Spontaneous Ischemic Infarction of the Spinal Cord with Traumatic Sequela

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Abstract:
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What is believed to have been spontaneous infarction of the spinal cord is reported in two individuals with known chronic cardiovascular disease apparently causing paraparesis while driving motor vehicles. Although blunt trauma to the spinal cord cannot be ruled out, there was no objective evidence of such. The history of spontaneous onset of paraplegia prior to trauma is clear in the first case but could only be inferred in the second case.

ADDITIONAL KEY WORDS vascular accidents cardiovascular disease paraplegia automobile accidents

Vascular accidents of the spinal cord have always been considered a rare occurrence. Blackwood found only nine examples of obstructive vascular lesions in a review of 3,737 postmortem reports from the National Hospital, Queens Square, London, in 1958. Innovations in diagnostic neuroradiology and microsurgery have initiated a resurgence of interest in spinal cord circulatory dynamics resulting in a more astute appreciation of vascular pathology and its clinical presentations. This has been reflected in a number of excellent reviews and case reports which indicate that the incidence of spinal cord vascular disease remains to be determined.

Two cases of apparent spontaneous spinal "stroke" are presented in this communication. In both instances the vascular accident appears to have been causally related to coincident automobile accidents.

CASE 1
A 67-year-old former seaman had a history of recent myocardial infarction, chronic "heart disease," and thrombophlebitis for which he was taking Coumadin. He was apparently doing well until, while driving on a clear day, he suddenly lost the use of his legs, causing the car to run off the road and crash. The accident did not involve another person or vehicle. At time of admission to the hospital, there was a complete paraplegia and partial paralysis of the upper extremities with C5 to 6 motor and sensory levels. The patient was slightly disoriented as to time and speech was somewhat slurred. External evidences of trauma were lacking and there was no complaint of head or neck pain. Skull and cervical spine x-rays were normal. Chest x-rays disclosed an enlarged heart with clear lung fields; serial EKGs were all abnormal, showing first degree AV and right bundle block, frequent premature ventricular contractions and left ventricular hypertrophy. On lumbar puncture the opening pressure was 65 mm H₂O, the fluid was clear, and manometrics were normal. Two cubic centimeters of Pantopaque (ethyl iodophenylundecanoate) were injected intrathecally and an incomplete myelographical block was seen in the T1 to 4 area suggesting spinal cord enlargement. Contrast media passed along the left lateral gutter to the cervical area (fig. 1).

Although there was no direct evidence of a neck injury, it was deemed wise to place the patient in Cone-Barton skull tongs with 8 lb traction. Exploratory decompressive laminectomy was felt to be indicated but was not performed because of a prothrombin time of 14% and a poor response to Mephyton (phytonadione) therapy. Repeat cervical spine x-rays with special views were normal and the tongs were removed on the ninth hospital day. On the thirteenth hospital day the patient became febrile to 103°F due to a gram negative urinary tract infection which responded...
FIGURE 1
Thoracic field of Pantopaque myelogram showing incomplete block beginning at arrow (T-6). This extended in irregular fashion to about the C-7 segment.

The severity of the hematological and infectious complications referred to above forced postponement of a C5 to T3 exploratory laminectomy until the fifty-seventh day of hospitalization during which time the neurological deficit remained unchanged. Upon opening the dura, the cord was found to be grossly swollen. Dorsal myelotomy revealed complete liquefaction of the cord to the T1 level. No old clot or hemosiderin staining was present.

The initial postoperative course was benign, but the patient began to run a low-grade fever with progressive cardiac and renal failure which did not respond to therapy, and he died on the ninety-third hospital day.

Autopsy revealed the cause of death to be pulmonary edema due to severe coronary arteriosclerosis, and acute myocardial infarction with extensive interstitial myocardial fibrosis. Sections of the spinal cord revealed very little tissue within the meninges identifiable as spinal cord from the C7 to T6 levels within the intact pia as it appeared grossly as a sac filled with thin milky fluid.

CASE 2
A 38-year-old Negro male USAF T Sargeant had a history of "pericarditis" 14, 12 and eight years prior to admission, as well as chronic hypertension and a known episode of atrial fibrillation. He was otherwise active and in good health until involvement in an automobile accident occurring under peculiar circumstances. While he was driving alone on a highway at noon on a clear and sunny day, his car suddenly left the road along a straight section, rolled down an embankment, and crashed. No cause for the accident could be found by the police.

The patient was initially admitted to another hospital where he was alert and oriented but amnesic regarding the accident. Frontal and occipital lacerations were sutured. The patient was paraplegic and unable to void and had been so to antibiotic therapy. Repeat myelography performed on the twenty-seventh hospital day revealed a filling pattern exactly similar to the pattern shown in figure 1.

The thoracic field of Pantopaque myelogram showing fusiform enlargement of spinal cord shadow at T5 to T6 levels.
since the accident. X-rays of the cervical and thoracic spine were normal.

He was referred to the USPHS Hospital, Seattle, the following day, where he was noted to be alert but slightly confused as to time and place. Blood pressure was 160/110 mm Hg and pulse was 104 bpm and regular. Other than the scalp lacerations and a trimalleolar fracture of the left ankle there were no other external evidences of trauma. Flaccid paraplegia and C5 to 6 to 7 arm weakness with a T4 sensory level were present with preservation of cremasteric reflexes. A Babinski sign was not present. EKG was abnormal with nonspecific T wave changes.

X-rays of the cervical, thoracic, and lumbar spine, including tomography, were normal. On lumbar puncture the opening pressure was 160 mm H$_2$O with grossly clear fluid and sluggish manometrics. Cell count revealed 4,200 RBCs and one WBC per cubic millimeter. Treatment with parenteral Dextran and intramuscular Decadron (dexamethasone) was instituted. Panmyelography performed on the second hospital day revealed a T5 to 6 defect (figure 2), consistent with intramedullary swelling of the spinal cord. The contrast media were not removed.

Four days later the patient was transferred to Madigan Army Hospital where repeat fluoroscopy revealed a complete myelographical block at T6 and an exploratory thoracic decompressive laminectomy was performed. Upon opening the dura, the spinal cord was found to be grossly swollen. Dorsal myelotomy revealed infarcted spinal cord of “toothpaste” consistency which extruded through the incision. No evidence of osseous-ligamentous trauma or hemorrhage was encountered.

Discussion

The first accurate accounts of spinal blood supply have been credited to Adamkiewicz and Kadyi. Although Bastian extended Reynold’s thoughts regarding the relationship between vascular occlusion and cerebral softening to include similar softenings in the cord, it was not until the development of arteriography (Moniz) that such relationships could be documented occasionally. But even with careful arteriography and postmortem examination the diagnosis of spinal stroke has very often remained only a clinical impression.

This is also due in part to the great amount of anatomical variation in the location and dynamics of the major feeding arteries to the spinal cord. Figure 3 illustrates diagrammatically the major arterial inputs to the spinal cord. The area in heavy dots represents the T4 level where the vascular supply is tenuous, creating a “watershed” zone most susceptible to ischemia.

In the cases presented it is believed that the spontaneous infarction of the cord occurred in the watershed zone, producing clinical deficit beginning at the uppermost level of involvement. The presentation of such infarctions in individuals with cardiovascular disease has been reviewed by Henson and Parsons and relates well to the cases presented here.

The history of spontaneous onset of paraplegia was clear from the history obtained in case 1, while it is inferred in case 2, due to the unusual circumstances of the accident. In both cases there was focal necrosis of the spinal cord in the watershed zone. Clinical examina-
tion and operative exploration revealed no evidence of gross trauma or tissue injury. No hemorrhage or hemosiderin staining of the spinal cord or its coverings was found in either case.

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References
12. Moniz E: L'encephalographie arterielle, son importance dans à la localization des tumeurs cerebrales. Rev Neurol 2: 72-90, 1927
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