WHEN MYOCARDIAL INFARCTION IS NOT THE ACTUAL DIAGNOSIS – DIAGNOSTIC CHALLENGES IN ACUTE SETTINGS

Kiedy zawał serca to nie zawał – diagnostyczne wyzwania w stanach nagłych

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Abstract

Aortic dissection is an acute state in which a tear in the intimal layer of the aortic wall allows blood to enter in between the intimal and medial layers. Risk factors include hypertension, smoking, dyslipidaemia, inflammatory diseases of the aorta and genetic connective tissue disorders. Severe, tearing chest pain is the main symptom of aortic dissection. The dissection may progress along the aorta, leading to several complications, such as myocardial infarction, acute aortic regurgitation, shock, stroke, acute kidney injury, abdominal organ ischaemia. Due to the high mortality rate, aortic dissection requires urgent diagnosis and rapid initiation of treatment. This article describes a case of a 78-year-old male admitted to the hospital with chest pain and neurological symptoms. Initially, the clinical presentation suggested a complicated myocardial infarction accompanied by cardiogenic shock; however, further diagnostic workup revealed an aortic dissection. The patient was transferred to the Cardiothoracic Surgery Operating Room, but he did not survive the surgery. The clinical presentation of aortic dissection can vary, with nonspecific symptoms due to possible multiple complications. Rapid, multidisciplinary diagnostic evaluation and immediate therapeutic intervention are essential, as the mortality rate increases rapidly over time.

Streszczenie

Rozwarstwienie aorty jest ostrym stanem, w którym w wyniku uszkodzenia błony wewnętrznej w ścianie aorty dochodzi do napływu krwi pomiędzy błonę wewnętrzną a środkową. Do czynników ryzyka rozwarstwienia należą: nadciśnienie tętnicze, palenie papierosów, dyslipidemia, choroby zapalne aorty i choroby genetyczne tkanki łącznej. Głównym objawem występującym w przypadku rozwarstwienia aorty piersiowej jest silny, rozdzierający ból w klatce piersiowej. Rozwarstwienie może postępować wzdłuż aorty, powodując liczne powikłania, m.in. zawał serca, ostrą niedomykalność aortalną, wstrząs, udar mózgu, ostre uszkodzenie nerek, niedokrwienie narządów jamy brzusznej. Ze względu na dużą śmiertelność jest to stan wymagający pilnej diagnostyki oraz jak najszybszego wdrożenia odpowiedniego postępowania leczniczego. W pracy przedstawiono opis przypadku 78-letniego mężczyzny przyjętego do szpitala z powodu bólu w klatce piersiowej i objawów neurologicznych. Obraz kliniczny przy przyjęciu w pierwszej kolejności wskazywał na powikłany zawał serca z towarzyszącym wstrząsem kardiogennym, natomiast w trakcie diagnostyki rozpoznano rozwarstwienie aorty. Pacjent po wstępnej stabilizacji stanu klinicznego trafił na Blok Operacyjny Kliniki Kardiochirurgii, gdzie zmarł w trakcie operacji. Obraz kliniczny rozwarstwienia aorty może być zróżnicowany, objawy mogą być nieswoiste ze względu na możliwe liczne powikłania. Wymagana jest jak najszybsza wieloprofilowa diagnostyka i niezwłoczne wdrożenie leczenia, ze względu na dużą śmiertelność, która dynamicznie wzrasta w czasie.

Keywords: aortic dissection; myocardial infarction; shock; stroke; complicated dissection

Słowa kluczowe: rozwarstwienie aorty; zawał serca; wstrząs; udar; powikłane rozwarstwienie

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Introduction

Aortic dissection (AD) is an acute aortic syndrome defined by an intimal tear that permits blood to enter the space between the intima and media, thereby forming a false lumen. AD is classified as type A (involving the ascending aorta) or type B (not involving the ascending aorta) according to the Stanford classification, as well as types I, II, IIIa, and IIIb (depending on the location of the dissection site) according to the DeBakey classification [1]. Hypertension (HT) is the most important and most common risk factor [2]. Cigarette smoking, dyslipidaemia, inflammatory aortic diseases, and genetic connective tissue disorders, such as Marfan syndrome, Ehlers–Danlos syndrome type 4, and Loeys–Dietz syndrome [3, 4], also play a significant role. The pooled incidence of all types of ADs is approximately 4.8 cases per 100,000 individuals per year, with the overall mortality rate reaching about 50% [5, 6]. Severe, excruciating chest pain unresponsive to nitrates that may radiate to the interscapular or lumbar regions, depending on the location of the lesion, is the main symptom of AD. Physical examination may reveal hyper- or hypotension, a murmur consistent with aortic regurgitation, and a pulse deficit in one limb. Complications of AD include aortic valve insufficiency, myocardial ischaemia or infarction, heart failure, shock, pericardial tamponade, limb or abdominal ischaemia, and neurological events such as stroke or transient cerebral ischaemia [3, 6].

According to the 2024 recommendations of the European Society of Cardiology, the diagnostic pathway depends on the risk of aortic dissection as assessed by the Aortic Dissection Detection Risk Score (ADD-RS). High-risk haemodynamically stable patients (ADD-RS ≥ 2) should have computed tomography (CT) of the aorta performed as soon as possible. This modality demonstrates the highest sensitivity (100%) and specificity (98%) among the available imaging techniques and is therefore the preferred diagnostic tool [7, 8]. In haemodynamically unstable patients who cannot be transported for CT, transthoracic and/or transoesophageal echocardiography represents an alternative diagnostic approach. In low-risk patients (ADD-RS < 2), an electrocardiogram, point-of-care ultrasound (POCUS), chest radiography, or D-dimer testing should be performed. If any raises suspicion of AD, CT should follow [4]. ICU admission for continuous monitoring is recommended. Surgical intervention is indicated for acute type A aortic dissection and complicated type B dissections, where open repair or thoracic endovascular aortic repair (TEVAR) may be considered based on arterial anatomy. TEVAR may also be considered in uncomplicated type B dissections with favourable anatomy and an estimated life expectancy of at least five years. In other cases, conservative management and outpatient follow-up are recommended.

Each case of aortic dissection presents a major diagnostic and therapeutic challenge requiring specialist, interdisciplinary care. This paper reports a case of AD with a particularly insidious and unfavourable clinical course.

Case report

A 78-year-old man with unknown medical history was transported by emergency medical team (EMT) to the emergency department (ED) with suspected ST-elevation myocardial infarction (STEMI). According to the EMT, the patient had been experiencing chest and epigastric pain accompanied by left-sided muscle weakness for several hours. An electrocardiogram performed by the EMT showed a junctional rhythm of 50 bpm, ST-segment elevation in leads II, III, and aVF, and ST-segment depression in lead V2. On admission, the patient was in serious condition, drowsy and confused, precluding medical history taking. Physical examination revealed left-sided weakness and pupillary asymmetry, with the right pupil dilated relative to the left. The patient was haemodynamically unstable (BP 80/50 mmHg), stabilised with a continuous dopamine infusion. Electrocardiographic findings suggestive of inferior STEMI prompted urgent transfer to the catheterization laboratory for coronary angiography. Due to haemodynamic instability from a junctional rhythm at 30 bpm, a temporary transvenous pacing lead was inserted in the first place, achieving capture at 80 bpm. Due to angiographic suspicion of cardiac tamponade, transthoracic echocardiography performed in the catheterization laboratory showed pericardial effusion with a maximum separation of 18 mm but no signs of tamponade. Coronary angiography demonstrated marked aortic dilatation with suspected dissection, and the procedure was therefore terminated. Emergency aortic CT confirmed a dissection involving the entire thoracic aorta (Fig. 1) and most likely the entire abdominal aorta, with possible extension into the iliac arteries. The dissection extended to the brachiocephalic trunk, the left common carotid artery (LCCA), the right common carotid artery, the left subclavian artery, and the left renal artery, with significant narrowing of the true lumen of the ascending aorta. A false lumen was identified in the LCCA, likely thrombosed, causing significant stenosis. The pericardial sac contained blood (approximately 15 mm), with bloody fluid measuring about 30 mm in both pleural cavities. The patient underwent cardiac surgical consultation and was scheduled for urgent surgical intervention. Upon admission to ICU, he remained in a serious general condition, drowsy and confused, with limited verbal and logical communication. Circulatory failure persisted despite continuous dobutamine infusion (blood pressure 90/55 mmHg, heart rate 80 bpm with ventricular pacing), along with respiratory failure requiring passive oxygen therapy via an oxygen mask. Pupillary asymmetry and left-sided muscle weakness persisted. Cardiac auscultation revealed a soft diastolic murmur. To stabilise haemodynamics, norepinephrine was added to the dobutamine infusion, and intravenous fluid therapy was initiated. Laboratory workup showed metabolic acidosis, elevated lactates (3.3 mmol/L), increased myocardial necrosis markers (hs-TnT 88 ng/L), impaired renal function (creatinine 1.5 mg/dL), markedly raised D-dimers (62.9 µg/mL), mild normocytic anaemia (haemoglobin 11.6 g/dL), and thrombocytopenia (100×10^3/µL). POCUS demonstrated segmental wall motion abnormalities, including akinesia of the proximal half of the inferior and inferolateral walls, with a reduced left ventricular ejection fraction of 45%. The ascending aorta was markedly dilated from the bulb, with a visible dissection membrane. The aortic valve was tricuspid with a dilated annulus and at least moderate regurgitation. Pericardial effusion was also noted, measuring up to 15 mm anterior to the right ventricle (with a visible fibrin layer) and up to 12 mm posterior to the left

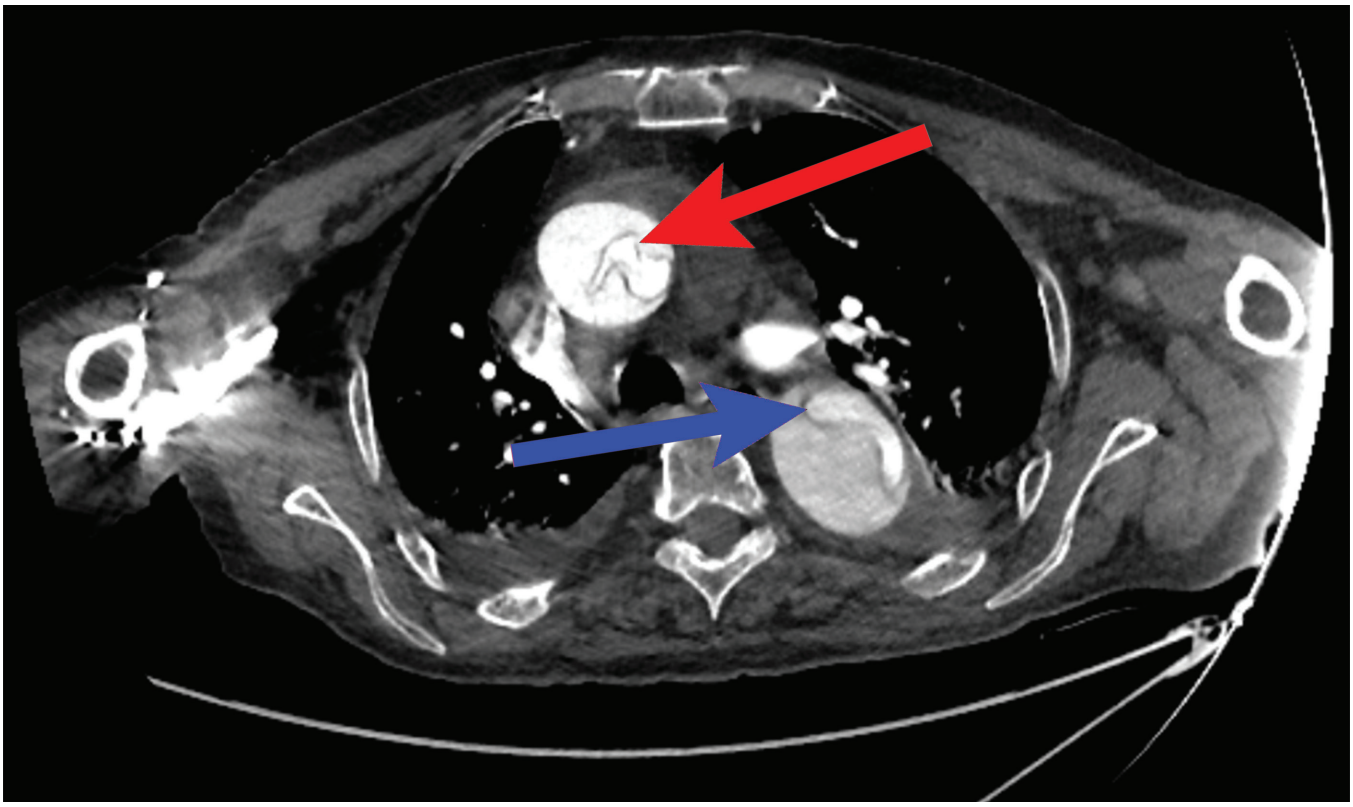

Figure 1. Evident dissection membrane in the ascending aorta (red arrow) and in the thoracic aorta (blue arrow)

ventricular posterior wall. The patient, in a critical condition, was transferred to the operating theatre of the Department of Cardiac Surgery for surgical intervention. Under cardiopulmonary bypass, median sternotomy was performed, relieving pericardial tamponade via a small incision, which provoked massive pericardial haemorrhage. Aortic rupture was palpated, with repeated unsuccessful attempts to place a transverse aortic clamp. Due to failure to establish effective cardiopulmonary bypass and control bleeding from the multidirectionally ruptured aorta, the patient died intraoperatively.

Discussion

Acute aortic dissection is a sudden, rapidly progressive emergency with high mortality, requiring prompt diagnosis and intervention. Acute aortic dissection often presents a diagnostic challenge, as its nonspecific symptoms can mimic other conditions, especially with organ involvement. This makes the presented case particularly noteworthy, as the patient exhibited multiple complications and features mimicking complicated MI rather than aortic dissection, thereby shaping the initial diagnostic and therapeutic strategy. Severe chest pain (type A in 79% and type B in 63% of cases), which may radiate to the interscapular and lumbar regions, is the primary symptom of AD [9]. Abdominal aorta involvement can cause abdominal pain, as seen in the presented case. Notably, abdominal pain may be the sole symptom of aortic dissection (AD) in 4.6% of patients, with such atypical presentations linked to delayed diagnosis and worse prognosis [10]. Chest pain is linked to many conditions and is a common cause of hospital admission, with atraumatic cases accounting for about 8% of ED visits [11]. AD is rarely the initially suspected cause of such symptoms, accounting for only 0.1% of cases (1 in 980) [11]. In the present patient, in addition to pain, the electrocardiogram demonstrated ST-segment elevation in leads II, III, and aVF, plus impaired automaticity typical of inferior wall MI. The clinical presentation strongly suggested STEMI, prompting initiation of targeted diagnostic and therapeutic measures. The patient was urgently transferred to the catheterization laboratory for coronary angiography. However, significant aortic dilatation and suspected pericardial effusion noted early in the examination prompted discontinuation of further assessment.

Acute aortic regurgitation, occurring in 40–75% of cases, which may lead to acute heart failure or even shock, is one of the most common cardiovascular complications of AD. STEMI secondary to aortic dissection is diagnosed in only 0.51% of cases [12]. Shock is a severe complication of AD, affecting 15.1–23.3% of patients and linked to worse short-term prognosis and higher in-hospital mortality [13, 14]. The described patient developed cardiogenic shock, potentially stemming from MI, acute aortic regurgitation, or progressive pericardial effusion. AD may also be complicated by hypovolemic shock, particularly in cases of aortic rupture with haemorrhage into the pleural cavities, mediastinum, or peritoneal cavity. Additionally, the patient presented with neurological symptoms suggestive of stroke and laboratory evidence of acute renal failure. These findings may have been secondary to shock or dissection involving the renal and cranial arteries, as demonstrated on CT.

The diagnostic challenges associated with AD have been well illustrated in a case report by Lasa-Berasain et al. [15].

The authors described a 60-year-old patient with abdominal AD managed with endovascular repair, who suddenly developed neurological symptoms suggestive of acute stroke, including extension of the left upper limb, drooping of the left corner of the mouth, and loss of consciousness. The patient subsequently experienced cardiac arrest; after successful resuscitation, he remained haemodynamically unstable and was therefore transferred to a referral hospital for further management. On admission to ED, physical examination revealed weak peripheral pulses, prolonged capillary refill time, and an early diastolic murmur over the aortic valve. The patient was hypotensive, with a blood pressure of 88/67 mmHg. Electrocardiography showed ST-segment elevation in the lateral leads, accompanied by ST-segment depression and tall T waves in the anterior leads. POCUS revealed, among other findings, a dissection membrane in the ascending aorta, along with aortic regurgitation. An urgent CT scan subsequently confirmed type A aortic dissection. In the authors' case, closely mirroring ours, the clinical presentation mimicked MI. Aortic dissection was suspected only after POCUS demonstrated the dissection membrane.

Chenkin [16] described a 69-year-old man presenting to ED with sudden-onset severe stabbing retrosternal pain that began 2 hours earlier during physical exertion. The pain was accompanied by dyspnoea and profuse sweating. Physical examination was largely unremarkable, except for markedly elevated blood pressure (185/101 mmHg). Electrocardiography revealed ST-segment elevation in leads aVR, V1, and V2, along with diffuse ST-segment depression in multiple other leads. Due to suspected acute coronary syndrome, the patient was scheduled for transfer to the catheterization laboratory. However, US performed in the emergency department revealed a dissection membrane in the ascending aorta along with severe aortic regurgitation. Pharmacological treatment was initiated, and an urgent CT scan was performed, which confirmed acute type A aortic dissection. During transport to the operating room, the patient developed cardiac arrest and died despite resuscitation efforts. Myocardial infarction masked AAD, posing a major diagnostic challenge due to their divergent treatment strategies.

Conclusions

As shown by our case report and other cited cases, acute aortic dissection produces a highly heterogeneous clinical picture, necessitating prompt multidisciplinary diagnostic evaluation for accurate diagnosis. In certain cases, subtly expressed symptoms or findings noted only on physical examination may prove crucial in raising suspicion of AD. Prompt and accurate diagnosis of AD is critical, as mortality rises by about 2% per hour of delay in initiating appropriate treatment [17]. Multi-organ complications can progressively alter the patient's clinical presentation and condition, leading to fatal outcomes despite advanced diagnosis and therapy.

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