Coarctation of the Abdominal Aorta
An Uncommon Cause of Arterial Hypertension and Stroke

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A 14-year-old male with no previous medical history presented with headache, left third cranial nerve palsy, and generalized tonic-clonic seizures. Magnetic resonance imaging of the brain revealed subarachnoid hemorrhage secondary to an aneurysm of the left posterior communicating artery. The patient underwent successful transcatheter coil occlusion of the aneurysm, and his neurological symptoms improved (Figure 1).

During admission, he remained hypertensive, with an arterial blood pressure of 170/96 mm Hg in the upper limbs despite medical treatment with atenolol, amlodipine, and enalapril. On physical examination, an abdominal systolic bruit was heard, and weak femoral pulses were noted. Blood pressure in the right and left arms was similar, but there was a significant differential pressure (50 mm Hg) compared with the lower limbs. Aortic coarctation and renal artery stenosis were considered to be the causes of arterial hypertension.

An echocardiogram showed moderate left ventricular hypertrophy and preserved systolic function. No abnormality in the aortic arch or proximal descending aorta was noted on echocardiography. An abdominal ultrasound showed a small abdominal aorta with diffuse narrowing and increased peak systolic velocity (509 cm/s). Presence of runoff flow throughout the entire diastole was also noted (Figure 2). Magnetic resonance angiography of the aorta confirmed the diagnosis of coarctation of the abdominal aorta and showed a narrowing of considerable length starting 5 cm below the diaphragm. This narrowing tapered to become a critical stenosis at the level of origin of the renal arteries (Figure 3). The abdominal aorta measured 2.8 mm at its smallest point and was consistently poorly opacified. Prominent collateral circulation from the mammary artery and the intercostal arteries to the abdomen was present.

At cardiac catheterization, there was a pullback gradient of 56 mm Hg from the ascending aorta to the descending aorta. Aortic coarctation was confirmed with pullback measurement of 56 mm Hg at the level of the renal arteries (Figure 3). At surgery, after resection of the coarctation segment, repair was performed with an arterioplasty device. The patient made a complete recovery and was discharged home on day 8 postoperatively.

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Figure 1. A, Cerebral angiogram demonstrating a large ruptured aneurysm (arrow) of the posterior communicating artery. B, Angiogram after endovascular coil embolization showing exclusion of the aneurysm.
abdominal aorta under general anesthesia. Balloon angioplasty of the stenotic segment was performed. Significant improvement in aortic and renal arteries diameters was noted after the procedure. An aortogram immediately after dilatation showed good opacification of the entire abdominal aorta (Figure 4). The gradient fell to 10 mm Hg. The patient has remained normotensive since the procedure.

Figure 2. A, Abdominal ultrasound showing a prolonged narrowing of the abdominal aorta. B, Continuous Doppler signal showing peak velocity of 500 cm/s and runoff throughout the entire diastole.

Figure 3. Magnetic resonance angiography of the aorta in sagittal (A) and coronal (B) planes showing a diffuse narrowing of the abdominal aorta that involved the origin of the celiac trunk, superior mesenteric artery, and both renal arteries. The stenosis was 5 cm long. The diameter of the aorta proximal to the stenosis was 10 mm; the minimum diameter of the aorta was 3 mm. Note the prominent mammary artery providing collateral circulation to the abdomen (upper arrow).
Coarctation of the abdominal aorta, also known as middle aortic syndrome or mid-aortic dysplastic syndrome, is a clinical condition caused by segmental narrowing of the abdominal or distal descending thoracic aorta secondary either to a congenital anomaly in the development of the abdominal aorta or to one of several acquired conditions. Acquired conditions include neurofibromatosis, retroperitoneal fibrosis, fibromuscular dysplasia, mucopolysaccharidosis, and Takayasu’s arteritis, all of which may result in narrowing of the abdominal aorta and other vessels. In Takayasu’s arteritis, the use of antiinflammatory agents may be useful.

This case highlights (1) the importance of a thorough physical examination, which in this case led to the discovery of an abdominal bruit and weak femoral pulses that raised the suspicion of aortic coarctation or renal artery stenosis as the cause of hypertension; (2) the association between anomalies of the aorta and cerebral arteries aneurysms (it has been reported that up to 10% of patients with aortic coarctation have cerebral aneurysms); and (3) that coarctation of the abdominal aorta, although rare (2% of all coarctations of the aorta) can be the cause of secondary hypertension and should be considered during the diagnostic workup of hypertension, especially in young patients.

Disclosures
None.

References

Figure 4. Aortogram before (A) and after (B) balloon dilatation of the abdominal aorta. After angioplasty, the diameter of the abdominal aorta was significantly increased.
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