Delayed neurologic deficit after endovascular abdominal aortic aneurysm repair

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Paraplegia or paraparesis secondary to spinal cord ischemia is an extremely rare complication after elective repair of abdominal aortic aneurysm. We report delayed paraparesis after endovascular abdominal aortic aneurysm repair in which one hypogastric artery was unintentionally occluded due to atheroembolism. A spinal catheter was immediately inserted after onset of paraplegia to promote cerebrospinal fluid drainage, which partially reversed the neurologic deficit. Our case underscores both the importance of the critical spinal collateral supply from the hypogastric artery and the role of spinal fluid drainage to maximize spinal cord perfusion in the setting of spinal cord ischemia. (J Vasc Surg 2003;37:690-2.)

Paraplegia or paraparesis secondary to spinal cord ischemia is an extremely uncommon but devastating complication after elective repair of abdominal aortic aneurysm (AAA). The incidence of this complication has been estimated at 1 in 400 after repair because of aneurysmal disease and 1 in 5000 after arterial reconstruction because of occlusive aortoilliac disease.[1] Causes of spinal cord ischemia after aortic surgery have been linked to prolonged aortic clamping, systemic hypoperfusion, direct interruption of the cord blood supply, and atheroembolism of critical spinal collateral vessels.[2,3] Blood flow to the lumbar spinal cord is derived from numerous sources, including the iliolumbar, lateral sacral, and hypogastric arteries. We report delayed paraplegia after endovascular AAA repair in which one hypogastric artery was inadvertently occluded due to atheroembolism. The patient regained partial neurologic recovery after cerebrospinal fluid (CSF) drainage. The cause of and management for this catastrophic complication of endovascular AAA treatment are discussed.

CASE REPORT

A 75-year-old man with a history of diabetes mellitus, emphysema, peripheral vascular disease, coronary artery disease, chronic renal failure, and congestive heart failure (ejection fraction, 35%) was seen for evaluation of a 5.4 cm infrarenal AAA found at computed tomography of the chest, abdomen, and pelvis. He previously had undergone coronary artery bypass grafting and bovine aortic valve replacement complicated by sternal dehiscence and mediastinitis requiring muscle flap repair. Abdominal aortography revealed an infrarenal AAA with adequate proximal aortic neck and distal iliac landing zones, and bilateral patent hypogastric arteries (Fig 1). Neither the inferior mesenteric artery nor the lumbar arteries were visualized. Because of his past medical comorbid conditions, a decision was made to treat the AAA endoluminally.

Endovascular repair with a modular AneuRx stent graft system (Medtronix AVE, Santa Rosa, Calif) was performed in the operating room with the patient under local anesthesia. After bilateral groin cutdown to expose the common femoral arteries, a 28 mm × 13.5 cm main trunk was placed through the right common femoral artery, followed by a coaxial 16 mm × 8.5 cm right iliac extension. A 16 mm × 11.5 cm left iliac limb was placed through the left common femoral artery, followed by a coaxial 16 mm × 8.5 cm iliac extension. The distal iliac extensions were placed in the distal common iliac arteries bilaterally. A completion arteriogram revealed adequately placed endografts, with patent bilateral renal arteries without evidence of proximal or distal endovascular leak. The right hypogastric artery, however, was occluded (Fig 2). Total operative time was 2 hours. The patient tolerated the procedure well and remained hemodynamically stable throughout the operation.

Forty-eight hours postoperatively the patient was noted to have bilateral lower extremity paraparesis. Neurologic examination revealed left L2 and right T10 flaccid lower extremity paraparesis with decreased sensation to the L2 level. Bladder and bowel function were maintained, and the patient had palpable dorsalis pedis and posterior tibialis pulses bilaterally. Urgent magnetic resonance images did not reveal evidence of cord compression, hematoma, or infarction. A CSF drainage catheter was placed, and CSF pressure was maintained at less than 10 mm Hg. Intravenous solumedrol (5.4 mg/kg bolus and 30 mg/kg drip) and mannitol (25 g) were administered. Blood pressure was augmented with intravenous fluid and dopamine drip to keep mean arterial pressure between 80 and 90 mm Hg. CSF drainage was continued, along with steroid therapy, over the next 3 days, and moderate improvement was noted at neurologic examination. The patient was transferred to a spinal cord rehabilitation unit on postoperative day 12, where he continued to receive regimented physical therapy. Eight months later he had regained some thigh and calf flexion on the left and slight dorsiflexion and plataflexion on the right. Postoperative surveillance CT scans showed no evidence of endovascular leak or device migration.

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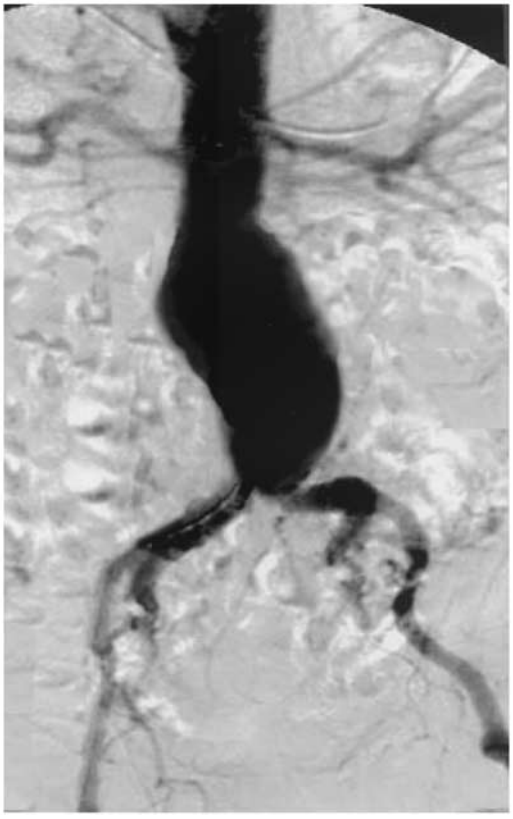

Fig 1. Preoperative angiogram reveals an infrarenal aortic aneurysm, which was suitable for endovascular repair, and patent bilateral hypogastric arteries.

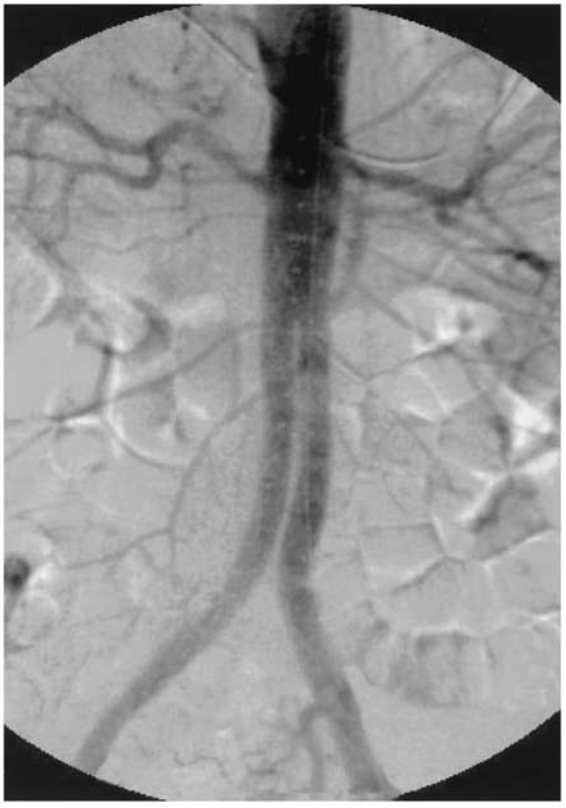

Fig 2. Postoperative angiogram shows successful exclusion of the infrarenal aortic aneurysm with aortic endograft, without evidence of endovascular leak. Right hypogastric artery was not visualized.

DISCUSSION

Development of delayed neurologic deficits after AAA repair is a rare and poorly understood phenomenon, and occurs in fewer than 0.3% of patients undergoing aortic aneurysm surgery.[1] Factors contributing to spinal cord ischemia after abdominal aortic surgery include interruption of the greater radicular artery (Adamkiewicz artery), prolonged aortic occlusion, intraoperative hypotension, atheromatous embolization, and interruption of collateral circulation arising from the lumbar and hypogastric artery circulation.[3]

Although placement of an aortic endograft uniformly occludes inflow of the inferior mesenteric artery and all infrarenal lumbar arteries, this is unlikely the sole mechanism responsible for our patient's paraparesis. A recent multi-institutional study that examined the efficacy of endovascular AAA repair noted no incidence of paraplegia or paraparesis after aortic endograft placement.[4]

Preoperative angiography in our patient revealed patent bilateral hypogastric arteries; however, CT scan reconstruction of the thorax and abdomen failed to visualize the greater radicular artery. Patients in whom the greater radicular artery arises from higher thoracic vertebral levels or those in whom this vessel is chronically occluded depend on lower lumbar arteries and hypogastric artery circulation to make a substantial contribution to the blood supply of the distal spinal cord.[5] We speculate that the unintentional occlusion of the right hypogastric artery may have in part compromised collateral flow to the spinal cord. The hypogastric artery provides collateral vessels to the spinal cord via the lateral sacral arteries and intrinsic spinal arteries at the level of the conus medullaris. Previous reports have linked the association of paraplegia with compromised pelvic perfusion by way of unilateral hypogastric ligation or embolization.[6] The presumed mechanism of hypogastric occlusion in our patient was extensive catheter-related manipulation along with passage of the endograft device in an atherosclerotic iliac artery. Despite placement of the right iliac endograft in the common iliac artery, as demonstrated on the completion angiogram, we postulate that occlusion of the right hypogastric artery was due to dissemination of atheroembolic material.

Rockman et al[7] reported two cases of lower extremity paraplegia after successful or attempted endovascular AAA repair.[7] In these two patients, atheroembolization to the spinal cord, due in part to catheter manipulation, was believed to be the primary cause of the spinal cord ischemia.

Atheroembolization is a well-known complication of endovascular surgery, in which atheroembolic materials typically disseminate to the pelvic and lower extremity circulation, resulting in either sigmoid colon necrosis or blue toe syndrome.[8,9] In addition, the endovascular AAA procedure has been associated with a greater degree of dissemination of particulate and gaseous emboli to the lower extremity compared with traditional open aneurysmorraphy, based on ultrasound scan evaluation.[10] With continual improvement of endovascular technique and devices, simplification of the endovascular AAA procedure may result in less catheter-related manipulation, which may lead to reduced risk for atheroembolization.

Our current understanding of delayed neurologic deficit after aortic surgery is largely derived from the experience of descending thoracic and thoracoabdominal aortic aneurysm repair. The treatment principles are primarily directed toward optimizing spinal cord perfusion and minimizing spinal cord edema. Supportive interventions to ameliorate spinal cord ischemia and improve functional outcome include CSF drainage, systemic steroid administration, permissive hypertension, diuresis, and intensive physical therapy. Among these techniques, CSF drainage is thought to have the most effect in reversing neurologic dysfunction in these patients.[11] Its beneficial effect of reversing spinal cord ischemia is thought to be due to increased blood flow in the spinal cord by means of decreasing CSF pressure. CSF drainage also reverses delayed-onset neurologic deficit after open aortic aneurysm repair.[12] A case report similar to ours was noted in whcih delayed onset of paraparesis occurred after endovascular AAA treatment. The patient regained partial neurologic function after immediate CSF drainage and subsequent physical therapy.[13]

In summary, neurologic dysfunction can occur after endograft placement in infrarenal aortic aneurysm. Institution of CSF drainage on the presence of neurologic deficit after endovascular AAA repair may improve the outcome. Further studies are needed to determine the pathogenesis and adjunctive treatment of paraplegia or paraparesis after endovascular AAA repair.

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