CASE REPORT

An Unusual Presentation of a Ruptured Abdominal Aortic Aneurysm

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Introduction

This is a case illustrating a leaking abdominal aortic aneurysm presenting with widespread microembolisation and paraplegia. Following it is a literature review and proposed explanation of events.

Case Report

A 70-year-old man was admitted to an accident and emergency department by a paramedic crew following a collapse at home. He complained of back pain and had suddenly lost all power and sensation in both legs. The past medical history revealed that two similar episodes had occurred over the past 12 months with complete resolution of symptoms within 6 h. On both occasions no medical intervention was sought.

On arrival in the accident and emergency department he was found to have a tender pulsatile abdominal swelling with evidence of widespread ecchymoses from the level of the umbilicus down to his feet. The lower limbs were flaccid and motor function to all muscle groups was undetectable. Sensation was absent from the T10 dermatome extending distally, knee and ankle reflexes were diminished.

The patient was taken to theatre for an emergency abdominal aortic aneurysm repair. At laparotomy an 8 cm infrarenal aortic aneurysm was found with a contained retroperitoneal rupture. Thrombus had disintegrated within the aneurysm sac. Two large patent lumbar arteries at the aneurysm neck were underrun with sutures, other lumbar arteries appeared thrombosed. The aneurysm was repaired with a 16 mm knitted Dacron tube graft. In the immediate postoperative period the patient was commenced on Dextrmethosone and an Iloprost (Schering) infusion. Throughout the patient's 6-day period on intensive care cardiovascular, respiratory and renal function remained stable.

Over the next 4 weeks following physiotherapy he regained some sensation to a level of L1 and motor power to the thigh and calf muscles but was unable to move against gravity. A post operative MRI scan of the lumbar and thoracic spinal cord showed a small right sided disc prolapse at L3/L4 with no bony destruction, which was not considered to be relevant to his present condition. A long term urethral catheter was passed for a distended atonic bladder and a sigmoid colostomy was fashioned as he had absent anal tone with faecal incontinence. After 3 weeks he was transferred for rehabilitation on a neurology ward. Subsequent review has shown gradual improvement in lower limb function but he is still unable to walk unaided.

Discussion

After an extensive literature search only 2 similar such cases have ever been published. The first of these detailing a leaking abdominal aortic aneurysm presenting with splenic rupture, duodenal obstruction and paraplegia1 (1973) and the second, a case presenting in a similar way to ours2 (1992). Acute aortic occlusion has been reported to mimic spinal cord or nerve root compression. A paper by Meager et al.
detailing 8 such cases described aneurysmal thrombosis as the aetiology for 2 of the cases with atherosclerosis and embolic disease being responsible for the other 6, but none were attributable to a ruptured abdominal aneurysm.

Transient paraplegia and/or paraparesis are rare but well documented complications following abdominal aortic surgery. The incidence rates have been quoted at 1:400. Since 1956, 111 English language cases have been reported, 35% occurring during ruptured aneurysm surgery and the remainder during elective repair or surgery for occlusive disease. A proposed mechanism for this pathology is thought to be one of or a combination of factors. These include interruption of the great segmental medullary artery, (the medullary artery of Adamkiewicz), prolonged cross clamping, intra operative hypotension and thrombotic embolisation via the lumbar arteries.

The blood supply to the lower thoracic and lumbar spinal cord is rather precarious. It is supplied by 2 small posterior arteries and a single anterior artery. The anterior spinal artery constitutes the majority (75%) of the total blood supply. These 3 arteries receive anatomically inconsistent radicular arteries at most of their segments. Additional enlarged segmental medullary arteries arise from the intercostal or lumbar vessels. The most prominent of these feeder vessels is known as the Adamkiewicz artery so named after the Polish pathologist (1850–1921). The anterior spinal artery requires additional supply from segmental arteries along its course to provide adequate perfusion of the distal spinal cord. This is due mainly to its long length and narrowing calibre. Studies on cadavers have shown the majority of humans to have only one such dominant artery. The artery has a varied level origin, 79% arising between T9–T12, 11% between T5–T8 and 10% between L1–L2. It is for this reason that spinal cord ischaemia is such a recognised complication following thoracic or abdominothoracic aneurysm repair. Attempts at identifying this artery are often carried out either perioperatively or postoperatively.

In our reported case it is proposed that transient episodes of spinal cord ischaemia had occurred in the past from thrombotic emboli arising from aneurysmal mural thrombus. These emboli had occluded an abnormally low Adamkiewicz artery. The initial presentation to our department of paraplegia was due either to a thrombotic embolus down a critical lumbar artery or a rapidly expanding haematoma obliterating the lumbar origins causing ischaemia of the anterior spinal cord and subsequent paralysis. Evidence of microembolic events occurring in the skin of the lower abdomen suggests that the former to be most likely.

References

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