Abdominal pain after stenting for aortic coarctation

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ABSTRACT

A 56-year-old man underwent stenting for treatment of coarctation of the aorta. He subsequently developed severe abdominal pain and paradoxical postoperative hypertension. Abdominal computed tomography revealed two long mesenteric artery lesions. After potent antihypertensive therapy, he did not develop intestinal necrosis, and he was discharged with no recurrent abdominal pain. Although postcoarctectomy syndrome is rare in the current era of nonsurgical intervention for coarctation of the aorta, it can still occur and should be carefully managed to prevent intestinal necrosis. (J Vasc Surg Cases and Innovative Techniques 2020;6:678-80.)

Keywords: Coarctation of aorta; Postcoarctectomy syndrome; Stent

Coarctation of the aorta (CoA) occurs in approximately 1 in 3000 live births,[1] and its surgical repair was introduced in the 1940s.[2] Surgery was the only treatment choice for patients in the ensuing decades until balloon angioplasty emerged as an alternative in the late 1970s.[3] Stenting is now the first-line treatment for most postpubertal patients.[4] We herein demonstrate a rare complication after nonsurgical intervention for CoA. The patient's consent was obtained for the publication of this report.

CASE REPORT

A 56-year-old man was referred to our hospital for paroxysmal atrial fibrillation. Physical examination showed high blood pressure in both arms and a grade 3 of 6 systolic murmur at the left upper sternal edge. Pulmonary valve stenosis was suspected by echocardiography. We then performed left and right heart catheterization. Although no pressure gradient was found between the right ventricle and pulmonary artery, a 78-mm Hg gradient was present between the ascending and descending aorta. Subsequent angiography confirmed a coarctation with a minimal lumen diameter of 5 mm in the descending aorta (Fig 1, *A*) and a subarterial ventricular septal defect.

After discussion among the heart team of our institution, we introduced a balloon catheter (18 mm × 4.0 cm) and a Cheatham-Platinum stent (18 mm × 4.0 cm) (Numed, Bronx, NY) in the coarctation site through a 14F right femoral sheath. Postprocedural angiography showed satisfactory positioning (Fig 1, *B*) and a left pressure gradient of only 5 mm Hg.

The patient reported an abrupt onset of severe abdominal pain with sweating 12 hours after the procedure. His body temperature was normal. Physical examination revealed an elevated blood pressure in both arms (180/100 mm Hg, compared with 163/79 mm Hg before the procedure), diffuse abdominal distention, and hypoactive bowel sounds. Laboratory evaluation showed a normal white blood cell count, neutrophil count, lactate dehydrogenase level, cardiac troponin I level, creatine kinase isoenzyme MB level, amylase level, and lipase level; however, the patient had a mildly elevated high-sensitivity C-reactive protein level (11.39 mg/L; reference range, 0-3 mg/L) and D-dimer level (0.45 mg/L in D-dimer units; reference range, 0.00-0.24 mg/L). To exclude artery damage and stent malposition, an enhanced computed tomography examination was performed 30 hours after the procedure. This examination revealed two long endovascular low-density lesions: one in the side branch of the superior mesenteric artery and the other extending from the ostium of the inferior mesenteric artery (Fig 2). The stent position was excellent, and no abnormalities associated with other organs were found.

Postcoarctectomy syndrome was suspected; therefore, we administered intravenous urapidil and papaverine with nil per os for 5 days to ensure complete bowel rest. The patient's abdominal pain gradually resolved in 6 hours, and his blood pressure substantially decreased to about 120/80 mm Hg. The patient reported paroxysmal severe abdominal pain each night at midnight for the next 4 days accompanied by elevated blood pressure, and potent antihypertensive therapy always effectively resolved the pain. However, biomarkers of bowel infarction, such as the lactate dehydrogenase level and white blood cell count, remained within the reference range throughout his clinical course, and his abdominal pain disappeared on the day 6 after the procedure.

The patient was discharged 8 days after the procedure. No abdominal pain had recurred at the 1-year follow-up, and he was able to gradually withdraw the antihypertensive drugs with a controlled blood pressure of about 130-140/80-90 mm Hg.

DISCUSSION

In the 1950s, some surgeons began to report patients with severe abdominal pain after surgical resection of CoA; this phenomenon was termed postcoarctectomy

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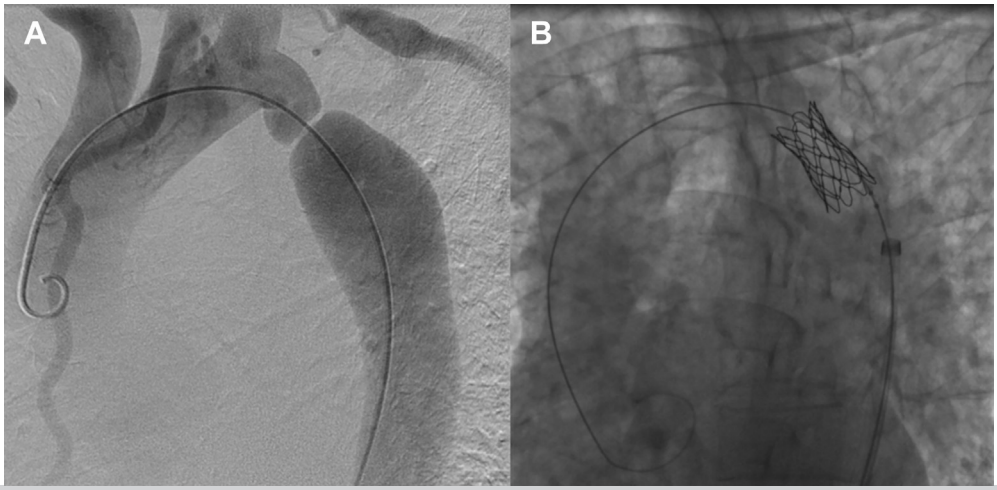

Fig 1. Procedural angiography. **A,** Before the procedure. **B,** After the procedure.

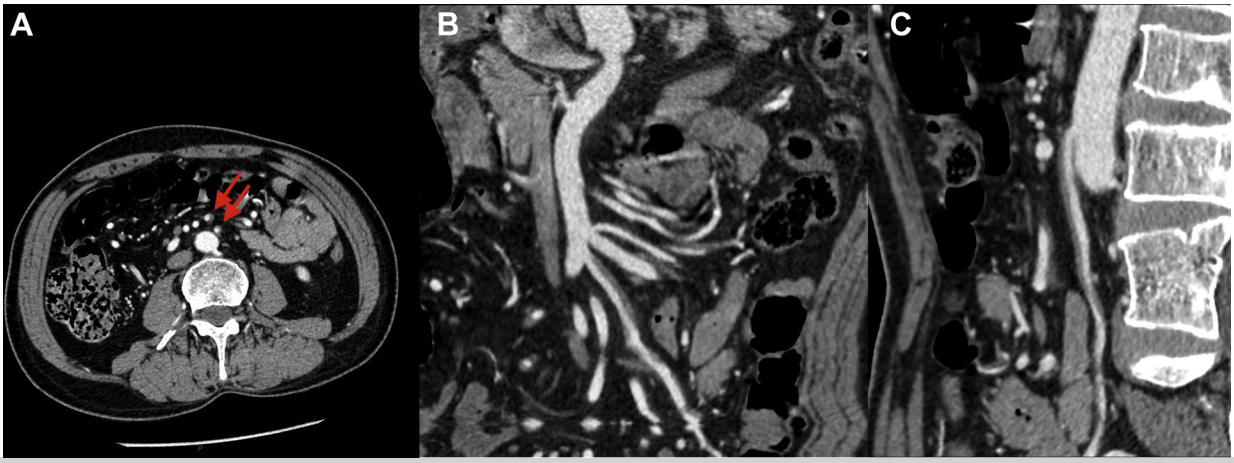

Fig 2. Postprocedural abdominal computed tomography. **A,** Endovascular crescent lesions in the superior and inferior mesenteric arteries (*red arrows*). **B,** Reconstruction of the superior mesenteric artery. **C,** Reconstruction of the inferior mesenteric artery.

syndrome.[5] In some extreme cases, even intestinal necrosis occurred. Mesenteric artery changes were confirmed by pathologic examination[5] and found to be reversible by angiographic examination.[6,7] This phenomenon is considered to originate from a disordered neuroendocrine reflex (carotid baroreceptors, sympathetic reflex, and renin-angiotensin-aldosterone system)[8-10] and to be closely correlated with postoperative paradoxical hypertension.[11]

Antihypertensive therapy before and after the procedure is very important to prevent and manage paradoxical hypertension or postcoarctectomy syndrome. Prophylactic propranolol was proven to control the perioperative blood pressure and plasma renin activity in the 1980s.[12] Short-acting intravenous medications, such as nitroprusside, nicardipine, esmolol, and urapidil, are optimal choices to balance high blood pressure and hypoperfusion.[10,13] Papaverine can relax the smooth muscles of the vessel wall and have been proven effective for mesenteric ischemia; additionally, one report showed its value in postcoarctectomy syndrome.[7]

To our knowledge, postcoarctectomy syndrome is extremely rare in the current era of nonsurgical intervention.[14] To our knowledge, the only such report is that of the COAST II trial, in which abdominal pain occurred in 1 of 158 patients without remarkable findings on abdominal computed tomography scans[15] compared with almost one-fifth of patients in the early era of surgical resection.[5] The main differences between surgery and nonsurgical intervention remain unclear, but they may involve the immediate and efficient blood pressure control[15,16] and the relatively little neuroendocrine disturbance[14] associated with nonsurgical intervention.

CONCLUSIONS

Although postcoarctectomy syndrome is rare in the current era of nonsurgical intervention, it still occurs.

Early recognition and appropriate blood pressure control are very important to prevent its progression.

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