SPONTANEOUS SUBARACHNOID HEMORRHAGE CAUSED BY EPENDYMOmA
OF FILUM TERMINAL

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(Received for publication May 20, 1957)

In 1951 Fincher1 presented a paper entitled “Spontaneous Subarachnoid Hemorrhage in Intradural Tumors of the Lumbar Sac,” based on 5 cases gathered from the literature and his personal records. The tumors in these cases, 3 ependymomas, 1 neurofibroma, and 1 neuroglioma, were manifested by abrupt onset of severe sciatica, headache, and the presence of subarachnoid blood on lumbar puncture. The similarity in the clinical picture of these cases prompted him to suggest that they represent a clinical syndrome. Roger et al.4 in 1949 presented 2 cases, not included by Fincher, but closely resembling his cases. One was a “neurospiongioma” (medullo-blastoma) without evidence of a primary intracranial lesion, and the other a meningo-gioma. Other authors have commented on bleeding into and around intraspinal neoplasms, giving rise to symptoms that do not fit the syndrome suggested by Fincher, and therefore not within the scope of this paper.2,3,5,6

Following is a report of a case of spontaneous subarachnoid hemorrhage caused by a tumor of the lumbar sac typical of the above syndrome.

CASE REPORT

A 25-year-old airman enjoyed excellent health until July 5, 1954, at which time he awoke with low backache. He had sustained no antecedent trauma other than having jumped down five steps of a stairway the day before, with no discomfort at the time. The low-back pain persisted, tending to radiate up to the dorsal region, and at times down the posterior aspect of both thighs. He was otherwise asymptomatic until July 8, 1954, when he awoke from a nap with a throbbing bifrontal headache, which persisted until admission at an Air Force Base Hospital 10 hours later. On admission there, the patient was found to be alert and oriented. Temperature was 99.2°F, pulse rate 70, and blood pressure 110/80. The only positive finding was low-back pain experienced on attempted flexion of the neck. Routine laboratory examination was normal. During the first hours in the hospital, the patient’s headache became much more severe, with onset of nuchal rigidity, nausea, and vomiting. Accordingly, a lumbar puncture was done. The opening pressure was 180 mm. of water. The fluid was grossly bloody in all three specimens. The centrifuged supernatant fluid showed marked xanthochromia. Cell counts and protein determination on the spinal fluid were not done. The patient was placed on bed rest. He required narcotics for the relief of headache. The patient became afebrile on the 4th hospital day, his temperature having been in the range of 100° to 101°F. By the 5th hospital day, the patient was asymptomatic and was transferred to a neurosurgical center. Because of a mix-up in his records, he was inadvertently discharged from that center without further studies being done. Up to that time, repeated neurologic examinations had been normal.

In October 1954, his original physician discovered that he had not had diagnostic studies at the neurosurgical center, and subsequently on Oct. 21, 1954, the patient was transferred to the Lackland Air Force Hospital. He had been asymptomatic from the time of discharge until admission at that time. General physical and neurologic findings were normal on ad-

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mission. On October 30, 1954, bilateral carotid angiography failed to show any abnormality. The patient was returned to duty on Nov. 1, 1954.

Shortly thereafter, he noted the return of mild low-back pain, not related to exertion. The pain increased gradually in frequency and severity, and after 4 days of outpatient physical therapy without relief he was admitted to his base hospital on Jan. 28, 1955. For several days prior to admission, the patient had noted aggravation of backache with coughing and deep inspiration. Neurologic findings were normal. Lumbar puncture showed grossly cloudy and bright yellow spinal fluid which clotted spontaneously. Jugular compression revealed no evidence of subarachnoid block. The spinal fluid contained 440 mg. per cent protein, 1840 erythrocytes, 10 lymphocytes, and 33 mg. per cent sugar. In view of these findings, the patient was transferred to the Neurosurgical Service at Lackland.

Examination. During his workup, bilateral sciatica developed, worse on the left than on the right, aggravated by cough and relieved by bed rest. He had no headache, and there were no subjective neurological symptoms referable to his bladder, lower extremities, or sexual organs. He walked with a limp because of pain in the back and legs. The neck was supple, but on flexion, low-back pain was aggravated. Jugular compression also aggravated the low-back pain. The normal lumbar lordosis was obliterated. There was tenderness to percussion over the lower lumbar spines. Flexion was limited to 45° because of pain. Straight leg raising was normal bilaterally. Neurological findings were normal except for equivocal diminution of the left patellar reflex.

Bleeding and clotting times were normal. Roentgenograms of the lumbar spine were normal. An electromyogram of the lower extremities was normal.

On Feb. 25, 1955, Pantopaque myelography was done. The needle was introduced at the lumbosacral level, and a partial block was demonstrated on jugular compression. The fluid was xanthochromic and clear and contained 500 mg. per cent protein. Five cc. of Pantopaque were introduced and an intradural block was shown at the L4 level. An additional 1 cc. of

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**Fig. 1.** Lumbar myelogram showing the intradural tumor outlined by dye introduced both below and above it.

**Fig. 2.** The dark circumscribed ependymoma removed at surgery.
Pantopaque was then introduced at the L2–3 level to outline the upper level of the obstruction, which was at the rostral level of L4 (Fig. 1).

Operation. On Mar. 1, 1955, lumbar laminectomy was performed. After opening the dura mater, the arachnoid was found to be markedly thickened and fiery red over the full extent of the exposure. A dark purple, ovoid tumor of the filum terminale, 4 cm. in length, was found filling the subarachnoid space (Fig. 2). After dividing the filum terminale, the tumor was lifted out with ease, since no nerve roots were attached to it.

Postoperative course was uneventful. Neurologic findings remained normal. The patient was discharged from the hospital on Mar. 16, 1955. He has continued to work and was asymptomatic when last heard of in October 1956.

Histopathologic diagnosis, confirmed by the Armed Forces Institute of Pathology, was papillary ependymoma. The marked vascularity of the stroma was noted (Fig. 3).

COMMENT

An ependymoma of the cauda equina gave rise to subarachnoid hemorrhage of such striking character as to prompt investigation by cerebral angiography. Since it is not uncommon for blood in the lumbar sac, from intracranial source, to produce low backache and sciatica, the patient’s original complaint of backache was ascribed to this phenomenon. Had laboratory examination of the original specimen of spinal fluid been done and shown marked elevation of the protein, as it did later, perhaps the spinal origin of the lesion might have been suspected earlier.

This case further documents the clinical syndrome proposed by Fincher.
REFERENCES


