and inconstant sensory deficits are often found. Water-soluble caudography and lumbar epidural phlebography may be negative or inconclusive. When lumbar spondylosis is seen on plain radiographs, there is a tendency to attribute the ischialgia to spondylotic lumbosacral radioculopathy. The present case illustrates that when ischialgia persists or becomes worse despite conservative therapy, the diagnosis may have to be reconsidered. Particularly when the pain involves more than one dermatome, regular reexaminations have to be performed to exclude rarer causes of ischialgia such as endometriosis,[4] retroperitoneal fibrosis,[5] retroperitoneal or intrapelvic tumors,[6] inflammatory processes,[7] or the "piriformis syndrome."[8]

A subgluteal lipoma extending to the pelvis through the ischiadic foramen—compressing the ischiadic nerve—must be rare, since there have been no other reports. Introduction of the CT body scanner may reveal analogous cases of lipomas or other benign tumors causing entrapment neuropathy of the ischiadic nerve. The prompt postoperative relief of pain in our case should stimulate further investigation of similar cases.

Acknowledgment

We wish to thank Dr. Gareth Davies for his advice.

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Accepted for publication September 4, 1979.

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Horner syndrome with causalgia

Article abstract—A previously healthy man presented with burning pain in the chest wall and arm; there was Horner syndrome on the same side. After extensive investigation, the disorder was attributed to a foraminal osteophyte involving the left T1 spinal root.

NEUROLOGY 30: 534-535, **May 1980**

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Case report. The association of Horner syndrome and causalgia of nonneoplastic etiology involving one arm is distinctly unusual. This 49-year-old man presented with pain in the left anterior chest wall. Onset followed a respiratory infection. The pain was not related to activity, coughing, or position. It appeared to arise from "deep within the arm." After 1 week it was localized in the left subscapular region and left elbow, and then radiated distally to involve the entire arm to the wrist. Two weeks after onset, the patient noted weakness in the left hand with burning paresthesias along the inner surface of the forearm, which became unbearable with minimal contact from clothing.

Three weeks after onset, a complete left Horner syndrome was noted, with mild ptosis, miosis, and decreased sweating on the left. When he raised both arms over the head or moved his head, the paresthesias worsened. Strength could not be evaluated because of pain. Reflexes showed mild decrease in the left biceps jerk on the left. Perception of touch and pain was impaired along the inner aspect of the left arm, with hyperpathia upon rubbing next to the same area. The remainder of the neurologic and general physical examination was normal.

Laboratory studies were normal with the exception of a cerebrospinal fluid (CSF) protein of 71 mg per deciliter, and routine cervical spine films revealed narrowing of the intervertebral disk spaces from C6 to T2. Flexion views showed good physiologic motion of the cervical spine. Polytomography studies of cervical and upper thoracic spine showed degenerative changes on the left; specifically, a large osteophyte was seen encroaching upon the neural foramen between T1 and T2 (figure). ^{99m}technetium bone scan revealed increased activity consistent with degenerative changes in the cervical spine; no evidence of metastasis was noted. A cervical myelogram showed an extradural root sleeve defect at T1 to T2, corresponding to the foraminal osteophyte seen on polytomography. Electromyography gave evidence of active denervation in muscles innervated by nerve roots

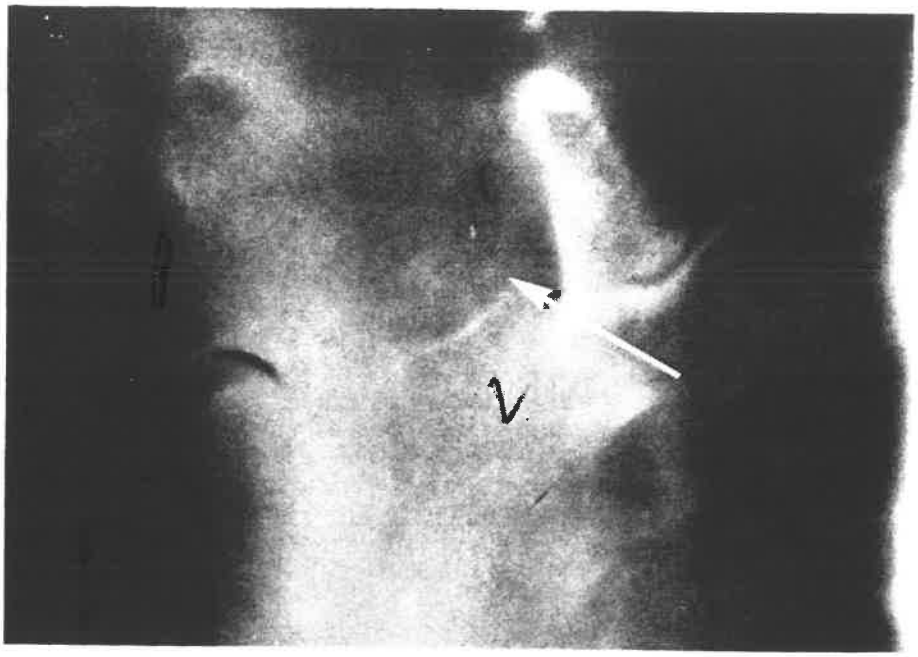

Figure. Polytomography revealing a large osteophyte encroaching upon the neural foramen on the left between T1 and T2.

C8 and T1. Motor and sensory nerve conduction studies were normal. There was no pupillary dilation after 4% cocaine hydrochloride administration on the left, but normal dilation on the right was noted. One percent hydroxyamphetamine application resulted in dilation of the pupils.[1,2] Pharmacologic testing of the pupils was consistent with Horner syndrome and localized to a preganglionic lesion.

The patient was treated with analgesics, bed rest, and hot packs to the neck. He gradually improved and 6 months later had no pain, but hyperalgesia remained along the medial aspect of the forearm, with decreased pain perception in the fourth and fifth fingers. Occasionally, when manipulating a small object such as a screw, he had cramps in the small muscles of the left hand. The Horner syndrome persisted with no other changes for 3 years.

Discussion. Cervical spine disease has been infrequently described with Horner syndrome. It has been reported with spinal concussion resulting from vertebral dislocation in two cases,[3] as a transient complication after an anterior C3-C6 fusion,[4] and after severe cervical trauma.[5,6] In our patient, the upper thoracic osteophyte probably interrupted the sympathetic innervation to the eye by affecting the peripheral connections that leave the cord with T1 and T2 roots.

Causalgia is most commonly seen after partial injury to a major nerve trunk; is defined as severe persistent pain, usually with a burning quality felt in the distribution of the involved nerve(s); and may be accentuated by various environmental and emotional stimuli.[7-9] The pathogenesis is still uncertain, but it has been proposed that causalgia results from an interaction between efferent sympathetic and afferent sensory fibers,[7] and sympathectomy is often found to be an effective treatment.[8]

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Accepted for publication August 24, 1979.

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