

Case Reports

Acute Ischemic Stroke From Fibrocartilaginous Embolism to the Middle Cerebral Artery

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Background and Purpose: Fibrocartilaginous embolism from the nucleus pulposus has been reported as a rare cause of spinal cord ischemia. We were unable to find previous reports of embolism from this source to cerebral arteries.

Case Description: A previously healthy 17-year-old girl fell during a basketball game. Left hemiparesis and unresponsiveness developed followed by signs of right uncal herniation and death over a 3-day period. There was no evidence of neck, head, or spine trauma, and cardiac evaluation was normal. Neuropathological examination showed extensive ischemic infarction of the right middle cerebral artery territory, brain edema, and herniation. Complete embolic occlusion of the right middle cerebral artery by fibrocartilaginous material, consistent with nucleus pulposus, was documented. Small, terminal coronary artery branches also showed embolism by the same material and limited areas of myocardial infarction.

Conclusions: Acute cerebral embolism after minor trauma in a young patient may be rarely due to fibrocartilaginous embolism from the nucleus pulposus. The pathogenesis of this problem remains poorly understood, but systemic embolism appeared to have occurred in this case. (*Stroke* 1993;24:738-740)

KEY WORDS • cerebral ischemia • embolism • trauma

Sudden onset of paraplegia from spinal cord infarction caused by embolism of fibrocartilaginous material from the nucleus pulposus is a relatively exceptional occurrence. In 1961, Naiman and colleagues¹ reported the first such case in a 15-year-old boy who developed paraplegia and died shortly after sustaining a minor injury during a basketball game. Since then, a total of 25 cases of fibrocartilaginous embolization to the spinal cord have been reported.² Involvement of the cervical spinal cord was reported in 21 of those cases, and in six of them extension of the ischemia to the medulla oblongata was present.² However, an extensive literature search failed to reveal instances of fibrocartilaginous embolization from the nucleus pulposus to the cerebral arteries. The present case documents a fatal case of embolic occlusion of the middle cerebral artery by fibrocartilaginous material resulting in extensive ischemic brain infarction in a 17-year-old girl.

Case Report

This previously healthy 17-year-old girl was engaged in intense physical activity during a basketball game

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when she fell, apparently with brief loss of consciousness. Two to 3 hours later, she presented with progressive weakness of the left side of her body along with decrease in the level of consciousness and was then admitted to a rural hospital. Three days later, after she failed to regain consciousness, transfer to the National University Hospital in Bogotá, Colombia, was obtained. Review of systems, past medical history, and family history were unremarkable. On admission, physical examination disclosed a well-nourished girl who was afebrile with a blood pressure of 120/70 mm Hg, a pulse of 75 beats per minute, and respirations at 26 breaths per minute. There were no skin lesions and no evidence of head, neck, or spine trauma. Cardiopulmonary examination was normal, as was the remainder of her physical examination. Neurological examination showed a comatose patient responding to painful stimulation with decerebrate rigidity. The right pupil was dilated and fixed with complete right oculomotor paralysis. A left hemiplegia was present with spontaneous bilateral Babinski's sign. The patient was diagnosed as having suffered from the possible rupture of an intracranial aneurysm. A number of laboratory tests were noncontributory. Computerized tomography of the brain could not be obtained. Cardiorespiratory arrest ensued promptly, and she died after unsuccessful resuscitation maneuvers.

Postmortem examination showed an extensive ischemic infarction in the territory of the right middle cerebral artery caused by embolism and total occlusion of the initial segment of the artery (Figure 1). On sectioning the stem of the right middle cerebral artery was totally occluded, extending beyond the origin of its first branches. The brain was edematous and weighed

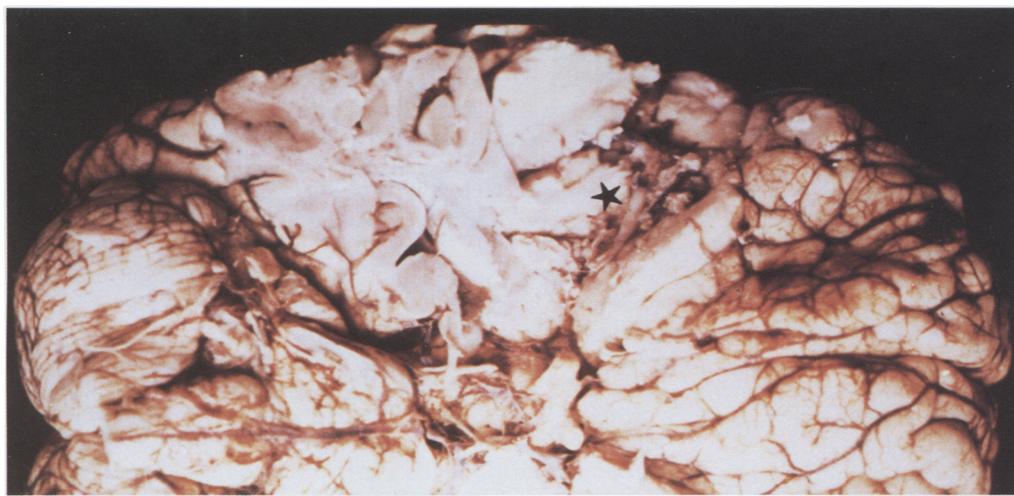


FIGURE 1. Basal view of fixed brain demonstrating presence of occlusion of stem of right middle cerebral artery (MCA). Bulging artery has been sectioned (star) to show occlusion of lumen. Temporal lobe has been removed to reveal MCA in situ and to demonstrate softening and loss of definition of tissue in infarcted MCA territory.

1,875 g. Flattening of the convolutions and bilateral uncal herniation, more severe on the right, resulting in compression of the right third cranial nerve, were observed. On histological examination the embolus consisted of fibrocartilaginous material completely filling the lumen of the right middle cerebral artery (Figure 2A). The identity of this material was consistent with nucleus pulposus on periodic acid-Schiff (PAS) staining (Figure 2B). The heart weighed 300 g and showed absence of valvular lesions, patent foramen ovale, or malformations. Two small distal coronary arteries showed occlusion by emboli of fibrocartilaginous material. Small areas of ischemic infarction were found in the myocardium in the vicinity of the areas of embolism. The remainder of the postmortem examination was unremarkable.

Discussion

Fibrocartilaginous embolism, probably originating from the nucleus pulposus, was demonstrated on postmortem examination as the cause of the embolic occlusion resulting in extensive ischemic infarction of the right middle cerebral artery territory in this patient. The young age of the patient and the history of physical

exercise during a basketball game preceding the ictus resemble quite closely the circumstances surrounding the onset of paralysis in the first case description of spinal cord embolism from this source.¹

Previously reported cases of lower brain stem infarction resulting from fibrocartilaginous embolism have been caused by occlusion of the anterior spinal artery, resulting in simultaneous infarction of the medulla oblongata and upper cervical cord.³⁻⁵ More exceptional is the occurrence of bilateral medial medullary infarction, reported by Kase and colleagues,⁶ as a result of embolic occlusion of the distal anteromedial and anterolateral branches of the anterior spinal artery in their intramedullary trajectory.

The nature of the emboli in the cerebral and coronary arteries in the case reported here appears to be fibrocartilaginous material consistent with nucleus pulposus. The arteries were totally occluded by a mixture of amorphous, partly fibrillary substance that stained soft pink on hematoxylin and eosin (Figure 2A) but stained negative for fibrin on phosphotungstic acid-hematoxylin, thereby ruling out a thrombus. The material was confirmed as being consistent with loose fibrocartilaginous ground substance by PAS-positive staining (Figure

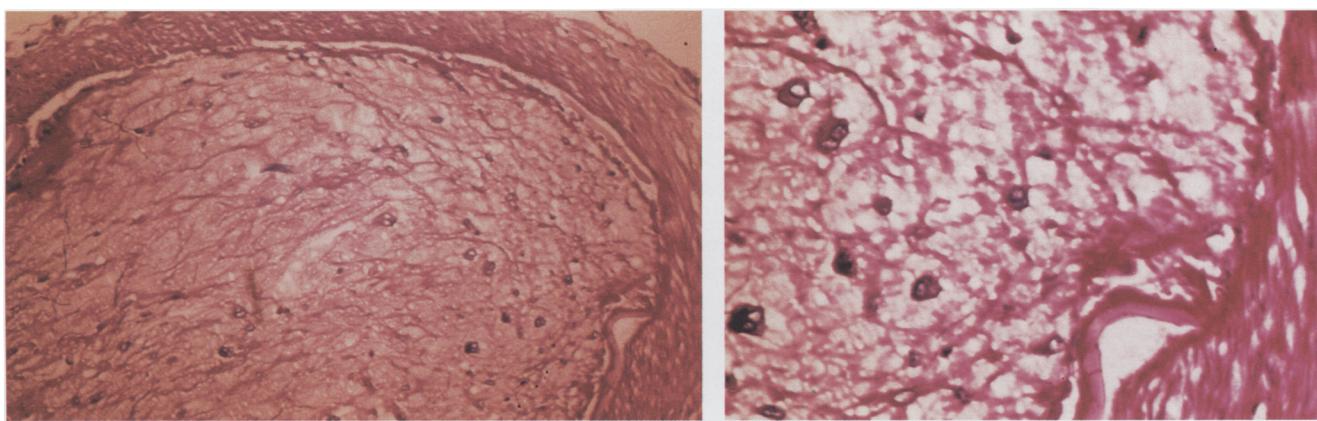


FIGURE 2. Panel A: Histological section of occluded middle cerebral artery illustrating presence of amorphous, fibrillary substance (hematoxylin and eosin stain, $\times 100$). Panel B: Higher magnification of right lower quadrant of panel A shows positive periodic acid-Schiff staining of ground substance and presence of round cell nuclei consistent with chondrocytes (periodic acid-Schiff stain, $\times 250$). Histological images demonstrate fibrocartilaginous nature of arterial embolus.

2B) and green staining with Gomori's trichrome. Furthermore, nuclei of a few chondrocytes were observed within the fibrillary amorphous collagenous matrix (Figure 2B).

The mechanisms of embolization of nucleus pulposus into spinal arteries or veins remain conjectural. The nucleus pulposus is a semifluid cushion of fibrocartilaginous material regarded as a remnant of the embryonic notochord. In children, the nucleus pulposus is soft, gelatinous, relatively large, and consists of a mucoid collagenous matrix, rich in mucopolysaccharides, containing a few multinucleated notochordal cells that disappear by the end of the first decade of life.⁷ Later, there is a gradual replacement of the mucoid material by fibrocartilage. With age its elasticity is reduced, and after the second decade degenerative changes occur in the disks along with softening and weakening of the annulus fibrosus. The intervertebral nucleus pulposus is larger and better developed in the cervical and lumbar regions than in the thoracic spine. This could explain the presence of cervical spine lesions in 21 of the 25 reported cases.²

The age distribution of cases of spinal cord emboli from nucleus pulposus follows a bimodal distribution with a first peak at age 20 and a second at age 49 years, for a median age of 36 years.² The young age of patients in the first group suggests that a larger volume of nucleus pulposus could be a factor in the pathogenesis. The second group includes 10 of the 25 patients with an age range from 46 to 77 years, five of whom were older than 60 years of age.² Degenerative changes in the nucleus pulposus, annulus fibrosus, and vertebral bone probably play a role in this second group of older patients.

Naiman and colleagues¹ postulated three mechanisms of embolization: 1) traumatic injury of the annulus fibrosus, followed by extrusion of fragments of nucleus pulposus into the spinal arterial system; 2) increase of disk pressure, resulting in "injection" of disk material into small blood vessels, which may persist inside the nucleus pulposus until adolescence; and 3) presence of arteriovenous malformations. Feigin and coworkers³ suggested that in cases with predominant venous embolization extrusion of nucleus pulposus into sinusoids and veins of the vertebral body via Schmorl's nodes may be the mechanism of embolization. Srigley et al⁸ have suggested that retrograde venous flow from vertebral marrow sinusoids through Batson's plexus into the spinal veins could also explain some cases of embolism. However, the presence of fibrocartilaginous material exclusively inside arteries, or within arteries and veins, such as in the cases reported by Feigin et al³ and Hubert et al,⁹ indicates the existence of arteriovenous communications in the spinal cord circulation. Systemic embo-

lism was probably present in the case reported here because coronary and cerebral emboli were documented. Paradoxical embolization through a pulmonary arteriovenous fistula would need to be invoked in the absence of an interventricular communication at postmortem examination of the heart.

The possibility of a source of embolism different from the nucleus pulposus may also be considered, because fibrous cartilage is also found in the pubic symphysis, ligamentum teres of the femur, glenoid and cotoyloid ligaments, and in the interarticular cartilages in a number of major joints. The history of trauma during exercise favors this hypothesis. Fibrocartilaginous embolism to spinal cord vessels is also known to occur in animals, in particular in dogs, horses, lambs, swine, and cats.¹⁰ In most instances, nucleus pulposus is the source of embolization. However, in one report involving an adult sow, it was suggested that emboli may have derived from the cartilage of vertebral physis.¹¹ Although exceptional, the differential diagnosis of acute stroke in the young must include fibrocartilaginous embolism as a potential cause.

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References

1. Naiman JL, Donohue WL, Prichard JS: Fatal nucleus pulposus embolism of spinal cord after trauma. *Neurology* 1961;11:83-87
2. Bockenek WL, Bach JR: Fibrocartilaginous emboli to the spinal cord: A review of the literature. *J Am Paraplegia Soc* 1990;13:18-23
3. Feigin I, Popoff N, Adachi M: Fibrocartilaginous venous emboli to the spinal cord with necrotic myelopathy. *J Neuropathol Exp Neurol* 1965;24:63-74
4. Kepes JJ, Reynard JD: Infarction of spinal cord and medulla oblongata caused by fibrocartilaginous emboli: Report of case. *Virchows Arch A Pathol Anat Histopathol* 1973;361:185-193
5. Bots GThAM, Wattendorff AR, Buruma OJS, Roos RAC, Endtz LJ: Acute myelopathy caused by fibrocartilaginous emboli. *Neurology* 1981;31:1250-1256
6. Kase CS, Varakis JN, Stafford JR, Mohr JP: Medial medullary infarction from fibrocartilaginous embolism to the anterior spinal artery. *Stroke* 1983;14:413-418
7. Warwick R, Williams PL (eds): *Gray's Anatomy*, ed 35 (British). Philadelphia, Pa, WB Saunders Co, 1973, pp 412-413
8. Srigley JR, Lambert CD, Bilbao JM, Pritzker KPH: Spinal cord infarction secondary to intervertebral disc embolism. *Ann Neurol* 1981;9:296-301
9. Hubert J-P, Ectors M, Ketelbant-Balasse P, Flament-Durand J: Fibrocartilaginous venous and arterial emboli from the *nucleus pulposus* in the anterior spinal system: A clinicopathological observation. *Eur Neurol* 1974;11:164-171
10. Cook JR: Fibrocartilaginous embolism. *Vet Clin North Am Small Anim Pract* 1988;18:581-592
11. Pass DA: Posterior paralysis due to cartilaginous emboli in the spinal cord. *Aust Vet J* 1978;54:100-101