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Case Report of Infarction in the Region of the Posterior Spinal Arteries

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Summary. Spinal cord infarction in the region of the posterior spinal arteries is reported in a 62-year-old woman. The softening was restricted to the lumbosacral region involving two segments. Sectioning the affected and adjacent segments serially no occlusion was found in the posterior spinal arteries. Besides the circumscribed infarction the microscopic picture of the spinal cord was characteristic of vascular myelopathy. The underlying disorders of the previously reported cases and the predisposing factors contributing to the development of infarction are discussed. It is concluded that an insufficient anastomotic network plays the essential role in the pathogenesis of the spinal cord infarction.

Key words: Spinal cord infarction – Posterior spinal arteries

Introduction

Infarction of the spinal cord is extremely rare in contrast to that of the brain, and the occurrence of posterior spinal softening can be considered as especially exceptional. Some 15 cases of posterior spinal softening can be found in the literature, however in some cases, the lesion was more extensive [2, 9, 20]. The purpose of this paper is to present a further case of posterior spinal infarction with autopsy verification.

Case Report

A 62-year-old women was admitted to our Department on April 30, 1982 because of pain, numbness and weakness in her legs. These symptoms had occurred suddenly in association with urine retention the day before admission. She had been treated for hypertension for several years.

On admission her blood pressure was 170/100 mm Hg and pulse rate 88/min and a general physical examination revealed no abnormalities. Neurological examination disclosed a severe flaccid paraparesis which was more pronounced on the left side. The tendon reflexes of the lower extremities were diminished on the right and absent on the left side and there were pyramidal signs bilaterally. She had urine retention, bowel disturbance, loss of the deep sensibility and diminution of the other forms of sensation below the L-4 dermatome.

The results of routine laboratory findings including those of CSF were normal. Plain radiographs of the spine demonstrated a marked torsional scoliosis associated with severe spondylotic changes in the thoracic and lumbar regions. Amipaque myelography revealed multiple filling defects at the levels of intervertebral spaces in the same regions with no evidence of total or partial block.

The patient was treated with vasodilators and antibiotics. Her neurological condition improved slowly and she began to walk with little assistance. The patient died suddenly 3 weeks after admission from pulmonary thromboembolism.

Pathologic Findings

The general autopsy disclosed thrombosis of the left superficial femoral vein as the source of the massive bilateral pulmonary thromboembolism. The heart showed left ventricular hypertrophy and there was a general atherosclerosis of considerable degree affecting predominantly the abdominal aorta.

The brain weighed 1310 g, and the arteries of the circle of Willis showed a marked atherosclerosis. Apart from the moderate internal hydrocephalus the brain appeared to be normal.

Spinal Cord. On external examination there was slight opacity of the leptomeninges. The major spinal blood vessels appeared normal. Slicing the spinal cord transversely at each level softening of both posterior columns could be detected at the levels of L-5 and S-1. The other segments had a normal appearance.

Microscopic Investigation of the Spinal Cord. From the upper part of the L-5 spinal cord segment to the lower part of S-1 the posterior third of the spinal cord was infarcted. In the posterior part of L-5 several irregular infarcted areas could be seen involving the crossed pyramidal tracts, the posterior columns and the posterior horns. At the level of S-1 the softening was connected and destroyed both posterior columns completely (Fig. 1). On careful microscopic investigation of the affected and adjacent segments sectioned serially there was no evidence of occlusion in the posterior spinal arteries.

Microscopic investigation of the other segments of the spinal cord with predilection for the cervical region revealed moderate to severe spongy state of the white matter (Fig. 2), hyaline thickening of the walls of the small arteries (Fig. 3) and capillaries, moderate depletion of neurons with gliosis in the anterior horns and pyknosis to varying degrees in the remaining neurons (Fig. 4). A large number of corpora amylacea were seen throughout the gray and white matter.

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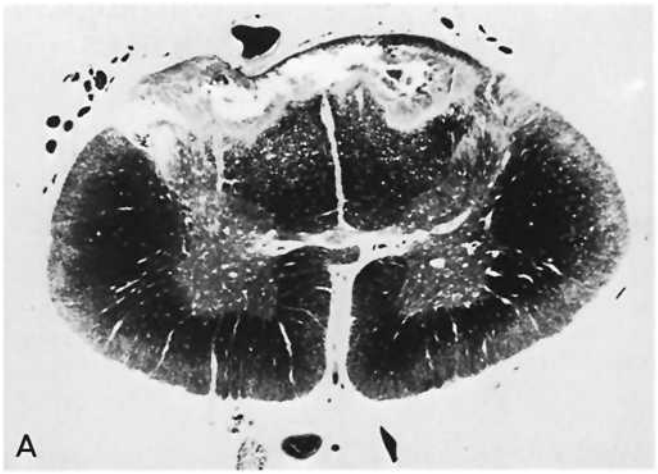

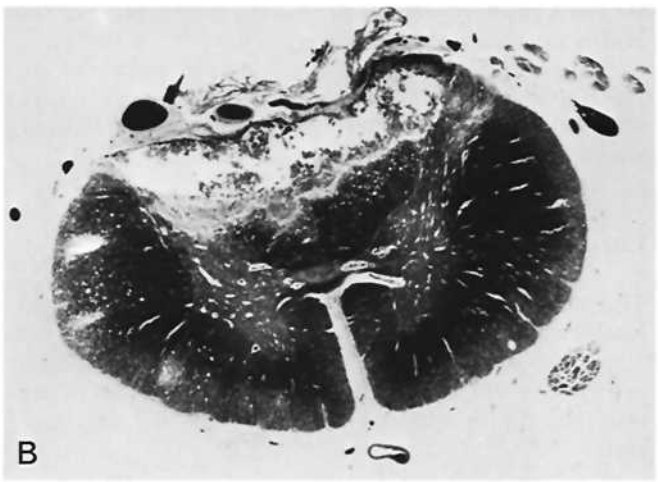

Fig. 1A, B. Transverse sections of the spinal cord show the distribution of infarction in the region of the posterior spinal arteries. (*A*) at the lower part of the L-5 segment; (**B**) at the middle part of the S-1 segment. Heidenhain stain for myelin × 8

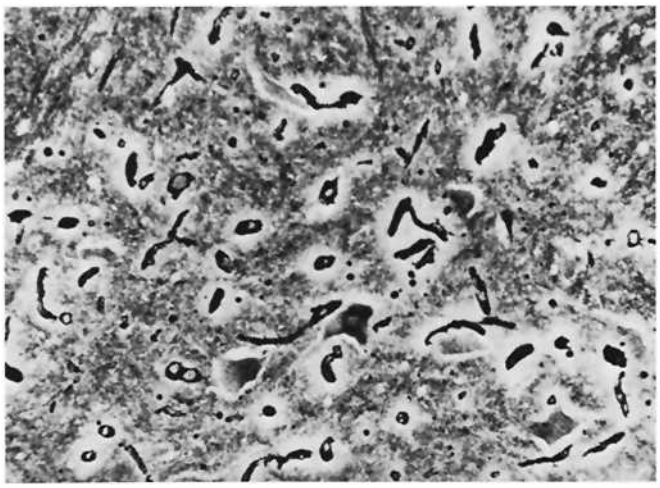

Fig. 3. Photomicrograph of the spinal cord at the upper part of the T-5 segment. The wall of the arterioles and capillaries are thickened in the anterior horn. Gömöri's method for reticulin × 100

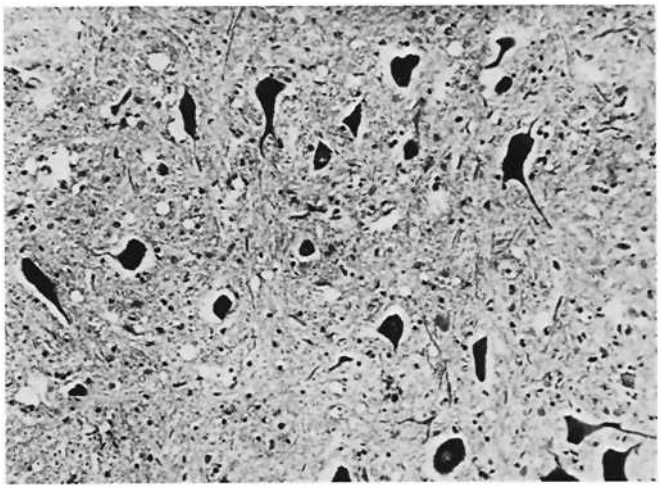

Fig. 4. Photomicrograph of the spinal cord at the upper part of the T-5 segment. Some of the neurons in the anterior horn are pyknotic and the number of glial cells is increased. Hematoxylin-eosin × 100

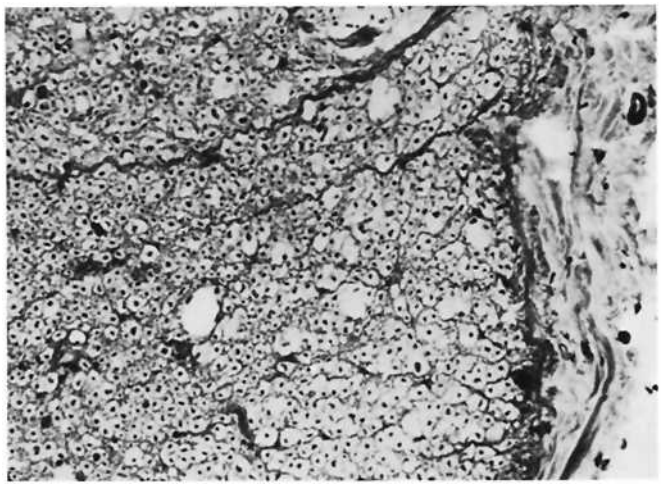

Fig. 2. Photomicrograph of the spinal cord at the lower part of the C-7 segment. The white matter shows severe spongy change. Hematoxylin-eosin × 100

The spinal arteries were free from atheroma. The thick-walled leptomeningeal veins were dilated and filled with fresh thrombus.

Discussion

According to Lazorthes [18] posterior spinal artery syndrome cannot occur in a pure form because of the variability of the area supplied by the posterior spinal arteries. The softening often involves—as in our case—the crossed pyramidal tracts and the posterior horns to varying degrees.

In the present case of posterior spinal softening there was also pathologic evidence of diffuse ischemic alterations considered characteristic of arteriosclerotic myelopathy [15, 17]. It is evident that the fairly severe aortic atherosclerosis and the pathologic changes of the parenchymal small blood vessels were responsible for this chronic ischemic change in the spinal cord. The marked deformity and spondylotic changes of the

vertebral column may have aggravated the chronic circulatory insufficiency.

Softening in the posterior third of the spinal cord has been generally observed together with thrombosis of the spinal arteries. Syphilitic arteritis [3, 13, 14], minor indirect trauma [20] and intrathecal phenol therapy [16] have been reported as predisposing factors. In two other cases the obstruction of the posterior spinal arteries was caused by embolization of atheromatous cholesterol material [20] and fragments of nucleus pulposus [2], respectively. Similarly to this presentation in some cases of posterior spinal infarction no occlusion of the spinal blood vessels could be found [1, 21, 23, 25].

It is noteworthy that even in cases of anterior spinal softening occlusion of the anterior spinal artery cannot always be observed [10, 12, 21]. In contrast to this, occlusion either of the posterior or anterior spinal arteries did not cause infarction in every case [8, 21, 22]. Moreover, obstruction of radicular arteries including the great anterior radicular artery of Adamkiewicz often did not impair the blood flow of the spinal cord [4, 8, 19]. On the other hand, several authors have emphasized that the extent of lesions can vary considerably in cases of occlusion in the spinal arteries, i.e., there is no consistent relationship between the extent of arterial occlusion and the size of infarction [21].

These different observations can probably be explained by the peculiar vascular supply of the spinal cord. According to the literature [7, 11, 18, 21, 24] the radicular arteries supply the anterior spinal artery and the paired posterior spinal arteries. However, not all radicular arteries reach the spinal cord. Most individuals have between 4 and 10 anterior radicular arteries as well as 10 to 23 posterior radicular arteries and generally they are found only on one side. The posterior radicular arteries are smaller in diameter and more evenly distributed than the anterior ones. In most cases the blood supply of the entire lumbosacral cord including the region of the posterior spinal arteries derives from the great anterior radicular artery of Akamkiewicz. It can be supplemented by other small arteries which presumably have no essential function under physiologic conditions. The anterior and posterior spinal arteries are interconnected by numerous anastomotic branches which form one or more complete circles around each spinal segment. The conus medullaris is surrounded by the most developed anastomotic ring. The main feeding arteries of the spinal cord can also be considered anastomotic channels consisting of the terminal branches of successive radicular arteries. In fact, the posterior spinal arteries are regarded rather as plexiform channels than separate arteries. Concerning the pattern of spinal blood vessels, there is considerable variation in different regions and among individual cases.

Except for the arterial circle surrounding the conus medullaris, the functional role of the pial arterial network is controversial [18, 24], although microangiographic studies have demonstrated a connection between the distribution of the posterior and anterior spinal arteries [5, 6]. It is likely that the efficacy of the spinal collateral circulation may also vary among individual cases.

On the basis of the apparently contradictory clinical and experimental observations we have to presume that it depends mainly on the functional state of the spinal collateral circulation whether infarction develops or not. A poorly developed anastomotic network at a given place may probably become insufficient resulting in infarction with simultaneous occurrence of some systemic factors such as severe aortic sclerosis, general hemodynamic failure and degenerative changes of the vertebral column even in the absence of actual occlusion of the spinal blood vessels. On the other hand, one or more point occlusions of the main spinal arteries or their sources most probably lead to infarction only in that case where the spinal anastomotic network is poorly developed in a circumscribed area. Systemic factors as predisposing causes must also be taken into consideration in the latter case. An underlying vascular disease such as arteritis of varied origin predisposing to thrombus formation may similarly contribute to the spinal collateral circulation becoming insufficient.

The pathologic changes of the intramedullary arteries also have an important role in the development of spinal cord infarction. The obstruction of these arteries can be compensated by collateral circulation only with difficulty. In a number of cases of spinal cord infarction, they were actually found to be occluded rather than the main spinal arteries obstructed [21].

It seems reasonable to presume that the development of an area of spinal cord infarction depends on many factors among which the functional state of anastomotic arterial network appears to be the most important. The discrepancy between the incidence of the pathogenetic factors mentioned above and that of spinal cord infarction occurs in the overwhelming majority of cases because of the capacity of the anastomotic arterial network of the spinal cord to maintain adequate collateral circulation even under pathologic conditions. Scrutinized microangiographic investigation in cases of spinal cord infarction would confirm the essential role of insufficient collateral circulation in the pathogenesis of the infarction.

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