



CASE REPORT

Spontaneously Sealed Aortic Dissection Presenting with Multiple Ischemic Strokes

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Introduction

Aortic dissection is a rare and potentially fatal disease. It usually presents with severe chest pain radiating to the back, isolated back pain, or abdominal pain.¹ Atypical symptoms such as dizziness, vertigo, syncope, weakness or numbness are uncommon. The prevalence of neurologic symptoms in aortic dissection account for up to 42% of cases.² One study revealed that 29% of patients with type A aortic dissection presented with neurologic manifestations, but only two-thirds of these patients reported chest pain.³ Based on these percentages, approximately 10% of patients present with neurologic symptoms and without chest pain. Neurologic manifestation in aortic dissection included ischemic stroke (16%), ischemic neuropathy (11%), syncope (6%), seizures (3%), hypoxic encephalopathy (2%), and spinal cord ischemia (1%).³

Case Report

A 21-year-old Caucasian male was transferred to our facility for workup of acute ischemic stroke. He was playing a video game when he suddenly felt a severe headache followed by diplopia, vomiting, confusion, and generalized weakness. He had no relevant past medical history. His family history was unremarkable for atherosclerotic or thromboembolic diseases.

The patient's cardiovascular examination was normal. He had no carotid bruit. He was

lethargic and confused. There was evidence of left-sided weakness, although a complete neurologic examination was limited by the decreased level of consciousness.

A CT scan showed an acute ischemic infarct involving the right temporal lobe. An MRI of the brain with contrast revealed an ischemic infarct involving the right occipital lobe and the posterior aspect of the right temporal lobe. Another infarct involved the left superior cerebellum. (See Figures 1 and 2.) A hypercoagulable workup on admission revealed positive antiphospholipid antibodies.

Given the presence of multiple strokes in different vascular territories, a cardioembolic phenomenon was suspected and the patient underwent a transesophageal echocardiography (TEE). The TEE was negative for cardiac emboli. However, it revealed the presence of a small dissection flap in the ascending aorta (Figure 3) and thickening of the intima of the descending aorta (Figure 4). These findings suggested an aortic dissection with a dissecting channel that healed spontaneously and left a small flap in the ascending aorta. An MRA of the head and neck was negative for aneurysm, dissection, or stenosis. A CTA of the chest showed no evidence of dissection or other abnormalities. The TEE was repeated one week later, as a preoperative workup for surgical repair of the dissection, and showed a complete resolution

of the dissection flap and decreased prominence of the intimal thickening in the descending aorta.

Surgery was deferred and the patient was treated conservatively with blood pressure control, physical therapy, and anticoagulation for possible antiphospholipid syndrome. He improved progressively and was discharged home to continue physical therapy. In a 6-month follow-up exam, he showed no evidence of neurologic deficit on the physical

examination. The TEE was negative. The antiphospholipid antibodies were normal indicating a false positive test initially. The diagnosis of antiphospholipid syndrome was unlikely because of the repeated negative antiphospholipid antibodies test and the absence of family history of hypercoagulability. Thus, anticoagulation was stopped and the embolic strokes were assumed to be secondary to the aortic dissection.

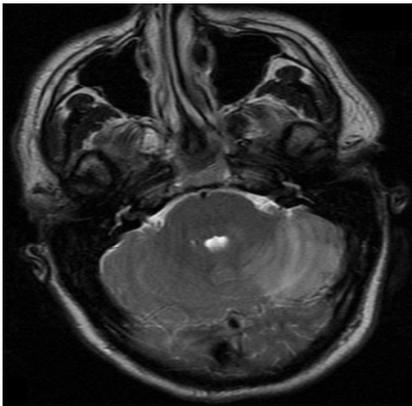


Figure 1. A T2 weighted MRI indicated an acute ischemic infarct involving the left superior cerebellum in the region of the left superior cerebellar artery.

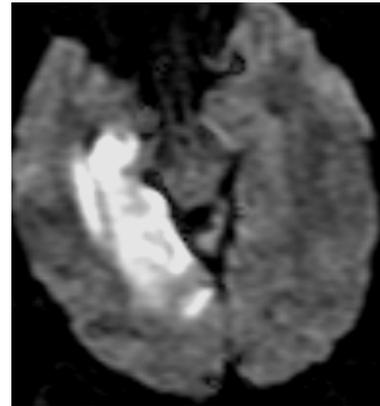


Figure 2. A diffusion weighted image showed acute ischemic infarcts of the right occipital and the posterior aspect of the right temporal lobe in the region of the right posterior cerebellar artery.



Figure 3. Two views of the ascending aorta on the trans-esophageal echocardiogram show the intimal flap just above the aortic valve (red arrows).



Figure 4. Intimal thickening of the descending aorta appears to represent, in the setting of the dissection flap in the ascending aorta, an aortic dissection that has sealed and healed spontaneously.

Conclusion

Cerebral infarcts in patients with aortic dissection are due to common carotid occlusion by progression of the false lumen with subsequent thrombosis or by intimal detachment.⁴ Another possible mechanism is an artery-to-artery embolism from a thrombus developed on the intimal surface of the dissected artery. It is difficult to know which mechanism is involved without an autopsy. In our case, the most likely mechanism was embolization, since there was no evidence of dissection in the carotids or cerebral arteries on the imaging. On the other hand, it is unusual for the dissection flap to follow two different pathways: the right posterior cerebral artery and the left superior cerebellar artery. These were the arteries corresponding to the vascular territories of the ischemic strokes in our patient.

The natural pathophysiology of aortic dissection involves the development of an intima-medial tear. The tear could be limited (incomplete dissection) or progress to form a dissecting channel and subsequently an aneurysm or rupture of the aorta.⁵ Spontaneous healing of aortic dissection is very rare. The mechanism of spontaneous healing of aortic dissection involves clotting of the hematoma followed by fibrosis. Another possible mechanism is endothelialization and obliteration of the dissecting channel.² Only a few cases of spontaneous healing of aortic dissection have been reported.^{2,5} A retrospective study revealed only four cases.⁵ A longitudinal study over 27 years identified five cases on autopsy.²

Transesophageal echocardiography is the best imaging modality for the diagnosis of

acute aortic dissection.⁶ It has high sensitivity (97-99%) and specificity (reaching 100% by the addition of M mode)^{6,7}, and is considered a class I indication.⁸ It is particularly important for the proximal ascending aorta and aortic valve. The CT scan has a good sensitivity (83-98%) and specificity (87-100%).⁶ However, the sensitivity to detect intimal flap is low (less than 75%).⁹ It is a class II indication⁸ and is superior for the imaging of the aortic arch vessels.¹⁰ MRI is comparable to the TEE in sensitivity and specificity, but usually is not performed in the acute settings.¹¹ It is the modality of choice for chronic dissection.⁸

Our case had many interesting features. First, the patient presented with ischemic stroke instead of typical chest pain and was completely free of chest pain during his hospitalization. The aortic dissection apparently was healed and sealed spontaneously at the time of diagnosis which is a rare outcome. In addition, the neurologic symptoms and signs resolved completely in a few months. The combination of these rare events in the same patient made this case unusual. To our knowledge, no similar cases have been reported in the literature.

Aortic dissection should be suspected in young patients with ischemic stroke, especially in the absence medical risk factors. This case also emphasized the importance of TEE as a diagnostic tool for the diagnosis of acute aortic dissection and as a workup for the patient with ischemic stroke. TEE should be interpreted carefully as a subtle abnormality, such as the intimal flap, may be missed easily.

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