

Why do emergency department clinicians miss acute aortic syndrome? A case series and descriptive analysis

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Abstract

The objective of this study was to understand why the diagnosis of Acute Aortic Syndrome (AAS) is missed in the ED, and to characterise the presenting features of cases in which a diagnosis of AAS was missed. A retrospective case series cohort study was performed, identifying and analysing cases where AAS was misdiagnosed in three UK EDs between 1st January 2011 and 31st December 2020. Forty-three cases were included, 22 of which were type A aortic dissections. The most common incorrect presumed diagnoses made were acute coronary syndrome (28%), pulmonary embolism (12%) and ‘non-specific chest pain’ (12%). In 31 cases (72%) there was no evidence from the notes that the clinician had considered AAS in the differential diagnosis. In 10 cases (23%), AAS was considered, but the clinician was falsely reassured by atypical or resolved symptoms, clinical examination, or normal chest x-ray. We conclude that ED clinicians may miss AAS by not considering it as a possibility, being falsely reassured by atypical or resolved symptoms, or mistaking it for other more common conditions. Further prospective work is necessary to establish the role of diagnostic aids and biomarkers in UK EDs.

Introduction

Acute Aortic Syndrome (AAS) is a life-threatening emergency affecting approximately 4000 people per year in the UK.¹ The term incorporates a spectrum of aortic pathology including penetrating aortic ulcers, which may progress to form an intramural haematoma. Ultimately this can lead to aortic dissection, where an intimal tear propagates between the intima and media with the potential to track proximally, distally, or both. Consequences may include cardiac tamponade, stroke, or ischaemia to other organs or limbs. AAS has been declared to boast a “lethal triad”: it is rare, lethal, and presents in atypical ways.² NHS Resolution recently identified AAS as a common cause of fatality-related negligence claims for the Emergency Department (ED).³ In 2020, the Healthcare Safety Investigation Branch (HSIB) reported on delayed recognition of AAS⁴ and identified that lack of diagnostic suspicion or attributing symptoms to another condition were particular risks for missing the diagnosis of AAS. It can therefore pose a significant diagnostic conundrum for the ED clinician. A significant number of patients in whom AAS is suspected show no evidence of it on CT, whilst an uncomfortable number of cases slip

through the net. Misdiagnosis rate is estimated to be as high as 38%, and around one quarter of cases are not diagnosed until 24 hours after presenting to the ED.⁵ The consequences of such error and delay can be fatal. Mortality follows a linear increase of up to 2% per hour of delay.⁶ However, over-investigation with too low a threshold for CT scanning of the thoracic aorta cannot be the solution, leading to low diagnostic yields^{7,8} whilst incurring significant costs and risks of ionising radiation.

Various risk stratification scores are in existence (some incorporating D-dimer) to help ED clinicians narrow down which patients require CT aortograms,^{1,9-14} though none has been validated in an undifferentiated ED population or in the UK. Even the best risk scoring system and diagnostic guidance is meaningless unless applied to the correct patients. Clinicians must understand which of the myriad of presentations with chest, back or abdominal pains, collapse, perfusion deficits or neurological compromise could be the 'needle in a haystack' manifestation of AAS to consider it as a differential diagnosis.

The aim of this study was to better understand *why* the diagnosis of AAS was missed during UK ED attendances, using a retrospective case series of patients from three UK EDs. Secondly, we aimed to characterise the presenting features of cases in which a diagnosis of AAS was missed.

Materials and Methods

This study was structured as a case series cohort study. Three UK EDs representing different hospital types and attendance sizes: the Royal Infirmary of Edinburgh (120,000 annual adult attendances), St John's Hospital Livingston (55,000 annual adult and paediatric attendances), and Wexham Park Hospital Slough (122,000 annual adult and paediatric attendances). Every patient in whom the diagnosis of AAS was found to have been missed during an ED presentation over a 10-year period (01/01/2011-31/12/2020) was included.

Data was collected about age, gender, presenting symptoms, alternative diagnosis, D-dimer result, CXR findings, time between ED presentation and AAS diagnosis, CT findings and patient outcome.

Cases were identified from one or more of the following sources: ED morbidity and mortality records, complaints records, post-mortem reports, and Electronic Patient Record (EPR) radiol-

ogy search reports of patients undergoing 'CT thoracic aorta' or 'CT aortic arch and carotids' requested by downstream inpatient teams after discharge from the ED, or on re-presentation to the ED.

Inclusion criteria were: episode occurred between 1/1/2011 and 31/12/2020; adult ≥ 18 years old; diagnosis of AAS made on CT scan within 7 days of discharge from the ED, whether discharged home or admitted under an inpatient specialty with an alternative suspected diagnosis; death from AAS (confirmed by PM report) occurring within 7 days of ED discharge.

Exclusion criteria were: diagnosis of AAS made on CT scan prior to discharge from the ED; alternative diagnosis found on CT (no radiological evidence of AAS); chronic aortic pathology/dissection only without acute changes; traumatic aortic dissection; no EPR available; pregnancy.

The study was deemed to be a service evaluation and therefore ethical approval was not required. The study was registered with the respective local Service Evaluation/Quality Improvement Project (QIP) databases. Patient representatives were not approached as part of this study, however The Aortic Dissection Charity Trust (TADCT) is a collaborator on our wider programme of work.

A pre-defined period of 10 years in three differing hospital types and attendance sizes was pre-selected for analysis. A study size was not pre-defined. The analysis of the data was conducted using Microsoft Excel. Unless otherwise stated, data are presented as median with Interquartile Range (IQR; 25th-75th percentile) for non-parametric continuous variables and as simple frequencies, proportions, and percentages for categorical variables.

Following identification of relevant cases, EPRs were accessed and a specifically designed data-extraction tool was completed by local emergency medicine clinicians. Where required information was not documented in the notes, it was sought by accessing radiology reports or laboratory results from the same ED attendance. Missing data, where irretrievable, was acknowledged in the data table and descriptive analysis. Chart elements were coded based on information in the patient EPRs. Descriptive analysis was performed on review of the ED notes regarding the reason for the missed diagnosis of AAS (Table 1).

Two reviewers independently analysed each case to establish a reason for the missed diagnosis. Pre-defined rules were designed to handle ambiguous elements including any disagreements being resolved by consensus.

Table 1. Reasons for missed AAS diagnosis in the ED (more than one reason for some cases).

Reason(s) determined for missed diagnosis	Number of cases
No evidence of consideration of AAS in differential diagnosis	31
Satisfied by alternative presumed diagnosis	19
Satisfied by exclusion of ACS	6
Diagnosis of AAS considered but not pursued as reassured by absence of radial-radial delay or BP differential	5
Diagnosis of AAS considered but not pursued as reassured by normal CXR	3
Diagnosis of AAS considered but not pursued due to atypical symptoms	1
Diagnosis of AAS considered but not pursued as reassured that pain had settled	1
Diagnosis of AAS suspected but CT misreported as being normal	1
Did not recognise widened mediastinum on CXR	1
Unknown (limited notes available)	2

Results

Forty-three cases were identified for inclusion. Figure 1 details their sources. The cases are detailed in the Supplementary Materials.

The median age was 68 years (IQR 58-78; range 27-89) and 60.5% were male. The most common type of AAS was Type A aortic dissection (51%; Figure 2). Cases were categorised into age groups for anonymisation purposes: <70y: 22 patients (19 male, 3 female); ≥70y: 21 patients (7 male, 14 female).

The most common site of pain was in the chest (27, 63%). Fourteen patients (32%) reported back pain, 7 of whom had chest pain radiating through to the back. Seventeen (16%) presented with neurological symptoms, and 9 (21%) had pre-syncope or collapse. Thirty percent had a combination of these symptoms. Sudden onset of symptoms was documented for 28 (65%) patients. D-dimer was measured for 11 patients, 10 of whom had a positive result. ECGs were variable in their findings with no theme identified and chest radiographs were normal in 16 (37%) patients.

The most common incorrect presumed diagnoses were Acute Coronary Syndrome (ACS) (28%), Pulmonary Embolism (PE; 12%) and 'non-specific chest pain' (12%). Figure 3 reports the presumed diagnoses made by ED clinicians. In 31 of 43 cases there was no evidence from the notes of consideration of AAS in the differential diagnosis. In 10 of 43 cases, AAS was clearly considered, but the clinician appears to have been falsely reassured by atypical or resolved symptoms, clinical findings or normal chest x-ray. Outcome was known in 40 patients, of whom 27 (67.5%) survived to hospital discharge (Figure 4). Of those with Type A dissection and discoverable outcome, 12 of 19 patients survived (63.2%; Figure 5).

The median time from ED presentation to diagnosis of AAS on CT or death was 15 hours (IQR 10-30; range 1.5-168 hours; n=39; 4 cases excluded due to out of hospital death with no precise time of death available).

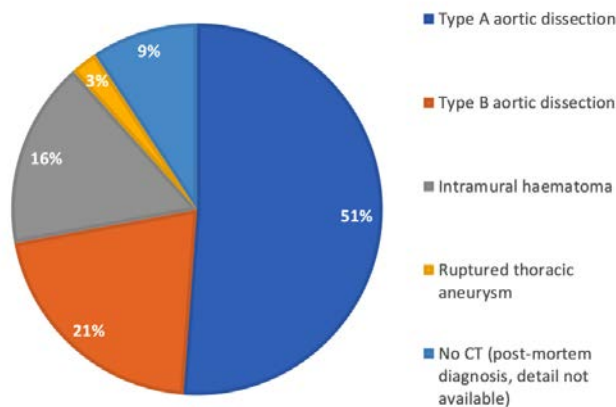


Figure 2. AAS diagnosis in all 43 cases.

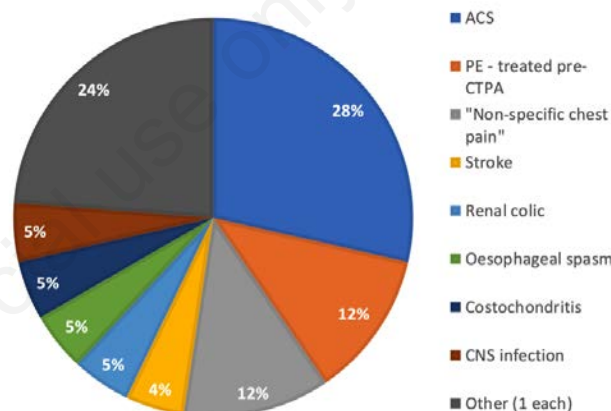


Figure 3. Alternative presumed diagnosis made in ED (n=42). 'Other' seizure cause includes: pyelonephritis, symptomatic aortic stenosis, vertebral crush fracture, lower respiratory tract infection, non-specific abdominal pain, pericarditis, decompensated heart failure, ischaemic pain secondary to fast AF, symptomatic complete heart block, gastritis.

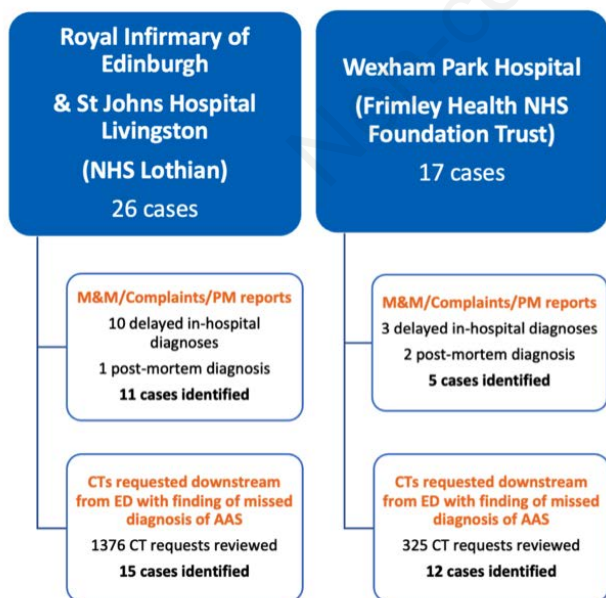


Figure 1. Details of the sources of all 43 cases were identified across the three sites.

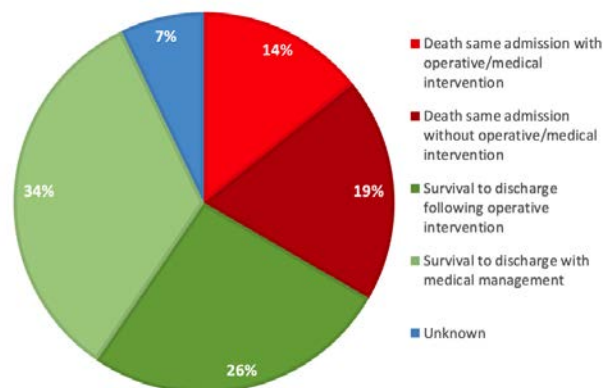


Figure 4. Outcomes of all AAS patients (n=43).

Discussion

The most common reason for ED clinicians missing the diagnosis of AAS was lack of consideration of it as a differential diagnosis (consistent with the HSIB report),⁴ sometimes even in the face of typical symptoms. This could be due to lack of clinician awareness or lack of experience. ED clinicians encounter a high number of chest pain presentations every shift, with the vast majority being safely discharged from the ED with no significant diagnosis made. Oversimplification of chest pain assessment, which may often be considered as “rule out ACS” or “rule out PE” can lead to AAS being overlooked. Similar issues affect non-chest-pain presentations of AAS, with other diagnoses being much more commonly encountered. Symptoms were noted as ‘sudden onset’ in 65% of our case series. Pain of “sudden onset with its worst severity being at its onset” has been reported to be the most discriminative feature of the chest pain of AAS.¹

Campaigns such as ‘Think Aorta,’¹⁵ launched in 2017, and RCEM Safety Alerts¹⁶ aim to increase clinician awareness through posters displayed in the clinical environment and maintenance of safety awareness initiatives. Awareness of the diagnosis is only the first hurdle. The next is to decide which patients need further investigation, and an appreciation of the possible atypical presentations.

In some cases, it is likely that the diagnosis was not considered due to atypical symptoms. For example, seven patients in our series had absence of pain (with neurological compromise or syncope only). Extensive aortic dissections can lead to shock with syncope and/or persisting reduced conscious level. History of preceding symptoms is likely to be very minimal in such patients, and detection will depend on the clinician considering dissection as a possibility. In other cases, the patient’s presenting features were more ‘textbook’ for another pathology, steering the diagnosis astray. Kurabayashi *et al.*¹⁷ published a retrospective analysis of 109 cases of aortic dissection in Japan in 2011. It was more likely to be missed when laboratory findings and symptomatology mimicked other diseases. In our case series, it was observed that the descriptive terms used to characterise chest pain were very variable. Interestingly, in 7 cases (16%) the pain was documented as either ‘pleuritic’ or ‘worse on deep inspiration’, which typically steers diagnostic momentum towards PE. Terms such as ‘crushing’, ‘tight’ or ‘heavy’ would rather sway clinicians towards a supposed ACS diagnosis. The inclination towards these erroneous diagnoses in such cases can be potentiated by ischaemic ECGs, raised troponins, or elevated D dimers, none of which should detract from the possibility of AAS. Notably, in a few cases, the onset of chest pain was during physical strain (such as lifting an object) and worsened by certain movements. This can divert suspicion, and falsely reassure the clinician towards a diagnosis of soft tissue or musculoskeletal injury.

Incorrect presumed diagnosis of PE can be particularly problematic as it may lead to thrombolysis prior to imaging in an unstable patient. Stable patients with presumed PE seen out-of-hours in the ED are usually anticoagulated and undergo CT pulmonary angiography the following day, as occurred in one patient in our series. Similarly, when AAS masquerades as an ischaemic stroke, there may be inappropriate administration of thrombolysis. There were no such cases found in this case series, but Bressler *et al.*¹⁸ reported a case in 2020 and recommended that AAS should at least be considered prior to thrombolysis. Usui *et al.*¹⁹ suggested that CT to exclude aortic dissection should be part of stroke protocol for ischaemic stroke, after reporting a single case study. Huang *et al.*²⁰ studied the characteristics of patients presenting

with painless AAS manifesting as stroke. They compared 200 patients with acute ischaemic stroke *without* AAS to 47 patients with acute ischaemic stroke secondary to AAS (4 from local stroke registry in Taiwan, 43 from literature search). Reported findings were that painless AAS stroke patients were likely to be younger with less comorbidity, and more often presented with sudden loss of consciousness, hypotension, bradycardia and had left-sided weakness on arrival to the ED.

Another observation from our study, is that AAS masqueraded as renal colic-type pain in two patients. Both were male, one aged over 70y; the other under 70y with Marfan’s syndrome. Al-Wahaibi *et al.*²¹ published a case report of AAS mimicking renal colic with microscopic haematuria. Particularly in older patients, the mantra of never assuming renal colic prior to ruling out aortic pathology is vital. Another lesson here is that there should be a very high index of suspicion for AAS in patients of any age with Marfan’s syndrome, who present with possibly attributable symptoms, as reflected by risk stratification scores such as ‘ADD-RS’²² and the Canadian Clinical Practice Guideline.¹³ The joint RCEM and RCR Best Practice Guideline¹⁶ recommends CTA if any high risk features are present (unless another cause for symptoms is identified and evidenced). The high-risk features are grouped into three categories of known high risk conditions (e.g. connective tissue disease), high risk pain features (e.g. abrupt onset) and high risk clinical findings (e.g. pulse deficit).

When it was evident that AAS was considered by the ED clinician, it appears they were inappropriately diverted from the diagnosis by falsely reassuring clinical findings such as symmetrical upper limb blood pressures, absence of radial-radial delay, or normal appearances of the mediastinal contour on chest x-ray. While the presence of such features would support the diagnosis, their absence is insufficient grounds to exclude it.^{13,22}

One case was identified in which there was erroneous radiology reporting. Similarly, Nagra *et al.*²³ in 2013 reported a case of type A aortic dissection missed by non-cardiac gated contrast-enhanced CT due to an aortic root dissection flap masquerading as an aortic valve apparatus. They warned that conventional spiral CT angiography suffers from motion artefact, and subtle dissections limited to the aortic root and proximal aorta could be dismissed as image artefacts. One patient in our series had ultrasound of the aorta as a method of assessment, but this is insufficient where AAS is a differential. The RCEM/RCR Best Practice Guideline¹⁶ imaging recommendations for AAS include that CT Aortogram (CTA) with its high diagnostic accuracy is the diagnostic modality of choice, imaging should include initial non-contrast and post contrast scans and that arterial phase acquisition should routinely be performed with ECG synchronisation (*i.e.* gated scan).²⁴

Rotella & Yeoh² emphasise that the busy and complex ED environment increases susceptibility to cognitive bias which may lead to delayed or missed diagnosis. They note that in a significant number of reported missed cases, there was failure to respond to clinical cues suggestive of aortic dissection, and attribute this to cognitive bias and error-producing conditions. Search satisfaction bias is a tendency to call off the search once one clue is found. Myriads of cognitive biases may be at play during any patient-clinician interaction in the ED,²⁵ and it is important that we are cognisant of these as possible to optimise the care we provide to our patients.

The diversity of guises in which AAS presents itself, its rarity and lethality, combined with the cognitive biases exacerbated by a busy ED, inundated by high numbers of patients with potentially attributable symptoms of chest/back/abdominal pain, syncope and/or perfusion deficits, create a significant challenge for the ED clinician. Too low a threshold for imaging is likely to lead to imag-

ing patients without pathology or who then have an “incidentaloma” discovered, over-burdening the radiology service which may impact on care received by other ED patients, and slowing patient flow through the ED (which in turn has a detrimental effect on overall patient safety). Yet under-investigating or misdiagnosis leads to patient harm. The RCEM/RCR guideline also noted that “Centres that have successfully addressed their TAD [thoracic aortic dissection, note of the authors] missed diagnosis rate by implementation of awareness raising programmes and increased access to CTA have reported a 3% pickup rate for TAD and 42% pickup rate for alternative diagnoses.”

Though as yet unvalidated in a UK ED population, risk stratification scoring systems such as ADD-RS in combination with D-dimer may prove useful in standardising the approach and helping clinicians to discern which patients to scan. The ADvISED study²⁴ (a multicentre observational study) prospectively assessed the performance of ADD-RS ≤ 1 plus D dimer < 500 ng/mL and reported a sensitivity of 98.8% (96.4-99.7%) with a specificity of 57.3%. This is not currently widely adopted into UK Emergency Medicine practice,²⁶ but subject to validation, may play an important role in improving our ability to select the correct patients to scan.

Limitations

We have only considered patients where the diagnosis was missed on initial ED presentation, and it is possible that these patients may differ from all AAS patients in some way. Our sites were self-selected but we believe our hospitals reflect the general scope of practice within UK EDs. The rarity of AAS and the relative rarity of missed cases, although catastrophic when they occur, makes prospective studies in this area challenging to conduct. This study is limited by the retrospective interpretation of EPRs and our assessment of whether AAS was considered is reliant upon documentation. Some missing data was irretrievable, though this only disabled evaluation of reason for missed diagnosis in 2 of the 43 cases. It is difficult to extrapolate conclusions about the helpfulness of D-dimer and there is a need for a prospective study looking at the use of D-dimer in this context.

Conclusions

AAS is a rare and potentially lethal condition. In our review of missed AAS diagnosis in three UK EDs, we found that in approximately three quarters of cases, AAS did not seem to have been considered in the differential diagnosis. When AAS was considered, too much weight was placed on misleading clinical or investigative findings. AAS can masquerade as other conditions (e.g. ACS and PE), and along with associated cognitive biases, this led to alternative incorrect diagnoses being made.

Campaigns such as ‘Think Aorta’ have taken on the important issue of raising awareness of AAS as a diagnosis amongst clinicians, yet its rarity in the haystack of presentations to ED leaves a challenge beyond that of mere awareness. Miss rates cannot be entirely attributed to ignorance - various cognitive biases such as availability bias, omission bias, confirmation bias, search satisfaction, or diagnostic momentum are also likely to be at play, leading to presumption of alternative incorrect diagnosis for a patient’s symptoms.² More research is needed to investigate the use of scoring systems to assist the clinician to determine risk level and which patients to scan. D-dimer may be helpful to refine this, with promising levels of sensitivity and improved specificity.^{22,27} The impact of the RCEM/RCR best practice guideline in terms of patient safety, scan rates, costs and benefit versus harm to patients

is yet to be quantified, and further prospective research will be beneficial, alongside evaluation of the utility of D-dimer in suspected AAS.

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