Obliterative arachnoiditis complicating lumbar spinal stenosis

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The authors report five patients with spinal stenosis who had a total myelographic block at the level of the obliterated subarachnoid space. Arachnoiditis had not been considered as a primary diagnosis until laminectomy revealed a non-pulsating, thickened dural sac that conformed to the internal configuration of the involved spinal canal. Two patients had stenosis complicated by spondyloarthrosis over multiple lumbar levels, one had a previous spinal fusion, another had degenerative spondylolisthesis, and the fifth had a large midline extruded disc at L2–3 that completely blocked the spinal canal. The dura was opened in two patients, confirming the lesion. Despite obliteration of the subarachnoid space, significant relief for approximately 1 year followed decompressive laminectomy, foraminotomy, and discectomy, with disappearance of neurogenic claudication in three patients. Postoperative erect films showed no caudad passage of contrast. While further observations are required, an awareness of this complication of spinal stenosis is important in the diagnosis and management of such patients and in evaluating their ultimate prognosis.

KEY WORDS • arachnoiditis • spinal stenosis • decompression • neurogenic claudication

SPINAL arachnoiditis is a protean disorder of multiple etiology. The most common causes include spinal anesthesia, the use of intrathecal medications, infectious and parasitic diseases, neoplasms, and trauma.1,3,6,16 At present, most cases are related to multiple myelographic and operative procedures.10,11,13,14,16 Unfortunately, etiological factors other than iatrogenic, with important diagnostic, therapeutic, and medicolegal implications, have been largely ignored. In particular, little evidence is available concerning the incidence, character, and clinical significance of arachnoiditis associated with various syndromes of spinal stenosis.

Five patients are presented who had cauda equina and nerve root compression related to stenosis of the lumbar spinal canal, complicated by spondyloarthrosis, degenerative spondylolisthesis with an intact neural arch, spinal fusion, and massive discal herniation. Complete obliteration of the spinal subarachnoid space was found in each, confined only to the involved areas. Each patient improved significantly after decompressive laminectomy, foraminotomy, and discectomy, indicating that relief of symptoms and
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FIG. 1. Case 1. Lateral (left) and posteroanterior (center) preoperative films of the lumbosacral spine show advanced spondylosis and arthrosis with relative stenosis of the entire canal. The iophendylate does not move below the midportion of the body of L-1. The oil was injected with difficulty 3 years before surgery after multiple lumbar punctures were unsuccessful at the lower levels. Right: After lumbar laminectomy, the subarachnoid space remained obliterated.

the associated effects can result in adequate restoration of function despite the changes in the pia-arachnoid.

Case Reports

Case 1

This 60-year-old man had suffered progressively severe pain in the low back and both legs for 10 years. Numbness, tingling, and episodic weakness in both his lower extremities occurred after standing and walking, and was relieved by sitting.

Examination. Hypesthesia was evident over the buttocks and perineum. The patellar reflexes were depressed, the ankle reflexes absent. There was atrophy of the buttocks, posterior thigh, calf, and anterior tibial muscle groups. The Lasègue sign was positive on the left at 20°. Peripheral pulses were normal. Lumbar spine films showed severe spinal stenosis complicated by spondyloarthrosis. Myelography confirmed the presence of stenosis with a block at L-1. The terminal end of the dural sac was brush-like rather than pointed, suggesting a complicating arachnoiditis (Fig. 1).

Operation. Laminectomy from L-1 to S-1 with bilateral foraminal decompression and medial facetectomy was performed. A herniated disc was removed from the left side at L3–4. The dural sac was thickened, rubbery, and non-pulsatile below the block.

Postoperative Course. The pain subsided and sensation returned to normal. The patient returned to work after 2½ months. Claudication did not recur.

Case 2

This 66-year-old woman had a 5-year history of low-back pain spreading down both legs. The pain was associated with walking and forced her to sit or lie down for relief. A myelogram was performed at the time of cervical discectomy 23 years earlier.

Examination. Straight-leg raising and back mobility were unrestricted. Patellar reflexes were normal but ankle reflexes were absent. There was marked weakness and atrophy of the hamstrings and gastrocnemius muscles, and moderate weakness of the anterior tibial and quadriceps groups. No significant sensory changes could be found. Pedal pulses were normal.

X-ray films of the lumbosacral spine disclosed spinal stenosis and spondyloarthrosis. No residual radiopaque oil was present (Fig. 2 upper). Lumbar puncture was impossible at the lower three levels. A normal flow was encountered only at the L1–2 level and 2 ml of iophendylate (Pantopaque) was injected. A
FIG. 2. Case 2. Upper: Plain films showing relative stenosis with spondylosis and advanced arthrosis. Lower: Myelogram shows a brush-like termination of the oil column at the level of the intervertebral disc at L2-3 (left). The tapered end of the oil column in the lateral view is typical of stenotic occlusion of the spinal canal. Postoperative films showed no further descent of residual Pantopaque.

Postoperative Course. The patient's pain subsided rapidly. Neurogenic claudication and numbness disappeared, and motor power improved. After 2 months, she was independent.

Case 3

This 69-year-old man underwent an uneventful lumbosacral discectomy and spinal fusion in 1962. Three months before the present hospitalization, he noted the gradual onset of back pain, followed by severe pain radiating to both lower extremities. Standing and walking caused increasing weakness and pain, which were relieved only by sitting and lying down.

Examination. Ankle reflexes were absent, and the patellar responses depressed. Sensation was decreased over the S-1 and S-2 dermatomes. There was atrophy of both lower limbs, with severe weakness of the anterior tibial, gastrocnemius, and hamstring muscles.

Lumbosacral films disclosed spinal stenosis and degenerative spondylolisthesis with an intact neural arch at L4-5 of approximately 1 cm with minimal olisthesis at L3-4. Advanced arthrosis was present. There was no residual iophendylate. Myelography showed a block at the upper third of the body of L-4 (Fig. 3). Arachnoiditis was a suspected complication.

Operation. Laminectomy of both L-4 and L-5 with removal of the lower half of the lamina of L-3 was followed by foraminal decompression and medial facetectomy at L4-5, where the foramina were narrowed dorsally by the arthrotic facets. The canal was markedly stenotic at the level of the ventral dislocation of the L-4 vertebra. The dura did not pulsate and was thick and rubbery, typical of obliteration of the subarachnoid space. It was not opened.

Postoperative Course. Claudication did not recur. After 1 month, the patient was free of pain and walked normally.

Case 4

A 57-year-old man with a history of 20 years of low-back pain had a spinal fusion from L-4 through S-1 in 1955. He was explored after myelography in 1974 because of recurrent low-back and extremity pain, but no relief was obtained. His left ankle reflex was absent and a sensory deficit was evident.
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FIG. 3. Case 3. Myelogram shows a total block in the midportion of the body of L-4 with spinal stenosis and degenerative spondylolisthesis with an intact neural arch at L4-5. Severe arthrosis of the posterior facets at this level is evident in the oblique (center) and lateral (right) views. In the posteroanterior view (left), the blunted terminal end of the dural sac is outlined by iophendylate through which prominent rootlets are apparent. In the oblique view (center), and the lateral view (right), the dural sac ends higher than is usually seen in lesions of this type, and the terminal end of the oil column is irregular without the usual dorsal indentation caused by hypertrophy of the facets. The block in the subarachnoid space remained after laminectomy.

over the L-5 and S-1 dermatomes. Weakness of both flexion and extension of the left foot was observed with diffuse atrophy of the buttocks, hamstrings, calf, and pretibial muscle groups.

X-ray films showed a thick bone plate covering the spinal canal, with marked stenosis beneath the fusion. The myelogram performed in 1974 showed a complete block at the upper level of the fusion with encystment of oil caudally (Fig. 4). It was not repeated.

Operation. Laminectomy with removal of the dense area of fusion down to the first sacral segment was followed by foraminal decompression and neurolysis. There was no pulsation of the exposed cast-like, thick, rubbery dura. Nerve roots were splayed out and heavily scarred. The dura was not opened.

Postoperative Course. Major pain was relieved. Two months later the patient returned to work without showing further deterioration.

Case 5

This 48-year-old man had a 20-year history of low-back pain with radiation down the right leg and, more recently, the left. Increasing weakness and atrophy of the lower extremities became apparent during the 3 months before admission. One month before hospitalization, he became totally disabled.

Examination. His back was rigid, with slight reversal of the lumbar curve. The Lasègue maneuver was positive at 40° bilaterally. Ankle jerks could not be elicited. The left knee jerk was depressed, the right absent. Both lower limbs were atrophic. Hypalgesia was present over both the L-5 and S-1 dermatomes. X-ray films disclosed narrowing at the L2-3 level; on myelography a complete block was noted at this level. The preoperative diagnosis included both herniated disc and tumor.

Operation. Laminectomy of L-2 and L-3 was performed, with foraminal decompression and the excision of a large midline herniated disc. The dura was opened before discectomy because of the suspicion of spinal cord tumor. Thickened arachnoid and matted nerve roots obliterated the subarachnoid space.

Postoperative Course. The patient improved rapidly and returned to essentially normal activity after 1 month.
Fig. 4. Case 4. Myelogram performed in 1974, after a Hibbs type spinal fusion from L-4 to the sacrum. The block on myelography begins at the upper border of the L-4 vertebra with an encysted globule of oil extending downward to the L4–5 interspace on the right side (left). The oblique view (right) shows the thick graft lying dorsal to the canal. Postoperative films disclosed no further descent of the radiopaque oil.

Discussion

Vincent, et al., in 1930, first described arachnoiditis complicating chronic arthritis of the spine. Rare cases of arachnoiditis found in association with spinal stenosis were reported by Blau and Logue, Ransford and Harries, and Verbiest. The reports of Blau and Logue, Ransford and Harries, and Falconer, et al., document patients with herniated discs without prior surgery or myelography who demonstrated arachnoiditis at the time of discectomy. Degenerative spondylothesis and spinal stenosis with neurogenic claudication were also present in several cases. Laminectomy and discectomy provided adequate relief despite the presence of arachnoiditis, the lesions being confirmed in all cases.

Epstein, et al., presented three patients with proven arachnoiditis of the terminal roots of the cauda equina. All had prior surgery and myelography for herniated discs. In two a recurrent discal extrusion was removed with relief of symptoms despite the arachnoiditis. The third patient with spondylosis and perineural scar was not improved. Arachnoiditis with persistent pain after multiple back operations virtually precludes successful long-term results from any procedure. The unique annular type of adhesive arachnoiditis producing myelopathy has responded to laminectomy and microdissection of constricting bands on the spinal cord. In the majority of cases, the process is diffuse and defies treatment. While adhesions may be separated, they tend to reform in uninvolved areas extending the pathology. The presence of widely disseminated arachnoiditis contraindicates surgery and further myelography.

In our small group of patients there were no clinical findings or x-ray characteristics diagnostic of arachnoiditis. However, myelograms suggested this complication in three patients. Pain and disability were pres-
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ent for 5 to 20 years in four patients and evident for only 3 months in the patient with degenerative spondylolisthesis. In the first two patients, the spinal canal over multiple segments was severely stenotic, and the residual space obliterated by advanced spondyloarthrosis. In the first and only patient with true radicular pain, an extruded disc was found below the level of the block on myelography. The patient with Hibbs fusion had undergone multiple operations and myelograms, with residual iophendylate evident on plain films. The last patient was included in this presentation because of the focal nature of the spinal stenosis and arachnoiditis caused by the midline disc extrusion confined to the L2–3 interspace blocking the spinal canal.

In all patients, the extent of the arachnoidal block was uniquely restricted to the levels of pathology. Two patients had no previous myelogram, and no patient had prior spinal anesthesia, evidence of infection, or subarachnoid hemorrhage. Two had had myelography 14 and 23 years before surgery, with no evidence of residual oil in the spinal canal. Lumbar puncture was impossible until the open dural sac was entered at the uppermost levels.

In addition to loss of symptoms of intermittent neurogenic claudication present in the first three patients, pain, motor deficit, and sensory disturbance resolved. The sustained response was similar to that obtained after decompression in patients with spinal stenosis and cauda equina radiculopathy without complicating arachnoiditis. Postoperative x-ray studies showed no evidence of subluxation or descent of retained iophendylate.

In no case was arachnoiditis the primary preoperative diagnosis. Obliteration of the subarachnoid space was confined to the regions compromised by stenosis. While the dura should never be opened in the treatment of “uncomplicated” spinal stenosis, two patients presented unique dural thickening that obscured the diagnosis. Biopsy in one patient showed fibrous tissue. Similar fibrous tissue was apparent grossly when the dura was opened in the second patient, who was suspected of having an intradural mass. This proved to be focal stenosis and obliteration of the subarachnoid space caused by an extruded midline disc. Despite the fact that the dura was opened and adhesions not disturbed, recovery was excellent. Retained iophendylate did not affect the result.

Decompression achieved by laminectomy, foraminotomy, neurolysis, and discectomy probably contributed to improvement in the intrinsic circulation of the cauda equina. Exploration must be thorough in areas not filled by contrast agent since no objective guide is available except clinical findings and signs of entrapment. These cases demonstrate the fact that arachnoiditis occurs as a complication of spinal stenosis with and without previous myelography and surgery, and does not necessarily indicate a poor prognosis.

The inexperienced surgeon must not be complacent in the diagnosis of arachnoiditis, since failure to recognize and treat an etiologically related spinal stenosis could deprive a patient of proper surgical relief.

References

5. Elkingston JS: Meningitis serosa circumscripta spinalis. (Spinal arachnoiditis.) Brain 59:181–203, 1936

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