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Intervertebral disc embolization resulting in spinal cord infarction

Case report

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✓ A case of spinal cord infarction resulting from embolization of fibrocartilaginous intervertebral disc material is presented. Cases from the literature are reviewed and the theories of pathogenesis are discussed. In all reported cases the diagnosis was not made until postmortem examination.

KEY WORDS • **embolization** • **nucleus pulposus** • **paraplegia** • **spinal cord infarction**

A CASE of spinal cord infarction as a result of embolization of intervertebral disc material is presented. Although it is rare, this entity has been reported in the pathology and neurology literature.[1–4,9–18] The published cases are reviewed and the theories of pathogenesis discussed.

Case Report

This 43-year-old right-handed man with a 3-week history of low backache was first evaluated in the emergency department of the referring hospital. On the morning of admission the patient was awakened by a severe ache in the buttocks and posterior aspect of the thighs which did not radiate below the knees. Numbness and weakness of the feet developed over the next few hours so that the patient could not walk or stand. One month prior to admission he had suffered a flu-like illness which had resolved. There was no history of trauma or strain to the back.

Examination and Hospital Course. The initial examination revealed bilateral loss of pinprick appreciation in a stocking distribution up to the midcalf. Vibration sensation was normal. There was complete paralysis of ankle plantar flexor and dorsiflexor muscles and weakness of the knee flexor, hip flexor, and hip extensor muscles. Ankle jerks were absent and the knee jerks were markedly decreased bilaterally. The cerebrospinal fluid (CSF) was normal except that the level of protein was elevated to 0.50 gm/liter. A computerized tomography (CT) scan of the spine from T-11 to the sacrum was normal. The provisional diagnosis was Guillain-Barré syndrome.

Two days after admission there was a sudden onset of severe lumbosacral pressure-like pain, followed (in less than 30 minutes) by complete paraplegia with a sensory level at T-10. Myelography and abdominal ultrasonography were performed that day with completely normal results. The CSF findings were unchanged. The paraplegia did not improve and the patient was transferred to the Toronto Western Hospital. On arrival, the complete paraplegia was still present. There was no motor or sensory function below T-10. The rest of the examination was unremarkable. The CT scan, myelogram, and abdominal ultrasound study were reviewed and considered to be normal. The clinical diagnosis was spinal cord infarction or hemorrhage.

Treatment in the hospital included dexamethasone, 4 mg, administered intravenously every 6 hours and

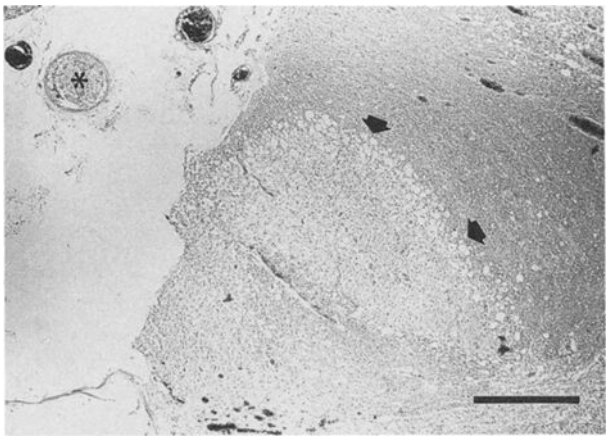

FIG. 1. Photomicrograph of a section of the spinal cord at the level of the lumbar nerve roots showing an area of infarction (outlined by *arrows*) and a nearby artery containing embolic material *(asterisk)*. HPS stain; bar = 250 μm.

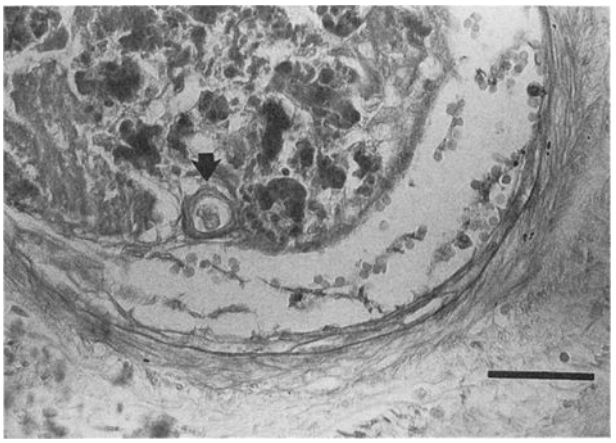

FIG. 2. High-power view of the embolic material which contains a chondrocyte in a lacuna *(arrow)*. Alcian blue stain; bar = 25 μm.

the weaning of antiembolism stockings. Nine days later there was some return of gross touch sensation on both upper thighs, but no motor recovery. Spine T_1- and T_2-weighted magnetic resonance (MR) images showed mild degenerative disc disease and Schmorl's nodes at T-9, T-10, T-12, and L-1. Two weeks after admission the patient became acutely short of breath and had a cardiopulmonary arrest. There had been no premonitory symptoms or signs of deep-vein thrombosis or of pulmonary disease.

Postmortem Examination. The general examination revealed large bilateral pulmonary emboli which were the cause of death. There was organizing thrombus of the right femoral vein. The aorta and vertebral column were grossly normal, although a detailed examination of the vertebrae and intervertebral discs was not performed. The brain was normal. The lumbar enlargement of the spinal cord was more prominent than usual, but was not striking.

Gross sections of the spinal cord after fixation revealed central dark discoloration and softening, beginning at the level of the T-12 nerve roots and extending to the tip of the conus. The changes were most extensive at the L-3 nerve root level where most of the cross-sectional area of the cord was discolored and softened. Microscopic examination of these levels revealed infarction which had a patchy distribution within the territories of both the anterior and the posterior spinal arteries. There was also infarction of the spinal nerve roots at several levels. The small arteries in the infarcted regions contained embolic material. This consisted of a predominantly acellular matrix, which stained with Alcian blue but not with fibrin stains (Fig. 1). Chondrocytes were found within the matrix in occasional emboli (Fig. 2). The emboli were in arteries of varying size, and none was found in the veins or capillaries. Emboli with the same staining characteristics were also found in small arteries associated with areas of infarction in the left psoas muscle. The popliteal and sural nerves of both legs were examined and were normal. Based on these findings, the cause of the antemortem paraplegia was believed to be spinal cord infarction secondary to embolization of the intervertebral disc material.

Discussion

Nucleus pulposus embolism was first reported by Naiman, *et al.*,[15] in 1961. They reported a 15-year-old boy who, after minor trauma (falling on his coccyx), died as a result of spinal cord and brain-stem infarction secondary to embolized disc material. Since then, several additional cases have been published.[1–4,9–14,16–18] There have been a number of reports in the veterinary literature.[5–8,20–22]

The 23 patients reported with this entity, including the case presented here, include eight men and 15 women. They ranged in age from 15 to 77 years. The clinical syndrome includes the acute onset of severe pain in the neck or back, followed within minutes by a neurological deficit referable to the brain stem or spinal cord. The spinal cord dysfunction may be complete or incomplete, is maximal within hours, and usually remains unchanged thereafter. The circumstances surrounding the illness have been reported for 14 of the 23 cases. Ten had minor head or neck trauma, but none had spinal fractures or dislocations or acute spinal cord injury. Infarction was found in the brain stem in five cases, in the cervical cord in 17 cases, in the thoracic cord in 10 cases, and in the lumbar cord in 4 cases. Investigations performed in the reported cases included plain radiographs of the spine in 10 patients, all of which were normal or showed mild degenerative changes. Myelograms are reported in eight patients, all with normal results. Magnetic resonance imaging, per-

formed in the patient reported here, did not help with the diagnosis. The CSF was examined in 11 cases; it was normal in nine and showed an elevated protein level in the other two cases. The diagnosis in all cases was made only at autopsy, although it was suspected antemortem in two of the cases reported by Bots, *et al.*[1]

Of the 23 patients, the emboli were found on the arterial side alone in 13, on the venous side alone in four, and on both sides in the remaining six. In the case of Srigley, *et al.*,[18] the emboli were found in the arterioles of the marrow of the C-3 vertebral body and in the arterioles supplying the spinal cord. Disc herniations at C2–3 and C3–4 and multiple microfractures of both vertebral endplates at the C2–3 level were found. The spinal column was examined in seven other cases, and was found to be normal in five and slightly degenerative in two.

The pathophysiology of this phenomenon is not definitely known. Any unifying theory must explain entrance of disc material into the vascular system and embolization from the point of entry to the arteries and veins of the spinal cord. Arteriovenous communications have been demonstrated in the epidural space of dogs and humans postmortem by intravascular injection of gelatinated China ink.[10,19] These communications are thought to explain the presence of emboli on either side of the circulation, regardless of whether the entry point is arterial or venous. None were noted in the 23 reported cases, but it is unlikely that they were specifically looked for.

Several theories of pathogenesis have been proposed. In their original paper, Naiman, *et al.*,[15] suggested that lateral rupture of an intervertebral disc could result in damage to a nearby radicular artery with entrance of nucleus pulposus fragments into the arterial circulation. This hypothesis was supported by Griffiths'[6] finding of a peridiscal artery containing embolic material in a dog. The vessel was located adjacent to a nucleus pulposus herniation. However, it is unlikely that this is a common mechanism because of the absence of hematomas in the reported cases, with the exception of one case in which a small organizing epidural clot was found at the appropriate level.[13]

Naiman, *et al.*,[15] also discussed the possible role of small arteries that penetrate into degenerative discs and the discs of young children. They suggested that a sudden rise in disc pressure (as would occur with an axial load) may result in the injection of nucleus pulposus material into these vessels and, via retrograde pulsion, into a radicular artery and thence to spinal arteries. This theory is plausible in their case of a young boy who fell to the ground, landing in a seated position just prior to the onset of symptoms. However, it would not explain cases in which there was little or no trauma.

A third mechanism was proposed by Feigin, *et al.*[4] They suggested that the fibrocartilage reached the spinal cord by the retrograde venous flow of intervertebral disc material which had herniated into the vertebral bodies (Schmorl's nodes). Srigley, *et al.*,[18] suggested the same mechanism, and their demonstration of fibrocartilaginous material in the vertebral body sinusoids in association with fractures of the endplates of the vertebral body is strong supportive evidence for this mechanism. In the present case, the demonstration on MR imaging of a Schmorl's node at the appropriate level for paraplegia in a patient with this syndrome is consistent with that proposed by Feigin, *et al.*,[4] but, because Schmorl's nodes are very common in the general population, we believe that this finding is of little diagnostic value.

In summary, nucleus pulposus embolization is an unusual cause of paraplegia. It should be included in the differential diagnosis when the clinical syndrome described here is observed. Antemortem diagnosis has not been reported.

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