Case Report. Spinal Cord Ischemia
Associated With Repair of a Ruptured Abdominal Aortic Aneurysm

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Abstract:
Spinal cord ischemia is a rare complication of rupture of an abdominal aortic aneurysm or its emergency surgical treatment; only seven cases have been reported previously. A patient with this complication is described. It was characterized by postoperative total loss of all forms of sensation in the legs and partial sparing of motor function. The patient had been in shock, but the only other organ to suffer ischemic changes was the bowel. The condition, therefore, was attributed to local interference with the blood supply of the lumbosacral cord which is derived principally from the abdominal aorta through varying numbers of lumbar arteries. Recovery was virtually complete.

Additional Key Words
lumbosacral cord, blood supply of Adamkiewicz, artery of

Impairment of the blood supply to the spinal cord is a potential but rare complication of rupture of an abdominal aortic aneurysm and its emergency surgical treatment; only seven cases had been recorded by 1969. Following emergency resuscitation he was operated upon by Dr. A. R. Downs. An infrarenal abdominal aortic aneurysm was replaced by a dacron prosthesis, the aorta being clamped below the renal arteries and the lumbar arteries oversewn and transected. There was a short subsequent period during which the aortic pulse was very feeble and frequent ventricular premature contractions were present, but pulmonary function was adequate and urine output good. The postoperative course was complicated by necrosis of the right side of the colon and terminal ileum, necessitating hemicolectomy. On the day after the initial operation the patient was observed to have strong but uncontrollable movements of the left leg and right stump. Examination showed good power, but loss of all forms of sensation below the twelfth thoracic segment. Sphincter control could not be assessed as bowel function had not returned and a Foley catheter had been inserted previously. By the following day, deep sensation and appreciation of pain had returned, but all other forms of sensation remained absent, the complete loss of position sense being particularly striking. The knee jerks were equal, the left ankle jerk normal and the left plantar reflex flexor. The patient was mentally clear and able to cooperate during all examinations. The pulses were present in the left leg postoperatively and the left foot was warm and normal in color. Full neurological examination

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had not been made preoperatively, but, using his prosthesis, the patient had been able to walk without difficulty and without any balance problems either in the light or in the dark.

There was gradual spontaneous improvement during subsequent days, although vibration sense remained impaired. Rehabilitation was not slowed appreciably as a consequence, although initial resumption of walking suggested some loss of power not evident when the patient was examined while in bed. When seen five months later, power was good in the left leg and right stump and he was able to walk without help. There was some residual hyperesthesia of the left sole and a feeling of tightness and numbness in the left calf. The only objective neurological abnormalities found were slight reduction of position sense in the left toes and absence of vibration sense at and below the anterior superior iliac spines. Precisely two years after the rupture, the patient died. His demise was sudden and occurred while visiting another city. No autopsy was obtained.

Discussion

Spinal cord damage following resection of an abdominal aortic aneurysm is extremely rare. Skillman and his colleagues1 were able to find records of only five such cases in the literature, adding two of their own. In the present case, the absence of neurological symptoms previously suggests that damage to the cord occurred either immediately before or at the time of operation, the subsequent recovery being compatible with a vascular lesion. Confinement of ischemic changes to spinal cord and bowel suggests local ischemia rather than general hypotensive effects and could have resulted from thrombosis, embolism, external pressure, dissection or surgical occlusion.

The presence of a segmental level indicates a cord lesion below the twelfth thoracic segment and the dissociated sensory loss suggests predominant involvement of the posterior quadrants. In contrast, retention of motor power, preservation of tendon and plantar reflexes, and the transient nature of the pain sense loss indicate relative sparing of the pyramidal and spinothalamic tracts and of the afferent inflow into the dorsal horns.

There are many variations in the arterial supply of the spinal cord, but regression of segmental vessels can result in dependence on a singular radicular artery. Kadyi's studies, summarized by Skillman et al.,1 showed that the most distant radicular artery, that of Adamkiewicz, is usually the largest and best developed, and that the caudal end of the spinal cord is largely dependent on blood supply from this vessel. It can arise from a lumbar artery at a lower level than the renal arteries. Therefore, the spinal cord may be vulnerable to a lesion of an infrarenal lumbar artery. Bolton2 showed that the lower posterior spinal arteries fill from terminal branches of the anterior. As the latter is unpaired, a single ischemic lesion can involve both halves of the cord. The posterior spinal arterial territory, "at the end of the line," is more vulnerable than the anterior and could become ischemic when the anterior territory is spared. The upward extent of the lesion would depend on the extent of the territory supplied by the radicular artery.

Therefore, the evidence suggests that the patient suffered an ischemic lesion of the lumbosacral spinal cord affecting predominantly the part supplied by the posterior spinal arteries.

References

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