Painless Intraspinal Leptomeningeal Carcinomatosis: A Myelographic Demonstration

Case Report

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Carcinomatosis of the leptomeninges in the absence of any associated gross focal metastasis within the central nervous system has been known since Siefert's description in 1902. An excellent review by Jacobs and Richland in 1951 concluded that dissemination was via the hematogenous route and discarded the theory of spreading through the "perineural lymph spaces." Intraspinal leptomeningeal carcinomatosis certainly is not uncommon, and cases of lymphosarcoma, melanoma, sarcoma, and Hodgkin's disease have been reported. Preoperative diagnosis has been difficult, however, especially in the early stage of dissemination.

We are reporting a case of intraspinal leptomeningeal carcinomatosis in which diagnosis was made by an early change in the Pantopaque myelogram and the spinal fluid.

Case Report

A 50-year-old woman was seen at the Mayo Clinic on August 16, 1968, for numbness and weakness of the legs of 3 months' duration. The numbness was noted over the posterolateral aspect of the legs and soles of her feet, more on the left side than on the right. She recalled having a mild dull ache over the lower part of the back in January, 1968.

At another hospital in June, 1968, examination had revealed weakness of the left hamstring and extensor hallucis longus muscles and the dorsiflexors of the left foot. The Achilles reflex was absent bilaterally. A lumbar Pantopaque myelogram made on August 2 was reported to show no evidence of abnormality (Fig. 1 left). Soon after this first myelogram constipation developed, and the patient became dependent on the use of laxatives for bowel movements. Weakness of the legs progressed rapidly.

In 1966 the right breast had been removed because of an infiltrating grade 4 adenocarcinoma with axillary nodal involvement. Subsequently the patient received a total dose of 2400 R deep therapy to the supraclavicular field and 3000 R to the anterior chest field. The patient had done well until the onset of neurological symptoms.

Examination. When first seen at this clinic on August 16, 1968, the patient was in a wheelchair. Bilateral flaccid paraparesis involving all the muscles of both legs was noted. Both ankle and knee jerks were absent bilaterally. The anal sphincter was patent. Sensory impairment over the lower extremities was significant, and saddle-shaped sensory loss was demonstrated extending bilaterally over the perineum and buttocks. Babinski's sign was absent. Straight-leg raising was limited to 70° on both sides. Plain x-ray films of the lumbar spine again did not show evidence of a bony lesion. An electromyogram revealed fibrillation and fasciculation with abnormal motor-unit potentials of a neurogenic type in the muscles of the distribution of the fifth lumbar and first sacral roots bilaterally. No fibrillation was obtainable from paraspinal muscles. Conduction velocity of the left peroneal nerve was 45 m/sec.

On August 19, Pantopaque myelography was performed for the second time; unusual, irregular granular filling defects limited only to the terminal part of the cul-de-sac (Fig. 1 right) were evident, more definite on the left side than on the right. The remainder of the subarachnoid space and the spinal cord was normal from the vertebral arteries to the cul-de-sac. The cerebrospinal fluid contained 1 lymphocyte, 8 polymorphonuclear leukocytes, and 13 red blood cells/cu mm with
468 mg of protein/100 ml and 47 mg of sugar/100 ml. The sedimentation rate of erythrocytes was 12 mm in the first hour (Westergren method); alkaline phosphatase measured 48 units/liter, and serum glutamic oxaloacetic transaminase (SGOT) 14 units/liter.

Operation. Immediately after myelography, exploratory lumbar laminectomy revealed that the first sacral root on the right was enlarged and hyperirritable, unusually firm, and compressed extradurally by a protruded lumbosacral disc. The protruded disc was removed. The dura was then opened, and opalescence of the arachnoid was noted immediately. After examination of a fresh frozen specimen of the arachnoid, the pathologist reported metastatic grade 4 adenocarcinoma. On close inspection the nerve roots in the cauda equina were found to be firm, and most roots were covered by fine, widely spread nodules less than 1 mm in size with considerable impairment of the vascular supply. A small piece of tissue from the left second sacral nerve root was removed for pathological examination. The nerve fibers and their myelin sheaths were greatly swollen, distorted, and degenerated by an extensive cancerous infiltration along the perineural and perivascular spaces (Fig. 2).

The wound was closed after decompressive laminectomy. At the time of the patient's discharge after 2500 R of deep therapy to the lumbar region, no appreciable change in neurological findings was demonstrable.

Comment

The shape and position of the terminal spinal cul-de-sac have been studied in normal and diseased subjects by Paleirac, et al.8 The normal cul-de-sac may be slit-like, or wide, funnel-shaped, blunted, or diverticular. It may be as high as the lumbosacral interspace or as low as the fourth sacral vertebra. No matter what kind of variation there may be, however, the normal arachnoid lining always is smooth. Extradural compression of the cul-de-sac also presents a smooth cut-off of the subarachnoid space.9 Therefore, irregu-