

## Seizure in the emergency department: Finding the zebra among the horses

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### ABSTRACT

Aortic dissection (AD) is a life-threatening emergency that mandates early and precise diagnosis. Seizure as an initial and exclusive manifestation of AD is extremely rare. The prevalence of this is sparsely available in the literature. We report the case of a 68-year-old male patient where the patient presented with seizures and discrepancy of blood pressures in the upper limbs. AD was suspected in this case with an atypical presentation.

**Key words:** Aortic dissection, Aortogram, Seizure

**A**ortic dissection (AD) is a life-threatening emergency that mandates early and precise diagnosis. Chest pain is the most common presenting complaint and it is classically described as sharp excruciating and having tearing or ripping quality with location migrating as the dissection extends along the aorta [1]. The frequency of neurological involvement is reported as between 17% and 40% [2]. Seizure as an exclusive manifestation of AD is very uncommon.

We report the case of a 68-year-old male with type A AD who presented with the complaint of a seizure.

### CASE REPORT

A 68-year-old male presented to the emergency department (ED) with a history of jerky movements of both upper limbs followed by unresponsiveness ½ h before arrival to the hospital. The tonic-clonic movements lasted for less than a minute. There was no history of headache, giddiness, chest pain, fever, or trauma. He was a known hypertensive and had suffered a cerebrovascular episode in the past for which he was on calcium channel blocker, beta-blocker, and angiotensin receptor blocker therapy. The patient did not have any history of seizures in the past.


On arrival to the ED, the patient was unresponsive, had noisy breathing, and a carotid pulse was not palpable on the right side. As per the basic life support algorithm, the decision to start compressions was taken; however, the carotid pulse when checked on the left side was present. In view of this, the chest

compressions were immediately withheld. On further assessment in the ED, it was noted that the patient had hypoxia (oxygen saturations 84% on room air), tachycardia (heart rate: 110/min), tachypnea (respiratory rate: 30/min), and the blood pressure recorded on the left side was 100/80 mmHg. No pulses or blood pressure could be recorded on the right side.

Resuscitation was initiated with high-flow oxygen and intravenous fluids. Intravenous levetiracetam 1 g was administered for seizures. His Glasgow coma score (GCS) was E2V2M5, moving all four limbs and pupils bilaterally equal and reacting to light. The clinical finding of asymmetric pulse and differential blood pressure reading on one side made the diagnosis of AD quite probable for this patient. There was no radiofemoral delay on examination.

Electrocardiogram revealed sinus tachycardia. Arterial blood gas was suggestive of metabolic acidosis with uncompensated respiratory alkalosis. In view of the low GCS, rapid sequence intubation was electively done in the ED. The patient was developing hypotension; hence, vasopressor support was initiated to maintain a mean arterial pressure of 65 mmHg. A portable chest radiograph revealed prominent superior mediastinum but was not conclusive (Fig. 1). A portable echocardiogram was suggestive of pericardial effusion with no signs of tamponade. Routine blood investigations were sent including hemogram and metabolic profile which were unremarkable. Further, the patient was shifted for computed tomography (CT) scan. CT brain was reported normal and CT aortogram revealed a Stanford type A intramural hematoma extending from the aortic root across the ascending aorta, arch, and the proximal descending aortic segment (Fig. 2) with hemopericardium (Fig. 3).

The cardiothoracic team explained the nature of the illness and grave prognosis of operative intervention in his case and the

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surgery was deferred by relatives as they refused the consent for the same. Over time, the pulselessness progressed to the right side as well as revealed by subsequent clinical examinations. The patient suffered a cardiac arrest later and expired within 6 h of ED presentation in the ICU.

## DISCUSSION

AD is the longitudinal splitting of the medial layer of the aortic wall provoked by the column of blood resulting in the formation of a false lumen within the dissecting plane. The literature reports the incidence of acute AD ranging from 2 to 3.5 cases/100 000 person-years which correspond to around 6000–10,000 cases annually in the United States [3]. The incidence is higher in men compared to women and increases with age [4]. True incidence is not easy to determine as the diagnosis of acute AD can be frequently missed at initial presentation and is a rapidly fatal condition the cause of death gets attributed to another cause. The most common risk factor attributing to this condition is poorly controlled blood pressure [1].

The most commonly used method for the classification of AD is Stanford type A or B depending on whether ascending aorta is involved or not [5]. The less commonly used DeBakey method classifies AD as Type I when involving ascending aorta, aortic arch, and descending aorta; Type II with ascending aorta involvement; and Type III with only involving descending aorta [6]. Accurate anatomical classification is essential as it

dictates the decision regarding operative versus non-operative management.

AD can mimic as acute coronary syndrome in ED and a high index of suspicion is required to confirm the diagnosis. Initial presentation of AD as neurological symptoms is infrequent but when present is often dramatic and can lead to obscuring of an underlying condition. Particularly when the presentation is primarily with neurological symptoms and associated with the absence of pain, the diagnosis can be difficult and delayed. The frequency of pain-free dissections is reported in the range of 5–15% [7]. The various neurological presentations as described in ADs are symptoms of acute ischemic stroke, disorders of consciousness, syncope, and epileptic seizures. These symptoms mostly appear at the onset of dissection and usually fade off to completely disappear in a short while of ED stay [2]. The fast recovery in such cases can be attributed to the transient arterial occlusion at the moment of dissection and its propagation.

Seizure as an initial manifestation of acute AD has been reported quite sparsely to date in the literature. One was reported by Mo *et al.* in a 46-year-old male who presented to ED with a sudden loss of consciousness and the right hemi-convulsive movements and later got diagnosed with type A AD [8]. The other case was reported by Finsterer *et al.* in an 88-year-old female as new-onset generalized tonic-clonic seizure in the absence of thoracic pain as the presenting sole complaint of type A AD [9]. This patient complained of brachialgia after awakening from the seizure episode which progressed to hemiplegia. AD was suspected and then confirmed on a CT aortogram. MR imaging of the brain was normal in this patient. Another case was reported by Rivera-Alvarez *et al.* in a 49-year-old male known hypertensive who presented in an unresponsive state after generalized seizures. Eventually, the patient developed hypotension, hence, CT aortogram was considered with neuroimaging. CT brain was normal but aortogram revealed a Stanford type A AD [10].

Our case also presented with a history of new-onset generalized tonic-clonic seizures and an absence of typical chest pain. The cause for seizure in our patient appeared to be cerebral hypoperfusion due to ongoing dissection. Another speculation for seizure could be a new cerebrovascular accident but that was ruled out by neuroimaging, wherein the CT scan of the brain was normal. The risk factor in our patient could be chronic hypertension which leads to medial degeneration of the aortic wall and predisposed the patient to AD. Differential readings of blood pressure and asymmetrically

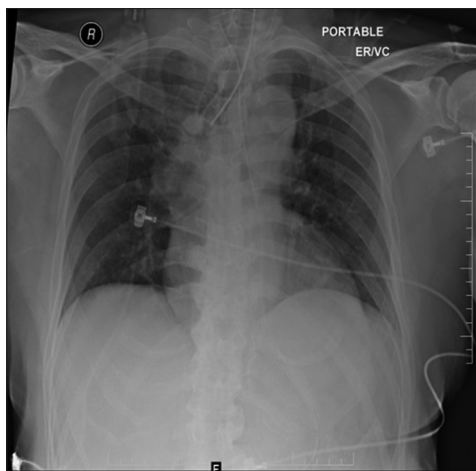


Figure 1: Chest radiograph showing possible widening of superior mediastinum

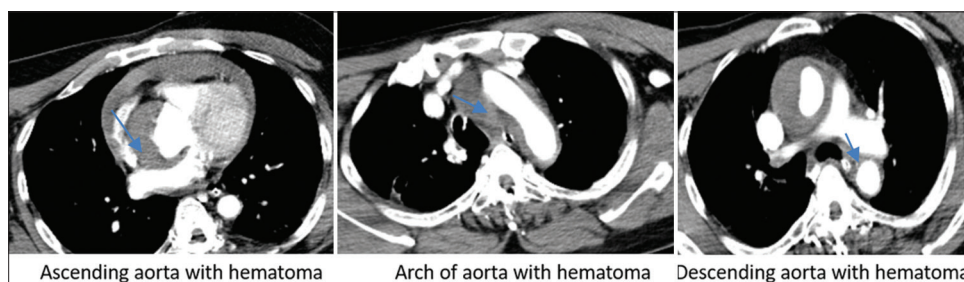
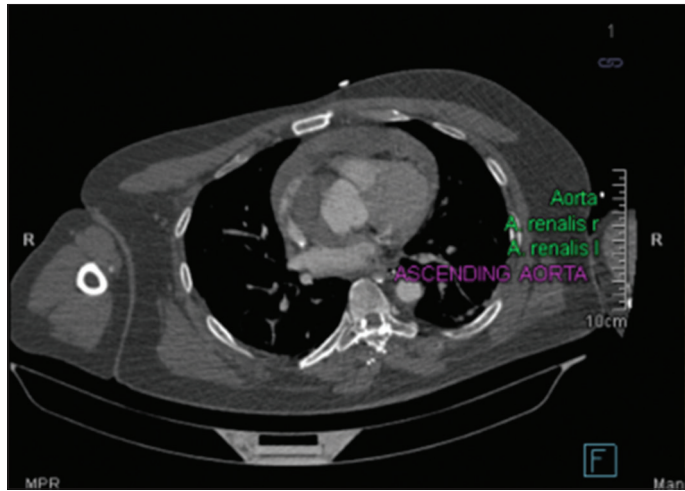


Figure 2: CT aortogram showing Stanford type A intramural hematoma extending from the aortic root across the ascending aorta, arch, and the proximal descending aortic segment



**Figure 3:** CT aortogram showing hemopericardium

palpable pulse provided the clue toward considering dissection as a probable diagnosis in our patient. Coarctation of aorta can present with pulselessness and radiofemoral delay while Takayasu's arteritis is also a pulseless disease due to large vessel arteritis. Further, the chest radiograph suggestive of the widened mediastinum and pericardial effusion on portable echo strongly prompted us to pursue additional definitive diagnostic aortic imaging and confirming the diagnosis of dissection.

## CONCLUSION

It is challenging yet pertinent for emergency physicians to unambiguously diagnose AD in a patient who has an atypical presentation to the ED with seizures and altered sensorium. This presentation can be a deterrent in obtaining an accurate and detailed clinical history. The differential diagnosis of AD should always be at the back of the mind of emergency physicians and the high index of clinical suspicion can prevent a missed diagnosis and unfavorable consequences.

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