Atherosclerosis-induced aortic coarctation: a case report

Abstract
Acquired aortic coarctations are uncommon but potentially life threatening. They are poorly reported in the literature, primarily due to their rarity. We describe a 36-year-old Asian woman with a longstanding history of moderate hypertension. She developed atherosclerosis in her abdominal aorta, in the region of the coeliac trunk and superior mesenteric arteries. Her condition deteriorated after a motor vehicle accident, upon which her blood pressure could no longer be controlled, despite the use of several antihypertensive medications. The patient experienced claudication and weakness in both of her legs. A CT angiogram revealed a near complete suprarenal aortic occlusion, which accounted for her presentation. A left axillo-femoral bypass was performed. The benefits of the surgery were seen immediately. Her blood pressure improved and her other symptoms diminished.

Case report
This case describes a 36-year-old Asian woman who was referred to a specialist for hypertension in 1999. Previously she had two episodes of pregnancy-induced hypertension in 1989 and 1994, treated with bed rest only. She had a relevant family history, as her mother had developed hypertension at age 60. The patient had no history of alcohol consumption or smoking, and no other medical history of note. Her clinic blood pressure in May 1999 was elevated at 160/90mmHg. A 24-hour ambulatory blood pressure monitor showed that her average blood pressure was also elevated at 142/86mmHg. This was consistent with mild hypertension, and the patient was prescribed lisinopril 20mg/hydrochlorothiazide 12.5mg, which brought her blood pressure under control at 124/80mmHg. At three-month follow-up her blood pressure was 135/80mmHg and she was discharged back to her family physician for ongoing management.

Five years later, in 2004, the patient was involved in a motor vehicle accident, immediately after which her blood pressure elevated to 180/100mmHg and could not be controlled with her current medication. She was hospitalised for one week after the accident. During this time, she began to experience pain in both lower extremities and had difficulty walking. She also complained of abdominal pain. Her physicians initially attributed these symptoms to her motor vehicle accident. In 2005, the patient was referred back to the specialist for her uncontrolled hypertension.

Secondary causes of hypertension such as adrenal tumour and renal artery stenosis were tested for. Tests showed a 24-hour vanillylmandelic acid (VMA) of 16, renin 5.48 and aldosterone 274, which were all within the normal range and ruled out an adrenal cause. A nuclear renal scan was performed and ruled out renal artery stenosis. Normal eGFR, creatinine, urea and electrolyte levels indicated normal renal function.

An echocardiogram was ordered to check her heart function under the duress of the acute hypertension. It revealed normal left ventricular (LV) function but borderline LV hypertrophy; her LV wall thickness and interventricular (IV) septum both measured 12mm. Essential hypertension was still the diagnosis at this time. Amlodipine 10mg once daily, atenolol 100mg once daily and losartan 100mg/hydrochlorothiazide 25mg once daily were prescribed to control her blood pressure. Propylthiouracil 50mg twice daily was also prescribed to control her hyperthyroidism, which had been recently diagnosed. After one month of
this regimen, her blood pressure remained elevated in excess of 180/100mmHg. Consequently, her medications were titrated upwards to amlodipine 10mg bid, atenolol 100mg once daily, terazosin 4mg bid, candesartan/hydrochlorothiazide 32/25mg once daily and aliskiren 300mg once daily. After one month of this new regimen, her blood pressure still remained elevated at 170/95mmHg.

Minoxidil 5mg bid and furosemide 40mg bid were added to the current regimen. Two weeks later her brachial blood pressure dropped to 135/75mmHg; however, she developed acute anuria and her creatinine levels rose dramatically (from 226 to 588µmol/L), indicating ischaemic nephropathy. The patient also complained of weakness, cramping, coldness and numbness in her feet and lower legs, and abdominal cramping. The patient’s leg and foot pulses were weak and blood pressure was unattainable in the lower extremities. Minoxidil and furosemide were ceased immediately. Her brachial blood pressure rose again to 185/105mmHg. Her creatinine level fell back to 202µmol/L, indicating that her kidney function had returned to normal.

An underlying vascular problem was suspected at this point. A Doppler ultrasound was performed, which indicated irregular blood flow within the abdominal aorta. The ankle/brachial indices were 0.47 for the right side and 0.46 for the left side (values <0.9 indicate arterial disease). An abdominal aortic ultrasound indicated occlusion of the distal aorta and both common iliac arteries. A CT angiogram showed severe atherosclerosis in the abdominal aorta.

The CT angiogram (Figures 1a and 1b) revealed (>90%) occlusion of the subdiaphragmatic aorta above the coeliac artery. There was extensive calcified plaque at the level of T12 with associated occlusions (90-99%) at the origins of the coeliac and superior mesenteric arteries. Collateral arteries were present. A hypertrophied inferior mesenteric artery and the hypogastric arteries supplied the entire bowel. The inferior mesenteric artery filled the arterial beds of the coeliac and superior mesenteric arteries through collateral vasculature. Extensive collateralisation from the intercostal internal mammarys to superior epigastric arteries and abdominal wall vessels allowed retrograde refilling of the iliac vessels and anterograde refilling of the femoro-popliteal system. No significant stenoses below the origin of the superior mesenteric artery were noted. This indicated that the patient had significant atherosclerotic disease of the aorta, resulting in a coarctation of the aorta. The physicians postulated that the blunt trauma she sustained during her motor vehicle accident created shear forces that dislodged plaque, resulting in acute subtotal occlusion of her aorta and ultimately the acute elevation of her blood pressure. Blunt trauma was felt to be the case because she did not have any typical risk factors for spontaneous thrombosis, such as heavy smoking or poorly controlled diabetes. There was also a temporal relationship between the motor vehicle accident and the acute rise in her blood pressure.

The patient was referred to a vascular surgeon. An aortic reconstruction would be precarious with a >20% mortality risk. There was also the risk of other complications such as paralysis, mesenteric infarction, and acute cortical necrosis of her kidneys. A left axillo-femoral bypass was performed instead. A bypass graft was placed subcutaneously along the side of her anterior chest and abdomen, joining the axillary and femoral arteries. Post-operatively, the patient’s symptoms improved considerably. Generally, she no longer has the claudication pain in her abdomen and legs. Upon exertion, she experiences claudication in her right leg due to the left axillo-femoral bypass. The right axillary blood vessels were preserved for future use in case the left graft occluded. Her physicians were able to reduce her blood pressure to 150/70mmHg without...
Coarctations of the aorta can result from congenital or acquired conditions. Clinical features such as uncontrolled hypertension, diminished or absent lower limb pulses, and intermittent claudication present in most cases. Diagnosis can be delayed due to the various locations of aortic coarctations and the potential formation of extended collateral vasculature. Surgical treatment is required. Close monitoring of the patient’s ankle/brachial index after surgery is essential.

Discussion

There are two types of aortic coarctation: congenital and acquired. Acquired aortic coarctations constitute only 2% of aortic coarctations. They can be caused by aortic dissection due to extension of a coronary artery dissection, Marfan syndrome, large-vessel vasculitis such as Takayasu arteritis, mycotic aneurysms, and severe atherosclerosis. Cases of abdominal aortic coarctations have been reported with anatomical variants, resulting in an array of clinical features. They do, however, commonly present with lower limb intermittent pain and ischaemia, differences in systolic blood pressure between upper and lower extremities, abdominal pain and hypertension. Other features may include spinal cord compression-like symptoms, urinary incontinence, impotence in men, inability to walk long distances, and mild sensory loss in the lower body. The variety of symptoms can make diagnosis difficult. Formation of extended collateral vasculature can maintain a basal perfusion and prevent the manifestation of acute ischaemia in the lower extremities for a long time, further delaying diagnosis.

Conclusion

Coarctations of the aorta can result from congenital or acquired conditions. Clinical features such as uncontrolled hypertension, diminished or absent lower limb pulses, and intermittent claudication present in most cases. Diagnosis can be delayed due to the various locations of aortic coarctations and the potential formation of extended collateral vasculature. Surgical treatment is required. Close monitoring of the patient’s ankle/brachial index after surgery is essential.

Further reading


References


