

A rare presentation of aortic dissection

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ABSTRACT

Introduction: Aortic dissection (AD) is a rare, life-threatening event that presents as acute chest pain, radiating to the back, often in the setting of hypertension. Aortic dissection is the most common fatal aortic pathology, yet the initial diagnosis is often missed. Additionally, a minority of patients present with either no symptoms or neurologic symptoms. This patient exhibited atypical presentation of AD and the purpose of this report is to raise awareness. **Case Report:** A 79-year-old woman with past medical history of hypothyroidism, osteoporosis, and chronic obstructive pulmonary disease (COPD) presented with bradycardia, acute chest pain, and generalized weakness. She was afebrile, her blood pressure was 100/54 mmHg, heart rate was normal sinus rhythm (NSR) at a rate of 81 bpm, respiratory rate 17 breaths per minute. Patient was AO × 3. Physical exam was unremarkable, except for trace pitting edema in bilateral lower extremities. Electrocardiogram (ECG) showed normal sinus rhythm at a rate of 86 with low voltage. Troponin I level was negative × 3. Chest X-ray (CXR) showed no acute cardiopulmonary findings. Transthoracic

and carotid echocardiogram revealed a dilated aortic root and dissection of the thoracic aorta. A computed tomography (CT) angiogram of chest, abdomen, and pelvis revealed a Type A AD of the thoracic and abdominal aorta, involving the common carotid arteries, left common iliac, and left external iliac arteries. **Conclusion:** Aortic dissection is a life-threatening emergency that requires early detection and treatment to decrease patient morbidity and mortality. However, it is difficult to diagnose as it can present with atypical findings and is frequently mistaken for other etiologies that cause chest pain such as acute coronary syndrome.

Keywords: Aortic dissection, Atypical, Bradycardia, Generalized weakness

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INTRODUCTION

Aortic dissection is a rare but life-threatening event that classically presents as acute chest pain, radiating to the back, often in the setting of hypertension. It is classified by the location of the dissection; Type A dissection involves the ascending aorta and Type B dissection involves the portion distal to the left subclavian artery [1]. In general, aortic dissection is the most common fatal aortic pathology, yet the initial diagnosis of aortic dissection often is missed [2]. The incidence of acute aortic dissection is 5–30 cases per one million cases per year [3]. Approximately 80–90% of patients

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experience acute onset of severe chest pain during acute aortic dissection; however, this constancy in symptoms does not translate to consistently correct diagnoses [4]. A retrospective study of 235 patients shows that although the majority of aortic dissection cases present with classical chest pain, an incorrect antemortem diagnosis is made 38% of the time [4]. Furthermore, a minority of patients present with either no symptoms or neurologic symptoms such as full body weakness, making dissection even more difficult to successfully diagnose [5, 6].

This patient exhibited an atypical presentation of aortic dissection, initially with bradycardia, temporary generalized weakness, and chest pain. The purpose of this case report is to raise awareness of atypical presentation of aortic dissection and lead to earlier diagnosis and treatment.

CASE REPORT

History and findings on admission

A 79-year-old woman with a past medical history significant for hypothyroidism, osteoporosis, and COPD presented initially to Hahnemann University Hospital by Emergency Medical Services (EMS) after being found to be bradycardic with acute chest pain and generalized weakness. She had no previous cardiac history. Past surgical history was significant for a root canal procedure one month prior to admission, a blepharoplasty in 2017, and a laparoscopic umbilical hernia repair in 2009. She had no known allergies. Medications included alendronate 70 mg per os (PO) weekly and levothyroxine 25 mg PO daily (patient reported same dose for >1 year), and she claimed compliance to her medications. The patient reported 1–2 alcoholic drinks per month and a 30 pack-year smoking history before quitting 25 years ago but denied any history of illicit drug use. Family history was significant for a father who died of a myocardial infarction in his 40s.

She reported initially a pulsatile chest pain that started after five minutes of brisk walking. The chest pain was pulsatile, nonradiating, and worsened with inspiration. She denied loss of consciousness but reported a feeling of full body weakness. When EMS arrived, the patient was found to be in sinus bradycardia in the 40s with no signs of ischemia. She was given sublingual nitroglycerin, aspirin, and atropine. Her heart rate responded appropriately to the 70s.

In the Emergency Department, she reported improvement to the chest pain, but reported chills and a headache; remaining review of systems was negative. She was afebrile, blood pressure 100/54 mmHg, heart rate was normal sinus rhythm at a rate of 81 bpm, respiratory rate 17 breaths per minute. The patient was alert and oriented to person, place, and time. Physical exam was unremarkable, except for trace pitting edema in bilateral lower extremities. Cardiac exam revealed normal S1/

S2, no murmurs, and no jugular venous distention. Neurological exam revealed no neurological deficits; strength was 5/5 in all extremities without any sensory deficits.

Initial results and diagnostic imaging

Electrocardiogram showed normal sinus rhythm at a rate of 86 with low voltage, possible left atrial enlargement, and anterior infarction that could not be ruled out. Troponin I level was negative $\times 3$. Prothrombin time and international normalized ratio (INR) were elevated to 15.6 and 1.36 seconds, respectively. Partial thromboplastin time was normal at 31.9 seconds. Thyroid stimulating hormone was elevated to 12.198 mIU/L and free T4 was within normal limit at 0.90 ng/dL. The complete metabolic panel showed decreased sodium at 135 mmol/L, increased creatinine of 1.13 mg/dL (unknown baseline), decreased albumin at 3.4 g/dL, increased aspartate aminotransferase to 37 IU/L, and normal alanine aminotransferase of 34 IU/L. A portable CXR showed a normal mediastinal width and no acute cardiopulmonary findings. The patient was admitted to a telemetry medical floor and electrophysiology was consulted for bradycardia.

The patient continued to report nausea overnight but endorsed near resolution of her chest pain. She reported new onset of worsening back pain that prevented her from sleeping. She described the back pain as constant, achy, and 4/10 severity that started between the scapulae and progressed down to her lower back. Tylenol did not relieve her back pain. Her vitals remained stable overnight with a heart rate ranging from 52 to 68 bpm and blood pressure ranging from 112/42 to 138/70 mmHg. ECG in the morning was unchanged from her previous one. Physical examination on day 1 of admission differed from her initial presentation as she was noted to have a nontender pulsatile mass in the right anterior neck, posterior to the sternocleidomastoid.

Transthoracic echocardiogram and carotid echocardiogram revealed a dilated aortic root and dissection of the thoracic aorta (Figure 1A–D). A CT angiogram of chest, abdomen, and pelvis was then ordered to further characterize the extent of the aortic dissection and showed a Type A dissection of the thoracic and abdominal aorta (Figure 2). The dissection began at the aortic root at the level of the sinotubular junction. The dissection extended into the right brachiocephalic, the visualized proximal right common carotid, left common carotid, and base of the left subclavian arteries. The dissection extended distally into the left common iliac and terminated at the level of the left external iliac artery. The right common iliac was spared. The celiac, superior mesenteric, and right renal arteries arose from the true lumen. The left renal artery and inferior mesenteric artery (IMA) arose from the false lumen. The ascending aorta was mildly ectatic, measuring up to 3.8 cm in diameter.

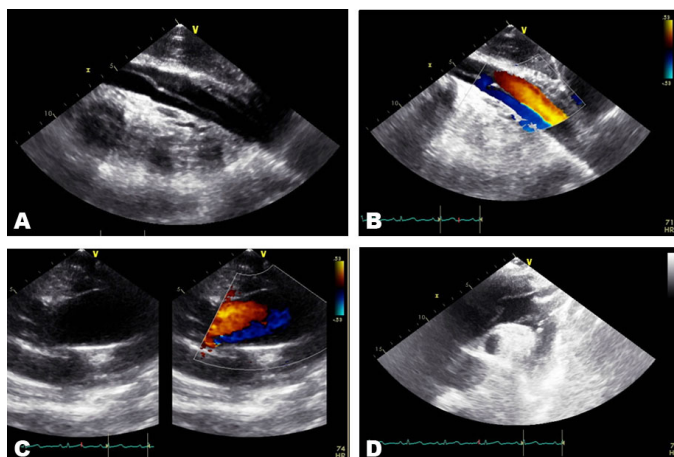


Figure 1: (A) Transthoracic echocardiogram demonstrating the descending aorta dissection flap from a subcostal angle. (B) Transthoracic echocardiogram with color Doppler showing dual flow at the descending aortic dissection. Blue represents false lumen. Red represents true lumen and entry point. (C) Parasternal long axis angle transthoracic echocardiogram with (right) and without (left) color Doppler showing dual flow and dilation of ascending aorta to 3.8 cm. Blue represents false lumen. Red represents true lumen. Note, body surface area: 1.4 m². (D) Transthoracic echocardiogram from a suprasternal angle demonstrating the dissection in the arch of the aorta involving the branch vessels.

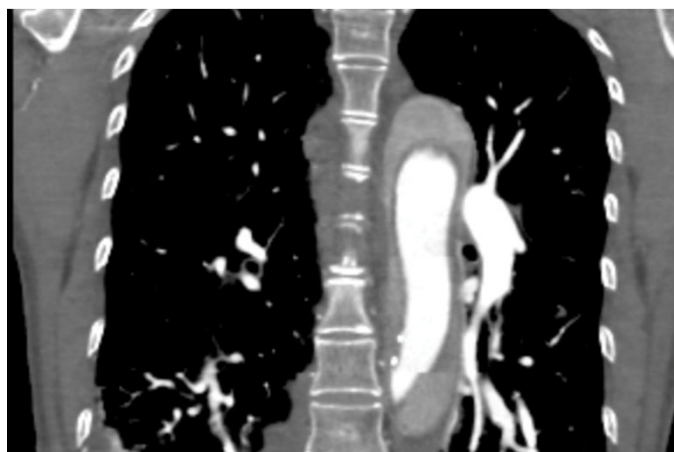


Figure 2: CT chest with intravenous contrast demonstrating true and false lumen of descending thoracic aorta.

Follow-up and outcome

Given the severity and extent of the aortic dissection, CT surgery was contacted, and the patient was emergently transferred to Hospital of the University of Pennsylvania where she underwent surgery.

DISCUSSION

Type A aortic dissection is a life-threatening emergency that requires early detection and treatment to decrease patient morbidity and mortality. However, it is difficult to diagnose as it can present with atypical findings as demonstrated in the case presented. Furthermore, aortic

dissections are frequently mistaken for other etiologies that cause chest pain such as acute coronary syndrome, making aortic dissections even more difficult to identify clinically [2, 7].

The classic presentation of aortic dissection includes acute onset of severe, sharp chest or back pain, rapid hemodynamic instability, tachycardia, and a widened aortic silhouette on CXR [8]. Depending on the extent of the dissection, the pain can radiate to the abdomen; however, a minority of patients report the abdomen alone as their only location of pain. A new onset heart murmur, often a diastolic decrescendo murmur of aortic regurgitation, is also a common finding in AD. Moreover, most patients with Type B AD initially present with hypertension, whereas Type A AD patients initially present with symptoms of hypotension as a result of aortic valve disruption [9].

Atypical symptoms of aortic dissection include lack of chest or back pain and/or the presence of neurologic symptoms. Patients who are more likely to have painless AD are greater than 70 years old, as in our patient, or those with a history of aortic aneurysms, diabetes mellitus, or cardiovascular surgery. One retrospective review determined that painless AD was often associated with hypotension and syncope [10]. Additionally, of all AD cases, only a tenth present with neurologic complaints, including weakness, syncope, or hoarseness [9].

Timely identification and diagnosis of AD is achieved through history and physical exam findings and is confirmed by imaging. The triad of features most indicative of AD include acute onset of ripping, tearing, or sharp chest and/or abdominal pain, an asymmetrical or absent pulse in extremities or neck, and a mediastinal widening on CXR. Unfortunately, our patient had none of these findings on initial presentation, which lead to a delay in diagnosis. We used computed tomographic angiography to visualize and confirm the dissection due to its availability, lower cost, and rapid acquisition of images, however, magnetic resonance angiography and multiplane transesophageal echocardiography are also readily used in institutions where CT is unavailable [11].

Initial management of AD includes medications to control for pain and antihypertensives to reduce the shearing forces on the wall of the aorta and minimize propagation of the dissecting vessel [1]. Patients with Type A AD, as in our patient, require urgent surgical correction regardless of the patient's hemodynamic status or the extent of the dissection. Type B AD is best managed with medical therapy rather than surgical intervention if the dissection is stable, the patient's hypertension does not persist, and if there is no major aortic branch occlusion leading to organ ischemia. Otherwise, these patients are also treated surgically. Following initial treatment for either Type A and B dissection, patients need life-long antihypertensive medications and serial vascular imaging studies, either MR or CT angiography, at 3, 6, 12 months and annually thereafter to monitor for disease progression.

The patient described in this case report displayed symptoms of chest pain, weakness, bradycardia, and later, back pain. While chest and back pain were cardinal symptoms of AD, bradycardia, and weakness lead us to delay the diagnosis. Neither the initial clinical exam nor ECG showed cardiac abnormalities, and the aortic dissection was discovered only after a new physical exam finding, a nontender pulsatile mass in the right anterior neck, resulted in an emergency carotid and transthoracic echocardiogram. Nevertheless, ultrasound technicians were able to capture an image of the dissected descending aorta, which allowed for swift action and surgical intervention that saved the patient's life.

CONCLUSION

Although this patient survived, future patients with similar presentations may not if the diagnosis is delayed further or missed altogether. This case should stand as an example that aortic dissection needs to remain high on the differential diagnosis of patients with acute chest pain, bradycardia and weakness, and additional noninvasive imaging such as ultrasound or CT must be used early to aid in the diagnosis.

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Author Contributions

Lauren Skerritt – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Lauren Musser – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Meet Parikh – Conception of the work, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Tahmid Rahman – Conception of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Chiduzie Madubata – Conception of the work, Acquisition of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Naitik Patel – Conception of the work, Design of the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission

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Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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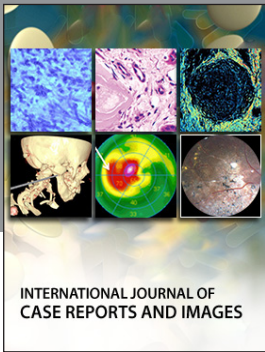
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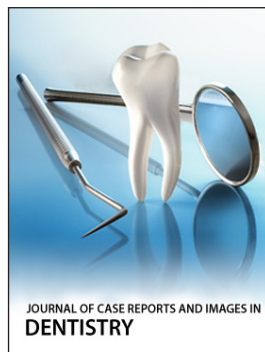
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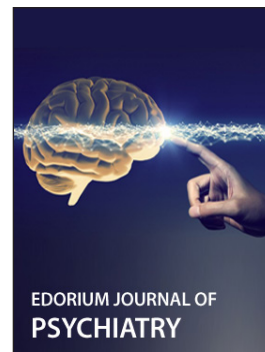
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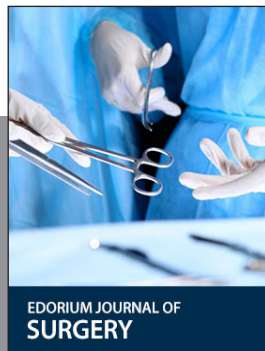
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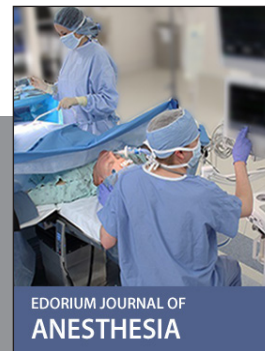
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