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Ischemic spinal cord infarction in children without vertebral fracture

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Abstract

Spinal cord infarction in children is a rare condition which is becoming more widely recognized. There are few reports in the pediatric literature characterizing etiology, diagnosis, treatment and prognosis. The risk factors for pediatric ischemic spinal cord infarction include obstruction of blood flow associated with cardiovascular compromise or malformation, iatrogenic or traumatic vascular injury, cerebellar herniation, thrombotic or embolic disease, infection, and vasculitis. In many children the cause of spinal cord ischemia in the absence of vertebral fracture is unknown. Imaging diagnosis of spinal cord ischemia is often difficult due to the small transverse area of the cord, cerebrospinal fluid artifact and inadequate resolution of MRI. Physical therapy is the most important treatment option. The prognosis is dependent on the level of spinal cord damage, early identification and reversal of ischemia, and follow-up with intensive physical therapy and medical support. In addition to summarizing the literature regarding spinal cord infarction in children without vertebral fracture, this review article adds two cases to the literature which highlight the difficulties and controversies in the management of this condition.

INTRODUCTION

Spinal cord infarctions in children can have devastating long-term consequences, but there are few descriptions of the etiologies, treatments, and outcomes of pediatric cord infarction in the literature. Spinal cord infarction occurs less frequently in children than cerebral infarction and can be difficult to distinguish from other myelopathies [1-4]. Cord infarction may be ischemic or hemorrhagic and the etiologies, with some exceptions, are similar to those in cerebral infarction. Here, we present two cases of ischemic spinal cord infarction in children, and provide a review of the literature, with discussion of pathogenesis, diagnosis, treatment and outcome.

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METHODS

PATIENT IDENTIFICATION

Patient cases at our institution were identified by survey of members of our pediatric neurology division and by ICD-9 search of the patient database between the years 1997 and 2005, using the following codes to identify patients with spinal cord infarction: 336.1 (acute infarction of spinal cord) and 336.9 (unspecified disease of spinal cord) [5]. Patients were included if they had focal hyperintensity on T2-weighted magnetic resonance imaging (MRI) of the spinal cord consistent with infarction and had no evidence of vertebral fracture on spinal x-ray or MRI. Of 65 patients initially identified by the search, two met criteria for inclusion.

LITERATURE REVIEW

Ovid MEDLINE and Ovid OLDMEDLINE were used to search the English-language literature using keywords “spinal cord” and “infarction”, and limiting the search to “all child 0-18”. Additional reports were identified in the reference lists of relevant articles.

REPORT OF CASES

CASE 1

The patient was a previously healthy 14-year-old female who began having difficulty breathing at school immediately after experiencing a burning sensation down her neck and back. In the nurses' office, she vomited, then rapidly became unable to move her arms and legs. She was taken to the emergency room, where she had progressive difficulty breathing. After losing her gag reflex and ability to vocalize, she was intubated. Neurological examination revealed weakness of facial muscles and absent cough. She was flaccid and areflexic in all extremities, although she did exhibit minimal withdrawal in her legs and responded to light touch throughout. MRI of the brain on admission was normal. Spinal MRI performed 48 hours after admission showed an irregular T2 hyperintensity in the lower medulla and anterior cord from C1-C7 and at T3. There was no enhancement. Diffusion-weighted imaging was not done. The patient spent the next 4 weeks in the intensive care unit. She underwent an extensive infectious disease work-up, which was negative. There was no information regarding thrombotic work-up, or history of birth control pills or shots in her medical record. Lumbar puncture was unremarkable, but she was given a presumptive diagnosis of transverse myelitis and was treated with steroids, plasmapheresis, and IVIG, without clinical response. She required tracheotomy and g-tube placement, and spent the next 6 weeks in intensive inpatient rehabilitation, with only minimal, inconsistent increase in the movement of her right arm. In the months after the event, the patient experienced severe anxiety and had difficulty sleeping. There was no history of any illness prior to the onset of the event.

MRI of the spine 6 months after the event (Figure 1) revealed linear T2 hyperintensity in the anterior cord from C2 to C5 and at T3. All intervertebral disks and vertebral bodies were normal. Her clinical course and MRI at 6 months were felt to be more consistent with spinal cord infarction in the anterior spinal artery territory than transverse myelitis. Eighteen months after the event, she was able to turn her head and talk, had minimal movement of her right hand, and decreased pinprick sensation in the C3 dermatome and below. Still incontinent of urine and stool, she remained wheelchair-bound and ventilator-dependent, with continued anxiety and difficulty sleeping.

CASE 2

The patient was a 17-year-old male football-player with a history of back pain and palpitations. He was started on beta-blockers by his cardiologist. At home he became dizzy, fell down the stairs, and was immediately quadriparetic, with severe neck pain and numbness of his right

face, left arm, and both legs. He was brought to the emergency room, where he regained movement of his upper extremities, and showed some movement of his proximal lower extremities within several hours. On exam several hours after admission, he had minimal flexion of the hips and knees. There was no voluntary movement of the feet or toes. Sensory exam revealed absence of all sensory modalities in the feet up to the shin. Reflexes were 2+ throughout the lower extremity bilaterally. Abdominal reflexes were brisk. The patient retained bowel and bladder function. Numbness of his face and arms resolved over the next few days. The initial MRI, performed on the first day of admission, was read as normal. Diffusion-weighted imaging was not done. A second MRI, one month after the event, showed a vague area of T2 hyperintensity in the thoracic cord, which was thought to be artifactual. T2-weighted axial images were not obtained. The patient continued to have lower extremity weakness and numbness with very slow improvement. He was discharged home four days later in a wheelchair with outpatient physical therapy three times weekly.

Three months after the event, MRI showed multiple Schmorl's nodes in the mid-to-lower thoracic spine, with desiccation of intervertebral disks. Subtle T2 hyperintensity was also noted within the central region of the cord extending from T5-T9, on both axial and sagittal T2 imaging (Figure 2). Another more subtle T2 hyperintensity was seen within the upper cervical cord at the C2 level. These lesions were seen on the high resolution screens in the neuroradiology department. Retrospective viewing of previous films showed Schmorl's nodes and similar T2 hyperintensity on T2 sagittal imaging, but no confirmation on T2 axial view was available. Thrombotic work-up revealed compound heterozygosity for the C677T and A1298C methylenetetrahydrofolate reductase (MTHFR) mutations, and the patient was started on daily aspirin and folic acid. The patient benefited from intensive physical therapy, which included two weeks at a rehabilitation hospital. Eight months following the event, the patient was able to ambulate with a cane. He continues to have decreased proprioceptive sense in his toes, and decreased vibratory sensation to the knee.

COMMENT

Spinal cord infarction in children is rare, and difficult to diagnose. The etiologies of spinal cord stroke in children are diverse (Table 1), and different from those commonly seen in adult patients.

Epidemiology

There is no comprehensive epidemiological description of ischemic spinal cord infarction in adults or children. In 1988, Sandson and Friedman reported spinal cord infarction as 1.2% of cases admitted for stroke at the Roger Williams General Hospital over a 4 year span [6]. In the adult population, spinal cord stroke is most commonly reported in the context of aortic surgery. Reported rates of peri- and post-operative spinal cord "complications", many or most of which appear to be of vascular origin, range from 0.41% in aortic coarctation repair [7] to 12% in repair of type IIIb dissecting aortic aneurysms [8]. There are few large studies of spinal cord ischemia in children. Of 900 infants autopsied after death before 4 weeks, 21 showed evidence of hypoxic/ischemic spinal cord damage [9]. Spinal cord injury without radiographic abnormality (SCIWORA) represents a mean incidence of 34.8% of traumatic pediatric spinal cord injuries [10]. Spinal cord ischemia is one of four proposed mechanisms for SCIWORA [11]. The number of cases of SCIWORA actually attributable to spinal cord ischemia is difficult to determine because MRI was not used to define the nature of spinal cord parenchymal damage in many children studied [11].

Spinal cord infarctions are more commonly reported in adults than children: why?

Spinal cord infarction is more commonly reported in adults than in children. This might be due to age-related processes affecting perfusion of the cord. The most common causes of spinal cord infarction in adults are vascular, including atherosclerosis, abdominal aortic disease/surgery, and hypotension [6,12-17]. Compromise of blood flow to the adult cord has also been associated with cervical spondylosis [18]. With the exception of hypotension, these are diseases that take years to develop.

There are subgroups of children who are predisposed to hypotension and bony compromise of the spinal cord. Sladky and Rourke discovered spinal cord infarction in 9 of 900 asphyxiated premature neonates who underwent autopsy after death before 4 days of life [9], and Singer et al. described spinal cord infarction in an additional 3 premature neonates with paraplegia [19]. They attributed the ischemia to poor autoregulation of spinal cord blood flow during systemic hypotension secondary to prematurity. Spinal cord infarction has been associated with cervical spine changes in two children with I-cell disease after minor trauma [20] and three children with achondroplasia, two under general anesthesia [21] and one during gym class [22]. However, in general the young spinal cord is less vulnerable to infarction. The spinal cord has extensive collateral blood flow [23]. It may be easier to miss the diagnosis of spinal cord infarction in children, due to the relatively smaller volume and cross-sectional area of the pediatric spinal cord, compared to both the adult spinal cord and to the pediatric brain; the ability of radiographic imaging to detect infarctions in a small cord is limited [24].

Diagnosing spinal cord infarction

MRI allows for increased sensitivity in detecting infarction within the spinal cord. Before MRI, the diagnosis of spinal cord infarction relied upon autopsy or documented vascular occlusion by angiography. Diffusion-weighted imaging, although not commonly used on the spinal cord, now allows identification of cord ischemia within hours of the insult [25]. The limitations of spinal cord MRI include increased artifact due to cord movement and CSF flow [26]. Expectation of this artifact may lead to dismissal of actual lesions, as in patient 2. Some small infarctions can only be appreciated on high-resolution viewing monitors. Correct diagnosis of spinal cord infarction requires that the entire spine be imaged in suspicious cases, and that T2-weighted axial and sagittal views are obtained, as well as diffusion-weighted images early in the course to document ischemia. In case 2, the lesions were difficult to see in the initial MRI images on film format. When the patient was re-imaged, we included T2-weighted axial views, which revealed the infarction in more detail.

Differentiating spinal cord infarction from other myelopathy

MRI imaging and clinical presentation of spinal cord infarction can be similar to other spinal cord diseases, particularly transverse myelitis. In addition to case 1, at least six other children have been described for whom the diagnosis of ischemic spinal cord infarction was difficult to distinguish from transverse myelitis [3,4]. The MRI lesions seen in all of these cases were in the anterior two-thirds of the spinal cord, suggesting that anterior spinal artery syndrome (ASAS) was the actual diagnosis. ASAS is characterized by the abrupt onset of symmetrical paralysis, impaired temperature and pain sensation, and loss of bowel and bladder function below the level of occlusion; vibration, light touch, and proprioceptive sensations are preserved [27]. Patient 1 clearly presented with these symptoms below the cervical level, which correlated with linear T2 hyperintensity of the anterior portion of the cervical spinal cord. The Transverse Myelitis Consortium Working Group defines inclusion and exclusion criteria for the diagnosis of acute idiopathic transverse myelitis, requiring that all inclusion criteria and no exclusion criteria be met in order to confirm diagnosis [28]. Diagnostic criteria for transverse myelitis include CSF pleocytosis or IgG index, or gadolinium enhancement representing inflammation of the spinal cord; progression of symptoms within 4 hours to 21 days following

onset; and findings on MRI that do not correspond to a vascular territory. Patient 1 had unremarkable cerebrospinal fluid findings, progression of symptoms within 30 minutes, and MRI findings in the anterior spinal artery territory.

Causes of spinal cord infarction in children

As in adults, ischemic spinal cord infarction in children is caused by decreased blood flow due to hypotension; vascular injury, compression, or other impairment; or thrombosis or embolization to spinal arteries. The underlying etiologies, however, can be quite different.

Systemic hypotension leading to spinal cord infarction in children can be caused by cardiac arrest [29] or cardiac tamponade [30], both of which can occur in adults. Locally decreased perfusion can be caused by arteriovenous malformations. Spinal arteriovenous malformations are rare in children, but have been associated with paraplegia in 5 patients with thoracic malformations [31] and vascular steal in one patient [32].

Instrumentation during procedures or surgery, or trauma can lead to vascular injury which precipitates cord infarction. Umbilical artery catheters can cause spinal cord infarction in neonates [33-36], usually due to disruption of the artery of Adamkiewicz, a major artery of the thoracic cord. Damage to this artery with resulting spinal cord infarction has also been reported in one child during the removal of a thoracic neuroblastoma [37]. Surgery on the aorta is a known cause of spinal cord infarction in adults [38]; children undergo aortic operations less frequently, and they are usually confined to the arch. Four children developed spinal cord infarction following repair of aortic coarctation [27,39]. Diseases of the abdominal aorta are rare in children, although there is one case of spinal cord ischemia with aortic dissection in an 18-year-old [40]. Minor trauma has also been associated with spinal cord infarction [41-48], but the exact etiology is not well understood. Pang et al. propose that in children, the relatively less elastic spinal cord is strained inside of the more flexible spinal column during hyperflexion injuries, and that this may result in reactive vasospasm of spinal cord arteries leading to ischemia [11]. The same mechanism is proposed in a case of cord infarction occurring in a child following operative removal of a pineal choriocarcinoma in the sitting position [49].

Cerebellar herniation was associated with cervical and thoracic cord infarction in three children with metabolic encephalopathy; two underwent lumbar puncture within 24 hours prior to herniation [50]. Cerebellar herniation associated with cord infarction is also suggested in children with meningitis who have undergone lumbar puncture [51-55].

There are many ways emboli and thrombi can form and impair cord perfusion. Emboli formation can be iatrogenic: embolic material to the spinal cord vasculature is reported in a pediatric patient undergoing sclerotherapy of esophageal varices [56]. Thrombotic vasculitis and/or occlusive arachnoiditis was implicated in cord infarction of many children with central nervous system infections [53,57-69]. Several thrombotic disorders have been reported in cases of spinal cord infarction [70-75]. Anterior spinal artery thrombosis is described in a child with neuroblastoma [76]. Systemic lupus erythematosus was associated with vasculitic ischemia of the spinal cord in one patient [17], and thrombosis in another [77].

Fibrocartilaginous embolism (FCE) is becoming an increasingly well characterized cause of childhood spinal cord infarction. This condition has been well characterized in animals, especially dogs [78,79], but also in cats [80,81], horses [82], swine [83], and turkeys [84]. The first human condition was described in a 15-year-old male in 1961; nucleus pulposus emboli were found within the spinal cord arteries at autopsy [85]. Patients usually experience sudden back pain with subsequent onset of rapid neurological deterioration following a "free interval" of minutes to 2 days [1]. FCE has been demonstrated, most often affecting the anterior spinal artery territory, at autopsy in 28 adults and four pediatric patients [1,86,87]. MRI has allowed

FCE to become better characterized non-invasively in surviving patients [1-3,88,89]. There are reports of FCE with varying presentations and MRI findings [2,3]. In animals, FCE can be suspected when radiography and myelography demonstrate intervertebral disk changes and cord swelling at a level correlating with clinical features of spinal cord ischemia [90]. Tosi et al. propose that similar criteria may be used to diagnose FCE in humans and that MRI findings may include collapse of intervertebral disk space or Schmorl's nodes [1]. It has been proposed that the vertical herniation of nucleus pulposus into vertebral bodies, as in the formation of Schmorl's nodes, may allow the entry of fibrocartilaginous material into the vasculature of the spinal cord [1-3]. We believe that the pathogenesis of cord infarction in patient 2 is most likely due to FCE, because his MRI showed Schmorl's nodes and disk changes adjacent to the area of thoracic T2 hyperintensity; presence of the MTHFR mutations may also have been contributory. This diagnosis is complicated by the immediate neurological deficit, unusual sensory distribution, and lack of neurological deterioration after onset.

For some children, the etiology of spinal cord infarction is unclear. Even after extensive work-up, the mechanism remains unclear [4,91] or is found to be multifactorial [21,51-55,71,73, 74,92] with no one clear cause. The etiology of Patient 1's spinal cord infarction is unknown. She has no history of trauma, or changes in her spine that would be consistent with FCE.

Treatment

Medical treatment of childhood spinal cord infarction has not been widely reported and it is unclear if it affects outcome. Anticoagulation with heparin and aspirin appeared to improve outcome in patients with spinal cord infarction and thrombotic disorders [70,71,73]. Patient 2 was treated with aspirin and folic acid for MTHFR heterozygosity; it is unclear if this has affected his outcome. Patient 1 and Patient 2 both underwent extensive physical therapy. Supportive care and rehabilitative physical therapy are key in the long-term treatment of children with spinal cord infarction. Both patients had improvement in their neurological function, although Patient 1's improvement was minimal. Patient 2 continues to show significant improvement with therapy.

Outcome after spinal cord infarction

Outcomes after spinal cord infarction have been more widely reported in adults than in children. A review of outcomes in 199 patients by Cheshire et al. showed 43 (22%) cases resulted in death, 47 (24%) cases were unimproved, 70 (35%) cases showed some improvement, and 39 (19%) cases were considered markedly improved [17]. This review included some children, but results were not reported with respect to age, cause or spinal cord region involved. Two series of adults with cord infarction found that outcome was poorer in patients with more profound disability at onset [12,16]. One follow-up study of 8 patients with cord infarction included two children: one had improvement in neurological function, and the other had deterioration [93]. There are reports of varied outcomes in children with spinal cord trauma [10,11,94-98], but there is no confirmation of spinal cord infarction and many of the children had vertebral fractures. Moffett and Berkowitz reviewed outcomes in 19 children with bacterial meningitis and spinal cord infarction, finding death in 5 patients, some improvement in 12 patients, and return to normal function in 2 patients [58].

Common medical and psychosocial complications of spinal cord infarction

The complications of spinal cord infarction include ventilator-dependence and decreased bowel/bladder function, depending on the areas of spinal cord infarction. Patients are more susceptible to respiratory infections and bladder infections from indwelling catheters or urinary retention; these infections can have significant morbidity [97]. Patient 1 remains ventilator-dependent and has a chronic Foley catheter that has led to several urinary tract complications,

including repeated infections and surgical removal of a bladder stone. She is followed by a pulmonologist and a urologist.

Chronic pain and spastic deformity are additional reported complications of cord infarction [16,19,93]. Patient 2 experienced chronic neuropathic pain and muscle pain associated with spasticity. He is taking medication to control this. Patient 1 receives regular botulinum injections for spasticity.

It is important to recognize and address the psychological impact of spinal cord infarction. Patient 1 has experienced anxiety, depression and insomnia as a result of her condition. Patient 2 has reported no long-term psychiatric difficulties, which may be related to his steady improvement.

CONCLUSION

The understanding of ischemic spinal cord infarction in children is an evolving area in pediatric neurology. Even as the causes become better characterized in children, our cases demonstrate the continued difficulty in diagnosis. The outcome of spinal cord infarction is difficult to change after the insult. Improvement in treatment requires more rapid and exact diagnosis, with both axial and sagittal T1, T2 and diffusion-weighted

MRI of the spinal cord to visualize both intrinsic and extrinsic lesions in suspected cases. Angiography, to assess for vascular occlusion and arteriovenous malformation, may be indicated in some cases. Thrombotic evaluation plays an important role in diagnosis, because thrombotic disorders can be medically treated. As MRI becomes more sensitive, and as new modalities of neuroimaging become available, we expect the diagnosis of spinal cord infarction to become better characterized and hopefully easier to manage. It is important to recognize and address the varied physical and psychosocial complications of spinal cord infarction in order to optimize outcome.

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Figure 1.
Patient 1. Digitally enhanced MRI (TR3650.0 / TE 104.0) at 6 months after the event, showing T2 hyperintensity (arrows) in the anterior medulla and along the anterior length of the cord from C2-C5 and at T3 consistent with infarction of the anterior spinal artery territory.

Figure 2.
Patient 2. Digitally enhanced MRI (TR 4616.7/ TE 101.184) three months after the event, showing

- A.** ill-defined central T2 hyperintensity is seen in axial view at mid T6-level
- B.** enlarged sagittal image of thoracic cord demonstrating central T2 hyperintensity from T5-T8 (arrows) and Schmorl's node (arrowhead). Infarction down to T10 can be seen on other cuts.

Table 1
POSSIBLE RISK FACTORS FOR SPINAL CORD ISCHEMIA IN CHILDREN WITHOUT VERTEBRAL FRACTURE

Risk Factors	# of Patients
Cardiac	
Aortic dissection[40]	1
Aortic coarctation repair[27,39]	4
Hypotension	
Cardiac arrest[29]	6
Cardiac tamponade[30]	1
Arteriovenous malformation	
Vascular steal[32]	1
Mechanism unknown[31]	5
Thrombotic disorders	
Prothrombin variant[70,71]	2
Protein S deficiency[75]	1
Primary antiphospholipid syndrome[72]	1
Systemic lupus erythematosus[17,77]	3
Infectious disease	
Bacterial meningitis[53,57-67]	15
Amebic meningoencephalitis[69]	1
Viral encephalitis[68]	1
Cerebellar herniation	
Metabolic encephalopathy[50]	3
Minor trauma[41-48]	18
Fibrocartilaginous embolism[1-3,85-89]	10
Cancer	
Anterior spinal artery thrombosis[76]	1
Atlanto-axial instability	
Achondroplasia[22]	1
I-cell disease[20]	1
Iatrogenic	
Sclerotherapy of esophageal varices[56]	1
Prolonged neck flexion during surgery in sitting position[49]	1
Umbilical arterial catheters[33-36]	8
Thoracic tumor resection[37]	2
Multifactorial	
Achondroplasia, surgery[21]	2
Meningitis, Factor V Leiden, Chiari I malformation[74]	1
Meningitis, lumbar puncture, cardiac arrest[51-55]	6
Weight-lifting, MTHFR homozygosity[73]	1
Umbilical artery catheter, arterial switch operation[92]	1
Protein S deficiency, spine surgery, epidural catheter[71]	1
Cause unknown/unclear	
Not reported[93]	2
Unclear[4,91]	6
Total #	108