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CASE REPORT

When Palpitations and Hand Weakness Point to the Aorta: An Atypical Case of Aortic Dissection

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Keywords

Atypical, Aortic dissection, Palpitations, Left-hand weakness, Hypertension

Abstract

Aortic dissection (AD) is a life-threatening condition often presenting with varied symptoms, complicating early diagnosis. We present a case of a 58-year-old woman with untreated hypertension and a remote history of cardiac ablation who presented with persistent palpitations and transient left-hand weakness, but without chest or back pain. An incidental AD was identified during imaging for a suspected transient ischemic attack. Effective blood pressure management and prompt cardiothoracic intervention, with successful surgical repair, enabled favorable outcomes. This case underscores the diagnostic challenges posed by atypical AD presentations, highlighting the importance of maintaining high index of suspicion in at-risk population.

Background

Aortic dissection (AD) is a life-threatening condition that requires prompt diagnosis and management. Key predisposing factors include structural weakness of the aortic wall and increased wall tension (1–3). It typically presents with sudden-onset severe chest or back pain, often described as tearing in nature (4). However, atypical presentations such as painless dissection, transient neurologic deficits, and palpitations have been reported (2, 5–8). Among painless dissections, left-sided neurologic deficits are the most common atypical manifestation (9).

Here, we present a case of extensive AD involving the ascending and descending aortas, incidentally discovered during imaging for a suspected transient ischemic attack (TIA).

Case Report

Presenting Symptom

A 58-year-old woman with untreated hypertension and a remote history of cardiac ablation presented to the emergency department (ED) with palpitations and a transient episode of left-hand weakness. She had no regular medical follow-up or medications.

Four days before ED presentation, while at a gas station, the patient developed sudden palpitations, lightheadedness, and a sensation of instability. These symptoms resolved quickly but the palpitations persisted. Later that same day, she noticed mild intermittent difficulties in holding her mobile phone with her left hand, which resolved completely by the following day and did not recur. Due to persistent palpitations, she sought care at an urgent care center and was immediately transferred to the ED for further evaluation.

Emergency Department Findings

On arrival, she was alert and in no distress. Blood pressure (BP) was 160/113 mm Hg, heart rate, 126 beats/min; oxygen saturation, 100% on room air; respiratory rate, 20 breaths/min; and temperature 36.6 °C. Body mass index was 33.2 kg/m^2. Besides the palpitations, systems review was completely unremarkable. Her only substance use included occasional alcohol and marijuana. She had no known family history of genetic

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Table 1. Emergency Department Laboratory Testing Showing Predominantly Normal Results But for Isolated Leukocytosis and Prediabetic Glycated Hemoglobin Level

Test	Reference Ranges and/or Units	Test Result
White blood cell count	4.3–10.0 K/μL	15.6
Hemoglobin level	11.8–14.8 g/dL	13.2
MCV	82– 99.0 fL	88.9
Platelet count	140–350 K/μL	243
Sodium	137–145 mmol/L	139
Potassium	3.4–5.1 mmol/L	3.6
Chloride	98–107 mmol/L	103
Carbon dioxide	22–30 mmol/L	26
BUN	7–17 mg/gL	12
Creatinine	0.5–1 mg/dL	0.80
Glucose	74–106 mg/dL	107
Calcium	8.4–10.2 mg/dL	9.5
eCrCl (C-Gault)	mL/min/kg	1.2
Troponin I	≤0.08 ng/ml	0.04
TSH	0.47–4.68 uIU/mL	0.53
Magnesium	1.6–2.6 mg/dL	2.4
HbA1c	≤5.6%	5.7

BUN = blood urea nitrogen; eCrCl = estimated creatinine clearance; HbA1c = glycated hemoglobin; TSH = thyroid-stimulating hormone; MCV = mean corpuscular volume; WBC = white blood cells.

conditions. Physical examination revealed an irregularly irregular rhythm but no murmurs or focal neurologic deficits.

Laboratory results showed isolated leukocytosis (15.6 k/μL). Troponin I, potassium, and magnesium were normal (Table 1). Her electrocardiogram demonstrated atrial fibrillation (A.fib) with rapid ventricular response (116 beats/min) and left ventricular hypertrophy, with no acute ST-segment changes. Her chest radiograph (Figure 1) was read as normal, but retrospective review after AD diagnosis suggested mediastinal widening. Noncontrast computed tomography (CT) scan of the head showed chronic microvascular ischemic changes with no acute findings. An immediate magnetic resonance imaging (MRI) of the head was scheduled, but it was deferred owing to claustrophobia and postponed to the next day. At this point, the working diagnosis was A.fib with a possible embolic transient ischemic attack. Aortic dissection was not considered, and D-dimer level testing was not ordered. Also, BP was measured only in the left arm.

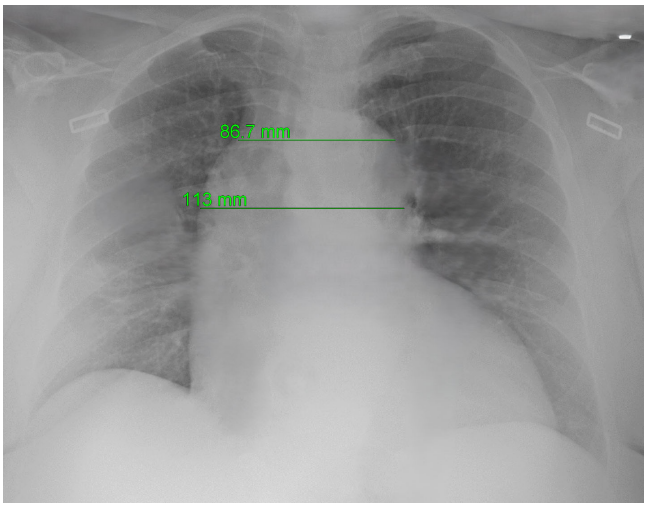

Figure 1. Chest radiograph obtained in the emergency department demonstrating mediastinal widening.

The patient received intravenous diltiazem for rate control and therapeutic single-dose enoxaparin for anticoagulation before being admitted to the telemetry unit approximately 2 hours later.

In-Hospital Course

Upon admission to the telemetry unit, a transthoracic echocardiogram (TTE) was conducted that revealed a left ventricular ejection fraction of 62%, with severe concentric left ventricular hypertrophy, a severely dilated left atrium, and a mildly dilated ascending aorta (AA) (4 cm), but normal aortic root. These findings did not raise suspicion for AD.

On hospital day 2, the patient was evaluated by a cardiologist, who transitioned diltiazem to oral administration and initiated apixaban. The patient again declined MRI despite being offered sedation to facilitate the procedure. Given her stability and lack of symptoms, discharge planning was initiated. A neurology consultation was arranged to facilitate outpatient MRI scheduling and follow-up.

On hospital day 3, the patient was evaluated by the neurologist, who found no neurologic deficits. As part of the stroke/TIA work-up, the neurologist ordered outpatient brain MRI using an open scanner as well as CT angiography (CTA) of the head and neck to evaluate for vascular stenosis given the patient's cardiovascular risk factors. The CTA, conducted the same day, revealed an incidental finding of a thoracic AD extending into the great vessels (Figure 2). Cardiothoracic surgery was urgently consulted, and plans were made for surgical intervention after at least 1 day to allow apixaban clearance.

Blood pressure control was initiated, with a target of less than 120/80 mm Hg. No bilateral BP measurements were performed postdiagnosis. A dedicated CTA of the aorta showed that dissection originated from the AA with aneurysmal dilation of the AA, extending into the arch, descending aorta, abdomen, and iliac arteries. The true lumen supplied visceral organs. A high-grade stenosis was observed in the true lumen supplying the right common carotid artery. Both the true and false lumens extended into the left common carotid artery, which remained patent. The dissection involved the left subclavian artery as well (Figure 3). She was transferred to the cardiovascular intensive care unit for preoperative optimization.

Surgical Management and Recovery

On hospital day 5, she had surgical repair. Intraoperative findings confirmed a dissection from the sinotubular junction to the arch, involving brachiocephalic vessels. She had AA replacement as a hemiarch with a 24-mm Hemashield graft, aortic valve repair

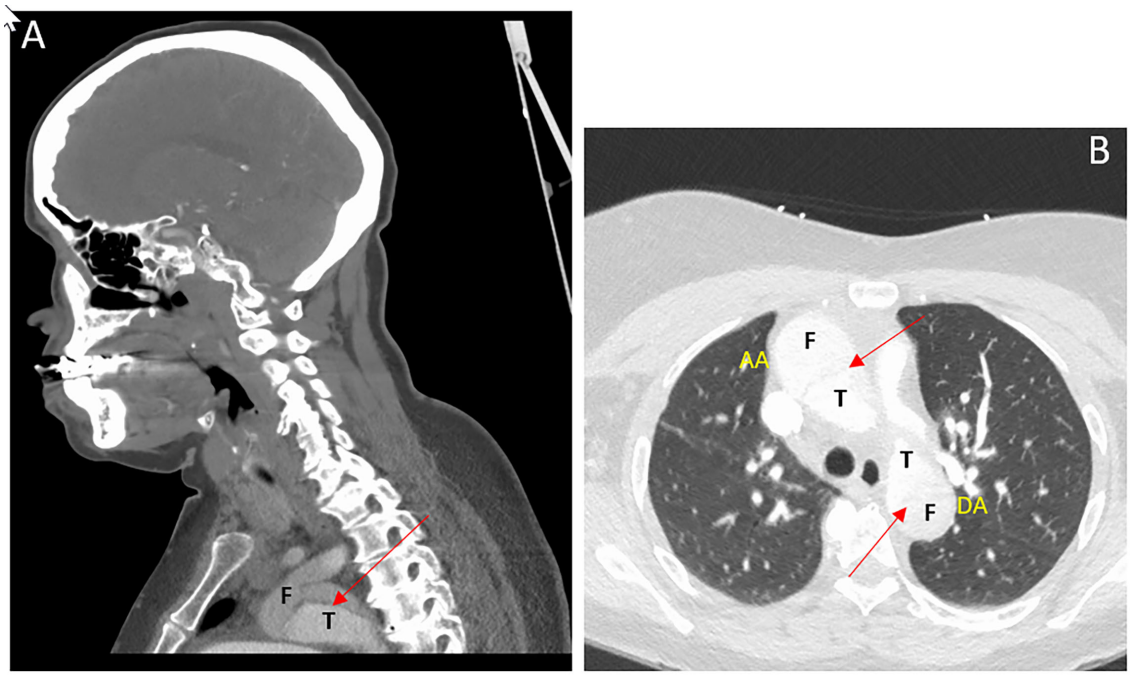

Figure 2. **Computed tomography angiography of the head and neck demonstrating an aortic dissection involving the aortic arch (*A*) as well as the ascending and descending aortas (*B*). *Red arrows* indicate the dissection flaps. AA = ascending aorta; DA = descending aorta; F = false lumen; T = true lumen.**

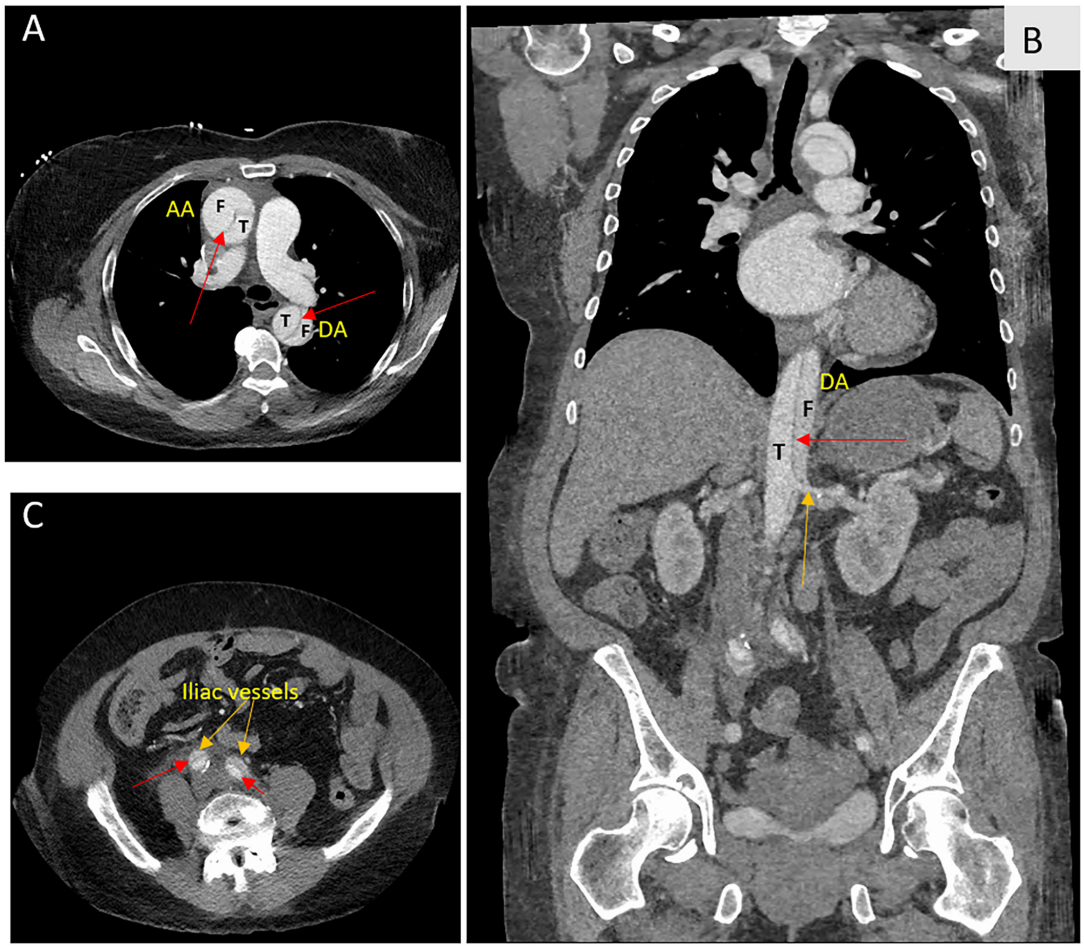

Figure 3. **Dedicated computed tomography angiography of the aorta demonstrating an aortic dissection involving the ascending and descending thoracic aortas (*A*), the abdominal aorta (*B*), and the iliac vessels (*C*). *Red arrows* indicate the dissection flaps. The orange arrow in (*B*) highlights the left renal artery arising from the true lumen of the abdominal aorta. AA = ascending aorta; DA = descending aorta; F = false lumen; T = true lumen.**

with valve resuspension, and left atrial appendage ligation. Postoperatively, she returned to the cardiovascular intensive care unit. An immediate postoperative TTE showed normal biventricular function, no dissection flap, and mild mitral and tricuspid regurgitation. Nicardipine was used for BP control. Her postoperative course was uncomplicated, except for a discharge TTE that revealed a reduced left ventricular ejection fraction (37%).

She was discharged on day 12 with a comprehensive regimen: apixaban, amiodarone, carvedilol, atorvastatin, aspirin, and diuretics. Instructions included medication adherence, a heart-healthy diet, activity modification, and close outpatient follow-up. At 10 weeks, a repeat TTE showed full recovery of left ventricular ejection fraction (64%) and normal diastolic function. She remained adherent to guideline-directed medical therapy, which included spironolactone, dapagliflozin, and carvedilol.

Discussion

This case underscores the diagnostic challenge of atypical AD presentations, particularly when pain is absent. Our patient presented with A.fib and a transient neurologic event—two findings that diverted attention from an underlying dissection. The assumption of A.fib-associated embolic TIA led to diagnostic anchoring and the absence of chest or back pain further reduced suspicion for AD. However, missed opportunities in which the AD could have been suspected earlier should be acknowledged:

1. **Clinical reasoning**: Anchoring on the diagnosis of A.fib-related TIA likely contributed to premature diagnostic closure. Considering a broader differential diagnosis that included vascular pathologies may have prompted simple, high-yield bedside assessments—such as bilateral BP measurements—ultimately leading to D-dimer testing and earlier CTA imaging.
2. **Imaging**: The chest radiograph was read as normal, but retrospective review suggested mediastinal widening—a key finding in AD. This serves as a reminder for clinicians to personally review imaging in the appropriate clinical context.
3. **Initial echocardiogram**: The TTE noted an AA diameter of 4 cm, a finding that was not pursued. Although not diagnostic, in the setting of hypertension, A.fib, and neurologic symptoms, this finding warranted further vascular imaging.

This case reinforces the importance of maintaining a high index of suspicion for AD in patients with neurologic symptoms, particularly when accompanied by risk factors such as hypertension. While rare, painless AD is well documented and often associated with worse outcomes due to delayed diagnosis (5, 6, 8, 10, 11).

Finally, the incidental diagnosis on head and neck CTA—ordered as part of a stroke work-up—highlights the value of thorough vascular imaging in select patients. This serendipitous discovery likely prevented catastrophic outcomes, as the dissection extended into multiple vascular territories, including cerebral vessels.

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