CASE REPORT

Use of heparin in aortic dissection: beware the misdiagnosis of acute pulmonary embolism

Yashashwi Sinha, Mahdi Saleh, Daniel Weinberg

Keele University Medical School, Stoke-on-Trent, UK

Correspondence to
Yash Sinha,
yashashwisinha@hotmail.com

SUMMARY

We present a case which highlights the diagnostic difficulties between a Stanford type A aortic dissection (AD) and a pulmonary embolism (PE) and the impact it has on subsequent management. A 75-year-old man presenting with chest pain, shortness of breath and dizziness was initially suspected of having a PE and started on low-molecular-weight-heparin (LMWH). The patient was correctly diagnosed afterwards with CT of the chest to have an aortic dissection. The detrimental use of LMWH may have caused a propagation of the dissection and delayed surgical intervention of an acutely life-threatening condition. When the diagnosis is unclear, the early use of CT can help differentiate AD from PE. This in turn can guide the management as well as the use of LMWH, which should be avoided until the correct diagnosis is confirmed.

BACKGROUND

Aortic dissection (AD) and pulmonary embolism (PE) are both serious conditions that present to the emergency department. AD is one of the most common emergencies involving the aorta. The incidence of AD has been reported to be between 2000 and 3000 new cases per year and it has an estimated 1–2% per hour death rate in the first 24 h.^{1 2} People with a type A AD, who manage to receive in-hospital treatment still have a high rate of mortality, despite surgical (27% mortality) and medical (56% mortality) management.¹

AD can be misdiagnosed at presentation in up to 38% of patients and can be painless in up to 15%.³ As there are no validated clinical diagnostic tools to diagnose an AD, it is a matter of clinical judgment as well as clinician experience. The diagnosis is made further challenging given the myriad of clinical presentations. The classic 'interscapular tearing thoracic back pain' is present in only 51% of patients.⁴

Similarly, acute PE may be present with or without the typical preceding signs of a deep venous thrombosis (DVT).⁵ Therefore, overlapping presentations must be taken into account when considering these two differentials of chest pain. In accordance with the National Institute for Health and Clinical Excellence (NICE) guidelines, clinicians should offer low-molecular-weight-heparin (LMWH) to treat confirmed cases of PE taking into account any contraindications.⁶

AD can be fatal if misdiagnosed as a PE. The two entities may present similarly, which makes it difficult to differentiate based on history and examination alone. Additionally, initial investigations may yield similar results such as an elevated D-dimer,

which can be misleading. However, a CT scan is an effective tool in distinguishing between the two pathologies with a 98% sensitivity and 95% specificity at diagnosing an AD.⁷

CASE PRESENTATION

A 75-year-old Caucasian gentleman had a sudden onset of chest pain with associated dizziness and severe shortness of breath (SOB) that had lasted for 6 h. The incident occurred while gardening and there was no history of trauma. He described it as the worst pain he had felt with a severity of 10/10. The pain did not radiate. Nothing alleviated or worsened the pain. The patient noted a similar, but far milder, episode occurring 6 months previously.

He had a background of benign prostatic hyperplasia, hypertension and gastro-oesophageal reflux disease. The patient was a non-smoker and non-drinker. He was a widower, lived alone and was independent with his activities of daily living. He was active and had a balanced diet. No relevant family history to note.

His drug history consisted of doxazosin, ramipril, amlodipine, bisoprolol fumerate, omeprazole and ranitidine.

On examination, the patient was alert. His Glasgow Coma Score was 15. He had normal heart sounds with no added sounds or murmurs. Jugular venous pressure was not raised. On auscultation of his lungs, he had good air entry and vesicular breath sounds bilaterally. His abdomen was soft, non-tender and bowel sounds were present. His vital signs were as follows: blood pressure 120/85, heart rate 92, respiratory rate 22, temperature 36.5°C and O₂ saturation of 90% on 60% oxygen.

INVESTIGATIONS

His routine blood tests demonstrated increased urea (12.4 mmol/l) and creatine (185 µmol/l), a low estimated glomerular filtration rate (eGFR) 31 mL/min and no other significant abnormalities. His arterial blood gases were within normal limits. His D-dimer was elevated at 720 ng/mL. ECG was regular with sinus rhythm. A transthoracic echocardiogram (ECHO) showed well-functioning right and left ventricles and no signs of pericardial effusion (see figure 1).

The patient was initially diagnosed with a PE, and LMWH was initiated. He was sent for a CT scan of his chest to confirm the diagnosis when an AD was noted and a Stanford type A AD was diagnosed instead. A CT pulmonary angiogram was performed to further assess the extent of the pathology and the LMWH was stopped (see figures 2 and 3).

To cite: Sinha Y, Saleh M, Weinberg D. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-009367

Standard Values		Indexed Values	
Standard Mode		LV	Normal Values
Ejection Fraction	[%]	82	56 - 78
Myocardial Mass ED	[g]	142.17	118 - 238
Stroke Volume	[ml]	122.59	51 - 133
ED Volume	[ml]	149.45	77 - 195
ES Volume	[ml]	26.86	19 - 72
Cursor Volume	[ml]	113.92	
Cardiac Output	[l/min]	8.09	2.82 - 8.82

Figure 1 Patient's echocardiogram results, detailing left-ventricular function.

On the CT scan, no filling defects were identified within the pulmonary arteries or their branches to suggest pulmonary emboli. There was a Stanford type A AD extending from the aortic root into the aortic arch and onwards along the entire length of the descending aorta and into the right common iliac artery. The flap involved the brachiocephalic artery and extended into the right common iliac artery. There was evidence of multiple foci of infarction in the right renal kidney because of the false lumen. No further features were present to suggest additional end-organ damage.

A renal ultrasound scan (USS) showed no evidence of significant decrease in blood flow to the kidneys because of stenosis.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis was a key step in the management of this patient and his triage in the emergency department. Given some cumulative factors, a diagnosis of PE was decided upon. Important differentials to be considered on presentation of acute chest pain with shortness of breath, and especially in this patient, were as follows:

- ▶ Acute coronary syndrome (ACS);
- ▶ Aortic dissection;
- ▶ Unstable or ruptured thoracic aneurysms.

TREATMENT

Surgery did not take place immediately and was delayed for 48 h to allow his renal function to improve and to reduce the impact of LMWH; this time became further prolonged due to the development of nosocomial pneumonia and a urinary tract infection.

Figure 2 Patient's CT scan showing the aortic dissection with the true lumen in the Patient's aortic arch.

Figure 3 Patient's CT scan showing the aortic dissection with the true lumen in the Patient's ascending and descending aorta.

OUTCOME AND FOLLOW-UP

The patient later stabilised; upon further discussion, it was decided that the patient was to be transferred to a specialist centre for his surgery.

DISCUSSION

The pathogenesis of AD is well established. An initiating event and a structural weakness of the arterial wall contribute to arterial intimal wall tearing.⁸ Several predisposing risk factors play an important role in the development of AD. The patient's risk factors included his arterial hypertension and old age. In this case, the underlying cause of the AD was most likely secondary to the patient's chronic hypertension. Once the tear had occurred, blood flow under arterial pressure dissected the tunica media creating a second 'false' lumen.

The retroperitoneal location of the aorta in addition to the tearing of intersecting nerve fibres in the intima results in severe chest pain with or without the classical interscapular pain.⁸ Additionally, during systole, the ascending aorta is exposed to maximal pressure fluctuations, and therefore maximal aortic expansion and relaxation increasing the risk of intimal tearing. Other symptoms that may accompany chest pain in AD, which were present in this case, include SOB (with elevated RR) and dizziness. This may be due to the effects of the false lumen and its impact on blood flow via the lungs.⁹

Similarly, acute PE may also present with chest pain accompanying SOB with an abrupt onset, chest pain (alone), fainting (or syncope) and haemoptysis in the order of decreasing frequency.⁵ Additional preceding signs and risk factors that help differentiate PE from AD clinically include unilateral swelling of the leg, calf tenderness, recent travel and obesity as well as a sedentary lifestyle. However, all or none of these signs and risk factors may be present in a patient with acute PE.¹⁰ Therefore, the overlap of symptoms that exist between these two pathologies must be appreciated and an index of high suspicion maintained for AD whenever a patient presents with an acute onset of chest pain and dyspnoea. Use of the 'Wells' score for estimating the probability of a PE can help diagnosis and guide management.⁶

The key to making the correct diagnosis in vague presentations, however, are repeated examinations and investigations that help delineate the two conditions. Simple examination findings such as the patient's vital signs that can be used to differentiate between AD and PE may not be helpful. A vital sign that often changes in acute AD is arterial BP; however, the patient was normotensive and this is the case in 50% of AD and as such, it is not always helpful diagnostically.¹¹

Table 1 Results from a literature search in PubMed for the term '(Aortic Dissection OR Aorta Dissection) AND (Pulmonary Embolism OR Pulmonary Embolus)' yielded 223 results, of which 10 articles had published cases of either misdiagnosis or dual pathology of an aortic dissection and pulmonary embolus at the time of presentation

Author(s)	Year	Feature of article	Reference
Halefoglu AM	2012	Aortic dissection misdiagnosed as PE. Correct diagnosis made on CT scan	18
Blanke P <i>et al</i>	2012	PE coexistent with aortic dissection. Correct diagnosis made on CT scan	19
Tomaszuk-Kazberuk A <i>et al</i>	2010	PE coexistent with aortic dissection. Correct diagnosis made on CT scan	20
Seguro <i>et al</i>	2009	PE coexistent with aortic dissection. Correct diagnosis made on CT scan	21
Stougiannos P N <i>et al</i>	2009	Aortic dissection misdiagnosed as PE. Correct diagnosis made on CT scan	22
Elmali M <i>et al</i>	2008	PE coexistent with aortic dissection. Correct diagnosis made on CT scan	23
Radwan K <i>et al</i>	2007	Aortic dissection misdiagnosed as ACS with PE. Correct diagnosis made on CT scan	24
Gupta R, <i>et al</i>	2003	PE coexistent with aortic dissection. Correct diagnosis made on CT scan	25
Thoongsuwan N and Stern EJ	2002	Retrospective case study on 130 patients with clinically suspected aortic dissection. PE misdiagnosed for aortic dissection in two cases. Correct diagnosis made on CT scan	26

PE, pulmonary embolism; ACS, acute coronary syndrome.

Performing a D-dimer test yields high false-positive rates for both conditions as fibrinolytic activity is high in pathologies where blood clot degradation occurs, but is a poor investigation for differentiating between AD and PE.¹² Data from the Prospective Investigation of Pulmonary Embolism Diagnosis study suggest that blood gas analysis are of insufficient discriminating value to rule out a diagnosis of PE.¹³

If clinical assessment and a resting ECG (or serial ECGs) are not suggestive of an ACS, NICE guidelines recommend considering other life-threatening acute conditions, specifically AD and PE.¹⁴ The ECG changes in acute AD are usually non-specific; however, 1–2% of patients have been noted to have ST-segment elevation.¹⁵ McGinn and White were the first to describe the association between PE and the S1Q3T3 pattern. Several other associations have been made since then, including a normal sinus tachycardia, complete and incomplete right-bundle-branch-block, axis changes, ST-segment and T-wave changes, P-pulmonale and atrial arrhythmias. Despite this, there is a low sensitivity of the ECG in diagnosing a PE.¹⁶

A transthoracic ECHO was used, but it has a much lower sensitivity and specificity for diagnosing AD compared with a transoesophageal ECHO, which may have identified the diagnosis at an earlier stage. A transoesophageal ECHO may be able to identify anatomic features and any signs of rupture related to the dissection in many cases.¹⁵ Guidelines from the British Thoracic Society suggest the use of ECHO in patients with major central PE to establish diagnosis and exclude other differentials. One of the main signs of PE on ECHO is right ventricular dilation and hypokinesis (McConnell's sign); however, ECHO changes only occur with significant obstruction to pulmonary circulation and small emboli may be missed.¹⁷

Recently, similar cases have shown that it is not uncommon to misdiagnose AD for an acute PE instead that the gold standard investigation to confirm diagnosis is CT (table 1).^{18–27} The use of CT as an investigation when in suspicion of an AD is also in line with NICE guidelines.¹⁴ CTPA may show filling defects in the pulmonary arteries when performed with angiography consistent with a PE, whereas a large and extending haematoma that is present in the aorta is more in line with AD. Additional useful findings, more diagnostic of AD, included the patient's urea, creatine and eGFR, which indicated renal damage, ultimately diagnosed as being due to extension of the false lumen over the renal artery.

According to NICE guidelines, the patient should have been offered LMWH or fondaparinux after confirmation of his PE.⁶ In terms of management, LMWH that is indicated in the

treatment of an acute PE is contraindicated in AD. Stanford type A dissections require urgent surgery as the dissection can move more proximally causing myocardial infarction. Naturally occurring thrombus formation in the false lumen, which may stop disease progression, can be inhibited by the accidental use of LMWH.²⁸ The use of LMWH in misdiagnosed cases may exacerbate and quicken the propagation of the false lumen due to its anticoagulant effects, which can be disastrous for the patient.²⁹

The delayed use of CT, the time taken for renal function normalisation alongside reducing the impact of LMWH, delayed the surgical intervention in this patient. This delay was prolonged by the acquirement of nosocomial infections. It is therefore crucial to arrive at the correct diagnosis in order to avoid the detrimental effects to the false lumen because of LMWH usage. Additionally, there should be a low-threshold for ordering an early CT of the chest in a patient with acute chest pain to rule out or diagnose important differentials of PE or AD.¹⁴

Learning points

- Early use of CT can help differentiate aortic dissection (AD) from pulmonary embolism (PE), when in doubt of the diagnosis.
- Low-molecular weight heparin should not be used until the diagnosis is confirmed with a CT.
- Appreciate the overlap of presentations between AD versus PE that may make diagnosis difficult.

Contributors Yashashwi Sinha has given the final approval of the submitted manuscript for which I take public responsibility. I collated the case and my coauthors helped me write it.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Mehta RH, Suzuki T, Hagan PG, *et al*. Predicting death in patients with acute type a aortic dissection. *Circulation* 2002;105:200–6.
- 2 Nienaber CA, Fattori R. Aortic diseases—do we need MR techniques? *Herz* 2000;25:331–41.

- 3 Spittell PC, Spittell JA Jr, Joyce JW, *et al.* Clinical features and differential diagnosis of aortic dissection: experience with 236 cases (1980 through 1990). *Mayo Clin Proc* 1993;68:642–51.
- 4 Pineault J, Ouimet D, Pichette V, *et al.* A case of an aortic dissection in a young adult: a refresher of the literature of this 'great masquerader'. *Int J Gen Med* 2011;4:889–93.
- 5 Miniati M, Cenci C, Monti S, *et al.* Clinical presentation of acute pulmonary embolism: survey of 800 cases. *PLoS One* 2012;7:e30891.
- 6 National Institute for Health and Clinical Excellence. *Venous thromboembolic disease: the management of venous thromboembolic diseases and the role of thrombophilia testing*. [CG144]. London: National Institute for Health and Clinical Excellence, 2012.
- 7 Nienaber CA, Eagle KA. Aortic dissection: new frontiers in diagnosis and management: Part II: therapeutic management and follow-up. *Circulation* 2003;108:772–8.
- 8 Pretre R, Von Segesser LK. Aortic dissection. *Lancet* 1997;349:1461–4.
- 9 Yasuda K, Nishibe T. [Pain characteristics in disease of heart and aorta]. *Nihon Rinsho* 2001;59:1727–32.
- 10 Abunasser J, Tejada JP, Foley RJ. The diagnosis and management of pulmonary embolism. *Conn Med* 2012;76:5–14.
- 11 Khan IA, Nair CK. Clinical, diagnostic, and management perspectives of aortic dissection. *Chest* 2002;122:311–28.
- 12 Brown MD, Rowe BH, Reeves MJ, *et al.* The accuracy of the enzyme-linked immunosorbent assay D-dimer test in the diagnosis of pulmonary embolism: a meta-analysis. *Ann Emerg Med* 2002;40:133–44.
- 13 Stein PD, Goldhaber SZ, Henry JW, *et al.* Arterial blood gas analysis in the assessment of suspected acute pulmonary embolism. *Chest* 1996;109:78–81.
- 14 National Institute for Health and Clinical Excellence. *Chest pain of recent onset: assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin*. [CG95]. London: National Institute for Health and Clinical Excellence, 2012.
- 15 Braverman AC. Acute aortic dissection: clinician update. *Circulation* 2010;122:184–8.
- 16 Todd K, Simpson CS, Redfearn DP, *et al.* ECG for the diagnosis of pulmonary embolism when conventional imaging cannot be utilized: a case report and review of the literature. *Indian Pacing Electrophysiol J.* 2009;9(5):268–75.
- 17 British Thoracic Society Standards of Care Committee. Suspected acute pulmonary embolism: a practical approach. *Thorax* 1997;52(Suppl 4):S1–24.
- 18 Halefoglu AM. CT Diagnosis of a thoracic aort aneurysm with type B aortic dissection clinically misdiagnosed as acute pulmonary embolism. *Case Report Med* 2012;2012:720394.
- 19 Blanke P, Apfaltrer P, Ebersberger U, *et al.* CT detection of pulmonary embolism and aortic dissection. *Cardiol Clin* 2012;30:103–16.
- 20 Tomaszuk-Kazberuk A, Sobkowicz B, Lewczuk A, *et al.* Silent pulmonary embolism in a patient with acute myocardial infarction and type B acute aortic dissection—a case report. *Adv Med Sci* 2010;55:99–102.
- 21 Seguro LF, Ogawa TM, Castelli JB. Case 6: 80-year-old woman, with arterial hypertension and chronic dissection of the aorta, presenting intense dyspnea and chest pain. *Arq Bras Cardiol* 2009;93:652–9, 701–8.
- 22 Stougiannos PN, Mytas DZ, Pyrgakis VN. The changing faces of aortic dissection: an unusual presentation mimicking pulmonary embolism. *BMJ Case Rep* 2009;2009:bcr2006104414.
- 23 Elmali M, Gulel O, Bahcivan M. Coexistence of pulmonary embolism, aortic dissection, and persistent left superior vena cava in the same patient. *J Cardiovasc Med (Hagerstown)* 2008;9:1180–1.
- 24 Radwan K, Peszek-Przybyla E, Gruszka A, *et al.* Acute aortic dissection imitating ST elevation myocardial infarction with accompanying pulmonary embolism. *Cardiol J* 2007;14:595–6.
- 25 Gupta R, Uhrbrock D, Birnbaum Y. Images in cardiology: coexisting pulmonary embolism and abdominal aortic dissection. *Clin Cardiol* 2003;26:395.
- 26 Thoongsuwan N, Stern EJ. Chest CT scanning for clinical suspected thoracic aortic dissection: beware the alternate diagnosis. *Emerg Radiol* 2002;9:257–61.
- 27 Oliver TB, Reid JH. Thoracic aortic dissection that wasn't: CT demonstration of probable paradoxical embolus secondary to unsuspected pulmonary embolus. *Br J Radiol* 1997;70:840–2.
- 28 Trimarchi S, Tolenaar JL, Jonker FH, *et al.* Importance of false lumen thrombosis in type B aortic dissection prognosis. *J Thorac Cardiovasc Surg* 2012; pii: S0022–5223:01489–4.
- 29 Thrumurthy SG, Karthikesalingam A, Patterson BO, *et al.* The diagnosis and management of aortic dissection. *BMJ* 2011;344:d8290.

Copyright 2013 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow