Acute Aortic Dissection Presenting as Atypical Chest Pain in a Young Hypertensive Male: A Case Report

James R. Fletcher, MD[1] **Amara N. Osei**, MD, PhD[2] **Lena M. Hartmann**, MD[1]

[1]Department of Internal Medicine, Northgate University Hospital, London, UK
[2]Division of Cardiothoracic Surgery, St. Crispin Medical Centre, Manchester, UK

Correspondence: James R. Fletcher, MD • j.fletcher@northgate.nhs.uk
Received: 12 February 2025 • **Accepted:** 04 April 2025 • **Published:** 20 May 2025

Background: Acute aortic dissection (AAD) is a life-threatening emergency that carries a mortality rate exceeding 1% per hour if untreated. Atypical presentations—particularly in younger patients without classical risk factors—frequently delay diagnosis.

Case Presentation: A 34-year-old male with poorly controlled hypertension presented with progressive interscapular pain of 6-hour duration mischaracterised as musculoskeletal. Serial aortic computed tomography angiography (CTA) confirmed a type A dissection extending from the aortic root to the iliac bifurcation (DeBakey type I). Emergency open surgical repair with composite graft replacement of the ascending aorta was performed. The patient was discharged neurologically intact on day 12.

Conclusions: This case underscores the importance of maintaining a high index of suspicion for AAD in young hypertensive patients presenting with back or chest pain, even when the presentation is atypical. Prompt CTA and multidisciplinary involvement are essential for favourable outcomes.

Keywords: aortic dissection; hypertension; chest pain; computed tomography angiography; cardiac surgery

Introduction

Acute aortic dissection is one of the most catastrophic vascular emergencies encountered in clinical practice, with an estimated incidence of 3–5 per 100,000 persons per year [1]. The hallmark presentation—sudden, severe, tearing or ripping chest pain radiating to the back—is absent in up to 17% of cases, making diagnosis challenging [2].

Risk factors include hypertension (present in 72% of cases), Marfan syndrome, bicuspid aortic valve, and cocaine use [3]. When the diagnosis is missed in the emergency setting, in-hospital mortality may exceed 50% [4].

We report a case of DeBakey type I AAD in a young male presenting with interscapular pain initially attributed to a musculoskeletal cause, highlighting diagnostic pitfalls and the importance of early cross-sectional imaging.

Case Presentation

History

A 34-year-old male security officer with a 4-year history of poorly controlled essential hypertension presented to the Emergency Department at 03:22 with a 6-hour history of progressive, sharp, interscapular pain (rated 8/10 in severity). The pain had onset at rest, was non-radiating at first, and had since migrated toward the anterior chest. He denied dyspnoea, syncope, nausea, or neurological symptoms. He was a non-smoker with no family history of connective tissue disease. His only regular medication was amlodipine 5 mg once daily, which he admitted taking inconsistently.

Physical Examination

On arrival the patient appeared diaphoretic and anxious. Key findings were:

- Blood pressure: 178/102 mmHg (right arm), 142/88 mmHg (left arm) — differential of 36 mmHg
- Heart rate: 112 bpm; respiratory rate: 20 breaths/min; SpO_2: 97% on air
- Auscultation: early diastolic murmur at the left sternal border (grade II/VI)
- Bilateral radial pulses palpable; right femoral pulse diminished
- No signs of Marfanoid habitus; no lens dislocation

Vital Signs and Selected Laboratory Values

Parameter	Value	Reference Range
Blood pressure (R arm)	178/102 mmHg	<120/80 mmHg
Blood pressure (L arm)	142/88 mmHg	<120/80 mmHg
Heart rate	112 bpm	60–100 bpm
Temperature	37.4 °C	36.1–37.2 °C
SpO_2	97%	≥95%
Haemoglobin	13.8 g/dL	13.5–17.5 g/dL
Platelets	241 $\times 10^9$/L	150–400
White cell count	14.2 $\times 10^9$/L	4.0–11.0
Serum creatinine	118 µmol/L	62–106 µmol/L
D-dimer	4.8 µg/mL FEU	<0.5 µg/mL FEU
High-sensitivity troponin I	28 ng/L	<52 ng/L
CRP	6 mg/L	<5 mg/L
INR	1.1	0.8–1.2

Investigations

ECG showed sinus tachycardia with left ventricular hypertrophy by voltage criteria; no ST-segment changes or ischaemic features.

Chest X-ray demonstrated mediastinal widening (10.2 cm at the aortic knuckle) and a small left pleural effusion. This prompted urgent CTA of the aorta.

CTA of the thorax, abdomen, and pelvis revealed an intimal flap originating 2 cm above the aortic valve, extending through the arch, descending thoracic aorta, and terminating at the iliac bifurcation—consistent with DeBakey type I (Stanford type A) dissection. The true lumen supplied the right coronary and right renal arteries; the false lumen supplied the left renal artery with resultant hypoperfusion. No haemopericardium was identified.

Transoesophageal echocardiography confirmed moderate aortic regurgitation secondary to cusp prolapse into the false lumen.

Differential Diagnosis

Given the presenting symptoms, the following differentials were considered:

1. **Acute aortic dissection** (type A) — confirmed on CTA; supported by inter-arm BP differential and mediastinal widening.
2. **Acute coronary syndrome** — troponin normal on two serial measurements; ECG non-ischaemic.

3. **Pulmonary embolism** — D-dimer elevated but Wells score low (2 points); CT pulmonary angiogram negative for filling defect.
4. **Musculoskeletal pain** — excluded by imaging findings and haemodynamic profile.
5. **Oesophageal rupture (Boerhaave syndrome)** — no history of vomiting; excluded on CTA.

Management and Outcome

Immediate measures included intravenous esmolol infusion (target heart rate <60 bpm), sodium nitroprusside titrated to systolic BP 100–120 mmHg, and large-bore IV access with crossmatch of 6 units of packed red cells.

The cardiothoracic surgical team was activated within 30 minutes of CTA reporting. The patient was taken to the operating theatre 3 hours and 18 minutes after Emergency Department presentation. Under cardiopulmonary bypass with moderate hypothermic circulatory arrest (24 °C), the dissected ascending aorta and hemi-arch were replaced using a Dacron composite graft; the aortic valve was resuspended.

The operative time was 5 hours 42 minutes; total bypass time 210 minutes; circulatory arrest time 18 minutes.

Postoperative course was uneventful aside from a mild acute kidney injury (creatinine peak 198 µmol/L, day 2), which resolved with IV fluid optimisation. The patient was extubated at 14 hours and transferred from the intensive care unit on day 4. He was discharged home on day 12 on bisoprolol 5 mg, ramipril 5 mg, and aspirin 75 mg daily. Imaging at 3-month follow-up showed complete false-lumen thrombosis in the ascending segment with a patent true lumen throughout.

Discussion

This case illustrates several important teaching points. First, the inter-arm systolic BP differential of 36 mmHg—a finding with a positive likelihood ratio of 5.7 for AAD [5]—was identified only because bilateral measurements were taken. In many emergency settings this vital step is omitted.

Second, the initial attribution of interscapular pain to a musculoskeletal cause led to a 90-minute delay before imaging. Registry data confirm that diagnostic delay is an independent predictor of operative mortality [6]. The International Registry of Acute Aortic Dissection (IRAD) reported that the mean time from symptom onset to diagnosis was 4.3 hours [4].

Third, the role of serum D-dimer as a rule-out biomarker in low-probability cases merits attention. Studies have reported sensitivities of 96–100% for AAD at a cut-off of 0.5 µg/mL FEU [7]; however, positive predictive value is poor and a raised D-dimer cannot substitute for CTA in moderate- or high-probability presentations.

The favourable outcome in this patient was contingent on rapid multidisciplinary collaboration, adherence to target-heart-rate control before surgery, and familiarity with the DeBakey classification to guide surgical planning. Long-term surveillance with MRI or CT at 1, 3, and 5 years is recommended per European Society of Cardiology guidelines [1].

References

[1] Erbel R, Aboyans V, Boileau C, *et al*. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases. *Eur Heart J.* 2014;**35**(41):2873–2926. doi:10.1093/eurheartj/ehu281

[2] Mészáros I, Mórocz J, Szlávi J, *et al*. Epidemiology and clinicopathology of aortic dissection. *Chest.* 2000;**117**(5):1271–1278.

[3] Nienaber CA, Clough RE, Sakalihasan N, *et al*. Aortic dissection. *Nat Rev Dis Primers.* 2016;**2**:16053.

[4] Bossone E, LaBounty TM, Eagle KA. Acute aortic syndromes: diagnosis and management, an update. *Eur Heart J.* 2018;**39**(9):739–749.

[5] Klompas M. Does this patient have an acute thoracic aortic dissection? *JAMA.* 2002;**287**(17):2262–2272.

[6] Rampoldi V, Trimarchi S, Eagle KA, *et al*. Simple risk models to predict surgical mortality in acute type A aortic dissection: the International Registry of Acute Aortic Dissection score. *Ann Thorac Surg.* 2007;**83**(1):55–61.

[7] Suzuki T, Distante A, Zizza A, *et al*. Diagnosis of acute aortic dissection by D-dimer. *Circulation.* 2009;**119**(20):2702–2707.

Conflict of interest: The authors declare no competing interests.

Funding: No external funding was received.

Patient consent: Written informed consent was obtained from the patient prior to publication. All identifying details have been anonymised in accordance with COPE guidelines.