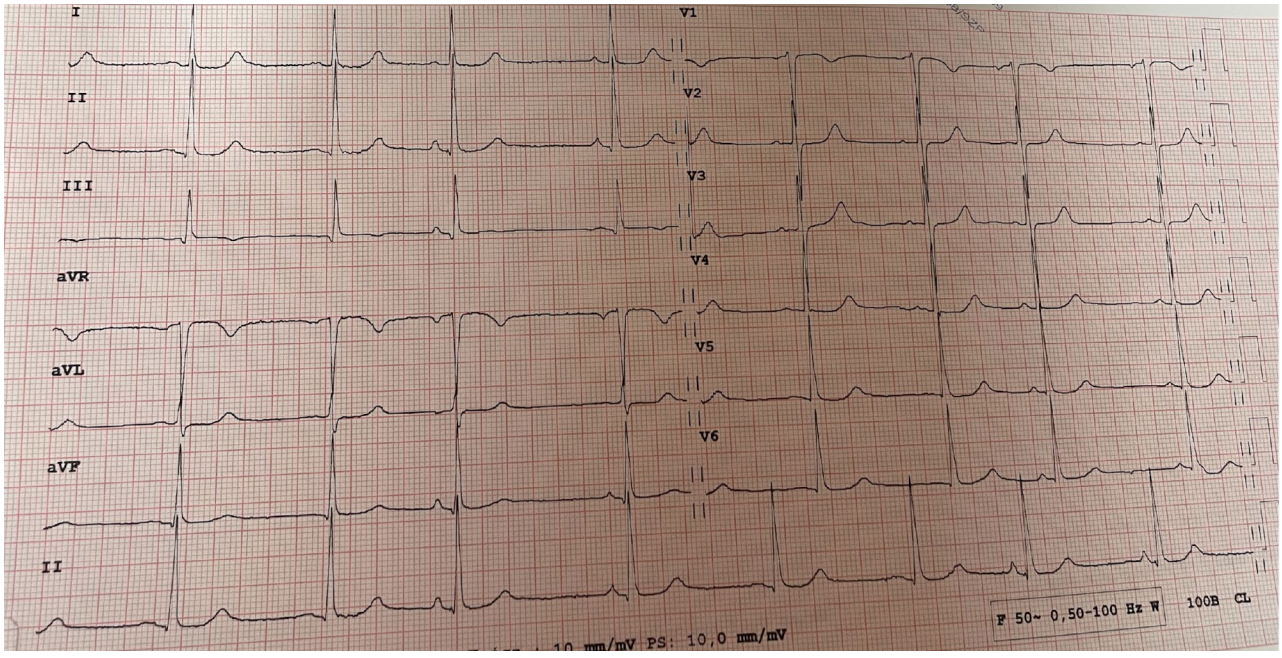

Fig. 1 ECG examination record – bradycardia 53 BPM with single premature supraventricular beats; voltage criteria of LVH

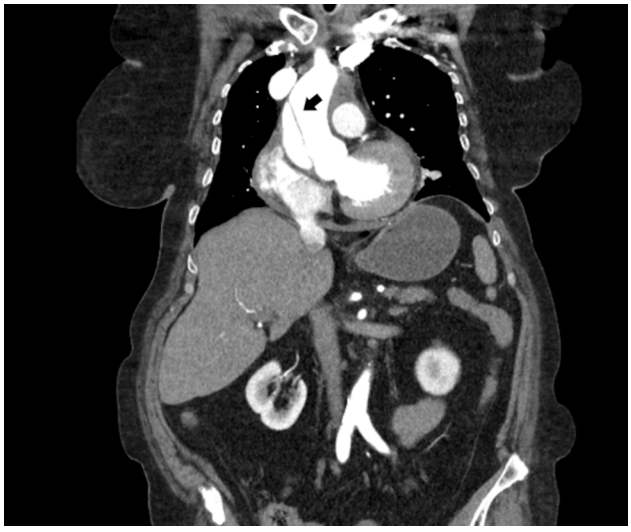

Fig. 2 Contrast-enhanced CT scan of the thoracic vasculature in the frontal projection: the ascending aorta is dilated at the dissection site, with intimal delamination (black arrow) and uniform enhancement of both true and false vascular lumina

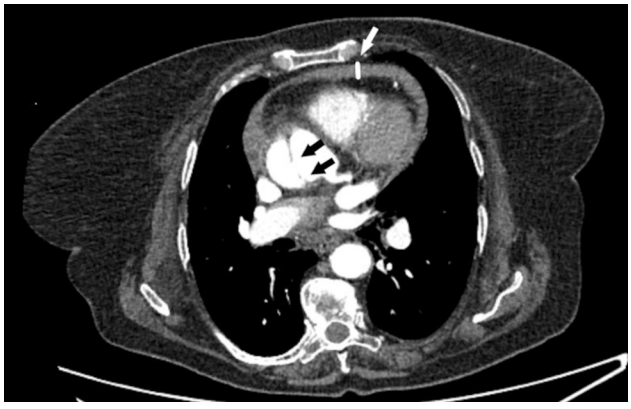

Fig. 3 Contrast-enhanced CT scan of the thoracic vasculature in the transverse projection: intimal delamination within the lumen of the ascending aorta (black arrows) and accumulation of fluid in the pericardial sac (white arrow)

10 mm). The diameter of the ascending aorta at the dissection level was dilated to 44 mm. The remaining aortic segments were undilated (Figs. 2 and 3).

The patient was urgently referred to the Department of Cardiac Surgery - she was qualified for emergency surgery. While waiting for the arrival of the transport ambulance, a poinf-of care transthoracic echocardiogram was performed (Figs. 4 and 5) and revealed ascending aortic dilatation of up to 4.8 cm with a prominent false lumen, intraventricular septal hypertrophy, and accumulation of fluid in the pericardial cavity, with no signs of life-threatening tamponade. Moreover, laboratory results were obtained (Table 1), which revealed no significant deviations in the inflammatory parameters, erythrocyte sedimentation rate (ESR), DD, haemahaematology, or

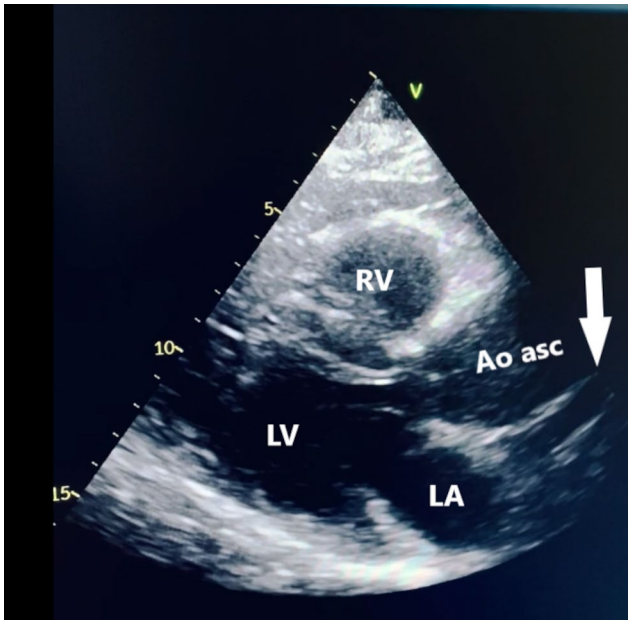

Fig. 4 Point-of-care echocardiography of the parasternal long-axis projection: the ascending aorta is dilated at the dissection site, with intimal delamination (white arrow), leading to the formation of true and false vascular lumina

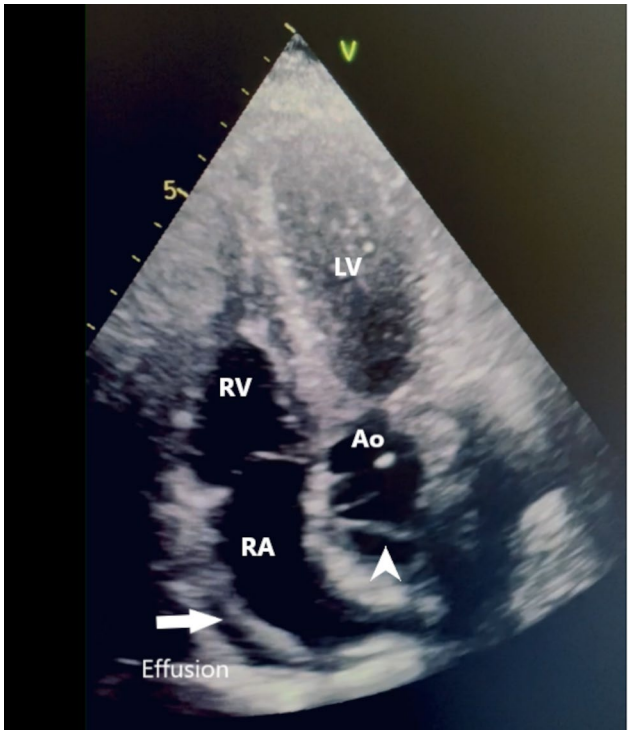

Fig. 5 Point-of-care echocardiography in modified apical view: accumulation of fluid in the pericardial sac at the right atrium of the heart (white arrow) and intimal delamination within the ascending aorta (white pointer)

Table 1 Patient's laboratory test results

Laboratory parameter	Result	Unit	Reference range
WBC	9,2	tys/ug	4,0–10,0
RBC	4,44	mln/ul	3,9 – 5,1
Ht	44,5	%	34,1–44,9
ESR	11	mm/h	0–20
DD	0,25	ug/ml	0,0–0,5
NT-pro -BNP	840	pg/ml	0,0-125
Troponin	0,011	ng/ml	0,000–0,014
TSH	2,4	ulU/ml	0,270-4,2
Creatinine	0,82	mg/dl	0,5 – 0,9
Na	142	mmol/l	136–145
K	4,1	mmol/l	3,5–5,1
CRP	2,0	mg/l	0,0–5,0

Abbreviations: WBC: White blood cells; RBC: Red blood cells; Ht: Hematocrit; ESR: Erythrocyte sedimentation rate; DD- D- dimer level; NT-pro -BNP: N-terminal pro b-type natriuretic peptide; TSH: Thyroid stimulating hormone; Na: Sodium; K: Potasium; CRP: C-reactive protein

troponin T measurements and a slight increase in the NT pro-BNP level.

While waiting for the transport ambulance, a continuous intravenous infusion of pressor amines was started due to increasing hypotension, despite this patient had suffered sudden cardiac arrest, and resuscitation was performed in accordance with the ERC guidelines spontaneous circulation could not be restored. Since the amount of effusion in the pericardium was not increasing in POCUS, pericardial aspiration was not performed. The autopsy confirmed hypovolemic shock due to a perforated aneurysm during aortic wall dissection into the left pleural cavity as the cause of patient death (only 150 ml of clotted blood was found in the paricardial sac) The total time elapsed between the initial contact with the emergency physician and the pronouncement of death was one hour.

Discussion

The objective of this report is to present the case of a patient who was diagnosed with high-risk AAD while presenting with unremarkable DD levels. This is an unusual presentation, as it has been shown that DD levels are significantly elevated even in cases of low-risk AAD [15]. The proposed diagnostic path for suspected AAD is very clearly presented in the figure in the ESC guidelines (Fig. 6) According to the ESC guidelines for the diagnosis and treatment of aortic diseases, ECG and laboratory tests should be considered for differential diagnosis or detection of complications in patients admitted to the hospital with chest pain and suspicion of AAD, including DD levels in patients at low risk of AAD (in addition to imaging studies, which are crucial for patients at high risk of AAD) [5]. D-dimer is a byproduct of blood clot degradation via the fibrinolytic system; hence, it is a marker of the coagulation system and fibrinolysis activation [6]. Elevated DD levels are observed in disorders associated with thrombosis, such as aortic dissection, acute coronary syndrome, and thromboembolic disease [7]. Notably, according to other studies, DDs are of the highest diagnostic value within the first hour from the onset of symptoms [5]. A number of studies in the literature have confirmed that DD levels > 500 ng/mL are very sensitive and reliable parameters for ruling out AAD [16]. In addition to DD, the levels of biomarkers such as troponin and CRP were found to be higher in patients with AAD than in controls [8]. DD, microRNA, and IL-6 have been identified as high-sensitivity markers for the diagnosis of AAD. Although the specificity of the DD assay is low, it is a reasonably good indicator of AAD when considered in the context of other biomarkers, especially as the symptoms of aortic dissection intensify [9, 17]. This was the case with the patient described by Caroline et al., who presented with a suspicion of epilepsy or transient ischaemic incident due to accompanying neurological symptoms. Although the patient had no chest pain or shortness of breath, a thoracic CT scan was performed due to the high DD level, which revealed AAD [12]. Similarly, in the case of a patient with persistent symptoms of a respiratory tract infection, dry cough, and pleuritic chest pain, an angio-CT scan was performed due to elevated serum D-dimer levels, revealing a penetrating aortic ulcer [18] In the case presented herein, the normal DD level could be misleading if the decision to perform a CT scan was based on its determination. Owing to the 95–97% sensitivity of DD assays, limitations exist regarding the use of DD as an ideal marker for the diagnosis of AAD [19]. In addition, false-negative DD results are observed in younger patients, patients with short dissection lengths, false lumen thrombosis, and some subtypes of acute aortic syndrome, such as intramural haematoma or incomplete dissection [10, 11]. In the case presented herein, no significant abnormalities were detected in the laboratory findings despite the patient's diagnosis of AAD. Although the chest pain reported by the patient had been present for more than an hour, no significant increase in the DD level was observed, with a level as low as 0.25 µg/mL. Thus, unremarkable DD levels do not always exclude the presence of serious aortic disease, even though a considerable number of patients with AAD present with DD levels > 500 ng/mL [20]. This confirms the need for performing imaging studies as quickly as possible, rather than relying on laboratory test results, when AAD is suspected. Imaging can reveal characteristic features of AAD, such as pericardial effusion. In this case, the doctors demonstrated appropriate diagnostic vigilance and, without waiting for laboratory test results, referred the patient for a CT scan. The most likely better solution would have been to urgently perform a POCUS examination, which can be conducted even in patients in

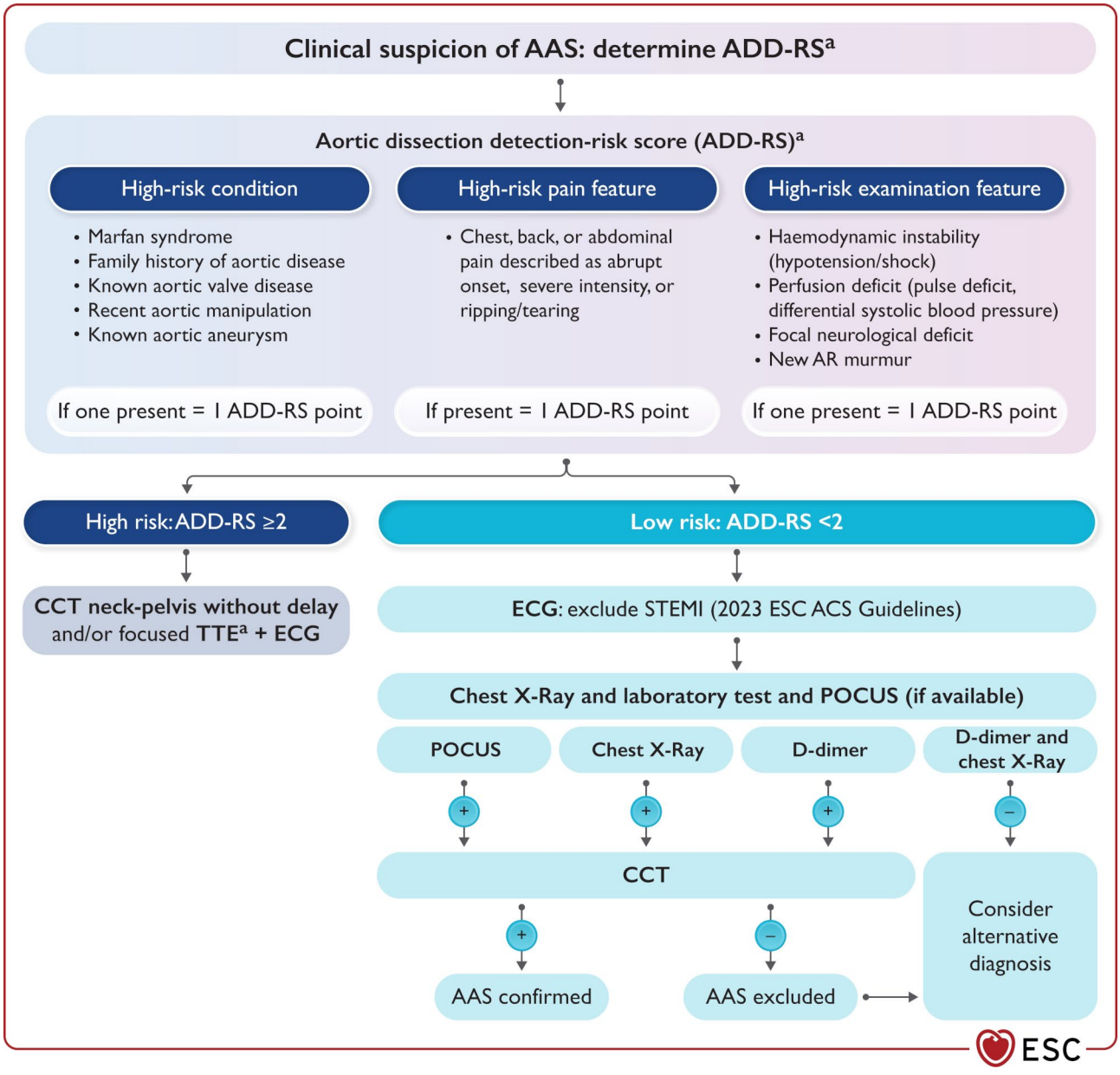

Fig. 6 Multiparametric diagnostic work-up of acute aortic syndrome – ESC Guidelines for the management of peripheral arterial and aortic diseases

a very critical condition who might face difficulties being transported for a CT scan.

Conclusion

In the case presented herein, the patient was eligible for urgent CT of the thoracic vessels and bedside transthoracic echo. Imaging studies are crucial for making a prompt and accurate diagnosis. Quick POCUS may highlight symptoms that may coexist with ADD, e.g. pericardial effusion, dilatation of the ascending aorta, aortic valve regurgitation. Notably, however, despite the imaging diagnosis of AAD, the results of the laboratory tests were normal, rather than elevated as would be expected in this case. Thus, unremarkable DD levels do not exclude the presence of a serious aortic disorder, in this case, AAD. It is important to remember that clinical suspicion should always take precedence over any laboratory parameter. In summary, the need to discover a more sensitive and specific marker for AAD and the dissemination of POCUS is highlighted as such a determination could prove helpful in situations where advanced diagnostic imaging exams are not feasible.

Acknowledgements
not applicable.

Author contributions
A.S and B.M wrote the main manuscript text and prepared all figuresA.W provided data about the patientW.I read the literature, completed the data and translated the textL.G provided substantive supervision over the entire case description processAll authors reviewed the manuscript.

Funding
Not applicable.

Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethical approval
Not applicable.

Competing interests
The authors declare no competing interests.

Received: 11 November 2024 / Accepted: 3 January 2025
Published online: 09 January 2025

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