Infrarenal aortic coarctation in a 15-year-old with claudication

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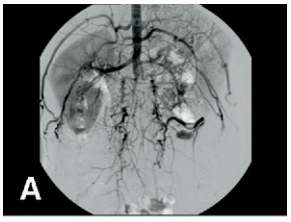
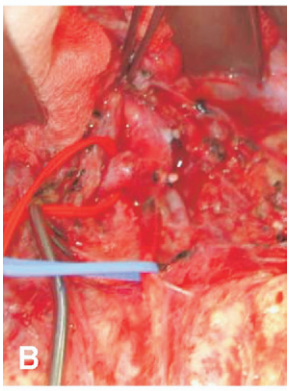
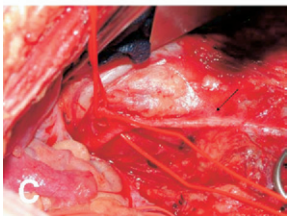
A 15-year-old girl with a history of hypertension requiring left nephrectomy at age 10 presented with a 1-year history of progressive bilateral lower extremity claudication. Ankle-brachial indexes were 0.6, and pulses were absent. Renal function was normal. Computed tomographic angiography showed a complete absence of the infrarenal aorta, with collateral flow via bilateral internal mammary arteries reconstituting normal iliofemoral systems via the epigastric arteries, referred to as the *Winslow pathway* (cover image).[1] The right renal artery appeared normal. Aortography demonstrated similar anatomy (A).







Operative findings included severe infrarenal aortic (B, *blue loop*) and common iliac artery (C, *arrow*) hypoplasia. The right renal (B, *red loop*), superior mesenteric, and lumbar arteries were widely patent. A 14 × 7-mm bifurcated polytetrafluoroethylene graft was interposed between the aortic stump and the iliac bifurcations. The patient's claudication resolved, pulses became palpable, and ankle-brachial indices measured 1.0.

Coarctation of the abdominal aorta is a rare entity of unknown etiology. Proposed mechanisms include anomalous development, infection, obliterative panarteritis, and fibromuscular dysplasia. Pathology usually reveals intimal fibroplasia. Patients typically present between the first and third decades of life with uncontrolled hypertension secondary to renal artery stenosis.[2] Claudication is less common and is seen in cases of infrarenal hypoplasia. The visceral arteries are involved in more than 20% of cases, but mesenteric ischemia is rare because of collateral formation. Physical examination reveals thoracoabdominal bruit and diminished lower extremity pulses. Arteriography aids in defining the location and extent of disease.

Four types of aortic coarctation have been described: type I, suprarenal coarctation and renal artery stenosis; type II, infrarenal coarctation and renal artery stenosis; type III, suprarenal coarctation and normal renal arteries; and type IV, infrarenal coarctation and normal renal arteries. For types I to III, surgical intervention is aimed at improving renal blood flow and preserving renal function. Repair may include thoracoabdominal aortic bypass, anatomic aortic reconstruction (grafting or patch angioplasty), renovisceral bypass, or renal autotransplantation. Surgical intervention for type IV coarctation is indicated in cases of severe claudication and consists of aortic reconstruction.[3]

Regardless of the anatomic variant, surgical intervention is required to alleviate uncontrolled hypertension and symptoms of claudication. Untreated patients experience premature death secondary to the morbid sequelae of uncontrolled hypertension. Long-term follow-up (in excess of 20 years) demonstrates reconstructive success, symptom relief, and normal quality of life.[4]

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