

Reflex Sympathetic Dystrophy Associated With Low Lumbar Disc Herniation

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Reflex sympathetic dystrophy can be associated with lumbar disc herniations. Both central and peripheral neuroanatomic pathways can be implicated in the development of this syndrome. Clinical findings of vasomotor instability in the leg supported by plain roentgenograms showing osteopenia, bone scan showing increased uptake, and a favorable response with sympathetic blocks suggest the diagnosis. Symptoms should be relieved with appropriate nerve root decompression but may require, in addition, a therapeutic lumbar sympathetic blockade. [Key words: low-back pain, herniated lumbar disc, reflex sympathetic dystrophy, therapy]

REFLEX SYMPATHETIC dystrophy is a painful clinical syndrome resulting from either significant or minor trauma to the peripheral and, less often, to the central nervous system. Clinically, the syndrome is characterized by burning pain in the involved extremity associated with vasomotor dysfunction and dystrophic changes in the skin, bone, and joints.^{6,12}

While the hallmark of this syndrome is an abnormal autonomic reflex, the pathogenesis is uncertain. This fact, coupled with the frequent disparity between the severity of the symptom complex manifest and the apparent precipitating event, has yielded a confusing and extensive litany of terms designating the syndrome.¹² In deference to Weir Mitchell, who originally described the syndrome back in 1864,⁹ Patman¹² has suggested the following useful classification: 1) causalgia (literally "burning pain" in Greek) should be used to denote syndrome occurrence secondary to a specific nerve injury; 2) mimo-causalgia (imitation of) should be used to designate the syndrome when it is due to injuries to the pe-

ripheral or central nervous system not mediated through a specific, well-defined peripheral nerve.

Both kinds of reflex sympathetic dystrophy have been described involving the upper and the lower extremities, with the majority of cases involving the hands. When the causalgia involves the lower extremities, the sciatic and tibial nerves are usually implicated.

A case of lower-extremity causalgia is presented secondary to a herniated nucleus pulposus encroaching on the L5 nerve root. The possible pathogenesis is examined in light of the currently entertained theories and the involved autonomic neuroanatomy. The clinical significance that may exist between reflex sympathetic dystrophy and a herniated nucleus pulposus is discussed and supplemented with a review of the literature.

CASE HISTORY

P.P. is a 73-year-old white female who was referred to the Pennsylvania Hospital with a 21-day history of excruciating left-leg and buttock pain. The patient had the sudden onset of left-buttock and left-leg pain while walking. Despite several weeks of bed rest, analgesics, and anti-inflammatory medication, she had persistent, severe symptoms. Position did not affect the pain pattern, and it was not associated with fever, chills, rashes, or arthralgias. The patient described severe increase in pain with any tactile stimulation of the leg and foot, a

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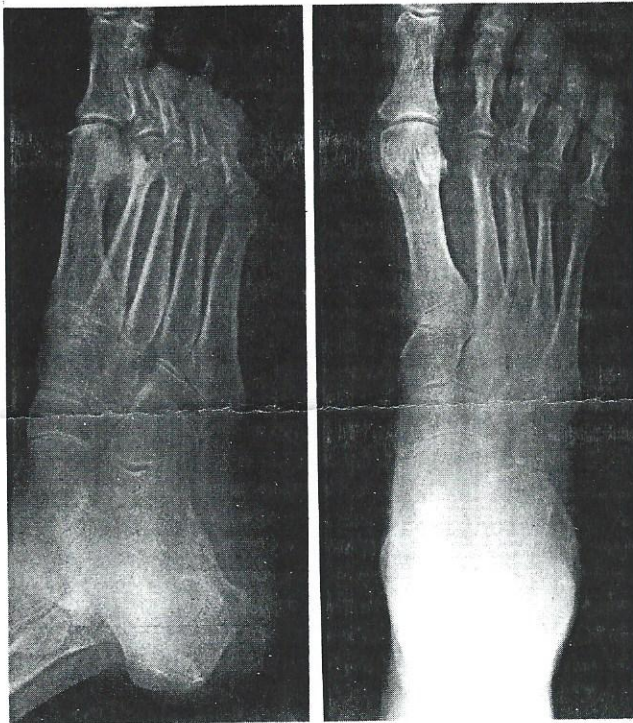


Fig 1. Diffuse osteopenia. Some midfoot soft-tissue swelling.

cold intolerance, and an inability to bear any weight on the left side.

Past medical history featured three term pregnancies, a uterine suspension procedure several years in the past, as well as a prolonged period of treatment for essential hypertension with various diuretic medications. The review of systems disclosed recent emotional depression precipitated by the unexpected death of her husband of 45 years. The patient denied smoking, alcohol consumption, and any known allergies. Physical examination displayed an oriented 73-year-old white woman in bed, obviously uncomfortable, but in no acute distress. Her gait was extremely difficult to test, due to instability and pain. It was limited only to a few steps associated with a great reluctance to bear any weight on the left side. The patient had tenderness over the lower lumbar area and marked left sciatic notch tenderness. Straight leg raising to 60° bilaterally did not increase her already severe leg pain. Reflexes were symmetrically diminished at the knee and at the ankle. There was a suggestion of mild extensor hallucis longus weakness on the left, but extreme pain on palpation of the left lower extremity prevented a reliable motor strength evaluation. There were burning dysesthetic complaints over the left leg and foot in a stocking distribution on sensory examination. The left foot and calf were pale, relatively cooler than the contralateral side, and hyperhydrotic. There was a suggestion of edema, particularly in the midfoot and around the ankle, and the ankle range of motion was significantly limited passively and actively. Rectal tone was intact, and the pulses were full and equal at the groin, the popliteal space, and about the ankle.

Routine laboratory appraisal disclosed no abnormal-

ities. Electrolytes were abnormal, with a hyponatremia of 127 attributed to antihypertensive medication, but this was corrected within 24 hours without change in the patient's complaints. Radiographs showed diffuse osteopenia involving the spine, pelvis, and left foot. Bone scan demonstrated increased uptake in the left tarsal bones (see Figures 1 and 2).

Hospital Course. Bedrest failed to diminish the significant and near constant left-leg pain. Due to the apparent inappropriate severity of the left-leg pain, the manifest vasomotor instability, the diffuse osteopenia on plain films, and the positive bone scan, a sympathetic lumbar nerve block was performed with 0.25% Marcaine. Clinically, the calf and left foot were much warmer (an increase of 4–5 degrees centigrade), and the patient's inappropriate sensitivity to palpation was, for all practical purposes relieved. The physical examination and pain pattern now appreciated were typical of an L5 radiculopathy, and a metrizamide myelogram demonstrated a left epidural defect lateral to the L5 nerve root (see Figure 3).

Since the patient had no improvement with subsequent epidural steroid injections, she underwent surgery for excision of an obviously extruded nucleus pulposus. Postoperatively, the patient had complete relief of leg pain, with minimal paresthesias remaining. Nine months following surgery, she was without complaint and/or any lower-extremity disability.

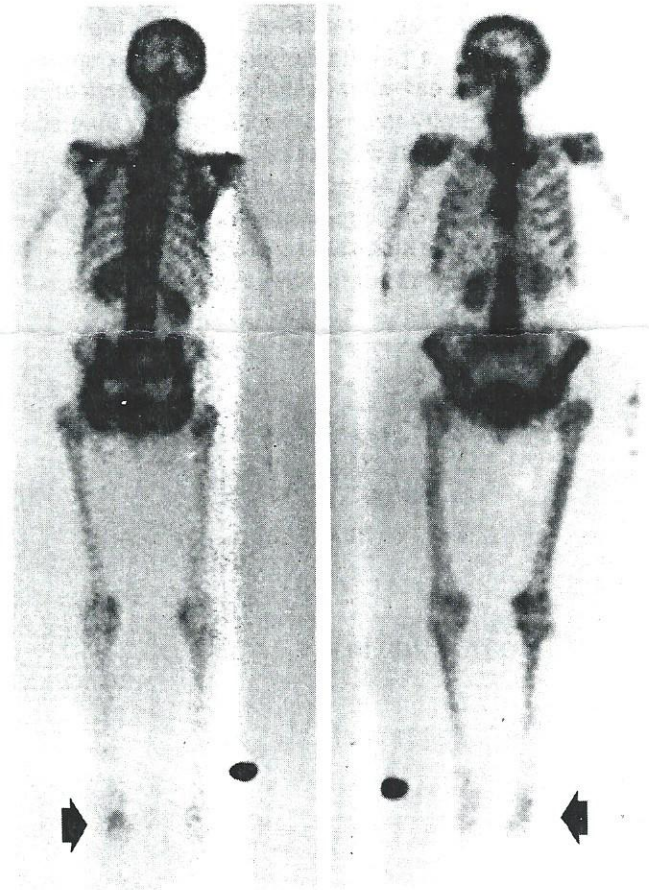


Fig 2. Technetium bone scan showing increased uptake in the mid-tarsal area of the left foot.

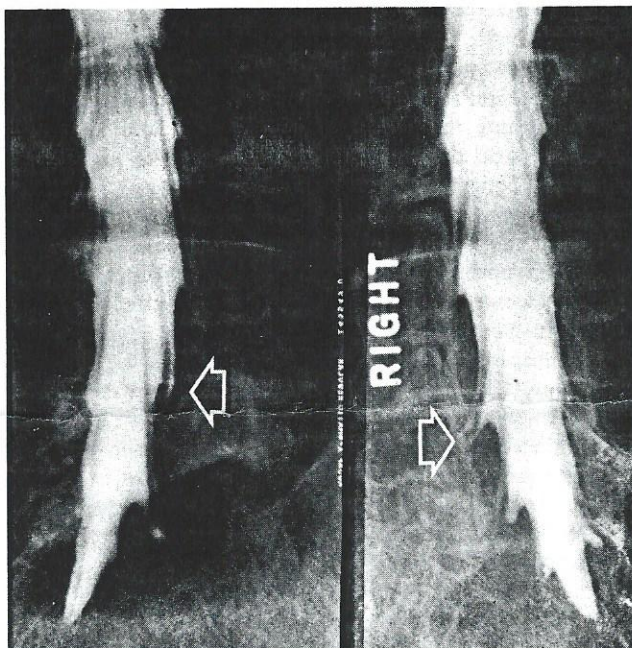


Fig 3. Metrizamide myelogram showing cutoff of the left L5 nerve root.

DISCUSSION

Several prerequisites have been suggested as necessary prior to the manifestation of a reflex sympathetic dystrophy.⁶ First, a painful lesion involving a specific mixed peripheral nerve precipitates an awareness of injury in the extremity involved. Second, rather than eliciting a normal sympathetic reflex in response to the injury, an abnormal autonomic reflex occurs. It is this latter prerequisite that represents the hallmark of the syndrome, but of which the exact mechanism through which it is realized is least understood. A third prerequi-

site postulated is what Lankford et al⁶ refer to as diathesis, or an unusually increased susceptibility to painful stimuli.

The pathogenesis of the syndrome is uncertain, but current etiologic concepts implicate both peripheral and central nervous system mechanisms. Doupe et al³ contend that abnormal or "short-circuited" synapses occur within the injured peripheral nerve between the efferent sympathetics and the afferent visceral and somatic fibers. This concept, however, relies upon the fact that peripheral nerves harbor these neural fibers in close proximity.

A more centrally mediated theory has been proposed by Livingston⁷ and modified by Melzack and Wall.⁸ Abnormal activity initiated by painful peripheral afferents occurs within the internuncial pool in the substance of the spinal cord itself, yielding persistent cortical perception of pain, as well as increased sympathetic efferent activity.

Both concepts could still function in cases of symptomatic lumbar disc herniation, even though the lumbar intravaginal dorsal and ventral roots, which would be implicated with disc herniation, differ anatomically from peripheral nerves. Indeed, neither proximal neural structure contains either preganglionic sympathetic fibers, as are found in all spinal nerves above L3,¹⁵ or a discrete peripherally directed postganglionic sympathetic fiber that would be involved in the classic causalgia produced when a mixed peripheral nerve is injured (see Figure 4).

The sinuvertebral nerves innervating the posterior longitudinal ligament and the annulus fibrosis, as well as the neurovascular contents of the epidural space, convey afferent sensory fibers through the posterior rami, as well as postganglionic sympathetic fibers¹³ (see

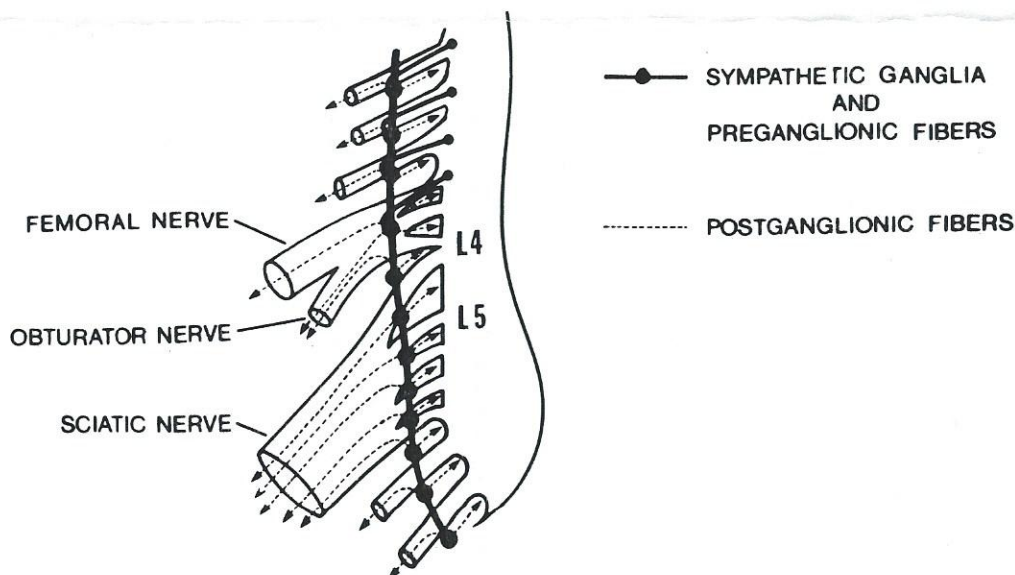


Fig 4. The cauda equina with its associated sympathetic nerves. Note that there are no preganglionic fibers below L3. All efferent fibers below L3 join the spinal nerves after they have left the neural foramina.

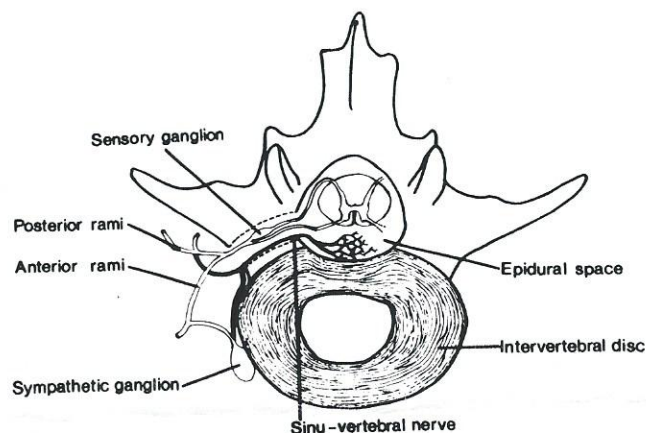


Fig 5. Sympathetic postganglionic fibers enter the neural canal along with sensory afferents from the posterior rami. This sinuvertebral nerve is in close proximity to both the annulus and the neural contents.

Figure 5). In the presence of an annular rent with disc prolapse and associated inflammatory reaction, the previously noted abnormal synapses occurring in a traumatized nerve can, therefore, theoretically occur within the confines of the spinal canal.

An injury to the intravaginal part of the dorsal and ventral roots or even to the true low lumbar spinal nerves just distal to the sensory ganglion would not result in direct sympathetic dysfunction since there are no preganglionic sympathetic fibers below L3. In this case, an increased cortical perception of pain would have to be implicated as precipitating the abnormal reflex autonomic activity. Melzack and Wall have suggested that this centrally mediated pathway could be implemented with inappropriately increased afferent activity in the internuncial pool in the spinal cord. Inappropriate sensation would then be conveyed to the brain through the ascending fibers of the substantia gelatinosa in the dorsal horns of the spinal cord and discharge an abnormal autonomic reflex. Since multiple sympathetic ganglia would be traversed in this sympathetic reflex arc, one should not be surprised to find a stocking-like distribution of pain and dysesthesia. This would result in a pain pattern not confined to the dermatome area supplied by the involved spinal nerve.¹²

The final prerequisite necessary to precipitate the syndrome, described as an increased susceptibility to the perception of pain, would also work centrally and prevent cognizant modification of the involved abnormal autonomic reflex. It is of interest that while our patient had no overall personality disorder prior to her symptom development, the recent death of her husband of many years may have been an aggravating factor. Indeed, Pak et al¹¹ reviewed 140 cases of reflex sympathetic dystrophy and found that approximately 37% of patients had apparent histories of psychiatric problems or emotional disturbances prior to the onset of the presenting complaint.

The relationship between herniated nucleus pulposus and the development of reflex sympathetic dystrophy has been rarely reported. Cayla and Rondier, in 1974,² reported on three cases, while Carlson et al, in 1977,¹ described two others. The syndrome has been reported following iatrogenic injury to lumbar spinal contents during diagnostic myelography¹⁰ and during chymopain therapy.¹⁶ The proposed mechanisms in both circumstances have been mechanical trauma from the spinal needle used and chemical injury due to the oil-based agent or the chymopain.

Our one case and the five cases in the literature do not allow for a description of a definitive syndrome for reflex sympathetic dystrophy secondary to disc herniations. Several characteristic features, however, derived from 23 cases of reflex sympathetic dystrophy secondary to various pelvic and lumbar spine lesions,² may be of some help, particularly since several were mirrored in our case history. First, the syndrome tends to occur more commonly in females and will be unilateral in presentation, particularly with ipsilateral lumbar or pelvic pathology. Causalgic pain is the presenting symptom and is associated with joint stiffness chiefly involving the ankle. The abnormal autonomic reflex activity, however, is not as flagrant as the upper-extremity cases of reflex sympathetic dystrophy.

A final characteristic of diagnostic importance is the frequent occurrence of osteopenic changes in the involved extremity. This abnormal reflex atrophy of bone, commonly referred to as Sudeck's atrophy,¹⁴ is most likely a manifestation of both local agents (hyperemia and decreased load demand due to the painful reflex dystrophy), as well as of systemic agents in the form of humeral factors affecting calcium and phosphorus metabolism.⁴

Fine-detail radiography has been used to document juxta articular osseous changes occurring in cases of reflex sympathetic dystrophy.^{4,5} It has been emphasized that various patterns of cortical erosion involving endosteal, periosteal, and intracortical bone, as well as trabecular disruption, can occur to a significant degree as early as six weeks after the onset of the sympathetic dystrophy, despite continual loading and the use of the extremity.⁵ Our patient's diffuse osteopenia lacked significant cortical erosion or trabecular collapse, since her symptoms were only of three weeks' duration. What was supportive of Sudeck's atrophy, however, was a positive technetium bone scan showing definite, increased uptake, particularly in the tarsal bones of the involved leg. Positive scans have been reported in involved extremities in 80-100% of patients with reflex sympathetic dystrophy. Furthermore, this scan may reflect bilaterality of the phenomenon before either plain roentgenographic films or patient symptoms would suggest the presence of a reflex sympathetic dystrophy.^{4,5} A similar finding was reported in Carlson's¹ cases, but an

explanation was uncertain, although both increased blood supply and actual increase in osteoblastic activity were suggested.

The importance of recognizing the syndrome, manifest clinically and supported roentgenographically, is obvious. The differential diagnosis involved in the case of leg pain, particularly when inappropriate, must include infections, neoplasms, metabolic and vascular disease, in addition to injury involving the extremity or lumbosacral spine. The coincidental presentation of sciatica with reflex sympathetic dystrophy may be confusing and delay diagnosis and subsequent therapy. While treatment should be aimed at the causative pathology, that is, the herniated nucleus pulposus, the use of sympathetic blocks for diagnostic purposes should not be overlooked and, of course, should be utilized therapeutically if symptoms persist despite appropriate lumbar surgery.

SUMMARY

Reflex sympathetic dystrophy is a well-recognized syndrome that may result from symptomatic low lumbar disc herniations. The current concepts concerning pathogenesis are discussed with their anatomic pathways. The features that may characterize the syndrome when it is caused by a disc herniation are discussed in light of our experience and of experiences reported in the literature. Clinical suspicion may be supported by fine-detailed radiography, scintigraphy, and diagnostic lumbar sympathetic blocks. The latter modality may also be helpful therapeutically if symptoms persist despite appropriate lumbar surgery.

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