Online Case Report

Ischaemic lumbosacral plexopathy following aorto-iliac bypass graft: case report and review of literature

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A 77-year-old man had aorto-iliac bypass for an abdominal aortic aneurysm (AAA). This was complicated by occlusion that needed extension of the graft to the right femoral artery. He was unable to move his right leg with numbness after surgery. This was caused by extensive lumbosacral plexopathy on the right side. Lumbosacral plexopathy is uncommon because the plexus has a rich blood supply. The incidence of ischaemic lumbosacral plexopathy is higher with re-operative and emergency AAA reconstruction. This may predispose the lumbosacral plexus to ischaemic injury. Consideration should be given to maintaining retrograde perfusion of the internal iliac artery.

Keywords: AAA repair – Lumbosacral plexus

A 77-year-old man who was non-smoker with no history of diabetes, hypertension, IHD or CVD was diagnosed with carcinoma of the prostate in October 2000. CT scan of the abdomen and pelvis showed an incidental 3.5-cm infrarenal abdominal aortic aneurysm (AAA). He was asymptomatic and followed up with 6-monthly abdominal ultrasonography. After 3 years, abdominal CT scan showed 4.5-cm AAA, 3-cm aneurysms of both common iliac arteries and internal iliac arteries aneurysms. Aorto-iliac reconstruction using bifurcated graft was advised.

Through a midline laparotomy incision, a 20-mm bifurcated graft was inserted after giving 3000 U of heparin. The upper end was anastomosed to the neck of the AAA and the bottom ends to the iliac bifurcation bilaterally. While the patient was in the recovery room, the right foot looked ischaemic. He was transferred to theatre where femoral embolectomy was performed. The

patient was transferred to the ITU where his right foot became ischaemic again. Because of persistent hypotension, the patient was returned to theatre again. The abdomen was explored with no obvious source of bleeding. The upper anastomosis was sound. The right groin incision was re-opened and the right limb of the bifurcated graft extended to the common femoral artery with improvement in the vascularity of the right foot.

On the following day, the patient complained of numbness in his right lower limb with flaccid paralysis (0/5). Physical examination showed complete absence of motor strength in all muscles of the right lower limb. CT scan of the epidural site was normal and MRI showed minor disc degeneration. EMG revealed severe denervation of the muscles of the right lower limb. These findings are compatible with severe lumbosacral plexopathy. In the first week, his motor power improved to 2/5 with partial sensory recovery. Over the following

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year, sensation returned to normal but the patient still had wasting in his right thigh for which he had regular physiotherapy. Power recovered but the patient needs the aid of a crutch.

Discussion

Ischaemic injury to the lumbosacral plexus after aorto-iliac reconstruction is rare. There are less than 80 patients reported in literature. The aetiology of ischaemic neuropathy is multifactorial, although the major single cause is the alteration in the blood supply to the spinal cord or the lumbosacral plexus. Hypoperfusion, interruption of critical arteries to the distal cord or lumbosacral roots or plexus may lead to permanent neurological deficit. Occlusion of the great radicular artery or the artery of Adamkiewicz, which supplies the anterior spinal artery in the thoracolumbar segment, has been mentioned in different studies.¹

As proposed by others,² we believe that interruption of the pelvic circulation is a major aetiological factor in the development of ischaemic injury in our patient. Almost all of the blood supply to the sacral plexus derives from the internal iliac artery. Both the lateral sacral and ilio-lumbar arteries, which originate from the internal iliac artery, send small branches into the anterior sacral foramina to supply the cauda equina.

The lumbar plexus is embedded in the posterior part of the psoas major muscle. The vasa nervosa to the plexus are derived from the blood supply to this muscle, almost all from the lumbar arteries. It has been established that vasa nervosa are essential for peripheral nerves as they cannot be sustained by axonoplasmic flow from the cell bodies.³ However, rich collaterals within the psoas usually protect the lumbar plexus from ischaemic injury even in the event of occlusion of most or all of the lumbar arteries.

Gloviczki *et al.*⁴ identified six types of injury resulting from partial or complete interruption of flow through the various components of the medullary blood supply. Differentiation between the types can be difficult and requires careful neurological evaluation supplemented by findings from MRI and EMG. Each type of injury has a different aetiology and a different prognosis.

Type I lesions involve complete infarction of the distal spinal cord and conus, supplied by the anterior and posterior spinal arteries. These injuries are characterised by flaccid paraplegia, corresponding sensory loss and poor recovery. Type II lesions correspond with anterior spinal artery syndrome in which the area supplied by the posterior spinal artery is preserved. Flaccid paraplegia is present with loss of pain and temperature sensations with intact proprioception and vibration senses. Type III lesions are due to bilateral root ischaemia with or without

patchy distal cord and conus ischaemia. Most of the cord functions are preserved. Type IV injury is unilateral plexus ischaemia caused by infarction of the lumbar or sacral plexus. Both types III and IV have favourable prognosis. Type V injury shows segmental spinal cord infarction. Patients present with spastic paraplegia because of preserved cord function distal to the infarction. Both this type and type II injury might be seen after thoracic or thoraco-abdominal aortic reconstruction. Type VI injury occurs as a result of infarction of the area supplied by the posterior spinal artery. These patients are characterised by loss of vibration and proprioception sensations.

In our patient, it seems likely that pelvic exclusion was responsible for the development of neurological ischaemic injuries especially after extending the graft to the femoral artery. His clinical picture was consistent with type IV lesions. In one report,⁵ simple interruption of one internal iliac artery during renal transplantation led to paraparesis. The other factor identified that may have played a role in the development of the neurological deficits in our patient was intra-operative hypotension.

Conclusions

Given the devastating nature of postoperative neurological injury, identifying means of prevention seems essential to decrease the incidence of these severe complications. Low flow because of hypotension, occlusion of critical radicular or pelvic arteries by thrombosis or athero-embolisation appear to be the most frequent causes of neurological injury. Measures such as avoidance of prolonged hypotension, minimising suprarenal cross-clamping, revascularisation of at least one internal iliac artery to maintain pelvic blood flow, use of heparin, and use of gentle technique to avoid embolisation are all sound measures to decrease ischaemic neurological injury.

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