

## CAUDA EQUINA COMPRESSION DUE TO NARROW SPINAL CANAL

Abdulvahap Gök\*

**Summary:** Eleven cases with signs and symptoms due to narrow spinal canal were presented. All the patients except one made almost complete recovery after surgery.

**Key words:** Narrow spinal canal, cauda equina, intermittent claudication, myelography.

At the beginning of the nineteenth centuries Lietaud and Portal reported narrowing of the vertebral canal. Portal explained this narrowing with vicious deviation of the spine (17). Many reports described the clinical pattern of cauda equina compression due to narrow spinal canal as neurogenic intermittent claudication, pseudoclaudication, cauda equina claudication (2, 3, 5, 8, 13, 19). Claudication though derived from the latin word for limp has by usage come to mean a discomfort and disability associated with exercises. Verbiest in his cases reported in 1954 (14) and 1955 (15) described the spinal stenosis in detail and attributed this phenomenon to abnormal shortness of the midsagittal diameters and thickness of the laminae.

In this article eleven patients were evaluated with reference to neurological presentation, myelographic features, operative findings and surgical results.

### CLINICAL MATERIAL

Eleven patients; five men and six women, ages range between twenty and sixtyfive were evaluated considering the symptomatology, neurological findings, myelographical appearance and surgical results (Table I). All the patients were examined neurologically and they had spine films. Contrast myelography was made by either lumbar or cisternal route. The surgical results were evaluated under the following criteria:

Excellent: Patient is able to do previous work, no complaints, full relief of discomfort.

Good: Patient is able to do previous work, but has mild discomfort as back or leg pain.

Fair: Partial relief of pain and partial improvement in motor and sensory loss.

Poor : Unable to get work and no improvement.

### DISCUSSION

All the patients in this series described backache at the beginning of their complaints. As the disease progress pain distributed to either buttocks or to legs as intermittent claudication or localized in one leg as root pain. Seven patients complained about intermittent claudication and the other four were included in the latter group. Pain occurred while standing up or walking a distance and at rest it disappeared.

---

\* Department of Neurosurgery, University of Gaziantep, School of Medicine, Gaziantep, Turkiye.

**Table I.** Operative Results in Spinal Stenosis

CASE NO	AGE YRS	DURATION OF SYMPTOMS	SYMPTOMS AND SIGNS	MYELOGRAPHIC FINDINGS	OPERATION	LENGTH OF FOLLOW UP	RESULTS
1	20	2 years	intermittent claudication no deficit straight leg raising bilateral +	narrowing between L <sub>1</sub> and L <sub>3</sub> total block at L <sub>3/4</sub>	laminectomy and partial facetectomy L <sub>2</sub> to L <sub>5</sub> both included	4 years	excellent
2	20	1 year	root pain in left leg no deficit straight leg raisin bilateral +	narrowing between L <sub>1</sub> and L <sub>4</sub> partial block at L <sub>4/5</sub>	laminectomy and partial facetectomy L <sub>2</sub> to L <sub>5</sub> both included discectomy at L <sub>4/5</sub>	4 years	excellent
3	20	1 year	root pain in right leg weakness in plantar flexion of right foot and hypoesthesia at L <sub>4</sub> , L <sub>5</sub> and S <sub>1</sub> denmatomes on the right side	narrowing between L <sub>3</sub> and L <sub>5</sub>	laminectomy and partial facetectomy L <sub>4</sub> to L <sub>5</sub> both included	4 years	good
4	47	2 years	intermittent claudication no deficit straight leg raising bilateral +	total block at L <sub>4/5</sub>	laminectomy and partial facetectomy at L <sub>4</sub> and L <sub>5</sub>	3 years	excellent
5	57	2 years	root pain in the right leg no deficit straight leg raising + on the right side	narrowing at L <sub>3</sub> and L <sub>4</sub>	laminectomy at L <sub>3</sub> and L <sub>4</sub> bilateral L <sub>3/4</sub> and L <sub>4/5</sub> partial facetectomy	2 years	excellent
6	65	10 years	intermittent claudication bilateral drop foot and bilateral weakness in dorsoflexion of foot. Hypoesthesia on both sides L <sub>5</sub> down to S <sub>5</sub> .	Partial block at L <sub>3/4</sub> and complete block at L <sub>4/5</sub>	laminectomy and partial facetectomy L <sub>3</sub> to L <sub>5</sub> both included	6 months	fair
7	61	4 years	intermittent claudication no deficit	partial block at L <sub>4/5</sub>	laminectomy and partial facetectomy at L <sub>4</sub> and L <sub>5</sub>	8 months	good
8	30	1 year	root pain in left leg no deficit	anterior defect at L <sub>4/5</sub> and complete block at L <sub>5</sub>	L <sub>5</sub> complete laminectomy L <sub>4/5</sub> discectomy	8 months	excellent
9	61	4 years	intermittent claudication not deficit	complete block at L <sub>4/5</sub>	laminectomy and partial facetectomy at L <sub>4</sub> and L <sub>5</sub>	7 months	good
10	65	15 years	intermittent claudication no deficit	lateral and posterior defect at L <sub>4/5</sub> and L <sub>5</sub> S <sub>1</sub>	laminectomy and partial facetectomy L <sub>3</sub> to L <sub>5</sub> both included	5 months	good
11	35	2 years	intermittent claudication bilateral weakness in dorsoflexion of foot. Bilateral hypoesthesia at L <sub>4</sub> , L <sub>5</sub> and S <sub>1</sub>	partial block at L <sub>4/5</sub>	laminectomy and partial facetectomy L <sub>3</sub> to L <sub>5</sub> (L <sub>5</sub> included) L <sub>4/5</sub> discectomy	2 months	good

red.

Wilson (18) divided the patients who have symptoms and signs due to cauda equina compression in two types; ischemic and postural. In both varieties the symptoms are the same. In postural type the pain is induced by body position lordotic posture, while in ischemic type muscular activity must be made to induce pain.

It must be differentiated from trombo occlusive vascular disease which shares the same symptoms with cauda equina compression. Some features are helpful in differential diagnosis. Pain in intermittent claudication due to vascular occlusive disease is produced by the same degree of exercise and completely relieved by a minute or more of rest. Bilateral aorta-iliac occlusive disease severe enough to produce disabling claudication is nearly always associated with impotence in males. Normal femoral and distal pulse and appearance of motor and sensory deficit during or after activity characterizes the patient with cauda equina compression.

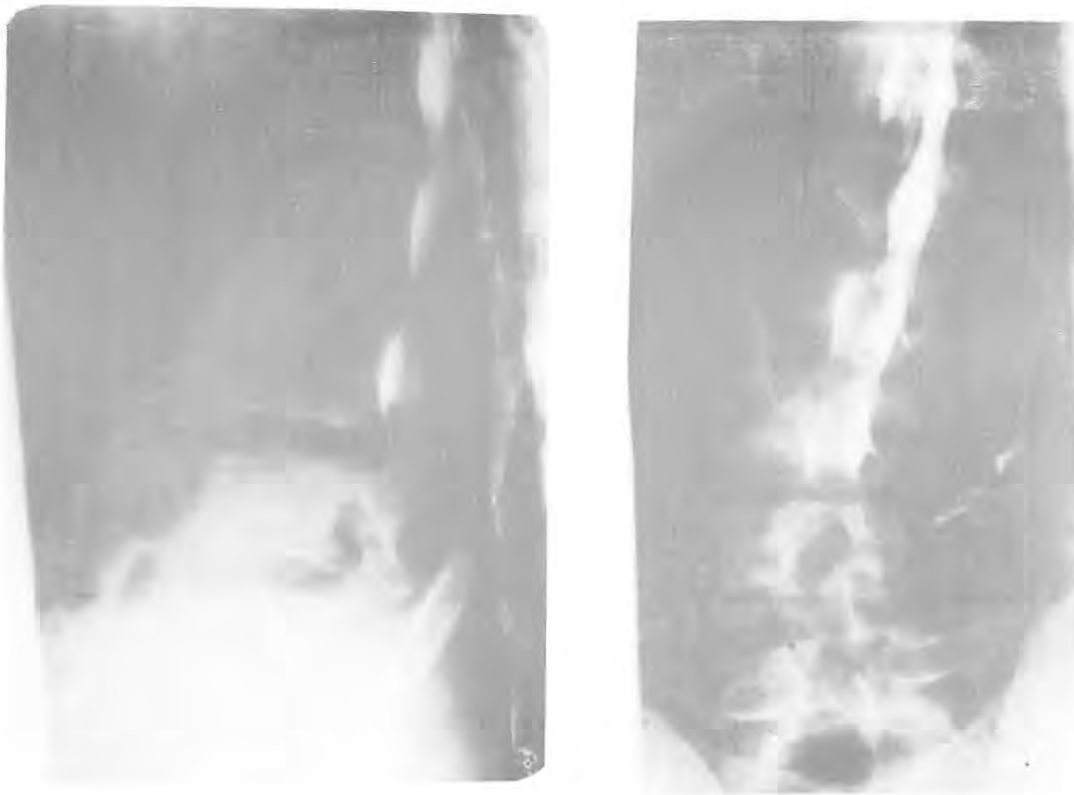
Two cases (case 6 and 11) showed severe and one case (case 3) showed mild motor and sensory deficit and also one (case 6) had urinary incontinence. The other patients showed no neurological abnormality. In all of the patients varying degrees of paravertebral muscle spasm, spinal movement restrictions and pain with spinal activity was noted. Some reports described the frequent absence of straight leg raising sign in spinal stenosis (12, 18). However this sign is seen more frequent in this series.

The most frequent findings on spine films of the patients were; scoliosis, shallowness of the interlaminar space and facets hypertrophy. These changes are known as helpful features in diagnosis of narrow spinal canal (11, 14). Measurements of interpeduncular distance

reported normal in narrow spinal canal (14). Two patients had degenerative spondylolisthesis at L<sub>4/5</sub> level. Narrow spinal canal was found in the listhetic area in these cases (Case 6 and 9). Disc herniation was found in three cases. The diagnosis of spinal stenosis was made by contrast myelography before the operation. In two patients it was not possible to make a lumbar puncture, so cisternal route was used. As a contrast by cisternal puncture iophendylate was given and in all the other patients lumbar myelography was made with iohexol (300 mg l/ml). Myelographic appearance demonstrated either uniform narrowing throughout the spinal canal, complete or partial block or posterior and posterolateral defects (Fig.1 - Fig.3).

It is stated that MRI may only help to demonstrate the presence of bulging disc, hypertrophic ligamentum flavum or long bony ridges as well as narrowness of dural sac and it is not diagnostic in spinal stenosis. However, CT is reported as the most definitive method for the diagnosis of spinal stenosis (16). Modic et al (9) in a prospective study by surface coil MRI, CT and myelography in canal stenosis and lumbar herniated disc disease found that, at the operative level there was 82.3 % agreement between MRI and surgical findings for both type and location of disease, 83.0 % agreement between CT and surgical findings and 71.4 % agreement between myelography and surgical findings.

The operative finding frequently seen was that dural sac was compressed by heavy laminae posteriorly, facets and ligamentum flavum laterally and posterolaterally. In this series the patients presented with a stenosis of canal underwent a total laminectomy, bilateral partial facetectomy and ligamentum flavum excision. No unstable back phenomena was seen in any patient after surgery. The



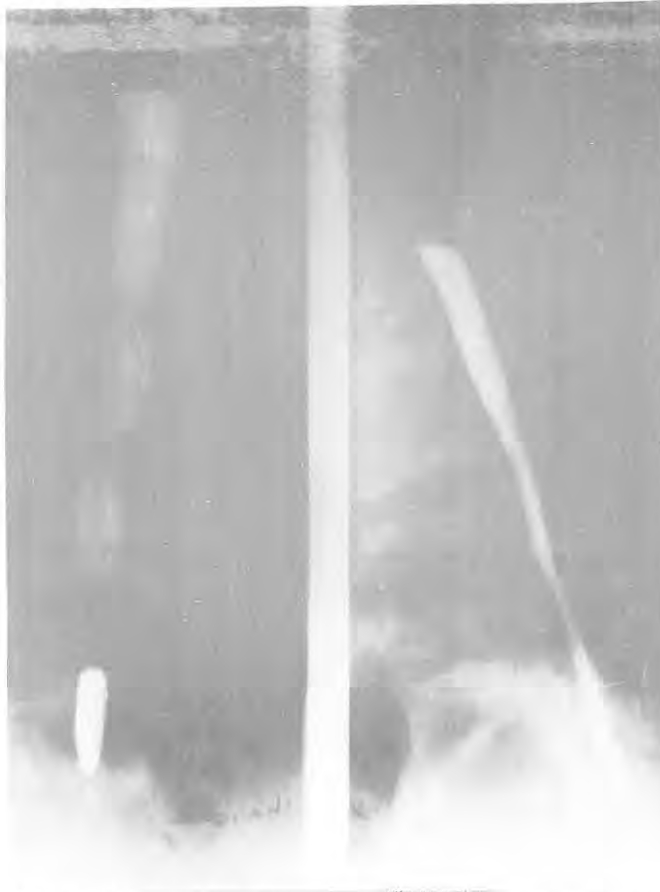
**Figure 1:** Iophendylate myelography (Case 1) A-P view shows complete obstruction at L<sub>3-4</sub> disc level. In the projection the uniform narrowing of the opaque fluid column seen from L<sub>1</sub> to L<sub>3</sub>.

protruded disc was removed in three patients

Five patients made excellent recovery (Case 1, 2, 4, 5 and 8) and they were completely relieved of symptoms. Five patients were in good and one patient (Case 6) was in fair improvement. This patient who improved fairly had severe neurological deficits before the operation.

The pathophysiologic mechanism of symptoms and signs due to cauda equina compression is not known yet. But there is an agreement in the reports that these symptoms and signs occur due to ischemic

phenomena (2, 5, 7, 8, 17, 18). Gilliat and Wilson (6) during an investigation of patients with median nerve compression at wrist found that ischemia in the affected arm resulted in intense median paresthesias and rapid onset of numbness where none had been present before. The increase in oxygen uptake of peripheral nerves while conducting impulses was determined by Cranfield et al (4). In an experimental work by Blau and Rushworth (1), it was found that the blood vessels became dilated in the ipsilateral half of the spinal cord and nerve roots of an animal related with extremity which was exercised.



**Figure 2:** Iophendylate myelography (Case 2) A-P view shows dural sac compression laterally at L<sub>2-3</sub> and L<sub>3-4</sub> level, and partial block at L<sub>4-5</sub>. Lateral projection shows narrowing of opaque fluid column from L<sub>2</sub> to L<sub>5</sub>.

Watanabe and Parke (17) at post mortem examination of a typical case of spinal stenosis of five years duration described so many changes including demyelination, pia arachnoid adhesions, interstitial fibrosis, empty axons, collapsed veins and functional radicular arteries at the stenotic area. The same histologic picture was found by Delemarter et al (5) in dogs of which cauda equina was constricted 75 % and followed for three months. They stated 50 % constriction was the critical point for neurological deficit and

histopathological abnormalities.

The neurological symptoms of a nerve root compression depends on whether the compression develops suddenly or chronically and also on the strength of the compressive force (10). The more rapid compression produce a dramatic deterioration in neurological findings (5). However is controversy between the neurological findings and structural changes in chronic compression (17).



**Figure 3:** Iohexol myelography (Case 6) A-P and lateral projection shows posterior and lateral dural compression at L<sub>3-4</sub> and complete obstruction at L<sub>4-5</sub>.

This controversy suggests; slowly developing compression may give opportunity to nerve to compensate and continue the essential functions for activity.

As a result it can be concluded that,

(1) While at rest compression of cauda equina bay narrow spinal canal does not give harm to root fibers to conduct impulses.

(2) When exercised the increased demand of nutrients of nerves was not supplied sufficiently by nerve vascular structure. This may lead to transient symptoms and signs.

(3) Under chronic compression interstitial fibrosis and destruction of the nerve structure may cause permanent deficits.

#### **References**

1. Blau JN, Rushjorth G: Observation on the blood vessels of the spinal cord and their responses to motor activity. *Brain* 81: 354-363, 1958.
2. Blau JN, Logue V: Intermittent claudication of the cauda equina. An unusual syndrome resulting from central protrusion of a lum-

- bar intervertebral disc. *Lancet* 20: 1081-1086, 1961.
3. Brish A, Lerner MA, BRaham J: Intermittent claudication from compression of cauda equina by a narrowed spinal canal. *J. Neurosurg* 21: 207-211, 1964.
  4. Cranefield PF, Brink F, Bronk DW: The oxygen uptake of the peripheral nerve of the rat. *J Neurochem* 1: 245-249, 1957.
  5. Delamerter RB, Bohlman HH, Dodge LD, Biro C: Experimental lumbar spinal stenosis. *J Bone Joint Surg.* 72-A, 1: 110-120, 1990.
  6. Gilliat RW, Wilson TG: A pneumatic-tourniquet test in the carpal-tunnel syndrome. *Lancet* 19: 595-597, 1953.
  7. Joffe R, Appleby A, ARgona V: Intermittent ischemia of the cauda equina due to stenosis of lumbar canal. *J Neurol. Neurosurg. Pshychiat.* 29: 315-318, 1966.
  8. Kavanaugh GJ, Svien HJ, Holman CB, Johnson RM: "Pseudoclaudication" syndrome produced by compression of the cauda equina. *JAMA* 206, 11: 2477-2481, 1968.
  9. Modic MT, Masaryk T, Boumphrey F, Gormastic M, Bell E: Lumbar herniated disc disease and canal stenosis: Prospective evaluation by surface coil MR, CT and Myelography. *AJR* 147: 757-765, 1986.
  10. Olmarker K, Holm S, Rydevik B: Importance of compression onset rate for the degree of impairment of impulse propagation in experimental compression injury of the porcine cauda equina. *Spine* 15, 5: 416-419, 1990.
  11. Schatzker J, Pennal GF: Spinal stenosis, a cause of cauda equina compression. *J Bone Joint Surg.* 50 B, 3: 606-618, 1968.
  12. Snyder EN, Mulfinger GL, Lambert RW: Claudication caused by compression of the cauda equina. *Am J Surg.* 130: 172-177, 1975.
  13. Sogaard I, Madsen FF: Neurogenic intermittent claudication. *Acta Neurochirurgica* 69: 195-203, 1983.
  14. Verbiest H: A radicular syndrome from developmental narrowing of the lumbar vertebral canal. *J Bone Joint Surg.* 36-B, 2: 230-237, 1954.
  15. Verbiest H: Further experiences on the pathological influence of a development narrowness of the bony lumbar vertebral canal. *J Bone Joint Surg.* 37-B, 4: 576-583, 1935.
  16. Verbiest H: Lumbar spine stenosis. Chapter 96. *Neurological Surgery.* Ed by JR Voumans. *WB saunders Company* 1990, pp. 2805-2855.
  17. Watanabe R, Parke WW: Vascular and neural pathology of lumbosacral spinal stenosis. *J Neurosurg.* 64: 64-70, 1986.
  18. Wilson CB: Significance of the small lumbar spinal canal: cauda equina compression syndromes due to spondylosis. *J Neurosurg.* 31: 499-506, 1969.
  19. Wiltse LL, Kirkaldy-Willis WH, McIvor MD: The treatment of spinal stenosis. *Clin Orthop.* 115: 83-91, 1976.