MECHANISMS AND PREVENTION OF ANTERIOR SPINAL ARTERY SYNDROME FOLLOWING ABDOMINAL AORTIC SURGERY

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Paraplegia or paraparesis occurring as a complication of thoracic or thoracoabdominal aortic aneurysm repair is a well known phenomenon, but the vast majority of elective abdominal aortic aneurysm repairs are performed without serious neurological complications. Nevertheless, there have been many reported cases of spinal cord ischaemia following the elective repair of abdominal aortic aneurysms (AAA); giving rise to paraplegia, sphincter incontinence and, often, dissociated sensory loss. According to the classification made by Gloviczki et al. (1991), this presentation is classified as type II spinal cord ischaemia, more commonly referred to as anterior spinal artery syndrome (ASAS). It is the most common neurological complication occurring following abdominal aortic surgery with an incidence of 0.1–0.2%.

Several aetiological factors, including intra-operative hypotension, embolisation and prolonged aortic cross-clamping, have been suggested to cause anterior spinal artery syndrome, but the principal cause has almost always been identified as an alteration in the blood supply to the spinal cord. A review of the literature on the anatomy of the vascular supply of the spinal cord highlights the significance of the anterior spinal artery as well as placing additional emphasis on the great radicular artery of Adamkiewicz (arteria radicularis magna) and the pelvic collateral circulation.

Although there have been reported cases of spontaneous recovery, complete recovery is uncommon and awareness and prevention remains the mainstay of treatment. However, being so tragically unpredictable and random, spinal cord ischaemia after abdominal aortic operations appears to be an unpreventable event.

Key words: *anterior apinal artery syndrome, spinal ischaemia, injury, AAA open repair, artery of Adakiewicz.*

INTRODUCTION

Spinal cord infarction following elective abdominal aortic aneurysm repair is a severe and catastrophic complication. Since it is rare, it is difficult to predict and the effects are devastating upon the patient, the patient's family and the surgeon. Type II spinal cord injury, also known as ante spinal artery syndrome, is one of the forms of spinal cord ischaemia that can occur following abdominal aortic surgery.

The purpose of this review is to explore the vascular anatomy of the spinal cord, identify mechanisms of ischaemic spinal cord injury, discuss possible aetiological factors, and highlight surgical guidelines that are believed to decrease the incidence of this tragic complication.

ARTERIAL SUPPLY OF THE SPINAL CORD

The arterial blood supply to the spinal cord consists of an intrinsic and an extrinsic network of arteries [1]. The intrinsic blood supply is composed of three longitudinal systems: one anterior and two posterior spinal arteries. These longitudinally orientated vessels arise at the level of the upper cervical spinal cord [1].

The posterior spinal arteries each arise from the posterior inferior cerebellar artery, a terminal branch of the vertebral artery, and form a plexus on the posterior surface of the spinal cord, the arterial vasocorona. These form anastomotic connections between the anterior and posterior spinal arteries, and provide an uninterrupted blood supply along the length of the spinal cord. However, these anastomoses are insufficient to sustain adequate spinal cord circulation [1–3].

The anterior spinal artery (ASA) originates from the union of two vessels, which arise from the vertebral arteries, at the level of the medulla, and lies in the anterior median fissure of the spinal cord. It is the principal artery of the three longitudinal systems and is responsible for 75% of the blood supply to the spinal cord. The ASA supplies the anterior two-thirds of the spinal cord, including the critical motor area [2, 4].

The extrinsic blood supply to the spinal cord consists of segmental spinal arteries that regularly feed the anterior spinal artery along its 40–45 cm length. These usually arise from subclavian, intercostal and upper lumbar arteries, and from branches of the internal iliac and middle sacral arteries. The lumbar and intercostal arteries give rise to approximately 12 posterior and eight anterior

radicular arteries. However, the largest of these radicular and segmental arteries is the arteria radicularis magna (ARM) or the great radicular artery of Adamkiewicz (Fig. 1), which reinforces the arterial blood supply to the distal cord [2, 5].

The ARM most commonly originates as a branch of the intercostal arteries [5], 70% of which have been identified to arise from left intercostal and/or lumbar arteries [6]. It arises between T9 and T12 in 75% of patients, T5 and T8 in 15%, and L1 and L2 in 10%. However, in the case of the great radicular artery of Adamkiewicz entering at the T5–T8 level, a complementary artery, the 'conus medullaris artery', may be present in the caudal area [7, 8]. Furthermore, at the caudal level, the anterior spinal artery and the two posterior spinal arteries anastomose around the conus medullaris in the arterial 'basket' (Fig. 1), referred to as the 'anastomotic ansa of the conus' by Lazorthes, et al [8].

Table

Classification of ischaemic injuries to the spinal cord and lumbosacral roots or plexus [17]

Classification	Site of Ischaemia	Neurological deficit
I	Distal thoraco-lumbar cord	Bilateral flaccid paraplegia and sensory loss. Bowel and bladder dysfunction.
II	Anterior two-thirds of the spinal cord (Anterior Spinal Artery Syndrome)	Bilateral flaccid paraplegia and loss of pain, temperature sensation; proprioception and vibratory sensation maintained.
III	Lumbosacral roots with or without patchy infarcts of cord	Bilateral asymmetric paraparesis with or without bowel and bladder incontinence.
IV	Lumbosacral plexus	Bilateral asymmetric paraparesis with or without bowel and bladder incontinence. Preservation of paraspinal muscle innervation on EMG.
V	Segmental infarction of the spinal cord	Bilateral spastic paraplegia with sensory loss.
VI	Posterior third of the spinal cord (Posterior Spinal Artery Syndrome)	Loss of proprioception and vibratory sensation.

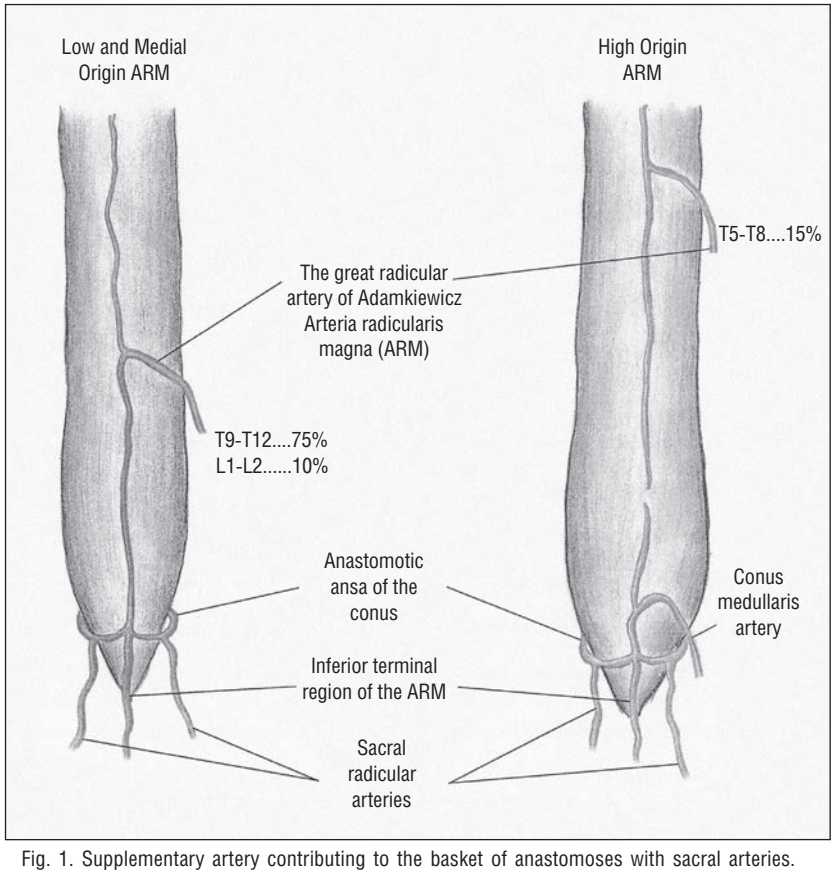

Fig. 1. Supplementary artery contributing to the basket of anastomoses with sacral arteries. Note the basket of terminal sacral arterioles around the conus of the spinal cord. Also note the supplementary artery, seen when the artery of Adamkiewicz takes off high in the thorax in 15% of patients. The significance of this conus blood supply in such patients is particularly important when the distal anterior spinal artery is stenotic or discontinuous. Adapted from Lazorthes, et. al. [8].

The distal spinal cord is perfused by vessels from the pelvic circulation, which are composed of the inferior mesenteric artery, branches of the profunda femoris and the internal iliac arteries and their iliolumbar and lateral sacral branches. Arising from the lumbar arteries (branch of the iliolumbar artery) and/or the lateral sacral arteries are ascending arteries from the filum terminale and the cauda equina, which anastomose with the intrinsic spinal arteries at the anastomotic ansa of the conus (Fig. 1) [9–11].

SPINAL CORD ISCHAEMIA

As shown by Table, Gloviczki, et al. [12] identified six types of ischaemic neurologic injury, based on the neurovascular anatomy of the distal spinal cord, nerve roots, and lumbosacral plexus; and correlated them with observed neurologic deficits.

Type I spinal cord ischaemia is characterized by bilateral flaccid paraplegia and sensory loss, with bowel and bladder dysfunction. It occurs as a result of global ischaemia to the distal spinal cord and conus, and has very poor long-term recovery [1, 12].

Anterior spinal artery syndrome (ASAS) is classified as type II spinal cord ischaemia and results from a lack of arterial blood supply to the anterior two-thirds of the distal cord. It is characterized by the immediate presentation of bilateral flaccid

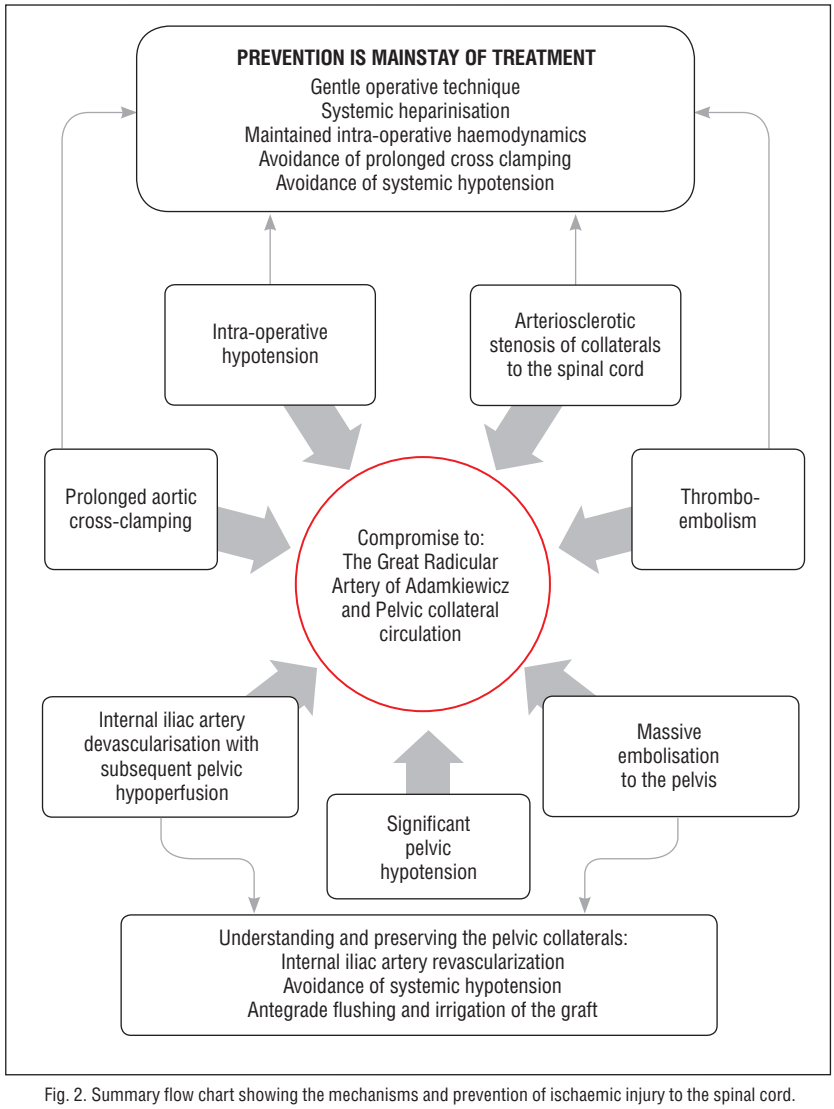

Fig. 2. Summary flow chart showing the mechanisms and prevention of ischaemic injury to the spinal cord.

paraplegia, with loss of pain and temperature sensation and preservation of proprioception and vibration. This type of injury is most commonly seen after thoracic or thoraco-abdominal aortic surgery. There is a minimal long-term improvement seen in some patients [3, 12].

Type III and IV spinal cord injury involve the nerve roots and lumbosacral plexus and often present with bilateral root ischaemia with or without patchy distal cord and conus ischaemia, and relative preservation of cord functions. Patients with type III and IV injuries have the better prognoses [12].

Patients with type V spinal cord ischaemia have segmental spinal cord infarction characterized by bilateral spastic paraplegia, due to preserved cord function distal to the infarction, with sensory loss [1].

Type VI spinal cord injury is the posterior spinal artery syndrome (PSAS), which occurs as a result of interruption of the posterior spinal artery to the posterior one-third of the spinal cord, and is characterized by the loss of proprioception and vibratory sensation [12].

MECHANISMS AND PREVENTION OF INJURY

Ischaemic injury to the spinal cord following abdominal aortic aneurysm surgery was first reported by McCune in 1956 [13], shortly followed by the first description of AAA-related ASAS by Mehrez, et al. in 1962 [14].

Although paraplegia occurs in up to 10% of patients following thoracic and thoracoabdominal aortic surgery, it has been estimated to occur in less than 1% following abdominal aortic surgery [7]. Nevertheless, isolated cases have been reported [3, 15–19], where incidence ranges from 1.4 to 2.0% for emergency AAA repair and 0.1 to 0.2% for elective repair [17]. According to Grace and Mattox [3], ASAS is the most common type of ischaemic injury which follows abdominal aortic surgery.

Studies looking at possible causes of spinal cord ischaemia following AAA repair have suggested a number of aetiological factors for ASAS. These include prolonged aortic cross clamping, intra-operative hypotension, thromboembolism, operative interference with the pelvic circulation and interference with a low origin great radicular artery (Fig. 2) [1, 17].

Early studies suggested that spinal cord ischaemia mainly occurred following operations for ruptured abdominal aortic aneurysms. This was consistent with the fact that these patients had significant periods of hypotension, often underwent supra-renal or supra-coeliac clamping and were usually not anticoagulated. However, in the 1999 review by Rosenthal [1], it was found that a majority of patients, who had developed spinal cord ischaemia, had undergone elective abdominal aortic surgery, suggesting that the factors associated with emergency repairs may not have been the most important causes.

The location and the duration of the aortic cross-clamping have been suggested as possible factors in the aetiology of spinal cord ischaemia. Cases with supra-coeliac, supra-renal and infra-renal aortic cross-clamping with associated hypotension have been reported [1, 19]. Particular emphasis has been put on maintaining flow through the great radicular artery of Adamkiewicz at all times; since as little as seven minutes of its total occlusion by supra-renal clamping, with hypotension, was found

to produce ASAS. Furthermore, routine infra-renal clamping for more than 20 mins, in cases of infra-renal abdominal aortic aneurysms, was also thought to cause permanent paraplegia [1, 19]. Defraigne, et al. [20] claimed that 45 mins of aortic cross-clamping was a safe limit in their study. However Rosenthal [1], in his review, found that patients who underwent infra-renal aortic cross-clamping and those who underwent supra-renal cross-clamping combined with anticoagulation, were at equal risk of developing a neurological deficit. Their patients, who had aortic cross-clamp times ranging from 24 to 97 mins, all developed paraparesis or paraplegia. Rosenthal [1], therefore, concluded that the location and duration of the aortic cross-clamp could not be implicated as a probable cause of spinal cord ischaemia.

Other proposed mechanisms whereby ischaemic spinal cord injury may occur includes arteriosclerotic stenosis of collaterals to the spinal cord and arteriosclerotic embolisation of the great radicular artery of Adamkiewicz, either by the pathological process itself or through surgical manipulation [3, 15]. Grace and Mattox [3] found that, in patients with neurological damage, there was a lack of lumbar artery backbleeding. Therefore they suggested that backbleeding from lumbar arteries may be an indication of satisfactory collateral blood supply to the spinal cord. Anatomical studies in patients with atherosclerosis have shown that origins of vessels leading to the anterior spinal artery are "commonly encroached upon by atherosclerotic plaques and debris." Jellinger [21] found that ostia of vessels supplying the spinal cord were impinged upon by atheroma and calcific plaques. He also highlighted that the origins of these vessels had an abrupt right angle take-off from the aorta, and that partial stenosis or occlusion of the orifice would only aggravate the hypotension in the ARM [15].

Many studies have suggested that intra-operative hypotension, with or without associated prolonged aortic cross-clamping, was an important factor leading to spinal cord ischaemia. It is believed that hypotension alone may be sufficient to cause spinal cord ischaemia if there is pre-existing compromise of distal spinal cord perfusion. Lynch, et al. [16] found that a mean arterial pressure below 55 mm Hg was associated with an increased risk of central nervous system ischaemia.

However, although there have been a few reported cases of ASAS which were thought to be caused by prolonged hypotension alone [19], it should be noted that most studies could not conclude that it was the sole or principal cause of spinal cord ischaemia. Nevertheless, the prevention of hypotension perioperatively, especially in patients with severe arteriosclerotic disease, is strongly recommended [1].

Hence, it is believed that the aetiology of spinal cord ischaemia is probably multifactorial, leading in all cases to be an alteration in the blood supply to the spinal cord [1]. As previously explained, the anterior spinal artery is the principal artery of spinal cord perfusion and interruption of its major segmental artery, the great radicular artery of Adamkiewicz, has been implicated as the principal cause of spinal cord ischaemia. In instances where the ARM originates abnormally high, between T5 and T8, or is chronically compromised, the extrinsic pelvic blood supply may be critically important. In 1985, Picone, et al. [18], identified several factors that may lead to spinal cord ischaemia as a result of interruption of the pelvic blood supply, which should be avoided if at all possible. These include: (1) internal iliac artery devascularization, with subsequent pelvic hypoperfusion, (2) significant hypotension, and (3) massive embolisation to the pelvis. Picone, et al. [18] suggested that these be avoided by revascularization of the internal iliac artery, by the avoidance of systemic hypotension, and by antegrade flushing and irrigation of the graft. Furthermore, they recommended that in instances where an internal iliac artery aneurysm must be oversewn, this should be performed from within the aneurysm wall to preserve the collateral flow to the lateral sacral and iliolumbar arteries. A similar technique was proposed by Wakabayashi and Connolly [22] in 1976, who suggested that the technique of preserving the intercostal arteries in thoracic aneurysms by incorporating the posterior aneurysm wall into the graft by diagonal aneurysmoplasty may be beneficial.

PROGNOSIS

Of the reported cases of paraplegia following abdominal aortic surgery, some patients have been reported to show improvement in motor function. This, however, was unrelated to the level of paraplegia, and recovery periods have been reported to range from four months to six years. Ischaemia, without necrosis, of the involved area of the spinal cord and the development of collaterals to the affected area have been suggested as possible mechanisms of recovery. Recovery is spontaneous and cannot be predicted. However, Grace and Mattox [3] have found that return of function was more likely if some recovery was noted early in the postoperative period.

Patients with spinal cord injury have been recommended to undergo MRI scanning in order to rule out a compressive lesion, since this is potentially curable. Also, EMG studies may identify the exact site of the lesion. This may be of help in predicting the outcome, as patients with type III and IV spinal cord injury have better prognoses [17].

CONCLUSION

Spinal cord ischaemia after abdominal aortic operations is unpredictable and random. The risk of it occurring may be minimised by gentle operative

techniques, systemic heparinisation, maintained intraoperative haemodynamics, avoidance of prolonged cross clamping, avoidance of systemic hypotension, internal iliac artery revascularization, and understanding and preservation of the pelvic collaterals. Nevertheless, spinal cord ischaemia after abdominal aortic operations remains a tragically unpredictable, random, and seemingly unpreventable event.

ACKNOWLEDGEMENTS

The author would like to thank Professor Harold Ellis and Mr. Stephen Brearley for their kind support and much appreciated guidance throughout the preparation of this manuscript and Ms. Serife Aydin for the illustrations.

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