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Journal of Medical Case Reports and Case Series ISSN:2692-9880

Research Article Volume 4 Issue 01

Aortic Dissection Masquerading as Pulmonary Embolism: A Case Series

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Received date: 07 December 2022; **Accepted date:** 19 December 2022; **Published date:** 17 January 2023

Citation: Gartly R, Kalgaonkar S, Isles C (2023) Aortic Dissection Masquerading as Pulmonary Embolism: A Case Series. J Med Case Rep and Case Series 4(01): https://doi.org/10.38207/JCMPHR/2023/JAN04010304

Abstract

Background

To investigate how often aortic dissection (AD) may masquerade as pulmonary embolism (PE).

Methods

Retrospective cohort study. The electronic records of 998 consecutive patients who underwent CT imaging of their aorta between 2016 and 2021 were analyzed. Those with aortic dissection were then examined to determine if CTPA had been requested before CT aortography.

Results

Thirty-one patients had AD. Five (16 %) AD patients underwent CTPA as their initial imaging modality mainly because they presented with pleuriticpain. All five had radiological signs suggestive of a false lumen which was confirmed subsequently in each case by CT aortography. All five had type B dissections. Four of the five had no evidence of PE, while one had PE and AD.

Discussion

We have shown that a significant proportion of patients with AD were misdiagnosed as PE initially.

clinicians should be aware that aorticdissection may masquerade as pulmonary embolism.

Keywords: aortic dissection, pulmonary embolism, CT pulmonary angiography, CT aortography

Introduction

Acute aortic dissection (AD) is an important cause of cardiac death, with an incidence estimated to lie between 5 and 30 cases per million per year **[1]**. It occurs when an intimal tear allows blood to penetrate the aortic media or when the rupture of vessels within the media resultsin separating the aortic wall layers **[2]**. Intramural hematoma, thoughsometimes regarded as a separate entity, is usually considered a formof AD **[1]**. ADs are generally categorized as type A or type B based on the Stanford classification: Type A dissections involve the ascending aorta, whereas type B dissections do not, regardless of the site of the entry tear **[2]**. Risk factors associated with dissection are hypertension, smoking, male sex, genetic connective tissue disorders, and positive family history of the aortic disease **[1,2]**.

AD usually presents with the sudden onset of severe anterior chest pain (more common in type A dissection) and back pain (more common in type B) **[2,3]**. The pain is typically described as ‘tearing’ or ‘ripping’ in character and may migrate as the dissection progresses **[2,4]**. However, AD may also be painless in 15 % of patients **[5]**. This and other atypical presentations frequently lead to delays in diagnosis. Spittell and colleagues found that the initial clinical impression was of an alternative diagnosis in up to 38 % of 235 patients with aortic dissection **[5]**. A Task Force of the European Society of Cardiology reported that up to 30 % of patients later found to have aortic dissection were initially suspected of having other conditions **[6]**. A retrospective study of 66 consecutive patients with the acute aortic syndrome, which included 43 patients with type A and 20 patients with type B dissection, revealed that 26 (39 %) were incorrectly diagnosed initially **[7]**.

The most common misdiagnosis in patients subsequently found to have AD is an acute coronary syndrome (ACS) **[7-9]**, the pain of which may radiate to the back, and which may also be a consequence of AD if the dissection extends proximally to involve the coronary vessels. Other presentations that may lead to a delayed diagnosis include suspected ischaemic stroke **[10,11]**, acute abdomen **[12]**, and PE **[13-17]**. Against this background, we undertook a study to investigate how often AD presents as suspected PE, based on the number of patients with AD in whom CTPA was requested before diagnostic CT Aortography.

Methods

This was a retrospective cohort study conducted in Dumfries and Galloway Royal Infirmary, which serves a population of 148,000 in southwest Scotland. Data were collected on 998 consecutive patients having CT aortography between November 2016 and October 2021. These patients underwent 1363 CT aortograms, as many had repeat or follow-up imaging within our timeframe. Each patient was counted once and classified according to their initial scan unless they subsequently developed an AD. We considered the following predefined disease categories: aortic dissection (AD), aortic disease other than dissection, peripheral vascular disease (PVD), and others.

For this study, we grouped patients with co-existing aortic disease and PVD as an aortic disease. We classified intramural hematomas as AD. AD was considered acute if first diagnosed between 2016 and 2021 and chronic if first diagnosed before 2016, and the patient underwent follow-up imaging during our study period. We reviewed the images of all thirty-one patients with AD to determine if diagnostic CT aortography had been preceded by CTPA examination.

Ethical considerations

We did not seek ethical approval as no patient-identifiable data were included in the analysis, in keeping with our health board's policy.

Results

Thirty-one of 998 consecutive patients who underwent CT aortography had an aortic dissection **(figure 1)**. Eleven (35 %) of these were type A, and twenty (65 %) were type B. Twenty-four (77 %) were acute, and seven (23 %) were chronic. Five (16 %) patients with AD were initially suspected of having PE and underwent CTPA scans before CT aortography. All five had type B dissections. Their median age was 72 years (range 54 to 76) at the time of diagnosis, four of the five were male, and two were hypertensive. D-dimer values at presentation in these five patients ranged from 1,151 to 11,027ng/ml (normal range < 500ng/ml). The clinical features which led the receiving physician to suspect PE as the most likely diagnosis at presentation were as follows.

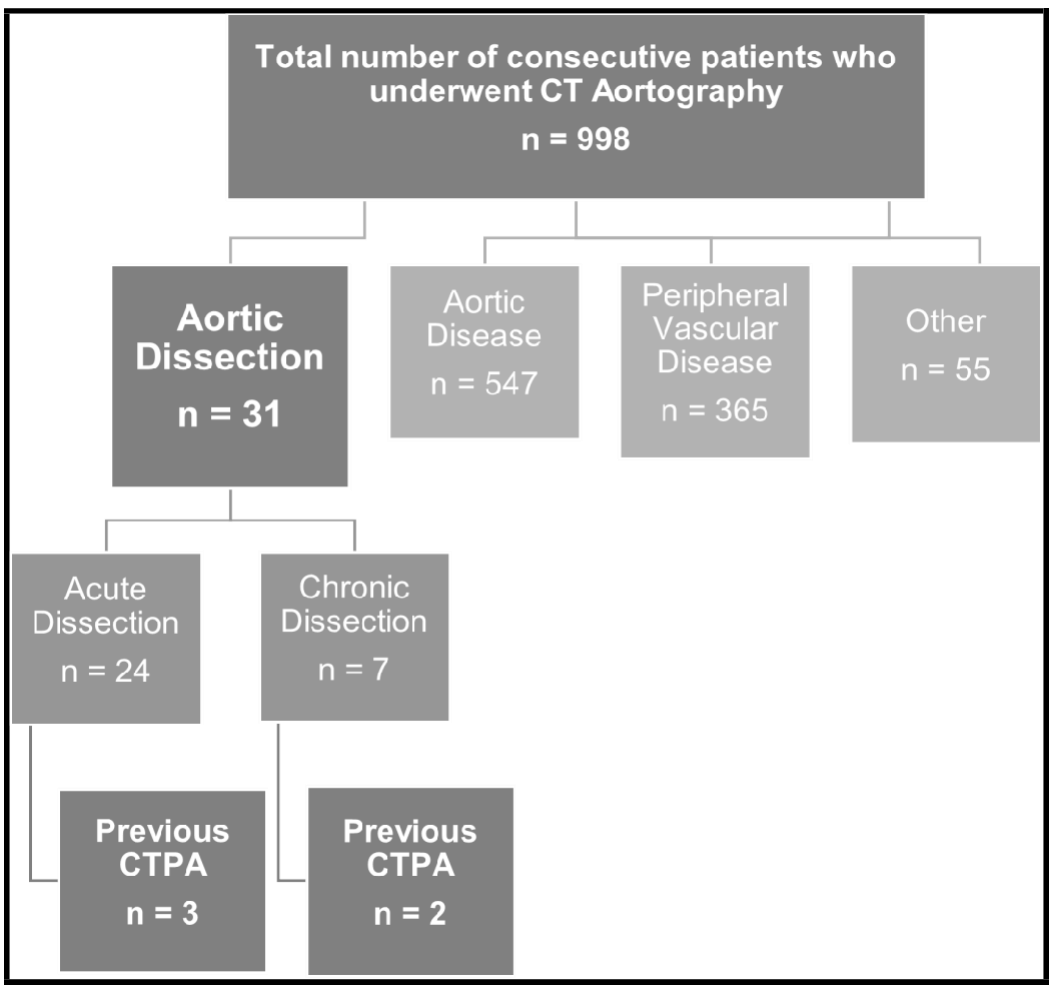

Figure 1: Study flow diagram showing the resulting numbers in each predefined disease category for those who underwent CT aortography. CTPA = CT Pulmonary Angiogram.

Case 1. A 76-year-old male woke with severe pleuritic central chest pain radiating to the right lower chest wall and the back. He had associated dyspnoea but no nausea, vomiting, or sweats. his blood pressure (BP) on admission was 124/77mmHg, and the electrocardiogram (ECG) was within normal limits. He was an ex-smoker with a medical history of asthma, giant cell arteritis (GCA), and transient ischaemic attack. D-dimer was raised at 1620ng/ml hence the referral for CTPA. This patient's AD was subsequently shown to be a consequence of GCA **[18]**.

Case 2. 66-year-old male presented with sudden onset central pleuritic chest pain radiating to the back. The pain started while he was showering and was associated with light-headedness, sweating, and shortness of breath (SOB). He gave a past medical history of angioneurotic edema and smoked 10-15/day. BP on admission was raised at 168/76mmHg. ECG was normal. His calves were tender on palpation, and this, in combination with a D-dimer of 2,480ng/ml and SOB, led to an initial suspicion of PE.

Case 3. 54-year-old male who complained of sudden onset pleuritic chest pain, which radiated to the upper abdomen and was associated with palpitations and sweating. He was not SOB and had no significant past medical history. The presentation followed a 10hr long-haul flight three days prior in which he hardly moved. BP on admission was 174/100mmHg. ECG was normal. Recent immobilization, pain worse on inspiration, and a D-dimer of 11,027ng/ml led to a request for CTPA. This patient was unusual in that he was found to have both PE and AD **[19]**.

Case 4. 73-year-old male who developed stabbing central chest pain following the death of his mother 24hrs previously. He had a background of PVD and smoked 10-15 cigarettes daily. He was hemodynamically stable with a BP of 136/69mmHg and little difference between his arms. ECG was normal, but D-dimer was raised at 1151ng/ml, hence the referral for CTPA.

Case 5. A 72-year-old female presented with a stabbing back pain that radiated to the chest and was worse on deep inspiration. She wasSOB, nauseated, and tender on palpation over the thoracic vertebrae and paravertebral muscles. This was on a background of chronic obstructive pulmonary disease, angina, myocardial infarction, and triple vessel coronary disease. She was an ex-smoker. BP on admission was 133/54mmHg with no difference between arms. Her ECG was expected, but D-dimer was raised at 1690ng/ml, hence the referral for CTPA.

All five AD patients underwent CTPA as their initial imaging modality. All had radiological signs suggestive of a false lumen **(figure 2 top panel)** which was subsequently confirmed in each case by CT aortography **(figure 2 lower panel)**. Four of the five had no evidence of PE, while one (case 3), somewhat unusually, had both. All five dissections were Stanford type B.

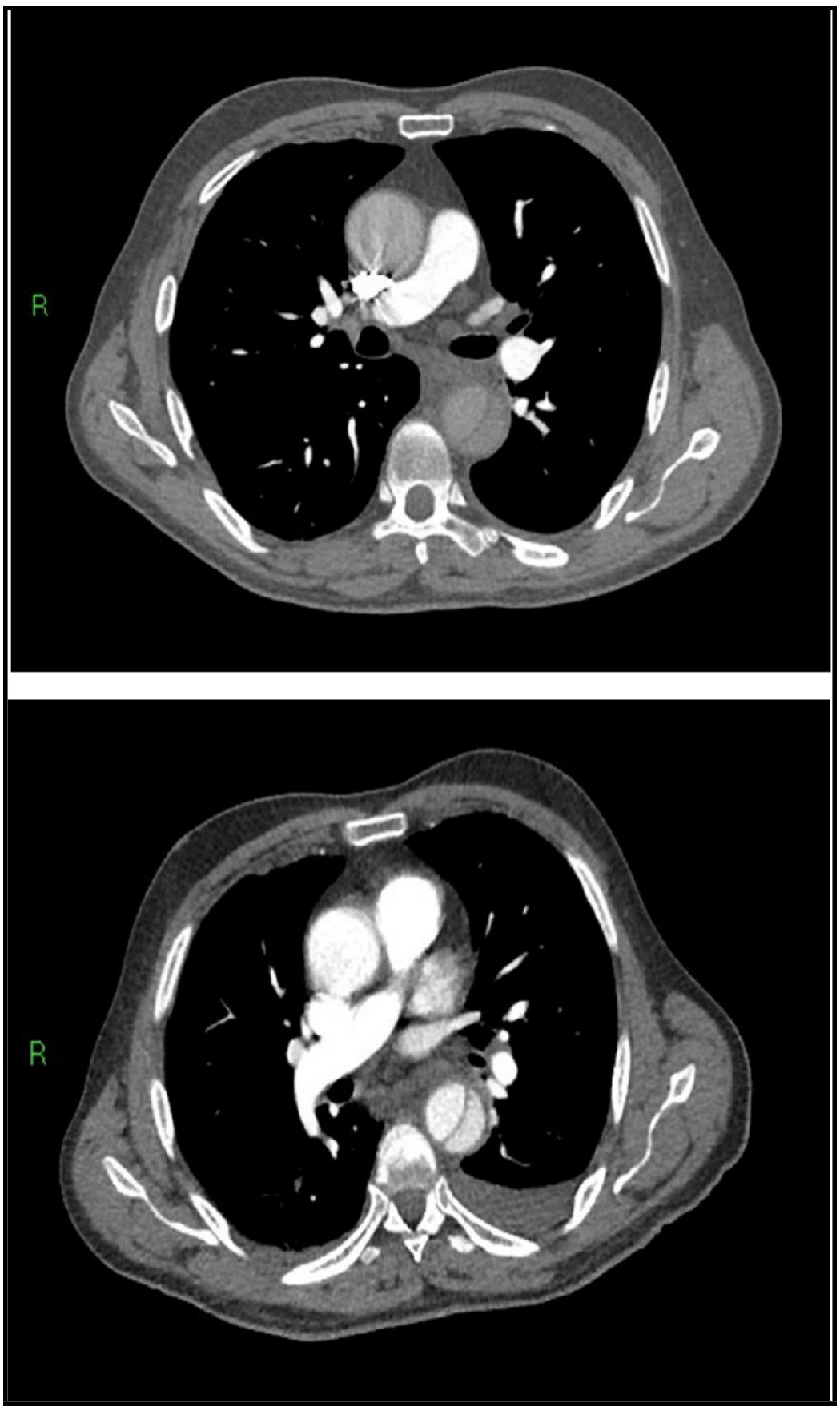

Figure 2: Representative example showing impression of false lumen in descending aorta on CTPA (upper panel) subsequently confirmed as AD by CT aortography (lower panel). CTPA = CT Pulmonary Angiogram. AD = Aortic Dissection.

Discussion

Our study highlights that a proportion of patients who develop AD are first thought to have PE. Misdiagnosis of AD as PE is less well recognized than misdiagnosis as ACS **[7-9]**, although several case reports have shown that AD may masquerade as PE **[13-17]**.

We and others have shown that occasional AD cases may be found in a consecutive series of patients undergoing CTPA for suspected PE. A retrospective review of 465 CTPA scans found AD in two **[20]**, while Lim and colleagues found two cases of AD among 85 patients with very high D-dimer whose CTPA was negative for PE **[21]**. To our knowledge, there are no previous estimates of the frequency with which patients whose final diagnosis is AD were first considered to have PE. We suspect the finding that 5(16 %) of 31 patients with AD had CTPA before CT aortography is a higher figure than most might have expected.

D-dimer is invariably elevated in PE and AD but lacks specificity for both **[21,22]**. These observations may nevertheless explain why some of our AD patients were thought to have PE initially. D-dimer can, however, be used as a rule-out test for AD. A prospective multicenter study **[23]** and a meta-analysis **[22]** concluded that D-dimer levels of less than 500ng/ml obtained within the first 24 hours of symptom onset could be used to identify patients who do not have AD.

Recent advances in CT imaging likely reduce the risk that AD is misdiagnosed as PE. Multidetector CT angiography is increasingly employed when evaluating the vascular system using higher quality and more efficient fast scanners in managing radiation dose **[24]**. Traditional CTPA protocols involve a bolus tracking technique with the region of interest placed on the pulmonary artery **[25]**. This gives excellent contrast within the pulmonary artery but limited evaluation of the aorta. A triple rule-out protocol involving an ECG-gated study can assess the pulmonary artery, aorta, and coronary vessels when the region of interest is placed on the ascending aorta **[24]**. Based on recent experience and review of these recent advances, radiologists in Dumfries and Galloway Royal Infirmary have adopted a non-ECG gated double rule-out protocol, requiring a slight increase in the radiation dose and volume of contrast, to image the pulmonary arterial tree and aorta simultaneously.

Strengths and limitations

This study included every CT aortogram performed within the specified time frame. The case information was available electronically for every patient. This ensured that no dissection was missed and avoided selection bias. There is controversy surrounding intramural hematomas and whether they result from dissection or develop into dissection **[1]**. We classified an intramural hematoma as a dissection for this study. Patients with dissection were demographically like those reported by others in that the majority were male and of older age **[1-4]**. It is likely that our results are not only representative of AD generally but also valid and applicable to the UK population.

A potential limitation of this study is that it was retrospective and observational. General physicians evaluated presenting symptoms subjectively rather than cardiologists with a particular interest in aortic diseases. The dissections which were first thought to be PE might have been diagnosed more rapidly had there been a greater awareness of the atypical presentations of AD **[5-8]**. It is also possible that clues to AD were present but not recognized or documented in the case record.

Conclusion

We have shown that a significant proportion of patients with AD were misdiagnosed as PE initially, mainly because they presented with pleuritic pain. Clinicians should be aware that aortic dissection may masquerade as pulmonary embolism.

Funding: None declared.

Competing interests: None declared.

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