Sudden onset of coma with anisocoria in a patient with type A aortic dissection: dilemma in management?

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Coma or stroke as a part of the clinical presentation of acute aortic dissection is generally considered to be a major contraindication for emergency surgery. Occasionally, clinical symptoms and signs may mislead us to an incorrect diagnosis or undue delay in management, which may cause a catastrophe in critically ill patients. In this study, we describe the abrupt onset of anisocoria and coma in a patient with aortic dissection that imparts a dilemma in subsequent management.

A 54-year-old man presented to the emergency ward with a history of syncopal attack and weakness in the bilateral lower limb in a medical emergency. There was no history of loss of consciousness, chest pain, shortness of breath, and seizures or trauma on admission. The patient was hypertensive for the past 1 year on irregular treatment. On examination, the patient was conscious, cooperative with bilateral equal and reactive pupils. He had a blood pressure of 130/50 mmHg in upper limbs and 140/55 mmHg in lower limbs. Chest auscultation showed diastolic murmur in the left third intercostal space. Bedside echocardiography showed severe aortic regurgitation and aortic dissection flap with severe left ventricular dysfunction (ejection fraction by modified Simpson's method=30%).

Immediately, the patient was transported for computed tomography (CT) angiogram to further delineate the dissection flap. CT angiography confirmed the presence of aortic dissection originating from the root of the ascending aorta and extending up to proximal common iliac arteries (Stanford type A) (Figs 1 and 2). Further, baseline investigations showed deranged coagulogram with prothrombin index (45%) and international normalized ratio of 2.06. Meanwhile, the patient had hemodynamic instability with a subsequent requirement of fluids, inotropes, and mechanical ventilation. So, the patient was planned for emergency exploratory surgical intervention. Surprisingly, it was observed that he had anisocoria with right side pupil 2 mm and left side pupil 4 mm in the ambient light while shifting the patient to the operation theater (Fig. 3). Neurological status of the patient was deteriorated to a score of 7 (M5E1VT) on the Glasgow coma scale. In view of the history of hypertension and deranged coagulogram, stroke was suspected. Noncontrast CT head was performed immediately, which showed normal brain parenchyma with no focus of ischemic or hemorrhagic stroke (Fig. 4). Again, CT angiography was carried out which revealed the presence of dissection flap extending into the supra-aortic vessels in addition to the previous findings.

After ensuring the absence of stroke, the patient was immediately shifted to the operating room and Bentall's procedure was performed under hypothermic cardiopulmonary bypass (CPB). He was weaned from CPB with milrinone 0.3 mcg/kg/min, adrenaline 0.05 mcg/kg/min, and noradrenalin 0.1 mcg/kg/min. The patient was transported to the cardiac surgical ICU and the trachea was extubated after the patient regained full consciousness. Postoperatively, anisocoria was further evaluated in both ambient and dim light for dilation lag, while in ambient light, right side pupil was 2 mm and the left

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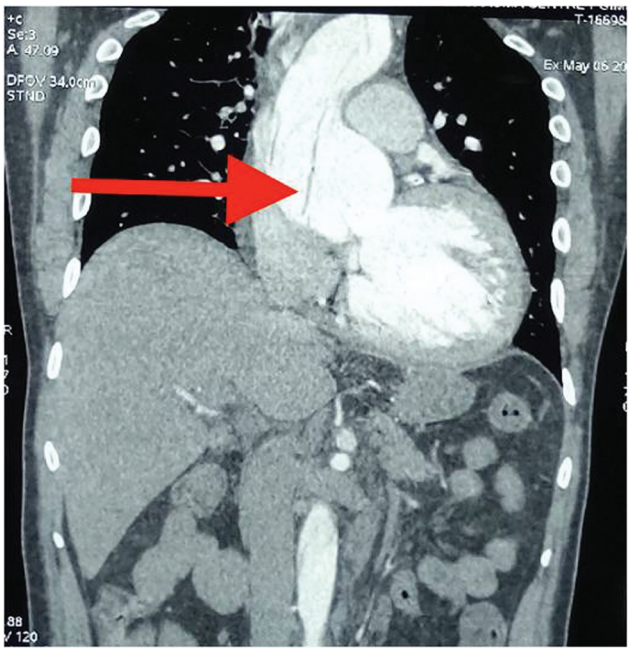
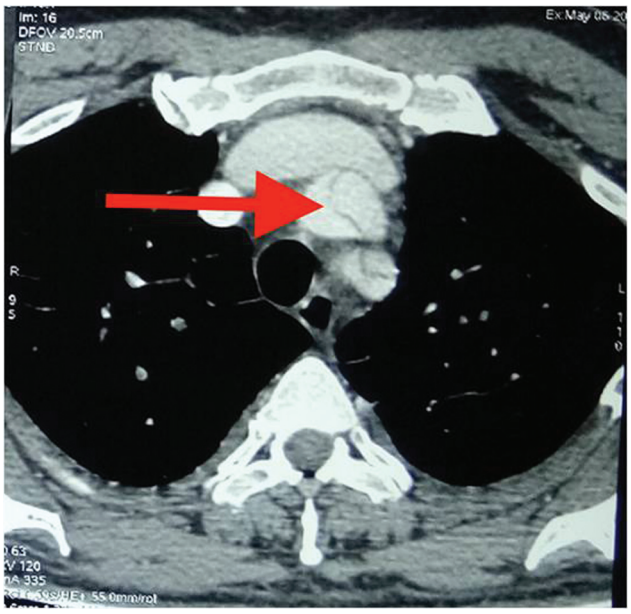
Figure 1



Computed tomography angiography of the aorta showing the dissection flap extending from the ascending aorta to bilateral common iliac arteries as shown by arrows.

Figure 2



Computed tomography angiography of the aorta showing dissection flap in the cross section of the aorta as shown by arrows.

side pupil was 4 mm, whereas in dim light, it was 3 and 6 mm, respectively. Warfarin and aspirin were started in the ICU immediately after surgery. Anisocoria persisted in the postoperative period for 5 days and disappeared later on. The patient was shifted to the ward on postoperative day 10 with no residual neurological deficit in the lower limbs. The patient was discharged from the hospital with minimal ptosis in the right eye.

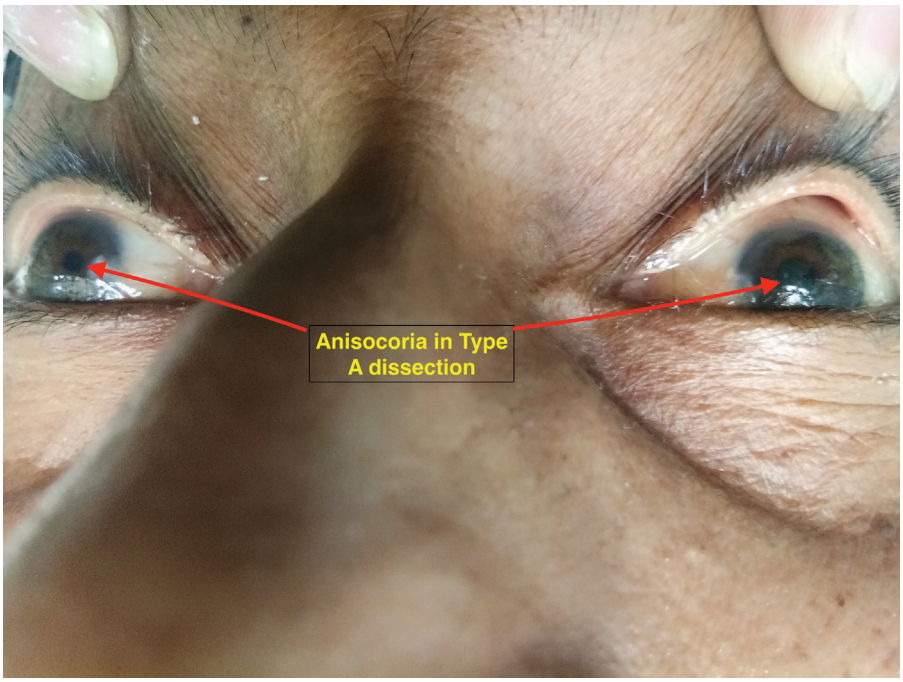
Acute aortic syndrome comprises acute aortic dissection and ruptured aortic aneurysm, which is an emergency life-threatening condition [1]. Knowledge of progression and extent of aortic disease is valuable because the treatment approach is highly reliant on the severity of the aortic disease. Although chest pain is the most common symptom in aortic dissection, frequency of pain-free dissections ranges between 5 and 15% [2]. Neurological symptoms may occur if the dissection involves carotid, vertebral arteries or arteries supplying the spinal cord. Previous studies mentioned that almost 10–40% of the patients had neurological symptoms in type A aortic dissection, with 50% being transient in nature [1,3]. Hence, clinicians should be alert in detecting aortic dissection in patients with neurological symptoms without having pain symptoms.

Stroke due to cerebral infarction and intracranial hemorrhage (ICH) is one of the causes for sudden onset of unconsciousness. Dissection is a cause of stroke in 0.4–4% of the general population, which predominantly involves the younger age group [4]. An abrupt onset of coma along with anisocoria in an aortic dissection patient with deranged coagulogram raised the suspicion of hemorrhagic stroke in our patient. Comatose patients with acute aortic dissection are generally considered moribund and are excluded from emergency surgery. Moreover, heparinization, CPB-related inflammation, and platelet dysfunction aggravate ICH and leads to poor survival of the patient [5]. Hence, it is important to rule out acute ICH before planning for emergency aortic surgery. However, the International Registry of Acute Aortic Dissection did not include coma as a risk factor for in-hospital death or as an impediment to long-term survival after surgical treatment of acute type A aortic dissection [5–7].

Risk of recurrent stroke is common in supra-aortic vessel dissections. Anticoagulation and antiplatelet drugs are commonly used to prevent recurrent stroke in patients having supra-aortic vessel dissection. If this therapy fails, balloon dilation and intravascular stent placement can be considered to restore luminal diameter of supra-aortic vessels [8].

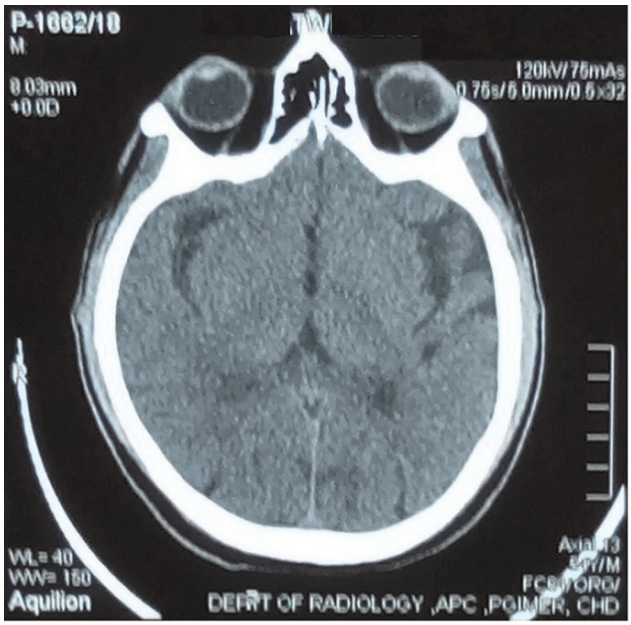
Anisocoria can be due to either physiological or pathological condition. Evaluating a comatose patient for anisocoria is highly challenging. Sudden onset of anisocoria and difference in size between pupils of more than 1 mm ruled out the possibility

Figure 3



Anisocoria in a patient with type A aortic dissection.

Figure 4



Noncontrast computed tomography head showing normal brain parenchyma with no signs of ischemia or bleeding.

of physiological anisocoria. Horner's syndrome is the one important cause for pathological anisocoria, which leads to ipsilateral miosis and ptosis. Again, identifying ptosis in the comatose patient is difficult. However, the presence of anisocoria with dilatation lag of the small pupil in our case was highly suggestive of Horner's syndrome. Moreover, in our case, miosis of the right eye ruled out the possibility of third cranial nerve paresis, which usually causes mydriasis. Finally, the involvement of the ipsilateral neck vessel in CT confirmed the above diagnosis. Transient, incomplete Horner's syndrome is an unusual complication following acute aortic dissection, extending into the carotid artery [9]. Probable reason for anisocoria in carotid dissection is due to localized inflammatory response, enlargement of the artery, and formation of hematoma which compress superior cervical ganglion.

Hence, it is important to keep in mind that sudden onset of coma or other neurological symptoms in aortic dissection might be due to the involvement of supra-aortic vessels, which would prevent unnecessary delay in emergency surgery.

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Conflicts of interest

There are no conflicts of interest.

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