A silent case of aortic dissection: a case report

Marco Passaretti,[1] Carlo Maino[2]

[1]Department of Biomedical Sciences, Humanitas University, Pieve Emanuele, Milan; [2]Head of Emergency Department Humanitas Mater Domini, Castellanza, Varese, Italy

Abstract

Type B aortic dissection is a relatively rare clinical condition with, however, a significant clinical impact and therefore an early diagnosis is necessary. The clinical presentation is highly variable depending on the aortic district involved and the presence of signs secondary to hypoperfusion. This peculiarity makes diagnosis even more difficult. We describe the case of a 52-year-old patient who presented to our emergency department with significant chest pain and crisis; his medical history was silent and there were no known cardiovascular risk factors. The ultrasonographic, electrocardiographic, laboratory and semeiological findings and the resulting diagnostic pathways were totally negative. Only advanced diagnostics allowed the diagnosis of aortic dissection. This case allows us to reflect on the interpretation of blood chemistry tests and the application of diagnostic scores/pathways in everyday clinical practice, especially in less defined clinical pictures.

Correspondence: Marco Passaretti, Department of Biomedical Sciences, Humanitas University, via Rita Levi Montalcini 4, 20072 Pieve Emanuele (MI), Italy,
E-mail: marco.passaretti@humanitas.it

Key words: aortic dissection, score, pathways, D-Dimer

Conflict of interest: the authors have no financial or proprietary interests in any material discussed in this article.

Ethics approval and consent to participate: no ethical committee approval was required for this case report by the Department, because this article does not contain any studies with human participants or animals. Informed consent was obtained from the patient included in this study.

Patient consent for publication: the patient gave his written consent to use his personal data for the publication of this case report and any accompanying images.

Availability of data and materials: all data underlying the findings are fully available.

Received: 4 March 2025.
Accepted: 11 July 2025.
Early view: 22 August 2025.

Emergency Care Journal 2025; 21:13751
doi:10.4081/ecj.2025.13751

Introduction

Acute aortic syndromes is a potentially fatal condition that requires early diagnosis and immediate targeted treatment. The epidemiology is still poorly understood due to the high pre-hospital mortality, the prevalence is estimated to be around 0.2-0.8%, while population-based studies report incidences of 2.5-7.2 per 100,000 population/year, underlining a progressively increasing trend in recent years;[1-4] it affects mainly the male sex (65%) with a peak around the 7th decade of life (63 years). The clinical presentation can be subtle, especially in the absence of pre-clinical risk factors (cardiovascular, collagenopathies, family history) and in the absence of blood test with high specificity and sensitivity. We present the case of a 52-year-old patient with very intense chest pain, non-suggestive laboratory findings, negative diagnostic scores and a diagnosis of "type B" aortic dissection.

Case Report

In September 2024, a 52-year-old Caucasian man was brought to the Emergency Department complaining of acute chest pain of severe intensity that had been present for about three hours. He reported waking up suddenly complaining of typical thoracoalgia (numerical rating scale, NRS 9/10), described as persistent, with a tearing quality, resembling a knife-like sensation and not modifiable by breath nor exacerbated by mobilisation. He reported that he had never experienced such pain. Full medical history was assessed and collected.

Patient works as a laborer in a construction company and had no significant family medical history. The patient's past medical history was also negative, he denied taking drugs, was non-smoker, and drank only socially. The patient presented conscious with hemodynamic stability. Very agitated and in pain (NRS 9/10). The vital signs were in the normal range (heart rate 66 bpm) with the exception of the blood pressure trending towards hypertension (180/90 mmHg) despite the administration of nitroglycerine (2 puffs of nitroglycerine, 0.60 mg) during transport. There were no signs of diaphoresis and fever. On physical examination there were no major alterations: the hearth sounds were rhythmic without cardiac murmurs, radial pulses were palpable, symmetrical and normosphygmic. In the lower limbs there were no signs of ongoing deep vein thrombosis. Due to the persistence of the severe chest pain, morphine 4mg was administered immediately plus 2 mg after one hour and due to the concomitant hypertensive blood pressure trend (PAS 200-180mmHg, PAD 85-95mmHg) a further 2 puffs of nitroglycerine were administered with partial benefit on the algia and a reduction in systolic values.

Initial diagnostics

The following diagnostic investigations were performed: i) Electrocardiogram (ECG): SR with HR of 60 bpm, left axis deviation, non specific and diffuse repolarization abnormalities; ii) ABG analysis (Arterial Blood Gas): ph 7,38, no electrolyte alterations, BE -0.4, Lat 0.7; iii) Point-of-Care ultrasound (POCUS, convex probe 5 MHz): lung sliding is present in all examined zones, no signs of pneumothorax, A-lines, no effusions, no free fluid at Morrison's, splenic-renal and Douglas pouch, no pericardial effusion, aorta with normal course and diameter (21 millimeters) (Figure 1); iv) cardiac echoscopy (sector probe 2.5 MHz): no pericardial effusion, no right section dilatation, no dyskinesias. No signs of dissection at aortic root level nor signs of aortic regurgitation (Figure 2). The haematochemical exams were as reported in Table 1. Negative troponin and D-dimer results are emphasised.

In view of the non-unambiguous interpretation of the clinical picture, particularly in view of the laboratory findings (negative troponin and D-dimer) and the persistence of the painful symptoms, the differential diagnosis of chest pain was made: i) cardiovascular: Acute Coronary Syndrome (ACS), acute aortic syndrome, Pulmonary Thromboembolism (PTE); ii) pulmonary: pulmonary infarction, pleurisy, pneumothorax; iv) gastrointestinal: oesophagitis, Gastro-Oesophageal Reflux Disease (GERD), "pill oesophagitis", gastric/duodenal ulcer; v) mediastinal: mediastinitis; vi) "wall": osteomuscular; vii) psychiatric.

The following causes were excluded: pulmonary (negative point-of-care ultrasound, D-dimer and CRP not suggestive), gastrointestinal (negative history, symptoms not suggestive), mediastinal (clinic and bioumoral findings not suggestive), wall (negative history, symptomatology not suggestive) and, finally, psychiatric (negative history).

In the suspected life-threatening cardio-thoracic-vascular aetiology (ACS vs. PTE vs. acute aortic syndrome), the main diagnostic scores/pathways were calculated: i) HEART Pathway[5]: 3 points (low risk of 6 weeks MACE, 0.9-1.7%), negative troponine at one and three hours (rule-out for SCA); ii) Wells's Score[6]: 3 points (low risk), Geneva-R Score[7]): 0 points (low risk), YEARS Pathway[8]: rule-out for TEP (0.43%); iv) ADD-RS (Aortic Dissection Detection Risk Score)[3,4,9]: 1/3 with subsequent rule-out for negative D-dimer (414 ng/ml, RR 0-500 ng/ml FUE).

Approximately 4 hours after admission and a total of 12 mg of morphine, a significant improvement in the algic symptoms was observed (NRS 2/10) but pressure values above the limits persisted (mean systolic blood pressure 175 mmHg, mean diastolic blood pressure 95 mmHg, hearth rate 60-65 bpm) so ramipril 5 mg was administered.

In light of the marked improvement in clinical condition and the blood pressure, it was initially decided to discharge the patient. However, in view of the characteristics of the thoracic pain symptoms (intensity, type, duration, onset) and the need for large doses of a major opioid in a naive patient (12 mg of morphine) to resolve the algic symptoms, it was decided to perform second-level diagnostics by requesting chest CT angiography.

Chest CT angiography (CTA) revealed an intramural haematoma of the thoracic aorta extending from the aortic arch (distal to left subclavian artery's site of emergence, where there are also a few wall calcifications of the aortic arch), involving the entire descending aorta and currently exhausting at the thoraco-abdominal passage, maximum thickness 12 mm. At the level of the distal section of the thoracic aorta in the context of the intramural haematoma, there was an arterial blush of 1.5 cm compatible with the lesion of the intima in a Stanford B-type aortic dissection (Figure 3).

Because of the diagnostic finding, the patient was sent to a center with cardiac and vascular surgery for the continuation of the conservative versus endovascular/open medical treatment course.

Table 1. Haematochemical exams.

White blood cell	10.9/mmc
Haemoglobin	13.8 g/dl
Platelets	371000/mmc
Urea	34.5 mg/dl
Creatinine	0.82 mg/dl
LDH	207 IU/L
CRP	0.65 mg/dl
D-dimer (FEU)	414 ng/ml
Troponin (0h)	13.5 ng/ml
Troponin (1h)	12.26 ng/ml

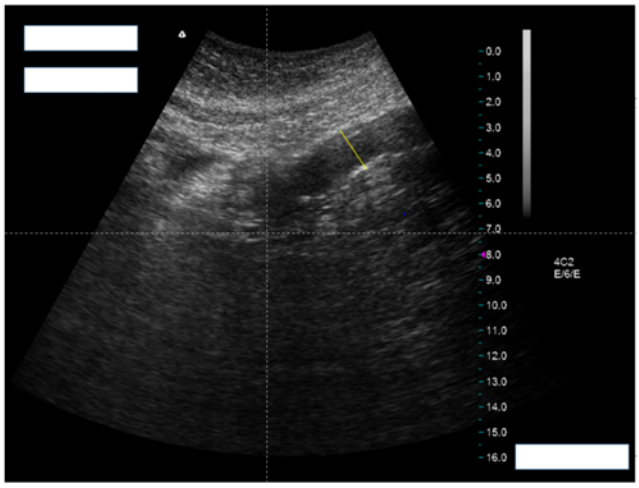

Figure 1. POCUS: aorta with normal course and diameter (21 millimeters).

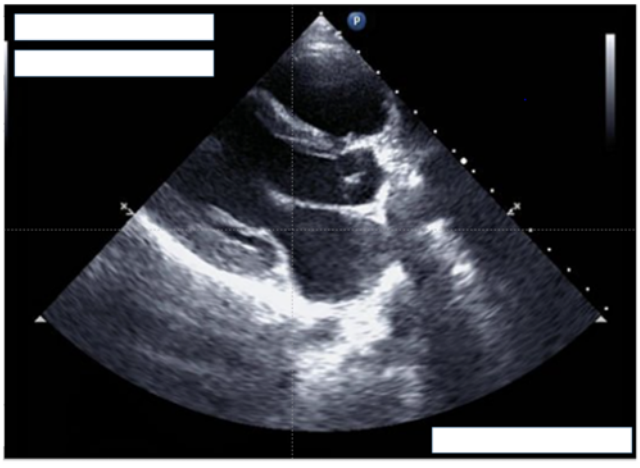

Figure 2. Cardiac echoscopy: no signs of dissection at aortic root level nor signs of aortic regurgitation.

Follow-up

In the light of type B of dissection and the absence of symptoms of hypoperfusion, after the emergency transfer of the patient to the HUB hospital, a conservative medical approach was chosen, initially through intravenous administration of labetalol for 72 hours, followed by gradual titration of oral antihypertensive therapy (Ramipril 5 mg bid, Doxazosin 2 mg bid, Nitroglycerin 15 mg 1/day, Nebivolol 5 mg die, Rosuvastatin 10 mg die, amlodipine 5 mg bid) with good control of blood pressure and planning close radiological follow-up.

Last evaluation with CT angiography (November 2024) revealed a markedly reduced hyperdensity of the thrombus in the pre-contrastographic phase (max thickness of about 6 mm on the left postero-lateral side of the descending thoracic aorta). Two small saccular regions of enhancement protruding from the aortic lumen in the context of the previous intramural haematoma, one on the left lateral side, the other on the right postero-lateral side (max. 8 mm at this level, where it causes small bulging of the media and the adventitia above) were observed: these formations are compatible with ULPs (ulcer like projections) of the intima. There is mild contrastographic enhancement of the adjacent thrombus, with hyperemia of the adventitial wall. Clinical vascular surgical follow-up is recommended.

Discussion

Type B aortic dissection according to Stanford's classification accounts for approximately 25-30% of acute aortic syndromes with a hospital mortality rate of medical treated patients of approximately 10% and the 3-year survival rate is 78% for those treated with medical management alone.[10,11] Chest pain accounts for 5-9% of admissions to the emergency department, and early detection and subsequent definition of the diagnostic-therapeutic course becomes of primary importance. We decided to present this case in order to emphasize the need for a rational use of diagnostic insights and decision-making algorithms. We focused, especially, on the use and interpretation of the D-dimer for diagnostic rule-out; a role highlighted in the EACTS/STS LG 2024: The D-dimer blood test is extremely helpful in the emergency setting. If the D-dimer is negative, the patient does not have an aortic dissection.[3] Indeed, circulating D-dimer levels increase in most patients with AAS.[12] Conversely, low levels of D-dimer argue against AAS, most strongly in patients at low PTP (Pre Test Probability), potentially allowing rule-out without further tests.

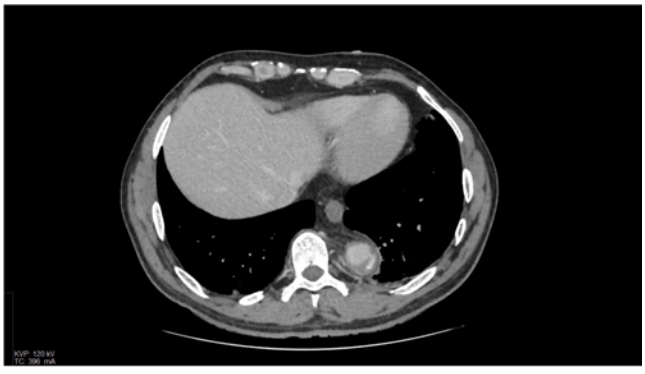

Figure 3. CT angiography: arterial blush of 1.5 cm compatible with the lesion of the intima in a Stanford B-type aortic dissection.

However, although D-dimer is a high-sensitivity test, it cannot be used alone to rule out acute aortic syndrome, as demonstrated by this case. In recent years, several studies have highlighted that in patients with low Pre-Test Probability (PTP), the integration of the Aortic Dissection Detection Risk Score (ADD-RS) and D-dimer testing can safely rule out acute aortic syndrome (AAS) with high sensitivity. Pooled sensitivity was 99.9% (95% confidence interval [CI]: 99.3% to 100%, $I^2 = 0$) for ADD-RS = 0 combined with D-dimer < 500 ng/mL or age-adjusted D-dimer; 98.9% (95% CI: 97.9% to 99.9%, $I^2 = 0$) and and LR- 0.02 for ADD-RS ≤ 1 and D-dimer < 500 ng/mL as in the present case.[13]

Furthermore, the diagnostic accuracy of integrating POCUS with pre-test probability (PTP) and D-dimer testing has also been investigated. This integrated approach has been shown to safely rule out Acute Aortic Syndrome (AAS): a low POCUS-integrated PTP combined with a D-dimer < 500 ng/mL yielded a sensitivity of 100% (95% CI: 97.9-100), while an ADD-RS ≤ 1 combined with a D-dimer < 500 ng/mL showed a sensitivity of 98.8% (95% CI: 97.1-100).[14]

However, this case highlights the need to avoid relying solely on the diagnostic accuracy of scoring systems and their combinations. It underscores the crucial importance of a multiparametric evaluation that incorporates laboratory findings, imaging results, and clinical presentation. Clinical assessment should take into account the nature of the pain, its temporal progression, and the patient's response to therapy.

Furthermore, it is worth highlighting as a potential subject of further investigation the temporal correlation between the onset of symptoms and the timing of D-dimer measurement. In this particular instance, the blood sample was taken five hours after symptom onset and was not subsequently repeated.

In this case, the absence of cardiovascular risk factors, negative initial test results, and low diagnostic scores led to a delayed clinical suspicion. The pre-test risk was low, and D-dimer was negative, which initially supported the decision to forgo further diagnostic imaging such as Computed Tomography Angiography (CTA).

Only the "human factor" understood as instinct mixed with experience made it possible to reach the correct diagnosis by means of advanced diagnostics, thus underlining the importance of always considering serious pathologies in the presence of intense and persistent chest pain, even in the absence of instrumental and above all laboratory alterations; highlighting how the presentation of aortic dissecative disease can be very varied and "clinically subdued".

Conclusions

This case emphasizes the importance of a comprehensive clinical evaluation, semeiotics and view of the patient as a whole regardless of laboratory findings, literature data and diagnostic scores. The latter, although necessary and of fundamental support, to date, cannot replace the clinic. As our fathers taught us, the clinic remains Queen.

References

1. Aboyans V, Boukhris M. Dissecting the epidemiology of aortic dissection. Eur Heart J Acute Cardiovasc Care 2021;10:710-1.
2. Pacini D, Di Marco L, Fortuna D, et al. Acute aortic dissection:

epidemiology and outcomes. Int J Cardiol 2013;167:2806–12.
3. EACTS/STS guidelines for diagnosing and treating acute and chronic syndromes of the aortic organ. Ann Thorac Surg 2024;118:5-115.
4. ESC Scientific Document Group. 2024 ESC Guidelines for the management of peripheral arterial and aortic diseases. Eur Heart J 2024;45:3538-7005.
5. Mahler SA, Riley RF, Hiestand BC, et al. The HEART Pathway randomized trial: identifying emergency department patients with acute chest pain for early discharge. Circ Cardiovasc Qual Outcomes 2015;8:195-203.
6. Anand SS, Wells PS, Hunt D et al. Does this patient have deep vein thrombosis? JAMA 1998;279:1094-9. Erratum in: JAMA 1998;280:328. Erratum in: JAMA 1998;279:1614
7. Le Gal G, Righini M, Roy PM, et al. Prediction of pulmonary embolism in the emergency department: the revised Geneva score. Ann Intern Med 2006;144:165-71.
8. Van der Hulle T, Cheung WY, Kooij S et al. Simplified diagnostic management of suspected pulmonary embolism (the YEARS study): a prospective, multicentre, cohort study. Lancet 2017;390:289-97. Erratum in: Lancet 2017;390:230.
9. Nazerian P, Mueller C, Soeiro AM et al. Diagnostic accuracy of the aortic dissection detection risk score plus d-dimer for acute aortic syndromes: the advised prospective Multicenter Study. Circulation 2018;137:250-8.
10. Tsai TT, Nienaber CA, Eagle KA. Acute aortic syndromes. Circulation 2005;112:3802-813.
11. Evangelista A, Isselbacher EM, Bossone E, et al. Insights from the international registry of acute aortic dissection: a 20-year experience of collaborative clinical research. Circulation 2018;137:1846-60.
12. Watanabe H, Horita N, Shibata Y, et al. Diagnostic test accuracy of D-dimer for acute aortic syndrome: systematic review and metaanalysis of 22 studies with 5000 subjects. Sci Rep 2016;6:26893.
13. Bima P, Pivetta E, Nazerian P, et al. Systematic review of aortic dissection detection risk score plus D-dimer for diagnostic rule-out of suspected acute aortic syndromes. Acad Emerg Med 2020;27:1013–27.
14. Morello F, Bima P, Castelli M, et al. Diagnosis of acute aortic syndromes with ultrasound and d-dimer: the PROFUNDUS study. Eur J Intern Med 2024;128:94-103.