

# Aortic dissection presenting with respiratory failure: case report and literature

Simone Gherardi,<sup>1</sup> Renato Ippolito,<sup>2</sup> Stefano Pasqualin,<sup>3</sup> Vito Cianci,<sup>2</sup> Matteo Paganini<sup>4</sup>

<sup>1</sup>Emergency Department and Emergency Medical Service, “San Bassiano” Hospital, ULSS 7 “Pedemontana”, Bassano del Grappa;

<sup>2</sup>Emergency Department, University Hospital of Padova; <sup>3</sup>Emergency Medicine Residency Program – Department of Medicine (DIMED), University of Padova; <sup>4</sup>Department of Biomedical Sciences, University of Padova, Italy

Correspondence: Simone Gherardi, Emergency Department and Emergency Medical Service, “San Bassiano” Hospital, ULSS 7 “Pedemontana”, via dei Lotti 40, Bassano del Grappa (VI), Italy.

Tel.: +39.0424.888404.

E-mail: simone.gherardi@aulss7.veneto.it

Key words: aortic dissection; aortic aneurysm; aortic rupture; emergency medicine.

Contributions: RI and SG separately reviewed the literature about aortic dissection and chose the articles to include. SP wrote the case report section, and SG wrote the scoping review section; MP and VC reviewed and finalized the paper draft. All the authors have read and approved the final version of the manuscript and agreed to be held accountable for all aspects of the work.

Conflict of interest: the authors declare no potential conflict of interest.

Funding: none.

Ethics approval and informed consent: as no personal data on the patient concerned are available and the case is presented for educational purposes only, no formal approval was sought from the local ethics committee.

Availability of data and materials: all data generated or analyzed during this study are included in this published article.

Acknowledgments: we thank the Radiology Department of the Hospital of Padova for providing the case images and the Department of Cardiothoracic Surgery for informing us about the patient’s clinical course. We thank all the staff on duty in the Emergency Department for their commitment.

The patient had passed away at the time the paper was submitted. Even though not formally required (see ethic approval), we sought her relatives’ permission to publish the case; we’re grateful to them for letting us share this case with the emergency medicine community to advance knowledge.

Received: 10 May 2024.

Accepted: 31 July 2024.

Early view: 2 September 2024.

This work is licensed under a Creative Commons Attribution 4.0 License (by-nc 4.0).

©Copyright: the Author(s), 2024

Licensee PAGEPress, Italy

Emergency Care Journal 2024; 20:12633

doi:10.4081/ecj.2024.12633

*Publisher's note: all claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article or claim that may be made by its manufacturer is not guaranteed or endorsed by the publisher.*

## Abstract

Due to its nonspecific symptoms, in several cases, Acute Aortic Dissection (AAD) is a difficult-to-diagnose urgent condition. The torn intimal layer initiates a false lumen, which can potentially propagate and cause life-threatening ruptures or organ ischemia. Intramural hematoma can rarely develop inside the false lumen, resulting in compression of surrounding structures and worsening the patient’s prognosis. We report a case of AAD - Stanford Type A complicated by an intramural hematoma compressing the pulmonary artery, accompanied by a discussion of relevant literature. This patient presented to the emergency department with acute respiratory distress; clinical and initial diagnostic findings mimicked pulmonary embolism. However, a systematic differential evaluation supported by point-of-care ultrasound allowed a prompt diagnosis of AAD and avoided fixation errors.

## Highlights

- Acute aortic dissection is a difficult-to-diagnose, must-not-miss condition in emergency medicine.
- Intramural hematoma is a rare but worrisome complication of aortic dissection, potentially compressing surrounding structures and worsening the patient’s prognosis.
- Rarely acute aortic dissection can present with shortness of breath. This presentation can be misleading and can be associated with worse prognosis.
- A case of Stanford A aortic dissection presenting with respiratory distress and mimicking pulmonary embolism – due to an intramural hematoma compressing the pulmonary artery – is reported here, along with relevant literature.

## Introduction

Acute Aortic Dissection (AAD) is a serious, difficult-to-diagnose medical emergency.<sup>1,2</sup>

Caused by traumatic injury or predisposed by a series of conditions (e.g., connective tissue disorders, hypertension, pre-existing aortic aneurysm, pregnancy, vasculitis, or atherosclerosis), AAD starts from a tear of the intimal layer which creates a false lumen within the aortic wall. As the most frequent acute aortic syndrome (85-95%),<sup>3</sup> the pathophysiology of AAD entails proximal or distal propagation of the false lumen, potentially resulting in complications such as organ ischemia – due to compression of the true lumen or branch vessel involvement and obliteration – or life-threatening ruptures. Treatments range from non-invasive medical

support to surgical management, according to the severity of the case. Symptoms of AAD vary from the classical severe chest and/or back pain with sudden or “tearing” features to atypical presentations; the absence of classical signs and symptoms characterize less than 5%.<sup>4</sup> Intramural hematoma is associated with a worse prognosis than AAD alone.<sup>5</sup>

### An overview of Acute Aortic Dissection

Chest pain is the most common symptom reported in patients affected by AAD,<sup>6</sup> but subtle or uncommon symptoms have been described as well.<sup>7</sup> A retrospective cohort study conducted in the UK suggests that emergency physicians initially consider AAD in the differential diagnosis but finally exclude it as symptoms resolve, especially in atypical presentations.<sup>8</sup> Overall, acute chest pain can potentially represent the manifestation of serious medical conditions but, unfortunately, is one of the most common reasons to attend the Emergency Department (ED). Chest pain accounts for approximately 10% of non-injury-related visits, with an incidence of 8-19 per 1000 person-years - higher in urban than rural hospitals - with a mean age of 52-61 years and mostly men (49-57%).<sup>7</sup> It is, therefore, tempting for the emergency medicine practitioner to take advantage of heuristic shortcuts, missing atypical manifestations among such a numerous and undifferentiated group.

According to the DASHED study,<sup>2</sup> of all the patients presenting in the United Kingdom’s EDs with potential AAD symptoms, only 0.3% have an acute aortic syndrome.

Some AADs are painless, presenting with syncope or acute neurological symptoms, usually affecting younger patients with fewer comorbidities. For these reasons, Usui *et al.* suggested that stroke patients should always have a Computed Tomography (CT) full-aortogram.<sup>9,10</sup> Among other possible findings, AADs can present with hypertension (especially in Stanford type B), while pulse deficits or asymmetry are uncommon. More than 20% of patients with confirmed AAD lacked abnormalities of the mediastinum or aortic contour on chest X-rays.<sup>6</sup> However, recent data showed that, when AAD was taken into differentials, ED physicians were falsely reassured by clinical findings such as symmetrical upper limbs blood pressures and radial pulses or normal appearances of the mediastinal contour on chest X-rays.<sup>11,12</sup>

Electrocardiogram (ECG) is inaccurate in the diagnosis of AAD. When altered, signs of myocardial ischemia or criteria for ventricular hypertrophy can bias the interpretation of the underlying disease. Specifically, in 5% of AADs - Stanford type A, signs of acute myocardial infarction have been described, caused by the dissection’s propagation back to a coronary ostium and the consequent compression of the proximal coronary artery exerted by an expanding false lumen. Since aortic dissection flaps usually originate from the right-anterior aspect of the ascending aorta, above the right coronary sinus, the right coronary artery is typically involved, therefore manifesting with an inferior ST-Elevation Myocardial Infarction (STEMI) pattern.<sup>13</sup> Isolated anterior STEMI drops the likelihood of aortic dissection but does not eliminate it.<sup>14</sup> Such variability also depends on the predominance pattern of coronary arteries, which is mostly a right pattern.

Among laboratory testing, D-dimer is promising, but supporting evidence is conflicting.<sup>2</sup>

Suzuki *et al.* suggested a possible role of D-dimer in risk-stratifying patients within the first 24 hours after symptom onset.<sup>15</sup> According to the ADVISED Prospective Multicenter Study, among patients presenting with D-dimer <500 ng/mL and an Aortic Dissection Detection (ADD) risk score of 0, 1 over 300 is missed, whereas with an ADD risk score >1, 4% of dissections are

missed.<sup>16</sup>

Many clinical decision tools are available for the ruling in/out of AAD, but none of them has been proven to be superior to clinical gestalt.<sup>2</sup>

Point-of-Care Ultrasound (POCUS) is currently fundamental in evaluating the undifferentiated patient in the ED,<sup>17</sup> and has dramatically changed the management of AAD patients.<sup>18</sup> In some series, the sensitivity of this tool in the diagnosis of ascending aortic dissection was reported to be 78-90% but only 31-55% in descending aortic dissection; similarly, specificity for Stanford type A aortic dissection ranged between 87 and 96%, and was lower for type B (60-83%).<sup>19</sup> The advantages of POCUS are the absence of radiation, a fast learning curve, the capacity to rapidly answer clinical questions, and the opportunity to perform the exam at the patient’s bedside or even in the prehospital arena. Among the different protocols suggested in emergency medicine, the Sonographic Protocol for the Emergent Evaluation of aortic Dissection (SPEED) is specifically meant for diagnosing acute aortic dissection, and it has been recently tested in a prospective observational study.<sup>20</sup> The Rapid Ultrasound for Shock and Hypotension (RUSH) protocol includes the visualization of the aortic root in the Parasternal Long Axis (PLAX) view and abdominal aorta, thus seems helpful in evaluating potential AAD.<sup>21</sup> Therefore, to improve the assessment of the aorta, its other segments should be visualized in the remaining Transthoracic Echocardiography (TTE) projections and completed with a trans-abdominal window extending the assessment to the abdominal aorta for the construction of a sort of aortic map in 4 points.<sup>19</sup> Moreover, POCUS allows the evaluation of other signs, such as pericardial effusions or aortic regurgitation, making differential diagnosis easier and faster. Transesophageal Echocardiography (TEE) is even better in diagnosing AAD, approaching 100% sensitivity and specificity in some series, but it is not routinely available in the ED.<sup>22,23</sup>

Finally, a CT angiogram of the aorta is currently considered the gold-standard diagnostic test for aortic dissection.<sup>1,24</sup>

### Materials and Methods

We report a case of AAD with intramural hematoma causing compression of the pulmonary artery and consequent pulmonary blood flow obstruction mimicking massive pulmonary embolism. The ultimate goal is to describe the peculiarities of this patient’s clinical presentation and first assessment to increase awareness by disseminating it among emergency medicine practitioners.

In the second part, we summarize the published literature about this rare condition, discussing the complexities of managing undifferentiated patients presenting to the ED complaining of dyspnea or chest pain.

A scoping review was performed through a search on Google Scholar or Pubmed for publications about aortic dissection with intramural hematoma compressing the pulmonary artery. The following keywords were searched in titles and abstracts: aortic dissection; acute aortic dissection; aortic syndrome; hypoxia; dyspnea; intramural hematoma; and pulmonary artery. Only articles with available full texts were included. Two authors independently screened titles and abstracts of all potential studies for inclusion, seeking a third opinion in case of conflict.

### The case

An 80-year-old woman was transported by ambulance to the

ED of the Azienda Ospedaliera-Università di Padova, complaining of acute chest pain, respiratory distress, nausea, vomiting, and right leg pain.

Her medical history was relevant for hypertension, hypothyroidism, and previous breast and kidney cancers in regular follow-up after remission. Daily therapy consisted of acetylsalicylic acid, levothyroxine, and an Angiotensin-Converting Enzyme (ACE) inhibitor.

After triage, the patient was promptly placed under complete monitoring, showing Peripheral Oxygen Saturation (SpO<sub>2</sub>) of 88% in room air; respiratory rate of 36 breaths per minute; non-invasive blood pressure of 170/80 mmHg; heart rate of 80 bpm, sinus rhythm. Oxygen was administered through a non-rebreather mask, slightly reducing respiratory rate and improving SpO<sub>2</sub> to 98%. Cardiac and pulmonary examinations were unremarkable. Interestingly, the right leg showed signs of hypoperfusion (temperature slightly colder and femoral and popliteal pulse strength severely reduced).

The ECG showed sinus rhythm with poor R wave progression in precordial leads but no alterations in depolarization or repolarization (Figure 1). An Arterial Blood Gas (ABG) analysis obtained immediately after the admission - before oxygen administration - was relevant for hypoxia and hypocapnia. POCUS demonstrated: a preserved lung sliding without pleural alterations or effusion; a collapsing inferior vena cava; no pericardial effusion; a hypertrophic left ventricle and both left and right chambers without signs of acute strain or wall motion abnormalities. Despite an aortic bulb diameter within the normal range, the parasternal long-axis view (supplemental material) of the heart showed an enlarged ascending aorta (46 mm) containing an intimal flap that continued through the aortic arch and the abdominal aorta. Compression ultrasound of lower extremity veins was normal, with the right femoral artery's flow present but reduced in amplitude compared

to the contralateral.

Intravenous analgesic therapy with 0.1 mg fentanyl and antiemetic treatment with 10 mg metoclopramide were administered, achieving a reduction of symptoms. A CT angiogram acquired from the supra-aortic to the iliac vessels confirmed a Type A aortic dissection extending from the valvular plane to the first tract of the common left carotid artery, left subclavian artery, down to both iliac arteries, with occlusion of the right iliac artery. Of note, the CT angiogram documented a hematoma of the aortic wall compressing the pulmonary artery lumen, with signs of active bleeding inside the wall of the ascending aorta. No lung and abdominal organ alterations were detected (Figure 2).

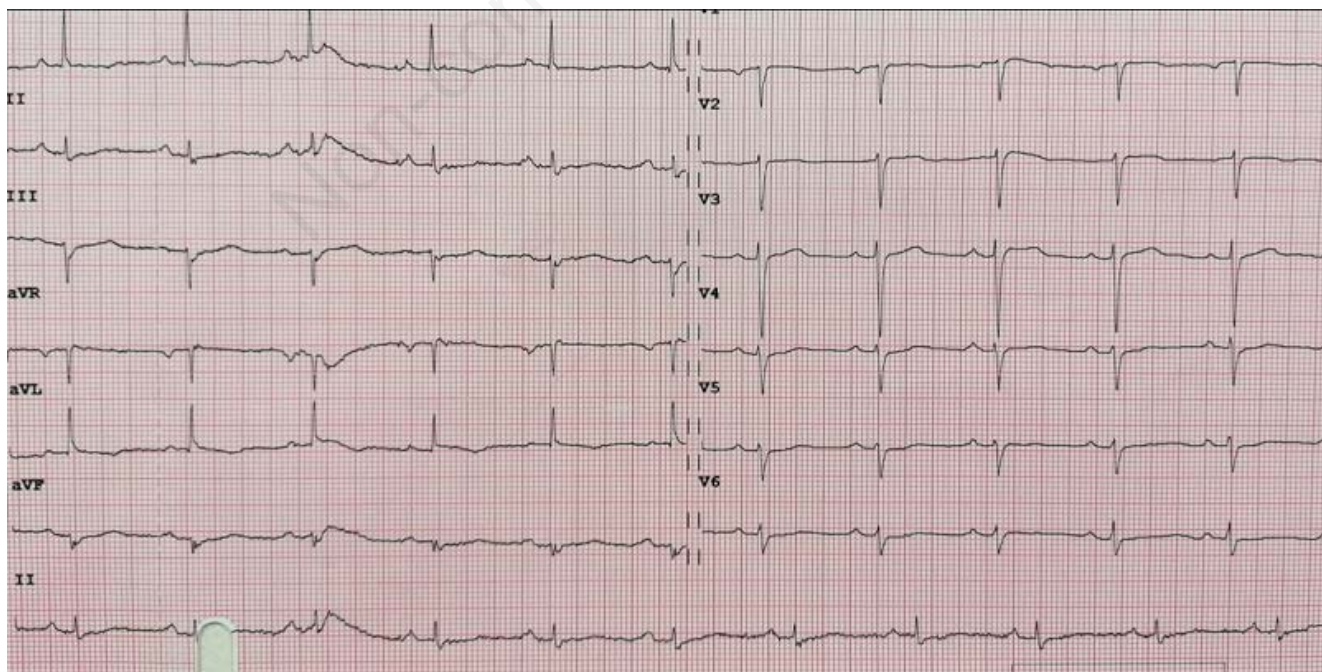
The elevated blood pressure was treated with a continuous infusion of labetalol and gradually titrated to achieve an invasively monitored arterial pressure of 120/60 mmHg. The patient was directly admitted to the cardiothoracic surgery operating room, where the ascending aorta was successfully replaced with an aortic prosthesis. After 14 days of admission, the patient was transferred to a cardiologic rehabilitation center.

## Results

After the literature search, we found one case series of 4 patients, 18 case reports, and a radiology retrospective cohort study focusing on the radiologic features (*Supplementary Table 1*).<sup>25-44</sup>

Only one article was published in an emergency medicine thematic journal,<sup>40</sup> while most were published in radiology or cardiothoracic surgery journals. Among the 22 described cases, chest pain was the most prevalent symptom, usually radiating to the back (17 patients). In this subset of patients, dyspnea was a fairly common symptom (9 patients).<sup>25,26,29,33-35,38,39,43</sup>

In 9 patients, the diagnosis was achieved with echocardiogra-



**Figure 1.** Patient's Electrocardiogram (ECG) at the presentation in the Emergency Department (ED). Poor R wave progression in the precordial leads could be associated with right ventricle overload.

phy (one with TEE), with echocardiographic findings reported. Specifically, pulmonary hypertension has been described; most patients presented with a Pulmonary Artery Systolic Pressure (PASP) greater than 70 mmHg. In particular, Dong *et al.* reported a PASP of 75 mmHg,<sup>45</sup> Sheu *et al.* 70 mmHg,<sup>30</sup> Can *et al.* 80 mmHg,<sup>33</sup> Okiwelu *et al.* 64 mmHg.<sup>36</sup> Podbregar reported a transtricuspidal gradient of 45 mmHg.<sup>34</sup> Gros-Gean reported 2 cases with normal-sized right-side chambers<sup>44</sup> and a case with an enlarged right ventricle at the CT scan. Masuo *et al.* reported elevated right ventricular pressure on right cardiac catheterization.<sup>25</sup> On ECG, only 3 cases reported ST abnormalities, 6 patients were tachycardic; none of the retrieved cases met STEMI criteria, nor were clear signs of right-side overload noted. D-dimer was reported to be >500 mg/L in 4 cases and normal in 2 cases. On chest X-ray, six cases reported a widened mediastinum.

## Discussion

The peculiarities of this case can be summarized in two overlapping features of AAD diagnosis in emergency medicine: its subtle nature and the importance of rule-out/rule-in clinical reasoning.

First, a patient presenting with respiratory distress could have diverted the attention of the treating team towards a “respiratory problem”, prompting a totally different case processing than that of an AAD. That could have led to inappropriate treatment or diagnostics, such as a contrast-enhanced CT scan for pulmonary embolism only, whose prevalence is higher than that of AAD and which has a presentation matching the clinical aspect and the arterial blood gas analysis. Considering that – in many centers – double rule-out CT scans for AAD and pulmonary embolism are not routinely performed, this fixation error could have led to a missed diagnosis. However, the presence of clinical signs con-

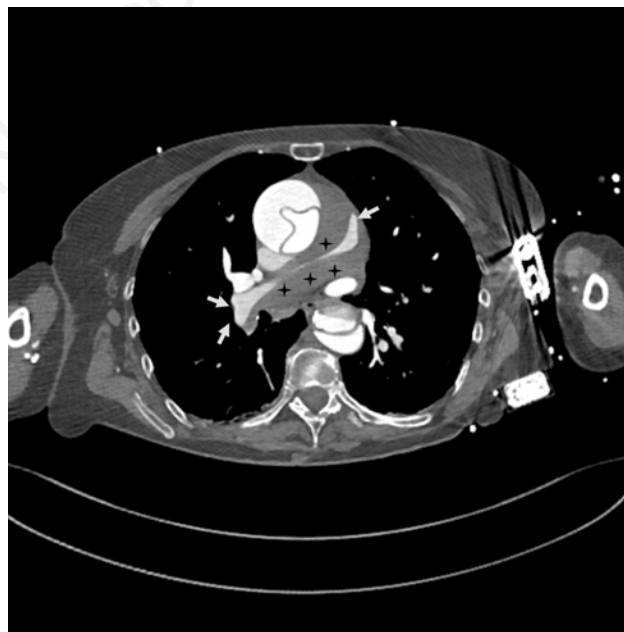
flicting with the suspect of pulmonary embolism and the ultrasound-integrated approach created an alternative hypothesis, which ultimately led to a correct and timely diagnosis.

Second, this case represents how fast thinking alone or pure heuristic reasoning, without an analytical re-check (slow thinking), could have perpetrated fixation errors and potentially translated into diagnostic delays in front of a masked medical emergency.<sup>46</sup>

Instead, a second reassessment of the patient’s clinical picture, challenging the initial suspected diagnoses, allowed the team to reveal an unexpected AAD.

Intramural hematoma is an infrequent complication of AADs, in which blood penetrates along the connective sheath or in the adventitia of the aorta. It has been reported as an independent risk factor for death.<sup>6,15</sup> Given the limited number of patients retrieved and the lack of guidelines specific to AADs with intramural hematoma compressing the pulmonary artery, it is difficult to elaborate valid recommendations; however, some suggestions can be abstracted from the present and the collected cases. In particular, while keeping emergency medicine reasoning accurate but fast, atypical conditions should be considered when the clinical picture and diagnostic data are conflicting, as demonstrated by the reasoning described in this case. Dyspnea was found as the most common presentation symptom within the collected cases, which is atypical for acute aortic syndromes, as is compression of the pulmonary artery by an intramural hematoma. In the broader context of AADs, dyspnea can be justified by acute aortic regurgitation, potentially present in Stanford type A dissections<sup>47</sup> and leading (if severe) to elevated left ventricle preload and filling pressure and eventually to acute heart failure. A respiratory compromise could also derive from cardiogenic shock – due to cardiac ischemia after coronary occlusion – or rare aortopulmonary artery fistula causing acute right heart failure.<sup>48</sup>

Instead, among the collected cases, only one had a Stanford type B dissection, although current literature reports that intra-



**Figure 2.** Computed Tomography (CT) aortogram performed in the emergency department. Note that the pulmonary artery (arrows) is compressed by the extravasation of blood (stars) from the dissected ascending aorta.

mural hematomas are more prevalent in this subtype of AADs (50-85%).<sup>49</sup> This discrepancy could be explained by the anatomic location of intramural hematoma compressing the pulmonary artery, which could happen more frequently in Stanford type A due to anatomical proximity. An intramural hematoma retrogradely propagating from a Stanford type B is unlikely to bypass the supra-aortic vessel roots and compress the pulmonary artery, and the summarized literature supports this hypothesis. Another explanation is the common adventitia at the root of the great vessels, anatomically shared by the ascending aorta and the pulmonary trunk.

Consequently, in Stanford type A aortic dissections, pulmonary arterial vessels can be involved in the process with resulting hypoxia and hemodynamic compromise, making the patient vulnerable to invasive procedures such as tracheal intubation and positive pressure ventilation or surgical repair of the aorta. Under this anatomic condition, blood from the dissected ascending aorta can extend beneath the adventitia of the main pulmonary artery and cross the barrier of the pulmonary hilum, potentially dissecting the broncho-vascular sheaths and reaching the pulmonary interstitium and alveoli. The accumulating blood may narrow the lumen of the pulmonary vessels because of their low pressure.<sup>11</sup> Consequently, hypoxia can be generated by blood extravasated inside alveoli or bronchial airways from ruptured pulmonary vessels, which could cause right to left intrapulmonary shunt. The “diffuse alveolar hemorrhage pattern” is the deadliest category, according to Sueyoshi *et al.*<sup>31</sup> Another explanation for this atypical dyspnea is pulmonary blood flow obstruction caused by intramural hematoma exerting its pressure on pulmonary arteries.<sup>50</sup> D-dimer dosage was reported only in 6 cases; in 2 of them, it turned out <500 mg/L, therefore underscoring the inconsistency of such parameter alone in posing the suspect or excluding intramural hematoma within AADs. Anyway, if pulmonary embolism is included in the differential of cases presenting with acute right heart failure and the described clinical picture, it is reasonable to think that intramural hematoma compressing the pulmonary artery could have lower D-dimer values than those found in pulmonary embolisms causing acute right ventricular strain. Also, acute pulmonary hypertension (*e.g.*, pulmonary embolism) typically manifests with a PASP <60 mmHg.<sup>51,52</sup> Where reported, in our series, we found severely higher PASP values, probably due to different mechanisms causing the acute obstruction, happening from outside the vessels in case of intramural hematoma. Anyway, it is possible that some of the patients included in these reports had different degrees of pulmonary hypertension, thus limiting the value of this finding.

Regarding the outcome, nine patients (out of 22) survived the acute phase, but real mortality can be underestimated due to potential complications affecting patients treated for AADs and publication bias.

## Conclusions

As an atypical manifestation, AAD developing an intramural hematoma compressing the pulmonary artery is a subtle but potentially deadly condition.

Findings like shortness of breath or respiratory failure –present in this case – could initially divert the clinician from considering acute aortic pathologies. Signs of right ventricle overload found with POCUS could also support an incorrect suspect of pulmonary embolism.

Emergency clinicians must know that POCUS can effectively

guide the diagnostic approach in life-threatening conditions.<sup>53</sup>

However, conflicting clinical picture and diagnostics results should prompt further evaluations for atypical and “must-not-miss” pathologies in emergency medicine.

## References

1. Isselbacher EM, Preventza O, Hamilton Black J, et al. 2022 ACC/AHA Guideline for the diagnosis and management of aortic disease: a report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardio* 2022;80:e223-393.
2. McLatchie R, Reed MJ, Freeman N, et al. Diagnosis of Acute Aortic Syndrome in the Emergency Department ( DASHED ) study : an observational cohort study of people attending the emergency department with symptoms consistent with acute aortic syndrome. *Emerg Med J.* 2024;41:136-44.
3. Baliyan V, Parakh A, Prabhakar AM, Hedgire S. Acute aortic syndromes and aortic emergencies. *Cardiovasc Diagn Ther* 2018;8:S82-96.
4. Bossone E, Labounty TM, Eagle KA. Acute aortic syndromes: diagnosis and management, an update. *Eur Heart J* 2018;39:739-49.
5. 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.
6. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA* 2000;283:897-903.
7. Stepinska J, Lettino M, Ahrens I, et al. Diagnosis and risk stratification of chest pain patients in the emergency department: focus on acute coronary syndromes. A position paper of the Acute Cardiovascular Care Association. *Eur Hear J Acute Cardiovasc Care* 2020;9:76-89.
8. Vercelli A, Berardi E, Poggiali E. Comments on “Why do emergency department clinicians miss acute aortic syndrome? A case series and descriptive analysis”. *Emerg Care J* 2023;19:11433.
9. Huang YC, Sung SF, Liu KT. Painless Acute Aortic Dissection may present as a stroke; risky markers that could be identified on hospital arrival. *J Acute Med* 2017;7:93.
10. Usui T, Suzuki K, Niinami H, Sakai S. Aortic dissection diagnosed on stroke computed tomography protocol: a case report. *J Med Case Rep* 2021;15:4-9.
11. Harris KM, Strauss CE, Eagle KA, et al. Correlates of delayed recognition and treatment of acute type a aortic dissection: the International Registry Of Acute Aortic Dissection (IRAD). *Circulation* 2011;124:1911-8.
12. McLatchie R, Wilson S, Reed MJ, et al. Why do emergency department clinicians miss acute aortic syndrome? A case series and descriptive analysis. *Emerg Care J* 2023;19:11153.
13. Sasaki S, Watanabe H, Shibayama K, et al. Three-dimensional transesophageal echocardiographic evaluation of coronary involvement in patients with Acute Type A Aortic Dissection. *J Am Soc Echocardiogr* 2013;26:837-45.
14. Kawahito K, Adachi H, Murata SI, et al. Coronary malperfusion due to Type A Aortic Dissection: mechanism and surgical management. *Ann Thorac Surg* 2003;76:1471-6.
15. Suzuki T, Distanto A, Zizza A, et al. Diagnosis of acute aortic

- dissection by D-dimer: the International Registry Of Acute Aortic Dissection substudy on Biomarkers (IRAD-bio) experience. *Circulation* 2009;119:2702-7.
16. Nazerian P, Mueller C, De Matos Soeiro A, 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.
  17. Wang Y, Yu H, Cao Y, Wan Z. Early screening for aortic dissection with Point-of-Care Ultrasound by emergency physicians: a prospective pilot study. *J Ultrasound Med* 2020;39:1309-15.
  18. Fojtik JP, Costantino TG, Dean AJ. The diagnosis of aortic dissection by emergency medicine ultrasound. *J Emerg Med* 2007;32:191-6.
  19. Evangelista A, Maldonado G, Gruosso D, et al. The current role of echocardiography in acute aortic syndrome. *Echo Res Pract* 2019;6:R53-63.
  20. Gibbons RC, Smith D, Feig R, et al. The Sonographic Protocol for the Emergent Evaluation of Aortic Dissections (SPEED protocol): a multicenter, prospective, observational study. *Acad Emerg Med* 2024;31:112-8.
  21. Ghane MR, Gharib MH, Ebrahimi A, et al. Accuracy of Rapid Ultrasound in Shock (RUSH) exam for diagnosis of shock in critically ill patients. *Trauma Mon* 2015;20:20095.
  22. Meredith EL, Masani ND. Echocardiography in the emergency assessment of acute aortic syndromes. *Eur J Echocardiogr* 2009;10:i31-9.
  23. Flachskampf FA, Wouters PF, Edvardsen T, et al. Recommendations for transoesophageal echocardiography: EACVI update 2014. *Eur Heart J - Cardiovasc Imag* 2014;15:353-65.
  24. Boileau C, Bossone E, Di Bartolomeo R, et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases. *Eur Heart J* 2014;35:2873-926.
  25. Masuo M, Takano H, Takamoto S, et al. Pulmonary artery obstruction caused by thoracic aortic dissection - a case with unique pathological findings. *Circ J* 2004;68:392-5.
  26. Dong HK, Sang WR, Choi YS, Ahn BH. Aortic dissection presenting with secondary pulmonary hypertension caused by compression of the pulmonary artery by dissecting hematoma: a case report. *Korean J Radiol* 2004;5:139-42.
  27. De Silva RJ, Hosseinpour R, Scream N, et al. Right pulmonary artery occlusion by an acute dissecting aneurysm of the ascending aorta. *J Cardiothorac Surg* 2006;1:1-3.
  28. Munín M, Goerner MS, Lombardero M, et al. Thickening of the pulmonary artery wall in acute intramural hematoma of the ascending aorta. *Cardiovasc Ultrasound* 2007;5:1-3.
  29. Sueyoshi E, Sakamoto I, Uetani M, et al. CT findings of ruptured intramural hematoma of the aorta extending along the pulmonary artery. *Cardiovasc Intervent Radiol* 2007;30:321-3.
  30. Sheu JJ, Fu M, Lee FY, et al. Ascending aortic dissecting aneurysm with isolated right pulmonary arterial medial intramural hematoma. *Ann Thorac Surg* 2008;85:2127-8.
  31. Sueyoshi E, Matsuoka Y, Sakamoto I, Uetani M. CT and clinical features of hemorrhage extending along the pulmonary artery due to ruptured aortic dissection. *Eur Radiol* 2009;19:1166-74.
  32. Inoue Y, Takahashi R, Kashima I, Tsutsumi K. Mediastinal hematoma: another lethal sign of aortic dissection. *Interact Cardiovasc Thorac Surg* 2009;8:275-6.
  33. Can M, Tanboga IH, Akgun T, et al. Type A intramural hematoma presenting with acute right heart failure caused by compression of the main pulmonary artery: a case report. *Ann Vasc Surg* 2009;23:535.e11-4.
  34. Podbregar M, Gabrijelčič T. Acute right heart overload due to pulmonary artery obstruction caused by ruptured aortic dissection. *Eur Heart J* 2011;32:517.
  35. Miele V, Merola MG, Roncacci A, Ianniello S. Pulmonary artery intramural hematoma associated with Stanford type B aortic dissection. *J Cardiovasc Comput Tomogr* 2015;9:466-8.
  36. Okiwelu N, Finn C, Vanden Driesen R, et al. Ruptured penetrating ulcer of the ascending aorta with pulmonary artery compression. *Asian Cardiovasc Thorac Ann* 2016;24:266-8.
  37. Wakakuri H, Hyodo H, Ohara T, Yasutake M. Perivascular hematoma along pulmonary artery complicating aortic dissection. *J Nippon Med Sch* 2016;83:182-3.
  38. Talla K, Dmytriw AA, Nguyen E. Acute Type A Aortic Dissection with mediastinal hematoma mimicking massive pulmonary embolus. *Int J Cardiovasc Imaging* 2017;33:259-60.
  39. Yamazaki S, Funakoshi H. Pulmonary artery stenosis due to aortic dissection. *Eur Heart J - Cardiovasc Imaging* 2019;20:1019.
  40. Chong WH, Saha BK, Wang C, Beegle S. Type A aortic dissection mimicking saddle pulmonary embolism on CT imaging. *J Am Coll Emerg Physicians Open* 2020;1:132.
  41. Bradette S, Béland M, Lalonde C. Pulmonary artery compression by hematoma, a rare but life-threatening complication of Stanford type A aortic dissection. *J Card Surg* 2020;35:2808-10.
  42. Warisawa T, Kotoku A, Miyauchi R, et al. Pulmonary artery occlusion due to abrupt pinhole rupture of aortic arch aneurysm. *JACC Cardiovasc Interv* 2021;14:e15-6.
  43. Hirata K, Ishimine T, Nakayama I, et al. Unilateral left pulmonary edema caused by contained rupture of the ascending aortic dissection. *Intern Med* 2021;60:751-3.
  44. Gros-Gean J, Lebecque O, Nchimi A, Vlad MM. Case series: pulmonary artery intramural hematoma in Stanford Type A Acute Aortic Dissection. *J Belgian Soc Radiol* 2021;105:34.
  45. Dong HK, Sang WR, Choi YS, Ahn BH. Aortic dissection presenting with secondary pulmonary hypertension caused by compression of the pulmonary artery by dissecting hematoma: a case report. *Korean J Radiol* 2004;5:139-42.
  46. Bedolla J, Pines JM. Cognitive biases and mitigation strategies in emergency diagnosis. In *Evidence-Based Emergency Care: Diagnostic Testing and Clinical Decision Rules, Third Edition*, Pines JM, Belollo F, Carpenter CR, Raja AS (eds.), John Wiley & Sons Ltd, 2023. Available from: <https://onlinelibrary.wiley.com/doi/abs/10.1002/9781119616870.ch60>
  47. Patel PA, Bavaria JE, Ghadimi K, et al. Aortic regurgitation in Acute Type-A Aortic Dissection: a clinical classification for the perioperative echocardiographer in the era of the functional aortic annulus. *J Cardiothorac Vasc Anesth* 2018;32:586-97.
  48. Kagiya N, Otsuki Y, Tsunoda M, Yoshitaka H. Multimodality imaging in aortic arch aneurysm ruptured into the pulmonary artery. *Ann Thorac Surg* 2019;107:e217.
  49. Kanaan T, Abdelrahman AS, Jaber J, et al. Type A aortic dissection with intramural hematoma: a challenging diagnosis. *Cureus* 2023;15:e33300.
  50. Plack D, Rehfeldt K, Nelson J, Kauss M. Pulmonary artery sheath hematoma. *Ann Card Anaesth* 2021;24:232.
  51. Konstantinides SV, Humbert M, Sian C, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). *Eur Heart J* 2020;41:543-603.

52. Humbert M, Hoeper MM, Berger RMF, et al. 2022 ESC / ERS Guidelines for the diagnosis and treatment of pulmonary hypertension developed by the task force for the diagnosis and treatment of (ESC) and the European Respiratory Society (ERS). *Eur Heart J* 2022;43:3618-731.
53. 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:S0953-6205(24)00234-6.

---

*Online Supplementary Materials*

*Supplementary Table 1. Retrieved papers.*

*Video. A conventional parasternal long axis view of the patient's heart. On the right of the screen the sino-tubular and ascending aorta. Note that the ascending aorta is enlarged and a mio-intimal dissection is present.*

Non-commercial use only