Delayed traumatic myelopathy following transfixion of the spinal cord by a knife blade

Case report

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A 35-year-old man who was stabbed in the back suffered only transient paresthesia in one leg. He was asymptomatic until 21 years later when, following a minor automobile accident, progressive myelopathy developed. Eight years later he showed a Brown-Sequard syndrome with a T-7 sensory level. At T-6 a knife blade was found that had transfixxed the spinal cord; its removal was followed by some improvement in the neurological deficit. Comparable cases are reviewed. A second usually minor trauma may trigger a rapidly progressive neurological deficit.

KEY WORDS · delayed myelopathy · spinal cord injury

MYELOPATHY associated with, but occurring many years after, spinal trauma has occasionally been reported following fracture, gunshot and stab wounds of the spine. The patient may completely recover from the initial acute traumatic disability only to present a clinical picture years later, after the trauma has almost been forgotten, which may suggest inflammatory, degenerative, vascular, neoplastic, or other types of progressive spinal cord disease.

We have had the opportunity recently to see an unusual example of delayed traumatic myelopathy in a man who had been stabbed in the back 33 years earlier, developed a progressive myelopathy 21 years later, and whose spinal cord was found to be transfixxed by a knife blade.

Case Report

This 68-year-old man was stabbed in the back in 1939. He experienced only transient tingling and numbness in the left leg for 3 weeks. There was no weakness and he was hospitalized for only 1 day. His assailant later sent him a message of apology, saying that he was not, in fact, the intended victim and informing him that the tip of the knife blade must be in his back since it was not on the end of the knife. He worked steadily and actively as a house painter and had no gait difficulty or other neurological symptoms. In October, 1960, when 56 years old, he was riding in a car that was struck from the rear, and hit the back of his head on the window but did not lose consciousness. When examined at another hospital he complained of low back pain on movement of the lumbo-
sacral spine. Plantar responses were noted to be flexor. However, a week later, a left Babinski sign and decreased pinprick perception over the right leg were observed. Within 1 year a spastic paraparesis most marked on the left, with bilateral Babinski signs had developed, and he was forced to begin using a cane. He denied any difficulty with urination or defecation, but felt that since the automobile accident his sexual potency had declined.

Examination. When examined in March, 1969, the patient was alert and well developed, with a spastic gait. The deep tendon reflexes were normal in the arms, hyperactive in the legs. Abdominal and cremasteric reflexes were absent. Both plantar responses were extensor. Strength and muscle tone in the arms were normal. There was a severe spastic paraparesis, much worse in the left leg. There was a decrease of pain and temperature sensation below T-7 on the right; there was also some decreased perception of pinprick in the left leg, but much less than the right. Vibratory sensation was diminished below T-10 bilaterally. Position sense was impaired in the left foot. Rectal examination revealed some laxity of the anal sphincter. X-ray films of the thoracic spine demonstrated the following (Fig. 1). The knife blade was in two pieces. The point (approximately 2 cm long) was buried in the center of the body of the sixth dorsal vertebra. The remainder (approximately 3.2 cm long) was in the center of the bony spinal canal at the same level, separated from the point by a 2 mm gap. A faint area of radiolucency of the bone surrounding the end of the larger segment indicated that it probably was not fixed in the bone. Myelography (Fig. 2) showed the blade passing through the center of the spinal canal with slight widening of the cord at this level. Cerebrospinal fluid examination showed clear fluid, a protein content of 83 mg%, a normal sugar and cell count, and negative serology. Complete blood count, urinalysis, and blood sugar were normal. Venereal disease determination (VDRL) was negative but a blood fluorescent Treponema antibody (FTA) determination was weakly reactive.

Operation. Laminectomy was performed by Dr. R. Smith on April 23, 1969. The rusty knife blade that had been in this man’s spinal cord for 30 years had pierced the lamina very close to the midline and lay so as to transfix the dura and the spinal cord. The fragment had passed very nearly through the midline of the cord in a sagittal plane; it was fairly firmly fixed to the tissues and to the bone beneath, but was removed with only moderate traction and no bleeding. Sur-

Fig. 1. Left: X-ray film of the dorsal spine showing the knife blade at the T-6 level. Right: Laminogram of the dorsal spine showing two parts of the knife blade.
Delayed traumatic myelopathy following transfixion of spinal cord

rounding the retained fragment, which was approximately 1 in. long × ⅛ in. wide × ⅛ in. in thickness, was approximately ⅛ in. of scar tissue and iron pigment within the substance of the spinal cord. The patient tolerated the procedure well and left the operating room in good condition. On return to the recovery room he was fully awake and moving his legs as well as preoperatively. Three years postoperatively the patient showed improvement as compared with the preoperative state. He was able to walk with a leg brace and one cane. While in the hospital he was given a course of penicillin for treatment of probable late latent syphilis.

Pathological Examination. Hematoxylin and cosin stained sections of the decalcified material surrounding the knife blade were studied. The specimen consisted of dense collagenous connective tissue and bone. The collagenous connective tissue was infiltrated with a few lymphocytes, plasma cells, macrophages and multinucleated giant cells of the foreign body type. There was a considerable amount of foreign material which varied greatly in size and color. The color ranged from bright yellow and refractile to almost black. A few of the small blood vessels within the connective tissue were moderately sclerotic. With a Prussian blue stain, the foreign material gave a positive reaction. No neural tissue was identified.

Discussion

In 1931 Guillain and Garcin reported the case of a 25-year-old painter who was stabbed in the back with resultant weakness of one leg. Two years later he was inducted into the French Army, where a typical Brown-Séquard syndrome was discovered and an x-ray showed a broken knife blade in the spinal canal. Surgery was refused; 5 years later he became paraplegic. The paraplegia disappeared following removal of the broken blade, but a Brown-Séquard syndrome persisted.

Antonelli in 1932 reported the case of a man who had been stabbed at age 16, and developed paraplegia at age 50. No improvement followed removal of the knife blade from the spinal canal.

Jones in 1943 reported a 25-year-old man stabbed in the thoracic spine without neurological symptoms. When he developed a spastic paraparesis 18 years later, a knife blade was found in the spinal canal. No improvement followed surgery and the patient died 4 months later. The spinal cord was found to be infiltrated with iron fragments and showed gliosis and atrophy.

Schneider in 1959 reported a man who became paraplegic after being shot in the upper thoracic region. He made a complete recovery after several weeks. Thirty-two years later he fell down a flight of stairs, landing on his buttocks. Progressive spastic paraparesis then developed with a T-5 sensory level. X-rays showed a bullet in the spinal canal at T-4.

MacCarty in 1954 reported the extraordinary case of a 66-year-old man who was shot in the neck in 1901 and recovered uneventfully without disability. Fifty years later while cranking a motor he felt something snap in the back of his neck. During the next month he developed pain, tenderness, and swelling in this area. A large osteomyelitic cavity was discovered around a bullet in the
body of the first cervical vertebra. Excellent recovery followed surgery.

In all of these cases the foreign body was within the spinal canal, but, to our knowledge, there has been only one other case in which the object associated with the delayed traumatic myelopathy actually penetrated and transfixed the cord. This was Case 3 of Schneider which has many similarities to our patient's story. A 43-year-old man was stabbed in the back 23 years previously without subsequent pain or other disability except for slight weakness of the left leg which disappeared 3 months following the injury. Thirteen years later he noted that coordination in the left leg, while dancing, was not as good as formerly. Stiffness and weakness of the left leg gradually progressed. Examination showed a spastic paresis of the left leg, and a sensory level at T-4. Spine films showed a knife blade in the spinal canal at T-4, which at operation was found to be transfixing the cord, surrounded by a mass of grayish black scar tissue. Immediately postoperatively the myelopathy became worse and then showed slow improvement.

In several of these cases of delayed traumatic myelopathy, the initial trauma, in spite of retention of a knife blade or bullet in the spinal canal, was followed by many asymptomatic years until a second relatively minor traumatic episode seemed to "trigger" within a short time a progressive deterioration at the site of the first lesion. Perhaps fibrous adhesions were loosened and the fragment became a mobile irritant to the cord.

The mechanisms causing the progressive myelopathy that ensues after a long period of time, either with or without the second traumatic event are uncertain. The spinal cord has been shown to move within the spinal canal with different body positions and it is easy to imagine small daily traumas and irritation of the cord, with consequent vascular and neural damage, gliosis, and arachnoiditis. Also there might be a progressive foreign body reaction to the retained fragment. All of the pathological descriptions following cord injury with a retained bullet or knife blade describe rusty fragments and iron pigmentation around the foreign body. However, this still does not explain both the delay of so many years during which the same influences were acting and during which the patient was asymptomatic, nor particularly why the myelopathy then becomes relatively rapidly progressive with severe disability within several years following the onset of symptoms.

Another mechanism of delayed posttraumatic myelopathy is that of development of a syrinx, as described by Klawans and Freeman. Since drainage of a cyst may produce significant improvement, this is an important possibility to consider. However, in the cases reviewed above, no evidence of syringomyelia was described.

The removal of a foreign object that has been imbedded in the spinal canal for many years has in some cases benefited the patient but in others there was either no change or deterioration. Because of this factor plus the fear of converting paresis into paraplegia, neurosurgical opinion is divided as to whether the knife blade should be removed. The critical determinant in our decision to operate was the consistent history that the myelopathy was disabling and progressive. Our patient was initially worse, but now, 3 years later is better than his preoperative state.

We concur with the recommendations of St. John and Rand that, in cases of spinal trauma, all foreign bodies and indriven bone fragments should be removed early, both to relieve pressure on the already damaged cord and to prevent later sequelae, such as adhesions, abscess formation, and iron encrustation. Chemotherapy should also be given in these cases.

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Delayed traumatic myelopathy following transfixion of spinal cord

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