SHORT REPORT

Spinal Cord Ischaemia after Endovascular Repair of a Ruptured Abdominal Aortic Aneurysm

J. P. P. M. de Vries1*, F. H. W. M. van der Heijden1, A. D. Montauban van Swijndregt2 and A. C. Vahl1

Departments of 1Vascular Surgery and 2Interventional Radiology, OLVG Hospital, Eerste Oosterparkstraat 279, 1090 HM, Amsterdam, The Netherlands

Key Words: Spinal cord ischaemia; Abdominal aortic aneurysm (AAA); Endoprosthesis; Rupture; Complication.

Introduction

Spinal cord ischaemia subsequent to infrarenal abdominal aortic aneurysm (AAA) surgery is rare. This complication has been reported in less than 120 patients in the English literature. Of these patients, only 39 were operated on for AAA rupture. The possible causes for this adverse neurological phenomenon appear to be multifactorial and part of one of the following events: (a) the length and level of perioperative ischaemia, (b) inability to restore blood flow to the spinal cord and (c) a reperfusion-like syndrome of the spinal cord after successful reconstruction.1

In recent years, endovascular repair of the AAA has gained ground, but also this treatment is not without neurological complications. So far, only three cases of spinal cord ischaemia after elective endovascular repair have been reported.2,3 Atheroembolization of the spinal cord vessels appears to be one of the major underlying causes.

We report the first case in which spinal cord ischaemia occurs after EVAR for a ruptured AAA. Use of an Aorto-Uni-Iliac endovascular device (and thus occlusion of the contralateral common iliac artery, which compromises the hypogastric inflow) appears a new major risk factor in addition to atheroembolization.

Case Report

A 77-year-old male was admitted to our emergency department with lower abdominal pain and collapse. Vascular risk factors included smoking, hypertension and diabetes mellitus. Physical examination revealed a painful, pulsatile abdominal mass, reduced femoral pulses and signs of hypovolaemic shock.

Spiral computed tomography (CT) showed that the maximum diameter of the abdominal aneurysm was 77 mm and that it had ruptured. There was a good neck just below the renal arteries which was suitable for endovascular stenting, with a length of more than 18 mm. The common iliac arteries were normal, but both hypogastric arteries showed calcified orifices, with occlusion of the more distal part of the left hypogastric artery and a patent right one (Fig. 1).

Because of clinical deterioration, endovascular repair was done urgently, using a Talent Aorto-Uni-Iliac graft (Medtronic AVE, Minneapolis, MN). This graft, with a proximal diameter of 30 mm and a distal diameter of 16 mm, was introduced via the left femoral artery. A 14 mm iliac extension was introduced to fix the endoprosthesis firmly in the left common iliac artery. Completion angiography showed two patent renal arteries and a mild type 2 endoleak from a lumbar artery. At that time, a patent internal iliac artery was seen only at the right side. Thereafter a 24 mm occluder was positioned in the origin of the right common iliac artery without problems. No angiogram was carried out after placement of the
occluder. An 8 mm femorofemoral bypass restored blood supply to the right lower leg.

During the first 14 h postoperatively systolic blood pressure fluctuated between 80 and 105 mmHg. Physical examination did not show any neurologic deficit. Thereafter the patient suffered from paralysis of both legs, decrease of vital and gnostic sensation from the L2 level and bladder retention. Drainage of cerebrospinal fluid was performed with a lumbar drainage catheter for three days with an overflow pressure of 10 cm of water. Unfortunately, no improvement in the neurological deficit took place. The patient was discharged on the tenth postoperative day to an outpatient revalidation clinic.

Three months postoperative surveillance showed no improvement in his neurological condition. CT scan demonstrated a successful exclusion of the infrarenal aneurysm with no signs of endoleak. Occlusion of the hypogastric artery on both sides had occurred (Fig. 2).

Discussion

Neurological deficit after AAA repair is so rare, a vascular surgeon will see this complication only once or twice in his/her professional career. The development of spinal cord ischaemia can be classified as immediate or delayed and the causes are multifactorial. In the literature, delayed neurological complications are reported up to 20 days after aortic surgery and are attributed to a reperfusion-like syndrome. Seldom, postoperative paraplegia is secondary to intra-medullary bleeding.

Our patient suffered from paralysis and bladder dysfunction 14 h postoperatively, which could be classified as early, but not acute ischaemia. Early ischaemia is considered to be secondary to hypoperfusion of the spinal cord. Blood supply to the spinal cord can be divided in intrinsic and extrinsic components and are mainly fed by the greater radicular artery (Adamkiewicz artery) and a collateral system from the lateral sacral and hypogastric circulation. The hypogastric arteries make an essential arterial contribution in the case of pre-existent occlusion or high thoracic origin of the greater radicular artery. Cadaver studies done by Priscoll show that in about 12% of the cases the Adamkiewicz artery originates in the L2-L3 segment where the aortic clamp is placed, which emphasizes the importance of the hypogastric collateral system.

To gain time, it is common to treat a ruptured abdominal aortic aneurysm using an Aorto-Uni-Iliac endovascular device, as in our case. This means occlusion of the opposite common iliac artery, which makes the hypogastric artery dependent on retrograde flow and it is therefore prone to occlusion. In our patient, the proximal part of the right hypogastric artery was occluded; collateral flow to the distal branches was not enough to perfuse the spinal cord.
sufficiently. Also, there is a risk of atheroembolization due to catheter-related manipulation of the ipsilateral hypogastric artery.

It is not very likely that the neurological deficit in our patient was caused by intramedullar bleeding. Therefore no medullary CT or MRI scan was carried out.

In retrospect, introduction of the endoprothesis in our patient via the less calcified right common femoral artery might have prevented neurological problems.

This is the first case report of a neurological deficit after endovascular treatment of a ruptured AAA. The use of a Mono-Uni-Iliac endovascular device in case of rupture gains time and therefore might save lives, but, as we speculate, it can be a risk factor in the occurrence of spinal cord ischaemia. Meticulous placing of the distal end of the uni-iliac device and the occluder must be carried out to avoid further unnecessary harm to the internal iliac arteries.

References


Accepted 6 November 2003