

Diffuse Reflex Sympathetic Dystrophy

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Reflex sympathetic dystrophy represents a group of disorders that include causalgia, algodystrophy, Sudeck's atrophy, and shoulder-hand syndrome. Early symptoms include pain, hyperesthesia, swelling, vasomotor instability, and limited range of motion, which may progress to trophic skin changes and osteoporosis.¹ Optimal treatment includes early mobilization and sympathetic blockade with a local anesthetic.^{1,2} The pathophysiology of reflex sympathetic dystrophy is thought to include excessive autonomic responses to various factors, including trauma (often seemingly trivial), arthritis, disc disease, myocardial infarction, herpes zoster, vasculitis, bone or joint infection, and tumor.³ Spread of symptoms to the opposite extremities has implicated central nervous system mediation of reflex sympathetic dystrophy.^{4,5} We describe a case in which acute exacerbation of chronic reflex sympathetic dystrophy resulted in a unique case of total-body involvement.

REPORT OF A CASE

A 43-year-old woman was referred to our Pain Clinic in 1975. Her past history included two lumbar laminectomies and spinal fusion. She complained of lumbosacral pain and hyperesthesia, hyperhidrosis, and pain of both legs. Muscle wasting and trophic changes of skin, hair, and nails of her lower extremities were evident. She had become immobilized, depressed, and was taking oxycodone and pentobarbital. A diagnosis of reflex sympathetic dystrophy was made and treatment initiated. Bupivacaine (0.5 per cent) injections into lumbar, buttock, and leg trigger points were only partially successful in alleviating the symptoms. Lumbar sympathetic block with 30 ml 0.5 per cent bupivacaine relieved pain, hyperhidrosis, and hyperesthesia in the legs and lumbar regions for two to three days. Bilateral pairs of lumbar sympathetic blocks (one-hour interval between sides) were repeated three times at three-day intervals. Physical therapy, biofeedback, and administration of doxepin, a tricyclic antidepressant, were also prescribed. The patient's condition improved markedly, and she was discharged and subsequently returned to work. She returned as neces-

sary for treatment, including lumbar sympathetic blocks, at average intervals of six weeks.

Three years later the patient experienced the acute onset of diffuse immobilizing arthralgias, edema, cold clammy extremities, and hyperesthesia of the entire body. A tentative diagnosis of systemic lupus erythematosus was subsequently ruled out by normal sedimentation rate, lupus erythematosus cell preparation, complement 3 and 4, antinuclear antibody screen, and rheumatoid factor.

Reflex sympathetic dystrophy was suspected, and a series of lumbar sympathetic blocks and stellate ganglion blocks was done, along with injections of paravertebral trigger points. In an attempt to provide continuous pain relief, these were repeated at two- or three-day intervals for several weeks. During pain-free periods following the blocks the patient received physical therapy for extremity mobilization. The patient's condition improved, and treatment frequency was tapered. She is now active, takes no medication, and needs only an occasional biofeedback session or sympathetic block.

DISCUSSION

The pathophysiology of reflex sympathetic dystrophy is thought to be initiated by damage to peripheral autonomic afferents or somatic sensory nerves, which for unknown reasons results in reflex aberration of trophic function in autonomic efferents and other nerves.¹ Neuronal trophic function maintains morphologic integrity of innervated tissue, apparently via axoplasmic transport of essential regulators and nutrient substances.⁶ Although short-circuiting of normal nerve impulses at the peripheral injury site has been suggested,⁷ mediation of trophic reflex and spread of symptoms implicate central nervous system mechanisms in the pathophysiology of reflex sympathetic dystrophy.⁴ A likely site is the spinal cord dorsal horn, where self-sustaining neuronal loops have been postulated to mediate symptoms of reflex sympathetic dystrophy.^{8,9} Normally, descending fibers modulate pain, evoking dorsal horn activity by enkephalinergic inhibition.¹⁰ Peripheral and central factors that enlist this inhibition are unclear, and may be impaired in the pathophysiology and spread of reflex sympathetic dystrophy.

Early recognition and aggressive treatment of reflex sympathetic dystrophy favor an optimal clinical outcome, since spontaneous remission is rare.^{1,11} Treatments have included sympathetic blockade, analgesics, physical therapy, beta-adrenergic blockade, corticosteroids, calcitonin, guanethidine, and surgical sympathectomy.¹² In our experience, minor forms of reflex sympathetic dystrophy, including myofascial

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syndrome, may be treated successfully with physical therapy and trigger point injections. If symptoms persist, more aggressive treatment, such as serial sympathetic blockade, is indicated to avoid a chronic, irreversible pain syndrome.

This case demonstrates that relapse may occur and that symptoms of reflex sympathetic dystrophy may spread diffusely. Spread of reflex sympathetic dystrophy to a bilateral or ipsilateral extremity is common, but our patient represents an apparently unique case of total-body involvement.

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The Effective Use of Epidural Morphine Sulfate for Postoperative Orthopedic Pain

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Postoperative pain following arthrotomy of the knee is usually intense. Patient cooperation with physical therapy after this procedure is usually less than optimal when standard methods of producing analgesia, such as parenteral administration of narcotics, are used. We report the successful use of morphine, given epidurally, for analgesia following a knee arthrotomy. The medical management of this case took place in the Jefferson Tower of the University Hospitals in Birmingham, Alabama.

REPORT OF A CASE

A 29-year-old Caucasian woman was admitted for an arthrotomy and repair of a traumatic disruption of the right knee. Medical, surgical, and previous anesthetic histories prior to this injury were unremarkable. Her only medication was norethindrone acetate,

for treatment of pelvic endometriosis. Premedication one hour before the surgical procedure consisted of diazepam, 10 mg, orally, and 30 ml of an antacid containing aluminum hydroxide, magnesium hydroxide, and simethicone. The patient had not received narcotics for more than 27 hours prior to operation.

The surgical procedure was conducted using continuous lumbar epidural analgesia with .75 per cent bupivacaine with 1:200,000 epinephrine, 20 ml. The sensory level of anesthesia was T9 bilaterally. During the first 24 hours postoperatively pain relief was achieved with 0.25 per cent bupivacaine with 1:200,000 epinephrine, 5 ml given epidurally every two hours to maintain adequate analgesia. Narcotics were not needed during this period.

Twenty-four hours after the operation the analgesia induced by bupivacaine was allowed to dissipate, and intense knee pain occurred. Morphine sulfate crystals, 10 mg, were dissolved in 10 ml of 0.9 per cent NaCl and passed through a Millipore® filter as specified by the hospital pharmacy.* Five milliliters of this preparation (morphine sulfate, 5 mg) were then administered through the epidural catheter following a negative aspiration for blood. Pain gradually decreased over the first hour, but was not completely relieved by two hours. A second dose of morphine sulfate, 4 mg (due to the loss of 1 ml, or 1 mg, in the Millipore filter), was then administered epidurally, after which the catheter was removed. Analgesia was complete an hour after the second dose,

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* The use of epidural morphine has been approved by the Human Use Committee of the University of Alabama in Birmingham. Informed consent was obtained from the patient.