ASCENDING SPINAL PARALYSIS

CASE PRESENTATION

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Ascending paralysis originating in the spinal cord has been a matter of concern to neurologists for a considerable period of time. Recently, there have emerged a number of clinical entities responsible for this condition. The interest derived in the so-called “whip-lash” injuries of the cervical spinal cord has tended to focus attention upon minor intraspinal lesions. We reported upon certain temporary effects which the contents of traumatic cystic cavities of the cervical spinal cord can have on certain functions of the spinal cord. At that time it was pointed out that such temporary signs constitute evidence of an incomplete lesion that might benefit from surgery. It is the purpose of this report to provide some details of circumstances wherein such a traumatic cystic cavity caused ascension of neurological signs.

CASE PRESENTATION

J.W.W. L309640. On Sept. 23, 1954, a 61-year-old white male was thrown from his riding horse when the latter stepped into a gopher hole. The patient noted immediate loss of sensation and motor ability in both of his legs. Within 2 hours of the time of the injury, the patient was operated upon in a local hospital. The laminae over D3, D4 and D5 were reported to have been removed, but the dura mater was not opened. He was treated with frequent turning, intermittent irrigation of the bladder, and enemas, and during the 4th postoperative week was made progressively ambulant in a wheelchair. Throughout this entire period from the time of injury the neurological level remained static at the 4th thoracic dermatome. He was then transferred to a Veterans Administration Hospital where ambulation with brace and crutch was begun. He progressed very well in this training, which was a quite remarkable accomplishment for a man of this age, and speaks well for the therapists involved. By the latter part of February, 1955 he was able to walk alone with brace and crutch. At this time he began to note that his sensation was being lost in his axillae and down the inner aspects of both arms. He also noted a decrease in his agility in ambulation with brace and crutch. At the same time he noted (for the first time) that spasms began to appear in both legs. The loss of sensation and motion in the arms progressed until he was entirely bedridden. The neurological consultant informed him that he had a form of ascending paralysis of unknown cause and implied that therapy would be to no avail.

At this point the patient’s family physician, Dr. William N. Horst of Crown Point, Indiana, came to me with the problem. I suggested that I would like to examine the patient, and he was brought into the office for that purpose. On April 15, 1955, neurological examination revealed partial sensory loss to C4 on the left and C8 on the right with bilateral anesthesia below T4. There was marked bilateral ulnar motor weakness, triceps weakness, and right biceps weakness, as well as bilateral radial weakness. There was atrophy of both hypothenar eminences as well as of the interossei. No ciliopinal reflex could be demonstrated. There was a minor amount of flexor-adductor spasm. The bladder had remained automatic. It was the impression of Dr. Horst that the hypesthesia as high as C4 on the left was a recent development and that the other changes from the original level of T4 had been fairly gradual. Since Dr. Horst had been present at the original operation, he was able to add the information
that there appeared to be no great amount of blood under the dura mater at that time.

It was hypothesized that the only possible lesion that would explain the progression noted was cystic cavitation of the spinal cord. The patient returned home to await admission to the hospital, but on April 22, 1955, Dr. Horst called to say that in his opinion the level was ascending more on the right side and that the patient appeared to be in moderate respiratory distress. He was therefore admitted as an emergency.

Operation was performed shortly after admission. It was found that only the dorsal spines of D8 and D5 and the lamina of D4 had been removed at the previous procedure. The laminae of D3, D3, D2 and D1 were then removed. The dura mater was opened in the midline and was found to be rather densely adherent to the cord posteriorly. The spinal cord appeared to be normal in size and coloration except for a small area at a point directly over the interspace between D4 and D5. A 25-gauge needle was inserted into the cord at this point, and 1.5 cc. of cloudy white fluid was gently aspirated. The blood vessels of the pia mater were moderately tortuous. The pia mater was incised in the midline for a distance of several centimeters. A blunt probe was then passed longitudinally in the midline, entering a cystic cavity which measured 25 mm. in length and 6 mm. in both width and depth. The cavity was irrigated thoroughly with a gentle stream of warm saline. It was estimated that perhaps one-third of the neural tissue still remained and that the cavity occupied primarily the central portion of the cord. Inspection of the upper part of the spinal cord showed that in addition to pulsation normally seen, the cord at the superior edge of the laminectomy pulsated in a fluctuant manner. Consequently, the laminectomy was extended upward through C6. A 25-gauge needle was then placed in the cord in the midline at approximately the C6 level. Again fluid was aspirated in the amount of approximately 1.5 cc. The pia mater was incised for a distance of 2 cm. in the midline. At a depth of 1 mm. beneath the pia mater, a cystic cavity was entered. Irrigation brought out what appeared to be necrotic cellular debris. Here the amount of spinal cord tissue remaining represented approximately one-half the total normally present. It appeared that the edges of the incised cord would reapproximate. Therefore, 3 mm.-size openings were punched out of the dorsal columns at the bottom of each of the cysts. In further irrigation, it was noted that the two cystic cavities apparently communicated quite freely. The point of maximal initial trauma was then further inspected and the disc material between T4 and T5 was found to be markedly protruberant posteriorly. Since the material was quite firm and could not be pushed back in place readily, the dentate ligaments were cut for several levels above and below that point. The spinal cord now floated quite freely. The dura mater was closed and the remainder of the closure was carried out in routine fashion.

Postoperative Course. Examination of the patient immediately upon awakening from the anesthetic revealed that the sensory level was T2 on the right and T4 on the left with indications of complete motor return in the arms and hands. Within several days it was quite apparent that this motor return was complete, insofar as all muscle actions were represented. Two years later, there is good strong and full use of all muscles of both arms and hands and the sensory level is T4 on the left and T3 on the right. He has complained of pain in the T1 root on the right and has been given roentgen-ray therapy to the foramen with some relief.

DISCUSSION

Several salient points emerge from an analysis of this case history. First is the fact that the original surgeons were content to commit the patient to a surgical exploration of the back without definitive surgery upon the spinal cord. The factors that deterred these surgeons from making a definitive exploration of the tissue that had received the major force of the trauma can only be postulated. Perhaps palpation with the finger (the force of which, as has been shown in experimental work, may be sufficient to induce permanent paraplegia) or the more or less "normal" appearance of the investing membranes (which obscure proper visualization of the
injured tissue) gave a sense of satisfaction that the decompression of the spinal cord had been completed by the removal of the overlying bone. Indeed, one could gain a measure of satisfaction from the fact that disability may not have been increased by the operation. The subsequent course of events in which this patient with a high degree of determination succeeded in reaching a level of physical activity not generally commensurate with a person of his age with such severe disability is commendable. However, in light of subsequent developments, one could wonder whether more definitive surgery might have resulted in a considerable sparing of sensory and motor function.

Perhaps of more significance to this discussion would be the question of the necessity for splitting the pia mater and evacuating traumatized spinal cord in this case, since this would seem to be indicated from the findings from our laboratory. There certainly is ample evidence that when pial incisions are properly conducted, no functional deficit results from this procedure alone. Further, the evidence is quite conclusive that under the same conditions the evacuation of necrotic cord material and blood elements can result in retention of function in undamaged conducting elements.

The gradual ascent of the symptoms in this case in which the predisposing factor was known should have been a matter of considerable concern in the evaluation of the events. The slowness with which the progression took place indicated that fibrous scarring was involved or that cystic cavitation was advancing. If the pia mater were intact, as it probably was, then a traumatic cyst was likely in view of the severity of the trauma. A cyst of traumatic origin could act like a subdural hematoma and extend. The protein content of this fluid was 130 mg. per cent. Since scarring would limit caudal extension, the cyst would be free to expand along the cephalad line of least resistance. Certainly there would be no accurate means of ascertaining this without operation.

The mere fact that surgical evacuation of a collection of fluid within the spinal canal with proper provisions for its continued drainage has resulted in the complete return of all function lost in the gradual progression indicates that the diagnosis and surgical therapy were correct.

SUMMARY

A case is presented to illustrate the potential of traumatic spinal cystic cavitation to produce signs of progression of symptomatology which can be reversed by surgical evacuation of the cyst.

REFERENCES