Traumatic infarction of the spinal cord in children

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Infarction of the spinal cord in childhood is rarely due to trauma. During a 15-year period (1971 to 1985), eight children were admitted to The Hospital for Sick Children, Toronto, with a diagnosis of traumatic infarction of the spinal cord. All of these patients had delayed onset of neurological signs varying between 2 hours and 4 days after their initial trauma. No bone abnormalities were seen on plain spine x-ray films. Myelography was carried out in seven of these children and found to be normal in all seven. Six patients who were paraplegic at the time of admission remained permanently paraplegic, but two with incomplete cord signs did show some improvement.

KEY WORDS • infarction • spinal cord injury • delayed paralysis • paraplegia

Infarction of the spinal cord may be encountered in association with dissecting aneurysms of the aorta, as a complication of surgery on the aorta and on the thoracic cavity, in myocardial infarction, in cardiac arrest, and in Stokes-Adams syndrome. It may be due to occlusion of the anterior spinal artery by syphilitic vasculitis, emboli from bacterial endocarditis, periarteritis nodosa, or cholesterol embolus.

In 1974, Keith reported on four children with traumatic infarction of the spinal cord who had been treated at The Hospital for Sick Children (HSC). In a review of spinal cord injuries seen at this institution over the past 15 years, we found four more patients. This paper presents a summary of all eight cases treated at this hospital.

Summary of Cases

During the 15-year period between 1971 and 1985, 95 children were admitted to the HSC with spinal-cord injuries. Of these, 34 had sustained an injury to their spinal cord without x-ray evidence of disruption of the spine. Eight of these 34 patients, four of whom have previously been reported by Keith, had a history in keeping with traumatic infarction of their spinal cord (Table 1). These eight patients (four boys and four girls) ranged in age from 1 to 10 years. Six patients were injured as the result of a motor-vehicle accident, five being pedestrians and one a passenger in an automobile. Two patients were injured as the result of a fall.

All patients had a latent period following injury which lasted from 2 hours to 4 days. Their neurological deficits developed following this latent period. Six patients very quickly progressed to a complete cord lesion at the thoracic level, and two patients had incomplete cord injuries at the upper lumbar level. All patients had normal spine x-ray films. One patient had a computed tomography metrizamide myelogram (CTMM) and a spinal angiogram. The CTMM showed localized cord atrophy of the mid and lower thoracic spinal cord, and the spinal angiogram showed occlusion of the anterior spinal artery ascending from the T-9 intercostal artery (Fig. 1). Seven other patients underwent myelograms shortly after their injury, the results of which were completely normal.

Two patients had exploratory laminectomies. In one patient, in whom the exploration was carried out several weeks after injury, the cord appeared atrophic, and in one patient, in whom the cord was examined shortly after the injury, the cord looked normal. In neither case was there evidence of local injury, swelling, or hemorrhage. Three patients sustained chest injuries and two of these patients showed marked swelling of the neck and chest. Two patients suffered a ruptured spleen; both of these required an exploratory laparotomy. Both patients were in a state of shock on arrival in the emergency department.

All patients have been followed for at least 1 year. The six patients who were totally paraplegic have remained so, whereas the two patients with incomplete...
Traumatic spinal cord infarction

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex, Age (yrs)</th>
<th>Cause of Injury*</th>
<th>Latent Period</th>
<th>Neurological Level</th>
<th>Myelography</th>
<th>Associated Injury</th>
<th>Final Outcome</th>
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<tbody>
<tr>
<td>1</td>
<td>F, 5</td>
<td>fall</td>
<td>24 hrs</td>
<td>T-6, complete</td>
<td>normal</td>
<td></td>
<td>not improved</td>
</tr>
<tr>
<td>2</td>
<td>F, 5</td>
<td>MVA (pedestrian)</td>
<td>6 hrs</td>
<td>T-2, complete</td>
<td>cord atrophy</td>
<td>chest injury, femur fracture</td>
<td>not improved</td>
</tr>
<tr>
<td>3</td>
<td>F, 1</td>
<td>MVA (pedestrian)</td>
<td>20 hrs</td>
<td>T-8, complete</td>
<td>normal</td>
<td>chest injury</td>
<td>not improved</td>
</tr>
<tr>
<td>4</td>
<td>M, 10</td>
<td>MVA (pedestrian)</td>
<td>11 hrs</td>
<td>T-10, complete</td>
<td>normal</td>
<td>chest injury</td>
<td>not improved</td>
</tr>
<tr>
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<td>M, 12</td>
<td>MVA (pedestrian)</td>
<td>4 hrs</td>
<td>T-12, complete</td>
<td>normal</td>
<td>abdominal injury (spleen rupture)</td>
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<td>F, 7</td>
<td>fall</td>
<td>2 hrs</td>
<td>T-11, complete</td>
<td>normal</td>
<td></td>
<td>not improved</td>
</tr>
<tr>
<td>7</td>
<td>M, 2</td>
<td>MVA (passenger)</td>
<td>48 hrs</td>
<td>L-1, incomplete</td>
<td>normal</td>
<td></td>
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</tr>
<tr>
<td>8</td>
<td>M, 2</td>
<td>MVA (pedestrian)</td>
<td>4 days</td>
<td>L-1, incomplete</td>
<td>normal</td>
<td>abdominal injury (spleen rupture)</td>
<td>improved</td>
</tr>
</tbody>
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* MVA = motor-vehicle accident.

Fig. 1. Spinal angiogram in Case 1. There is occlusion of the anterior spinal artery ascending from the T-9 intercostal artery.

Injuries have improved to the point of useful motor function.

Discussion

Infarction of the spinal cord without trauma is a well-known entity. In 1909, Spiller described thrombosis of the anterior spinal artery in a patient with syphilis, and in 1916, Reitter reported on a patient with infarction of the spinal cord secondary to a dissecting aortic aneurysm. Since that time, there have been numerous reports of infarction of the spinal cord associated with dissecting aneurysms and infarction secondary to surgery on the aorta and chest cavity.

In 1964, Hughes reported on a patient who sustained infarction of the spinal cord as a result of trauma. At postmortem examination, he found a transverse intimal tear encircling the aorta which was plugged by thrombus. In addition, there was a large mural hematoma which surrounded and compressed the origins of the second, third, fourth, and fifth intercostal arteries. The spinal cord was infarcted over spinal levels T3–7. In 1974, Keith reported on four of the patients in the present series.

Three of our eight patients had a chest injury, two of whom had marked swelling of the neck and cheeks suggestive of mediastinal hemorrhage which could compress the aorta and its branches. Two patients had retroperitoneal hematomas associated with rupture of the spleen; both of these patients were in shock on admission. The retroperitoneal hematoma could have interfered with the abdominal aorta and its branches and the hypotension would be an aggravating factor in these patients. One of our patients was shown on angiography to have occlusion of the anterior spinal artery branch ascending from the T-9 intercostal artery. Exploratory laminectomy was performed in two patients, one of whom showed an atrophic and one a grossly normal spinal cord.

In 1975, Ahmann et al. reported two cases of cervical cord infarction due to minor trauma in children. Both children had normal plain films and myelograms. Pathological studies revealed ischemic infarction involving the cervical cord and low medulla in one patient and the central gray matter of the low cervical cord in the other. They postulated that the vertebral arteries could have been temporarily occluded or thrown into spasm at the time of hyperextension, producing ischemia of the cervical cord.

Pang and Wilberger' presented 24 children with
spinal cord injury without radiological abnormalities and described the mechanism of neural damage in such cases, including longitudinal distraction and ischemia. They also pointed out that severe hypotension with suboptimal perfusion pressure to the traumatized cord could be an important factor in ischemic injury.

There are many excellent reviews and studies of the spinal cord blood supply and its variations in humans. 3,6-8 Most of the anterior and posterior radicular arteries supply only the anterior and posterior roots and dorsal root ganglia and are not significant suppliers of blood to the spinal cord itself. The middle and lower thoracic regions of the cord have the poorest segmental blood supply. This area of spinal cord is usually dependent on only one radicular artery, which most commonly arises from the T-7 intercostal artery, but can arise from T-8 or T-9.

The lower thoracic and lumbar regions are supplied almost exclusively by the unpaired great radicular artery of Adamkiewicz. Most of our patients sustained the damage in this vulnerable area. In Case 2, interference with the blood supply to the cord may have resulted from damage to the large branch from the costocervical trunk in the low cervical region or from destruction of one or more intercostal arteries.

Conclusions

Traumatic infarction of the spinal cord is a rare complication of spinal cord injury. However, it accounted for 8% of spinal cord injuries in children in our particular unit. The clinical picture is distinctive, with a blow to the trunk usually involving either the chest or abdomen. The children are typically neurologically intact immediately after the injury and then, following a latent period which varies from several hours to several days, they develop a profound and usually complete paraplegia which in most cases shows no evidence of recovery.

Plain spine x-ray films are normal in these patients and a myelogram shows no abnormality. Spinal angiography may show occlusion of important vessels supplying the spinal cord. There is no indication for exploratory laminectomy in any of these patients. Their outlook is poor and only if their spinal cord infarction is incomplete at the time of initial assessment will they show any degree of recovery.

References


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