Recurrent sciatica associated with herpes simplex

Case report

HAROLD H. MORRIS III, M.D., AND BRUCE H. PETERS, M.D.
University of Texas Medical Branch, Galveston, Texas

A case of recurrent sciatic pain associated with herpes simplex infection is presented and the characteristic differences from usual forms of sciatica discussed. Special features of this entity are recurrent episodes of neuralgic pain followed by zosteriform skin eruptions. Both rash and pain disappear within a few days. This entity should be considered when evaluating patients with recurrent sciatica.

Key Words • herpes simplex • sciatica • sacral plexus

The following case report emphasizes the possibility of herpes simplex as an unusual cause of sciatica.

Case Report

This 74-year-old woman suffered repeated episodes of severe dull pain in the right buttock and leg for 6 years, which also occasionally occurred on the left. The pain was worse after lying down or prolonged sitting; it was not exacerbated by sneezing or defecation, and was partially relieved by walking. There was no weakness or urinary abnormality. The pain reached its maximum a few hours after onset and was followed in 2 to 4 days by a rash, characterized by small vesicles, over the right or left buttock. Infrequently, paresthesias of the lateral aspect of the right foot accompanied the pain. The pain and rash always subsided after several days only to recur at approximately monthly intervals. She had no skin problems elsewhere on her body, and, except for carcinoma of the breast cured by mastectomy 11 years before, had enjoyed good general health.

Examination showed a healed left mastectomy scar with no evidence of recurrent carcinoma. Strength and muscle stretch reflexes were normal, and there were no pathological reflexes; hyperesthesia was noted over the first sacral dermatomes bilaterally. There were bilateral patches of small vesicles, on an erythematous base, adjacent to the first sacral vertebra and confined to its dermatome. The straight-leg-raising test was negative at 90° bilaterally. The impression of the consulting dermatologist was recurrent zosteriform herpes simplex. The following tests were normal: urinalysis, complete blood count, electrolytes, venereal disease (VDRL), blood urea nitrogen (BUN), serum glutamic oxaloacetic transaminase (SGOT), alkaline phos-
phatase, calcium, uric acid, and fasting blood sugar. Roentgenograms of the lumbo-
sacral spine showed no abnormalities.

Discussion

Our case is typical of the clinical picture produced by recurrent herpes simplex. Because of the history of recurrent back pain with dermatomal distribution, this entity could easily be confused with the herniated disc syndrome or other causes of lumbosacral radiculopathy. There are important distinguishing features. The patient had no history of back injury. She had suffered recurrent pain for years, but, except for minor sensory impairment, had no neurological deficit. She was aware that 2 to 4 days after each recurrent painful episode small "blisters" appeared on her buttocks. It must be emphasized that the skin lesions were not, of themselves, painful or troublesome, and were not mentioned as a part of her complaint until the physical examination prompted appropriate questions. The vesicles crusted, and the pain resolved over several days. Neither bed rest nor physical therapy diminished the length or severity of painful episodes. In fact, the pain seemed worse at night, and walking about provided some relief.

In 1950, Slavin and Ferguson showed that recurrent zosteriform skin eruptions, which our patient manifested, are due to herpes simplex virus. Since that report, other authors have emphasized this finding and that, since skin lesions produced by the varicella-zoster virus and herpes simplex may appear identical, one must rely on a history of recurrent eruptions, or viral cultures, to distinguish them. In our patient an unsuccessful attempt was made to culture the vesicle fluid. However, the lesions had crusted and were resolving when the culture was obtained.

Herpes simplex most frequently produces human disease of the oropharynx and genitalia, innervated by the trigeminal nerve and sacral plexus respectively. The most common sites for recurrent herpes simplex are in the distribution of the trigeminal nerve and in the sacral dermatomes. In animal experiments the virus has been shown to travel centrally from the site of inoculations via the peripheral nerve. Cushing noted the appearance of herpes following operations on the trigeminal ganglion. Carlton and Kilbourne reported that 16 of 17 patients developed herpes simplex after trigeminal sensory root section. Herpes simplex has been found present in the human trigeminal ganglion at routine autopsy. Although unproven in humans, it would be attractive to propose that the organism, after initial infection, could travel to the dorsal root ganglion where it would lie dormant. Periodic failure of those factors preventing viral replication could then cause the virus to reduplicate, producing recurrent symptoms.

Underlying debilitating disease, so commonly associated with herpes zoster eruptions, has seldom been reported in association with recurrent herpes simplex. Stroud reported one case of recurrent herpes simplex in a 68-year-old man under chronic steroid treatment for amyloidosis and the nephrotic syndrome. Most cases have occurred in otherwise healthy people.

There is no effective and safe therapy for recurrent herpes simplex. The pain may be severe, and symptomatic treatment is indicated. Because of the short duration of each episode, there should be little danger of addiction to narcotic analgesics prescribed only for the few days of intense discomfort.

Recurrent herpes simplex can result in perplexing sciatica. In cases with recurrent symptoms, careful questioning and inspection of the skin may lead to the precise clinical diagnosis. Unrewarding contrast studies are then avoided, and the patient can be reassured that the entity, although recurrent, is nonprogressive.

References


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Address reprint requests to: Harold H. Morris III, M.D., University of Texas Medical Branch, Galveston, Texas 77550.