Neurological Deficits Associated With Pelvic Fractures

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Disturbances of neurological function are an uncommon complication of pelvic fractures and reports in English have been rare. Neurological data accompanying these few published cases is generally fragmentary and hardly adequate for drawing more than tentative conclusions regarding the mechanism and site of the peripheral nerve injury.

Lam surveyed the literature and found mention of nerve injury in 0.75 per cent of 1889 pelvic fractures, but encountered 9 nerve injuries in his series of 100 cases. Bonn measures described associated nerve injuries in 11 per cent of his 44 patients. More recently, Patterson and Morton reported neurological complications in 1.2 per cent of 800 pelvic fractures. Reasons for this more than tenfold disparity between rates of neurological complications are not obvious.

No pattern of pelvic ring fracture seems to predominate in instances where nerve damage has occurred. However, nerve deficits appear to be more common with fractures of the sacrum than with pelvic fractures not involving that structure. Further comment will be made on this phenomenon later in the article. Patterson and Morton reported sacroiliac joint separation in over half of their patients with neural damage; sacroiliac subluxation, however, occurs in only 6 per cent of unselected pelvic injuries.

We have recently encountered 3 cases of nerve injury complicating pelvic fractures. All have undergone careful neurological evaluation, myelography, and surgical exploration. The data from these cases have increased our understanding of the nature and site of the neural damage.

Case Reports

Case 1. A 21-year-old white man was transferred to the Indiana University Medical Center 6 hours following an automobile accident. Immediately following the injury, he had been taken to another hospital where he was found to be lethargic, "somewhat shocky," and to have a deep laceration in the right buttock. Initial examination of this wound had allowed palpation of blood vessels between the sacrum and ilium. After profuse bleeding from the laceration was controlled by packing, an abdominal exploration was undertaken. Intraperitoneal and retroperitoneal hematomas were found but no bleeding point was identified. The patient was transferred to our hospital.

Examination. The patient was lethargic and the vital signs were within normal limits. He moved his legs and arms normally. A urethral catheter was in place. X-rays showed a compression fracture of the 12th thoracic vertebra and a comminuted transverse fracture of the sacral body with anterior displacement of the fragments.

Several days after admission, the urethral catheter was withdrawn but required replacement because the patient was unable to void. By this time he was alert, but had developed fecal incontinence and did not feel bladder fullness. He described numbness in his buttocks and both heels. Detailed neurological examination at this time revealed normal strength in all muscle groups in the legs. Knee jerks were brisk but only trace ankle jerks could be elicited. The bulbocavernosus reflex was absent. The anal sphincter was flaccid. There was a gibbus at T-12 and percussion of this area caused pain and "numbness" in his legs as well as an "electric sensation" down the spine. Sensation was diminished in the S-2, S-3, and S-4 dermatomes (Fig. 1). Several days later myelography was performed. There was a slight anterior indentation of the dye column at the site of the vertebral fracture, and the inferior margin of the dural sac within the sacrum was noticeably irregular.

Operation. A laminectomy of T-10 to L-2 was undertaken. The distal cord segments and nerve roots which originated from it were found to be normal. Although there was a hard ridge on the floor of the spinal canal at the T-12 level, the spinal cord was not compressed. A laminectomy of L-5 through S-2 was immediately performed. A hematoma 3 mm. in thickness was found in the epidural space at the S-2 level. At the S-2-S-3 level the sacrum was fragmental and the distal sacral segments were angulated 90° anteriorly. Both S-4 roots and the right S-3 root had been torn. Proximal stumps of these nerves were identified but the distal ends could not be found. The left S-3 root
was ragged, stretched, and thin, but still in continuity.

*Postoperative course.* Three weeks later the urethral catheter was withdrawn and the patient established a normal voiding pattern. Repeated catherization yielded no residual urine. Although the anal sphincter remained flaccid, the patient regained continence. One year following his injury the patient had normal bowel and bladder function. He is able to achieve erection and ejaculation.

*Comment.* This patient had an injury of the lower sacral roots at a level where they still lay within the sacral canal. There was extensive fragmentation of the lower portion of the sacral body below the alar processes; no other pelvic fractures were present. Among sacral fractures, this transverse comminuted type is unusual. The traumatic forces acting on the sacrum must have been applied directly and not by crush or sheer stresses acting elsewhere on the pelvic ring. As comminution and displacement occurred violently, the sacral roots passing through the fracture site were literally torn apart.

Meyer and Wiltberger reported 2 similar injuries. Their patients complained of sacral and posterior thigh pain and were unable to void, but had "no neurological deficit." Both patients were improved by surgical decompression of the S-2 nerve roots. In one the roots were tented and partially lacerated.

Most sacral fractures are caused by sheer and rotational stresses transmitted throughout the pelvic ring at the time of impact or crushing. Indirect sacral injuries of this sort are usually associated with at least one other pelvic fracture. Sacral fractures of this type are generally non-comminuted and commonly involve the alar process, passing through one or more foramina. Minimal displacement sometimes occurs. The most commonly reported neurological deficit complicating indirect sacral fracture is weakness of the glutei, hamstrings, and plantar flexors. Cutaneous sensory deficits involved various areas of the buttock, posterior aspect of the lower limb, and lateral aspect of the foot. Loss of power in the quadriceps, dorsiflexors and intrinsic muscles of the foot have been described as occurring in various combinations with the S-1 myotomal deficit.

*Case 2.* A 22-year-old white man fractured his pelvis and right leg in an automobile accident 4 years prior to admission to Indiana University Medical Center. Separation of the symphysis pubis and right sacroiliac joint had occurred. Since the injury, he had had considerable weak-
ness and diminished sensation in the right leg. He experienced occasional stress incontinence. Erection was poorly maintained although ejaculation was achieved.

Examination at the time of admission revealed a complete absence of muscle power in the right leg below the knee. There was considerable weakness and atrophy of the right hamstrings and glutei. Knee jerks were equal but the right ankle jerk could not be elicited. Anal sphincter tone was reduced. The cutaneous sensory deficit is illustrated in Fig. 2. Myelography demonstrated dural diverticuli extending laterally at the L-5 and S-1 levels (Fig. 3). At the L-1–L-2 level the column of contrast medium was indented anteriorly (Fig. 4).

Operation. Laminectomy of L-1–L-2 was performed. The arachnoid over the conus medullaris was opaque. All nerve roots were present and normal at this level but the right S-2, S-3 and S-4 roots were found to be somewhat smaller than their mates on the left. A small ridge was found on the floor of the spinal canal. Laminectomy of L-5 and S-1 was then carried out. The intra-arachnoid space was compartmentalized by translucent septa. The L-5 and S-1 roots on the right appeared normal. The right S-2, S-3 and S-4 roots were represented only by filamentous strands of greyish tissue.

Fig. 3. Myelographic demonstration of meningeal diverticula; postero-anterior projection.

Fig. 4. Case 2. Oblique projection showing narrowing of the contrast column at L-1–L-2.

The postoperative course was satisfactory but the patient remained neurologically unchanged.

Comment. This patient was left with a severe neurological impairment following traumatic disruption of his pelvic ring, including separation of the symphysis and sacroiliac joint. Unilateral sensory and motor deficits corresponding to the L-5 and S-1 segments were complete. These roots were structurally intact in the spinal canal. The large diverticuli extending laterally from their foramina, however, indicated that avulsive forces had acted on the nerves and dural sleeves just beyond those points.

The lower sacral roots were represented only by thin strands of tissue at the lumbo-sacral level but were relatively intact in the upper lumbar region. It seems likely that stretching of these nerves somehow occurred. Loss of functional continuity resulted.

If it is assumed that the violent injury stretched the sciatic nerve, the contributing spinal nerves could have been torn. Since the fracture-dislocation of the pelvis and fracture of the right leg occurred simultaneously, it is
bar region (Fig. 7). Cerebrospinal fluid protein was 69 mg. per cent.

Operation. Laminectomy of T-12 through L-3 was performed. The right superior articular process of the L-2 vertebra was lying freely in the right side of the spinal canal in a bed of granulation tissue. The dura was opened and the lower end of the spinal cord was found to be normal. The spinal roots were all present and normal except for a yellow discoloration of right L-2 root.

Postoperative course was uneventful and the patient was discharged with no change in his neurological deficit.

Comment. Virtually complete loss of motor and sensory function in one leg resulted from severe fracture-dislocation of the pelvis. Clinical evidence indicated injury to all roots contributing to the lumbosacral plexus at a level proximal to the origin of posterior primary rami. It seems unreasonable to attribute the deficit to injury of the cauda equina by fracture of the L-2 articular process. In that event, structural damage to the roots would certainly have been plainly visible when they

indeed conceivable that their combined forces may have resulted in traction on the ipsilateral sciatic nerve. Forceful displacement of the right hemipelvis at the moment of fracture may have allowed excessive traction on the nerve, normally prevented by the unyielding intact skeletal structures.

Case 3. A 31-year-old white man was admitted to the Indianapolis V.A. Hospital 5 months after injury in an automobile accident. He had sustained fractures of the right pubic ramus, ischium and ilium, as well as a severe dislocation of the right sacroiliac joint. Multiple rib fractures had occurred. There was also renal contusion with anuria and azotemia requiring peritoneal dialysis. There had been a total sensory and motor loss in the right leg since the time of injury. His convalescence had been satisfactory.

Examination disclosed complete flaccid paralysis of all muscle groups in the right leg except for a flicker of plantar and dorsiflexion of the foot and trace contraction in the hip adductors. There was virtually no sensory function in the right leg (Fig. 5). Electromyography showed fibrillation potentials in the right lower lumbar paravertebral muscles. Myelography disclosed a right-sided dural diverticulum in the L-5 and S-1 region (Fig. 6), as well as a deep indentation in the right side of the column of contrast medium in the upper lum-

Fig. 5. Case 3. Sensory loss. Stippling: hypalgesia; solid: analgesia.
were exposed at surgery. In addition, the dural diverticulum demonstrated by myelography indicates that avulsive disruption of the spinal nerves at the foraminal level had occurred.

As in Case 2, disruption of the pelvic ring had occurred and the mechanism of nerve injury may have been similar. However, traction on a single nerve could not be the complete explanation. Tearing of the L-5 and sacral roots may have occurred because of sciatic nerve traction. But one can speculate less confidently on the mechanism of injury to the lumbar plexus (L-1 to L-4). Simultaneous downward hyperextension of the hip and backward displacement of the right side of the broken pelvis may have been the explanation. The pelvic movement would have placed traction directly on the nerves arising from the lumbar plexus. In addition, the violently stretched ilio-psoas muscle could have produced a shearing action on the taut proximal components of the plexus as they pass between its fibers.

Discussion

No deficit as severe as that in Case 3 has been previously reported. Sensory and motor loss from structures innervated by all or part of the sciatic nerve have been recorded.\textsuperscript{7,10} Instances of incontinence, obturator nerve palsy, and quadriceps weakness have been described with and without associated "sciatic palsy."\textsuperscript{14} Myelography has been performed in only one published case;\textsuperscript{10} no abnormalities other than failure to fill an L-5 root sleeve were seen. The sciatic nerve can, of course, be stretched or lacerated by acetabular fracture-dislocations, but these more peripheral injuries are usually recognized as such and classified separately.

Traction injuries of the brachial plexus have been frequently described\textsuperscript{1,2} and are well understood. Violent stretching of the brachial plexus can cause either disruption of the spinal nerves distal to their foramina or avulsion of radicular elements from the spinal cord. In the latter instance, myelographic examination has usually demonstrated a cyst-like outpouching of the arachnoid at the site of root avulsion.\textsuperscript{11,13}

We could find only 1 report of lumbar root avulsion demonstrated by myelography\textsuperscript{6} and this evidence was not verified anatomically.

Cases 2 and 3 represent traction injuries to the lumbosacral plexus. Large irregular diverticuli near the lumbosacral level were disclosed by myelography in each instance. However, avulsion of roots from the spinal cord had not occurred in any of these cases. In one case nerve roots were found to be intact immediately proximal to the foramina at which diverticula were demonstrated. It must be assumed, then, that avulsion occurred at or just beyond the intervertebral foramina. One gains the impression from studying reported cases that the level of injury is commonly in the proximal lumbosacral plexus, but data are insufficient to draw a firm conclusion. Case 2 is an exception. The lower sacral roots showed evidence of intraspinal stretch damage, although they were still attached to the cord. It should be emphasized that in each of our cases, although multiple nerves had been torn, diverticula were present at only 1 or 2 levels.

The significance of myelographically demonstrable arachnoidal diverticula is
different at cervical and lumbosacral levels. Intraspinal root avulsion in the cervical region is usually, but not always, associated with irregular outpouching at the foramen. Diverticula have not been reported following extraspinal tearing of proximal components of the brachial plexus. In contrast, the lumbosacral diverticula in our patients were associated with a tear of the nerve near or beyond the foramina; diverticula were not related to avulsion of the root from the cord. The frequency with which the neurological deficits associated with pelvic fractures are caused by lumbosacral spinal nerve avulsion is unknown. Some sacral fractures cause root tearing by forceful shearing at fracture lines. We suggest that in others movement of part of a fractured or dislocated pelvic ring, occurring at the moment of injury, could result in traction on the nearby peripheral nerves. This could produce secondary disruptive tearing of proximal plexal elements. In other words, violent movement of a broken pelvis could well cause lumbosacral plexus avulsion, just as sudden force applied to the normally moveable shoulder can cause brachial plexus injury. A variety of further observations are necessary before this problem can be adequately understood. Anatomic observations are particularly important.

**Summary**

We have reported 3 cases with unusual neurological findings complicating fractures of the pelvis. Communion of the sacrum or severe pelvic fracture-dislocation may produce lumbosacral nerve avulsion at or beyond the foramen rather than at the spinal cord. We have proposed an explanation for this occurrence and have emphasized the diagnostic value of myelography in the demonstration of related dural diverticula.

**References**