## **Clinical Notes**

## An unusual descending aortic dissection with dramatic neurologic presentation

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cute aortic dissection (AAD) is a potentially life-Athreatening condition that requires rapid assessment and treatment. Classically, acute aortic dissection usually presents with an abrupt onset of severe pain in the chest, back, or abdomen. Patients often describe their pain as tearing or ripping.<sup>1</sup> Transient or permanent neurological symptoms at onset of aortic dissection are not only frequent (18-30% of the patients), but often dramatic and may mask the underlying condition.<sup>2</sup> Neurological symptoms are not necessarily associated with increased mortality, and occur mainly because of occlusion of carotid, vertebral, or spinal arteries, and vasa nervorum of peripheral nerves, or because of hypotension and related cerebral perfusion deficit.3 The most common symptoms of brain involvement include acute ischemic stroke, disordered consciousness, epileptic seizures, and syncope and are mainly related to ascending aortic dissection.<sup>4</sup> However, descending aortic dissection accompanying altered mental status is rare. In this report, a case of acute descending aortic dissection with dramatic presentation is described.

A 46-year-old male patient was admitted to our emergency department (ED) with paroxysmal vague pain in his low back. He arrived at the ED 6 days after the onset of symptoms. An abdominal CT scan performed in another hospital was normal. There was no history of any significant illness. On admission, he was alert and oriented (Glasgow Coma Scale score of 15/15). Routine physical examination did not reveal any abnormality except hypertension (160/98 mm Hg). His laboratory examination findings were almost within normal

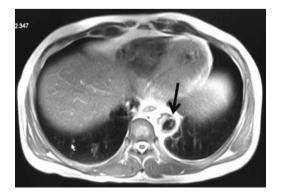


Figure 1 - An MRI enhanced scan in the coronal plane showing descending aortic dissection, and thrombogenesis in the pseudocoele.

limits. Three hours later he complained of severe low back pain and became drowsy followed by disturbance of consciousness (Glasgow Coma Scale score of 8/15, verbal response [2], eye opening [1], motor response to pain [5]), accompanied by blood pressure elevation to 198/122 mm Hg. The diameter of his bilateral pupils was around one mm, and the light reflex was dull. Funduscopic examination revealed neither papilledema nor hemorrhage. Neurologic examination was normal, and his mental status got better in the following one hour as his blood pressure dropped down after sodium nitroprusside treatment. An immediate head CT scan did not find any abnormality, but an MRI enhanced scan of the chest revealed a descending aortic dissection extending from the aortic isthmus to the right renal hilus level accompanying an intramural hematoma and an ulcer emerged in many segments (Figure 1). A toluidine red unheated serum test (TRUST) for syphilis screening was positive. A thoracoabdominal 3D reconstruction enhanced CT scan angiography was then performed to aid surgical planning. The CT images disclosed that the main branches of the arch aortic were not influenced, including innominate artery, left common carotid, and left subclavian. Shortly afterwards, he underwent cardiovascular surgery. Aortic aneurysm endovascular repair with stent-graft placement was performed and a week later he was discharged. There were no neurological symptoms on follow up after one month.

Acute aortic dissection is a cardiovascular emergency with poor outcome. Two factors, an initiating event and a structural weakness of the arterial wall, contribute to dissection of the aorta.<sup>5</sup> In our case, the syphilis related inflammation pathology may attribute the formation of aortic dissection. Classically, acute aortic dissection is described as a sudden onset of severe chest, back, or abdominal pain that is characterized as ripping or tearing in nature. Neurologic disorders have been associated with 18-30% of aortic dissection cases and may completely dominate the clinical picture.<sup>2</sup> These symptoms are due to general hypotension or dissection/occlusion of one or more aortic side branches supplying brain, spinal cord, or peripheral nerves. If there is no reason for the acute hypotension, symptoms of brain involvement are rare in descending aortic dissection because of no global cerebral malperfusion. The dramatic nature of the neurological deficit in this case was very unusual. In our case, head CT scan did not find ischemic stroke evidence and the enhanced CT scan angiography revealed no involvement of the main branches of the aortic arch. So his symptom of altered mental status was difficult to explain. We suppose that 2 factors may attribute to the neurologic symptoms: 1) the sudden

elevating blood pressure may lead to hypertension encephalopathy that will cause neurologic symptoms; 2) the focal brain lesions or global cerebral malperfusion as directly in consequence of cardiac disturbances or acute pain in aortic dissection.<sup>3</sup> Since acute aortic dissection is a process that may occur anywhere in the aorta, the clinical spectrum of presentation is broad and unpredictable. Any neurologic manifestations that occur unexplainably should also raise suspicion of aortic dissection especially with sudden pain. Although the clinical features of aortic dissection have gained wider appreciation, the diagnosis remains elusive in a substantial number of patients, necessitating clinical awareness and vigilance.

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