
Short report
Cauda equina compression presenting as spontaneous priapism

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Summary Disturbance of autonomic function is an unusual feature of compression of the cauda equina. A 61 year old man who had complete occlusion of the lumbar spinal canal with compression of the cauda equina from a large centrally prolapsed disc, had spontaneous priapism, precipitated by walking and relieved by resting. This symptom was comparable to claudication by compression of cauda equina. It subsided completely after surgical removal of a prolapsed L4–5 disc.

Compression of the cauda equina usually presents as pain in the lower back and legs. Symptoms are mostly related to position and exercise. Some patients develop immediate symptoms on standing up while others experience them after walking a certain distance. The syndrome is known as claudication from compression of the cauda equina (Dejerine, 1911; Verbiest, 1954; Snyder et al., 1975). Involvement of autonomic nervous function is very unusual. A patient who had spontaneous priapism on walking as the presenting symptom of cauda equina compression by a large central disc herniation is reported here.

Case report
A 66 year old Puerto Rican man had a back injury while at work on a car assembly line in 1973. His back was badly twisted as the result of a fall, after which he developed weakness of both lower limbs from the knees down. He underwent bed rest and physical therapy for about six weeks during which the weakness and back pain slowly cleared up. He stayed at work until 1974 and then retired for other reasons. Before retirement, he had varicose veins stripped from both lower limbs, and in 1967, he had undergone surgery for intractable peptic ulcer.

From 1977 onwards, he experienced numb sensation from the groin down, after walking more than about 200 yards. If he continued the exercise, the entire lower limbs became numb and dead as if anaesthetised. With rest, this would clear up with restoration of normal sensation. He started also to experience spontaneous penile erection on walking, which also subsided with rest. This became embarrassing for the patient who had to limit many of his outdoor activities. He had no bladder or bowel problems. There was no associated weakness or pain in the lower limbs.

Physical examination showed a well-developed male with an essentially normal system. His blood pressure was 126/78 mmHg.

Neurological examination showed no abnormality in the cranial nerves or upper limbs. The lower limbs showed marginal weakness of the right hamstring and calf muscles. He could balance well and walk on tiptoe and heel. There was no muscle wasting or tenderness. Straight leg raising was limited to about 45° on the right side and 60° on the left.

Pain sensation was dulled over the right leg from the knee down to the dorsum of the foot, sole, and along the back of the leg and thigh to the buttock (L5–S3 segments on the right side). Plantar responses were flexor bilaterally, and deep tendon reflexes were normal and easily elicited at the knees. With reinforcement, both ankle jerks were elicited and were symmetrical. The abdominal and cremasteric reflexes were intact. The anal sphincter showed normal tone.

The spine showed no gross abnormality, deformity, or tenderness. Arterial pulsations in the feet were intact, and no gross osteoarthritis was evident.

Routine laboratory tests including haemogram, blood chemistry, and urinalysis were normal.
Reiter protein reaction was nonreactive, and thyroid tests were normal. Serum B12 and folate levels were normal, and antinuclear antibody test was negative.

Chest radiography was normal; radiographs of the lumbar spine showed spondylolisthesis of L4 on L5 vertebra to about 15%. A lumbar myelogram showed complete block at the lower level of fourth lumbar vertebra (Figure). The spinal fluid was clear with no cells and a protein content of 0.38 g/l.

Electromyography with concentric needle electrodes was carried out bilaterally on tibialis anterior, extensor digitorum longus, peronei, gastrocnemius, and soleus and biceps femoris muscles. All muscles showed normal EMG patterns with no evidence of neurogenic or myogenic disorder. However, motor nerve conduction velocities (MNCV) of both common peroneal nerves were abnormal as shown in the Table.

He underwent lumbar laminectomy. Exploration showed considerable bulge of the annulus fibrosus at the L4–5 interspace and the nerve roots were oedematous and extremely tight within the canal. Bilateral excision of the L4–5 disc was made along with foraminotomy, thereby releasing the nerve roots.

The postoperative period was uneventful. When reviewed six weeks after surgery, he remained totally asymptomatic, tolerating physical activity well, especially walking. Penile erection and numbness of the lower limbs, both related to walking, had cleared up completely.

Motor nerve conduction velocity values of the common peroneal nerves had improved to 42.2 m/s on the right side and 45.4 m/s on the left. Ankle to extensor digitorum brevis (EDB) latency had come down to 6.5 ms on both sides.

**Discussion**

The usual aetiological mechanism of compression of the cauda equina is a large central disc herniation (Blau and Logue, 1961). A congenitally narrow spinal canal makes the contents more compressible by a relatively small enlargement of the disc (Verbiest, 1954). A typical patient with cauda equina compression complains of pain in the lower back, often radiating down the legs. Muscle weakness and paralysis are less frequent. Sensory and reflex changes are also common. However, sphincter disturbances and disturbed autonomic functions are extremely rare. This situation is not easy to explain, considering the parasympathetic outflow contained in the sacral nerve roots (S2–S4), forming part of the cauda equina. Clinical experience indicates that they usually escape serious injury by cauda equina compression.

The patient presented here had obvious cauda equina compression, as demonstrated by the myelogram and later confirmed by surgical exploration. His spinal cord was not directly or indirectly involved. However, one of his cardinal symptoms had been spontaneous priapism, directly related to exercise (walking). He had no bladder or bowel disturbance.

Penile erection results from parasympathetic stimulation. The parasympathetic fibres arise in the intermediolateral cells of the second, third, and fourth sacral segments of the spinal cord, and emerge in the pelvic nerves to form perivesicular, prostatic, and cavernous plexuses (Chusid and McDonald, 1964). These plexuses supply vasodilator fibres to the corpora cavernosa and motor fibres to the compressor urethrae, ischio-cavernous and bulbocavernous muscles, all concerned with penile erection and ejaculation.

Apart from penile tumescence caused by sexual excitation, a purely spinal reflex erection may follow stimulation of the glans penis. The patient
under discussion had spontaneous and recurrent priapism precipitated by walking with no possibility of sexual excitation or penile stimulation. Also, with rest this process invariably reversed, thereby resembling in its evolution and dissolution, intermittent claudication. There was no associated pain or discomfort. This syndrome completely subsided after removal of the offending prolapsed disc, and this strongly suggests a causal relationship.

References


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*J Neurol Neurosurg Psychiatry* 1979 42: 280-282
doi: 10.1136/jnnp.42.3.280

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