Chronic intramedullary abscess of the spinal cord

Case report

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The problems of diagnosis and treatment of a chronic intramedullary abscess of the spinal cord are illustrated by the following case, and by a review of previous case reports.

Case Report

This 15-year-old high school girl was admitted to this department on May 28, 1968, because of intermittent dorso-lumbar pain for 3 years and weakness and sensory loss in the right leg for 1 month. Lower abdominal radiation of the pain had led to appendectomy in 1965.

Examination. General physical examination was normal except for chronic tonsillitis. Temperature was 36.8°C, pulse rate 100, and blood pressure 120/80 mm Hg. Neurological examination showed weakness of the right leg, more evident distally. There was moderate bilateral spasticity of leg adductors and extensors, sustained ankle clonus and transient patellar clonus bilaterally. Knee and ankle jerks were hyperactive bilaterally. The upper and middle abdominal reflexes were normal; the lower ones were absent bilaterally. The Babinski sign was constantly present on the right, equivocal on the left. Heel-to-knee test was performed clumsily with the right leg due to motor weakness. Gait was bilaterally paretic and spastic, especially on the right. There was a moderate disturbance of light touch and pinprick sensation below T-10, but toe position and vibratory sense were normal. Bladder and rectal functions were also normal.

Laboratory Data. Examination of peripheral blood showed a hematocrit of 45%, and 5000 white blood cells per cu mm, with a normal differential cell count. Other routine laboratory studies, including urinalysis, blood urea nitrogen, fasting blood sugar, serum proteins, and sedimentation rate, were normal. Three blood tests for syphilis were negative. The antistreptolysin titer was 400 units per cu mm. The EEG, and skull and chest films were all normal. Spine films showed left L-5 sacralization but were otherwise normal. Electromyogram of the paravertebral muscles at the level of T-5 to L-1 were normal bilaterally. Lumbar puncture revealed a clear, colorless fluid containing 5 white cells per cu mm. The protein content was 35 mg/100 cc, and Pandy test, ++. Opening spinal fluid pressure was 120 mm of water; during straining and bilateral jugular compression there was a rapid rise (within 2 to 3 sec) to 200 and 350 mm respectively, followed by a rapid fall. After removal of 6 cc of fluid, the CSF pressure dropped to 20 mm of water; there was now a rapid rise to 150 mm during straining but during bilateral jugular compression there was a delayed and irregular rise (within 5 to
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10 sec) to 200 mm, followed by slow fall. The Queckenstedt test was therefore considered positive (see discussion). Spine scan with intravenous $^{99m}$Tc was normal. Contrast myelography with 6 cc of ethyl monoidostearate (Durolopaque Guerbet) showed a space-occupying intradural, intramedullary lesion at the level of T-7 (Fig. 1).

Operation (Dr. E. Tartarini). On June 6, 1968, under general anesthesia, a total laminectomy from T-7 to T-9 with partial removal of the T-10 lamina was performed. Epidural tissues, as well as the dura, were normal. The cord was enlarged and felt firm over the left side due to an intramedullary ovoid yellowish mass, visible under the pia at the T7-8 vertebral level. Pial vessels over the mass were distended and tortuous. The mass was crossed by the T-7 posterior root, which was cut. After coagulation of pial vessels and cutting of the pia, the mass bulged from surrounding tissue; incision of the mass yielded about 1 cc of a thick, green-yellowish purulent fluid. The mass was then separated from surrounding tissue, and completely removed by following planes of cleavage along a yellowish demarcating zone of reactive gliosis, bridged by small vessels penetrating the periphery of the mass (Fig. 2). The pus was not cultured. Histologic examination (Dr. A. Giampalmo) showed a capsule of a chronic encysted abscess, made up of a dense outer layer of collagenous tissue heavily infiltrated by inflammatory cells surrounding an inner layer of granulation tissue. There were no multinuclear giant cells (Fig. 3).

Postoperative Course. Recovery was satisfactory. The patient was given 500,000 units of penicillin, $\frac{1}{2}$ gr of streptomycin, and 12 mgr of prednisolone twice a day for 2 weeks. She had transient reflex voiding for 12 hours and bilateral loss of toe position sense and vibratory sense over the tibiae and toes. When discharged on July 7, 1968, the strength of the legs was nearly normal. The right Babinski sign was still present on the right and normal on the left. The gait was improved; cutaneous sense and proprioception below T-10 were slightly diminished. At a visit 8 months after surgery the patient had no pain. Gait and proprioception over the left leg were further improved. The left lower abdominal reflex had reappeared. Spinal fluid cytochemistries were normal; a manometry was normal both before and after fluid removal.

Discussion

The Queckenstedt test was interpreted as positive in that it pointed to a partial subarachnoid block. This was suggested by the

Fig. 1. Myelography, anteroposterior view with the head up shows a partial block at the level of T17, with widening of the medullary shadow characteristic of an intramedullary mass.

Fig. 2. Photograph of the entire mass removed from the cord. Scale in millimeters.
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Adson, Arzt, and Lepoire, et al., who found 46 published observations and added two of their own. Single cases have been subsequently reported, for a total of 51. Twelve of 19 patients survived the operation; prognosis has been definitely improved by antibacterial drugs, since 8 patients survived out of the 9 operated after 1948. The abscesses have been frequently found at the thoracic level and in the posterior portion of the cord. They usually extend longitudinally for two to three segments or more. Multiple cavities are rare. Infections enter the spinal tissue either directly, following penetrating wounds, as in the recent observation of Wright (traumatic abscess), or by contiguity, as in vertebral osteomyelitis, or through the blood stream (metastatic abscess). The source of mycotic embolus often remains unknown, but pulmonary suppurations, bacterial endocarditis, and genitourinary infections have been implicated. In these latter disease processes, retrograde thrombosis of spinal veins which are connected with pelvic and abdominal venous plexuses has been postulated. There was no x-ray or other evidence of tuberculosis in our case.

Acute cases evolve in a few days as a transverse myelitis; subacute and chronic cases mimic an intramedullary tumor of the posterior cord. General signs of infection can be totally lacking in chronic cases. Lumbar puncture shows cytochemical and manometric signs of spinal canal occlusion and, usually in acute cases, a secondary or associated leptomeningitis.

Cord abscesses evolve therefore as a focal lesion, sharing etiology and pathology with brain suppuration. The reduced volume of spinal cord tissue probably accounts for the relative rarity of spinal (compared to brain) abscess. Our surgery and autopsy records since 1936 list 32 cases of brain abscess and only this present case of spinal abscess. This frequency ratio is close to that of the brain/cord volume ratio, which is 44 to 1.5

The origin in our case was presumably metastatic, but the primary source remains unknown, although appendicitis and chronic tonsillitis must be suspected.

Summary
We have reported the case of a patient...
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who suddenly developed spastic paraparesis with sensory deficits after 2½ years of intermittent back pain. Surgery disclosed an encysted intramedullary abscess, which was excised. Postoperatively there was partial improvement in the neurological deficits. We have emphasized the value of a modified Queckenstedt test in cases of this sort.

References

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