CIRCUMSCRIBED ADHESIVE SPINAL ARACHNOIDITIS

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Adhesive arachnoiditis is an uncommon cause of progressive dysfunction of the spinal cord. The lesion rarely is diagnosed pre-operatively and its surgical management is dependent on the longitudinal extent of the lesion in the spinal subarachnoid space. Circumscribed adhesive arachnoiditis would appear to be encountered less frequently than the diffuse disease and is more amenable to surgical attack. The gross pathologic findings vary from milky-white discoloration and thickening of the arachnoid with constricting bands of arachnoid across the spinal cord to dense subdural masses obliterating the subarachnoid space. Microscopic characteristics include a paucity of blood vessels, dense connective tissue and leukocytic infiltration. In cases in which the entire spinal subarachnoid space is involved there may be intramedullary cavitation of the cord. Where constricting bands are present, arachnoid cysts may develop which behave as space-occupying lesions.

The etiology of the disease is protean. Infectious processes, trauma, radio-opaque contrast media, spinal anesthetic agents, toxins and blood have been implicated. Oftentimes, no etiology is apparent. There is no clear-cut way to differentiate clinically between circumscribed adhesive arachnoiditis and spinal-cord tumor. The diffuse form of the disease, however, frequently can be suggested by myelography. Therapy is directed to removal of a space-occupying lesion in cases of circumscribed arachnoiditis. In the diffuse form however, surgical therapy appears to offer little hope of neurological salvage.

The case presented herewith is of interest for several reasons. The author had the opportunity of evaluating the patient 4 months prior to the onset of spinal-cord dysfunction. She had a documented spontaneous subarachnoid hemorrhage at that time. A spinal anesthetic had been administered 11½ years prior to the onset of the cord symptoms. Which, if either, of these incidents had a role in the pathogenesis of her disease is speculative. The surgical endeavor outlined below was followed by spinal-cord edema which responded to intravenous urea. The latter adjunct was suggested by Poppen."

CASE REPORT

VBIH 101394. J.H., a 69-year-old white female, was first seen on May 4, 1960 because of sudden onset of headache, nuchal rigidity, nausea and vomiting. Lumbar puncture was performed and revealed grossly bloody cerebrospinal fluid which had a xanthochromic supernate after centrifugation. Neurological examination revealed only bilateral Babinski's toe sign. The patient recovered gradually and within 1 month was discharged from the hospital entirely well with a normal plantar reflex. In October 1960 she noted the gradual onset of numbness and weakness of the lower extremities. Progressive urinary hesitancy was noted at that time. She gradually became totally incontinent of urine. Ambulation became progressively difficult and on July 22, 1961 she was admitted to another hospital, barely able to walk without assistance. Lumbar puncture revealed xanthochromic cerebrospinal fluid with a complete block on jugular compression. Total protein was reported as 1800 mg. per cent. Her motor deficit increased after the diagnostic procedure. She was then referred for neurosurgical care at Vassar Brothers Hospital.

The only significant past history was the repair of a cystocele in February 1949 under spinal anesthesia using 10 mg. of Pontocaine. She had no complaints of weakness of her lower extremities or sphincteric disturbance in the ensuing years. No traumatic episode had occurred prior to the onset of the present illness.

Examination. Aug. 4, 1961. The patient was a 70-year-old white female complaining of numbness and weakness of her legs. There was no tenderness of the spine. There were no abnormalities in the cranial nerves. Ophthalmoscopic findings were normal. Tendon reflexes were lively and equal in both upper extremities. Knee jerks were hyperactive. The left ankle jerk could barely be elicited; the right was absent. The abdominal skin response was absent in all quadrants. There was a bilateral Babinski's toe sign. Appreciation of pinprick was diminished markedly from T6 down on the right including the saddle area. There was moderate diminution in appreciation of pinprick on the left beginning at T6 and becoming more severe as the lumbar dermatomes were reached. Light touch was involved similarly. Proprioception was abolished completely in the left great toe but was intact on the right. Tuning-fork appreciation was absent on the left and present on the right. There was paralysis of the left lower extremity and moderate paresis of the right lower extremity. In
summary this patient presented a modified Brown-Sequard syndrome with a level to T6.

Hemoglobin was 14.2 gm.; count of white blood cells was 5,280 with a normal differential. Blood urea nitrogen and blood sugar were normal. Roentgenograms of the chest were normal, as were those of the cervical, thoracic, and lumbar spine. Cystogram demonstrated an atomic bladder. Cisternal myelogram with 1 cc. of Pantopaque revealed a complete block at the level of T4.

Operation (R.M.W.). Laminectomy was performed from T3 to T7. There were no pulsations of the dura mater below the level of T4. Pulsations were normal above this. Upon opening the dura mater the arachnoid was found adherent to it and markedly thickened. It was pinkish-grey and measured approximately 3 mm. thick, encircling and compressing the spinal cord. At the caudal end of the lesion there was a tight band of arachnoid which closed the spinal cord. The dura mater was stripped from the subdural mass with ease and held back with stay sutures. The thickened arachnoid was first split down the middle, and peeled away from the cord. Each half was then excised laterally. The spinal cord had a yellowish discoloration. The constricting band was cut. A catheter then was passed caudally and cephalically in the subarachnoid space for a distance of 15 cm. with no obstruction. The dura mater was left open and sutured to the paravertebral muscle. The wound was closed in layers.

Microscopic Examination. Sections of the specimen showed it to consist of dense collagenous connective tissue with fresh hemorrhage and with very loose lymphocytic infiltration. One surface of the specimen showed a slightly looser construction of the connective tissue, and included multiple psammoma bodies (Fig. 1). At the border between this looser layer and the denser layer, there were a number of small blood vessels. Particles of hemosiderin were scattered through the tissue.

Postoperative Course. On the day following operation there was marked improvement in the motor deficit of the left lower extremity. Proprioception was now present in the left great toe, and the sensory deficit was less marked on the right. On the next day there was rapid increase in the paresis of the left lower extremity, proprioception was lost in the left great toe and the sensory deficit on the right was more profound. Ninety gm. of urea were given intravenously. A diuresis occurred and within 4 hours there was marked improvement in the motor power of the left lower extremity and sensory alteration on the right became less profound. From this point on the patient gradually improved and on the 12th postoperative day she was out of bed, ambulating with a walker. Seven months postoperatively, the patient was ambulatory with a cane. There were hyperactive reflexes in the lower extremities, and normal plantar responses. Proprioception, and appreciation of light touch and pinprick were normal. There was minimal spastic weakness of both lower extremities. At that time voluntary control of the bladder returned with a residual urine of 30 cc.

DISCUSSION

The presence of particles of hemosiderin in the surgical specimen would suggest that the subarachnoid hemorrhage had a role in the natural