Paresthesias after lumbar disc removal and their relationship to epidural hematoma

Report of two cases

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The authors present two cases of epidural hematoma causing paresthesias and paresis after lumbar disc surgery. Good recovery followed removal of the hematomas.

KEY WORDS epidural hematoma □ herniated lumbar disc □ paresthesia □ postoperative paresis □ disc surgery

During the last 10 years, no fewer than 950 cases of herniated lumbar disc have been operated on via the interlaminar approach in this department. In this paper, we report two patients who experienced worsening of their neurological status, and required a second operation 3 and 2 days, respectively, after the first.

Case Reports

Case 1

This 37-year-old man presented with a 1-month history of low-back pain radiating down the right leg. Neurological examination showed a slight decrease in pain sensibility over the right L-5 dermatome, and weakness in dorsiflexion of his right foot. Deep tendon reflexes were normal. Myelography showed an L4-5 disc herniation with compression of the right L-5 root. A prolapsed soft disc was easily removed via an interlaminar approach, and complete hemostasis was obtained at normal arterial pressure. Soon after the operation, the pain subsided and the patient showed no additional neurological deficit.

In the late afternoon of the same day, the patient began to complain of paresthesia, described as "buzzing" or "pseudocramp," in his right leg and foot. On the 2nd postoperative day, the paresthesia gradually increased, but no pain or other neurological sign was evident. On the 3rd postoperative day, paresthesia began to spread to the left leg. Neurological examination showed increase of preoperative paresis and hypesthesia over the L-5 and S-1 dermatomes bilaterally, with impaired bladder control. Myelography showed obstruction of contrast medium at the L4-5 level.

At emergency operation a clot compressing the right L-5 root and the dural sac was removed; this was the only obvious cause of compression. The source of the hematoma was believed to be the vascular network overlying the posterior longitudinal ligament. The dural sac was found to be collapsed. No cerebrospinal fluid (CSF) leakage was found in the operative field. After operation, the paresthesia cleared promptly and the patient's impairment of sensation and sphincter control recovered well. Six months later, slight paresis of right foot dorsiflexion was still present.

Case 2

This 36-year-old man was admitted for evaluation of low-back and right leg pain which had started 4 months before. Neurological examination was normal except for a reduction of the right ankle reflex and decreased pain sensibility over the right L-5 and S-1 dermatomes. Myelography demonstrated a herniated L5-S1 disc compressing the right nerve root at S-1. At surgery, a right interlaminar L5-S1 approach permitted removal of a partially extruded disc. Complete hemostasis was achieved at normal arterial blood pressure. Upon awakening from the anesthetic, the
patient reported relief of pain; there were no additional neurological signs.

The following morning, no changes in the neurological status were found. On the 2nd postoperative day, paresthesia was noted over the right L-5 and S-1 dermatomes. Based on our experience with Case 1, we monitored the patient’s neurological status by repeated examination every hour, and paresis of the right foot was detected early. Upon reoperation, without previous myelography, a clot was found compressing the dural sac and the exposed root at the operative level. After its removal, minimal arterial bleeding from the surface of the posterior longitudinal ligament was evident. As in Case 1, the dural sac proved to be collapsed and no CSF leakage was evident. The patient made a complete recovery.

Discussion

Neural complications as a consequence of lumbar disc surgery are uncommon. Loew and Caspar 2 stated that they occur in about 1% of cases. The most frequent cause of postoperative deficit is direct stretch injury of the nerve root over a large extruded fragment of disc material. 2,4,6,9 Other less common causes are reported. 1,3,7,8,10

The rarity of lumbar epidural hematoma makes it difficult to explain the cause of this phenomenon. In our cases, a complete hemostasis was obtained at normal arterial pressure levels; no coagulation disorder was present; myelography was performed 4 days preoperatively (as we usually do to avoid the loss of the natural tamponade for bleeding by a decompressed dural sac); and no CSF leakage was seen in either operation. In both patients, after a symptom-free period of nearly 24 hours, painless paresthesia over the dermatome of the exposed root was the first symptom of this complication.

Ochoa and Torebjörk 5 experimented on the correlation between microneurographic recordings from peripheral nerves and the perception of postischemic paresthesias in awake volunteers. They found that paroxysmal discharges causing paresthesias originate from the myelinated fibers of the compressed nerves, and that pain perception is depressed by about 50% in the paresthetic area, probably because nociception is blocked centrally by the input from large myelinated fibers. In our cases, the hematomas might have initially caused partial ischemic compression of the exposed root, which responded with paroxysmal discharges, felt by the patients as “buzzing” or “pseudocramp.” Major neurological signs probably occurred when the root and the dural sac were totally compressed by further enlargement of the hematoma.

We believe that persistent painless paresthesia after a short symptom-free period following lumbar disc surgery might suggest partial root compression by a hematoma and not a simple irritation caused by stretching of the root during disc removal.

References


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