Delayed paralysis after cervical fracture-dislocation

Case report

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The authors describe the late development of ascending damage to the spinal cord after a cervicothoracic fracture-dislocation that produced no evidence of cord or nerve injury other than transient mild paresthesia in the arms. After 16 years, progressive quadriplegia developed with subsequent ascending dissociated sensory loss in the upper cervical dermatomes. The presence of a central syrinx was verified at operation 18 years after the injury.

KEY WORDS • spinal cord injuries • quadriplegia • arachnoiditis • syringomyelia

A late, progressive form of syringomyelia has been reported, usually preceded by a definite injury to the spinal cord. In the case presented here there was no evidence of nerve or cord injury other than transient mild paresthesia in the arms at the time of the accident.

Case Report

A 29-year-old naval aviator suffered a fracture-dislocation at the C7-T1 vertebral level in May, 1942, when his disabled plane nosed over on landing. There was no initial impairment of sensory or motor function. He underwent a period of immobilization by halter traction and collar, followed by physiotherapy, and the following January he was examined for advice regarding return to flight duty. Examination showed his neck to be angulated forward and slightly stiff, but there was no neurological evidence of cord or nerve damage except for residual mild tingling paresthesia confined to the arms. Lumbar puncture revealed no evidence of block. The spinal fluid protein was elevated to 250 mg%.

The original x-ray examination of the cervical spine showed an anterior fracture dislocation of C-7 on T-1; there was a fracture of the spinous process of C-7 and a compression fracture of T-3. There were flecks of calcification in the intervertebral space. Unfortunately, these films are no longer available. Since the effort to correct this 7-month-old deformity by skull traction had been unsuccessful, it was decided to attempt open
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reduction. This operation was performed on January 26, 1943, with the patient in the prone position with 40 lb of traction. Efforts to pry the dislocated articular facets into a better position proved fruitless, so a posterior bone fusion was done to stabilize the displaced vertebrae. Recovery was uneventful and a stable fusion resulted. After a course of physical rehabilitation the patient was discharged to return to active duty in May, 1943. By this time his brachial paresthesia had disappeared.

This officer then continued in active combat duty during the remainder of World War II. He was once forced to parachute out of his disabled plane, but suffered no injury. He reported that wrestling was the only activity he avoided.

In 1948, he began to develop clumsiness with some weakness of the muscles in his hands and mild tingling in the last two fingers. This progressed very slowly over the next 2 years, and, in 1950, he was admitted to the U.S. Naval Hospital in Bethesda. There he was found to have slight atrophy of the interossei with reduction of sensibility to pain and temperature in the area supplied by the C-8 and T-1 spinal nerves. There was no demonstrable defect otherwise. Exploratory laminectomy by Dr. Gayle Crutchfield disclosed epidural scarring around these nerves, which he decompressed out into their intervertebral foramina.

Despite minimal residual weakness of the intrinsic muscles of his hands the patient again returned to active duty testing out new types of planes. He noticed no further disability for the next 8 years, and then he suffered a crippling progressive weakness of his arms and legs with spasticity and hyperactive reflexes in the lower extremities. Another exploratory operation at Bethesda was performed in January, 1960, on myelographic evidence of a complete spinal block at the T-1 level. This showed a severe arachnoiditis. Postoperative sepsis developed and the incision was drained. In addition, "the dura was opened and its edges stitched back." The reason for this was not given in this health record. In April, 1960, he was paraparetic but able to walk and had wasting in the intrinsic muscles of the hands. In November, his gait worsened and at an exploration of the same area of the spinal cord in January, 1961, the cervical cord was found to be swollen and fluctuant with a large pseudomeningocele. On aspiration of 6 cc of fluid from this syrinx it collapsed. Postoperatively the paraplegia and sensory loss were complete below the nipple line and there was sensory and motor loss in the C8-T1 distribution.

Since then the patient has been permanently hospitalized on the Spinal Cord Injury Service at the Veterans Administration Hospital in West Roxbury, Massachusetts. He uses a wheelchair and has spasmodic contractions of his legs and an automatic bladder. The original paresis of the intrinsic hand muscles has become complete paralysis with severe weakness and atrophy extending to the forearm muscles. Muscular testing during this past decade has shown paralysis extending upward to the seventh spinal segment with involvement of all the forearm muscles on the left, but sparing of pronator radii teres, supinator, and flexor carpi radialis and ulnaris on the right. The upper arm muscles have retained normal strength with intact biceps jerk, but the triceps jerk has been lost on the left. There is a dissociated sensory loss in the left upper extremity extending as far rostrally as the third cervical dermatome (see Fig. 1). Facial and occipital sensation are normal and there is a bilateral Horner's syndrome. Facial sweating does not occur normally but does appear on the right side of the face during urination and on both sides during defecation. There is no nystagmus or abnormality of any cranial nerves.

It was recently proposed to the patient that if there is any further extension of muscular weakness permanent drainage of the syrinx should be tried despite the fact that previous aspiration of 6 cc in 1961 failed to benefit him.

Discussion

Attention was called to this complication by Gordon Holmes in 1915. In 50 postmortem examinations on World War I soldiers dying after complete transection or severe contusion of the spinal cord he found a number of central cavities extending from the site of injury, which he described in detail. Barnett, et al., have recently published a monograph on this rare, late, progressive form of syringomyelia. They record 21 probable cases reported before Holmes' paper, and 73 others since. In 35 of these
cases, cavitation was confirmed at operation or postmortem examination. These authors have included 17 cases they themselves have seen among the 1387 patients treated at the Canadian Paraplegic Center in Toronto. This amounts to an incidence of 0.8% in paraplegics and 0.2% in quadriplegics. They also quote the incidence at the British Center in Sheffield as only 0.1%. In our review of the literature we have been able to find only nine additional reported cases of this syndrome.6-11

In view of the recent excellent publication by Barnett, et al., 2 there is no need to review the literature here. Our case is reported because few neurosurgeons have had any personal experience with this unusual complication; this case is also rare because there was no initial evidence of definite injury to the

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**TABLE 1**

<table>
<thead>
<tr>
<th>Author</th>
<th>Age, Sex</th>
<th>Vertebral Lesion</th>
<th>Onset of Symptoms Postinjury</th>
<th>Extent of Late Impairment</th>
<th>Verification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schneider &amp; Knighton, 1959</td>
<td>42, M</td>
<td>whiplash injury of cervical spine without fracture</td>
<td>2½ yrs</td>
<td>dissociated sensory loss, bilateral, with pain, weakness &amp; atrophy of arms at 5 yrs</td>
<td>laminectomy C4-5 with evacuation &amp; tube drainage of cyst; definite improvement</td>
</tr>
<tr>
<td>Turnbull, cited by Barnett, 1973</td>
<td>37, M</td>
<td>compression fracture of T12-L1</td>
<td>22 yrs</td>
<td>dissociated sensory loss of rt hand &amp; lt leg with weakness, fasciculation &amp; atrophy of rt lower arm; rt Horner's syndrome</td>
<td>laminectomy T10-L1 with tube drainage of cyst; no improvement</td>
</tr>
<tr>
<td>Bischof &amp; Frowein, 1976</td>
<td>64, F</td>
<td>compression fracture of T-10</td>
<td>4 yrs</td>
<td>spastic paralysis of legs with dissociated sensory loss below L-1</td>
<td>surgical drainage of cystic cavity</td>
</tr>
</tbody>
</table>
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spinal cord. There are only three instances in which ascending cavitation developed in the absence of immediate paralysis.1,4,10 The findings in these three cases are summarized in Table I.

The interested reader should refer to the monograph of Barnett, et al.9 for a detailed review of the clinical and pathological manifestations, as well as the theories of development of this serious complication. In their discussion of neurological signs they describe the frequent complaint of pain in the neck and arms, especially on straining, and subjective numbness with dissociated loss of sensibility to pain and temperature in the cervical dermatomes. This is often unilateral, at least in the beginning. In most instances of thoracic and lumbar injury, sensibility is preserved between the initial level of injury and the dissociated loss of sensation in the cervical dermatomes. Increasing weakness of the arms is very commonly seen with wasting of the muscles, fasciculations, and loss of tendon reflexes, indicating a lower motor neuron lesion. In addition, there may be involvement of the descending trigeminal tract or the fibers of the hypoglossal nerve if the syrinx progresses up to the medulla. A unilateral Horner's sign has been reported, as well as rare instances of neuropathic joints. The condition advances rostrally, rarely caudally. The onset of this progressive extension of the cavity has become noticeable after periods ranging from 2 months to 22 years after the initial injury.

Postmortem examinations have shown a continuous cavity ascending from the level of injury all the way up to the cervical region or even to the lower medulla. The cavity is irregular in shape, reaching its maximum diameter in the cervical cord. In the thoracic region it is often too small to produce neurological signs. When it extends asymmetricaly along the base of one posterior horn, neurological signs may at first be purely unilateral. These cysts are rarely connected with the central canal. A severe arachnoiditis binding the cord and spinal nerves at the level of injury is usually present, as demonstrated in our patient.

The experience of Barnett and Jousse8 with surgical treatment is of special interest. They recommend continued drainage of the cyst. When destruction of the cord is incomplete they have inserted a plastic tube from the lower end of the cavity to the normal subarachnoid space, as commonly practiced in the spontaneous form of syringomyelia. When the lesion has produced complete paralysis, transection of the sclerotic cord at the lower end of the cavity with insertion of a drain from its lower end to the space between the retracted stumps has been their treatment of choice. They report that in seven trials "a satisfactory result was obtained in every instance." Progression was stopped over a follow-up period of 2 to 6 years. In one patient this occurred following a second trial after only temporary benefit from the first operation. Opening the cavity and inserting a thin rubber strip has resulted in improvement in all of the four cases reported by one of us (ABR).11 In two patients there was a return of lost reflexes with considerable motor and sensory recovery. The other two were relieved of their former radicular pain. Laha, et al.7 reported neurological improvement in two of three cases in which the cyst was drained. Despite the progressive numbness and weakness of his arms, our patient has refused further surgery, because his condition has deteriorated after each of his last two operations.

The patient we have described had the characteristic signs listed above except for the absence of pain. The reason for presenting this case is the rarity of posttraumatic ascending cavitation in the absence of initial obvious damage to the spinal cord. Sixteen years after his initial cervicothoracic fracture he developed a progressive quadriplegia that worsened after surgery and has continued to progress asymmetrically in his upper extremities.

References


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