SPONTANEOUS SUBARACHNOID HEMORRHAGE IN INTRADURAL TUMORS OF THE LUMBAR SAC

A CLINICAL SYNDROME*

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This is a neurosurgical postulate in which there is an abrupt onset of intractable sciatica, a concomitant violent headache and the presence of a liberal amount of fresh blood in the spinal fluid. The total experience on which this study is based is perhaps insufficient for finality. The clinical parallelism in these cases, however, is so striking as to suggest that with a few additional contributions one may confirm the clinical characterization of certain tumors within the dura of the lumbar limits of the spinal canal. The diagnosis of these tumors is completely dependent on contrast myelo-graphic studies of this spinal area. Unless these investigations are inaugurated early, intermittent attacks of radicular distress and cephalalgia can be expected. With present-day attention centered on the “ruptured intervertebral disk” in accounting for lumbago-sciatica handicaps, and the enthusiasm for cerebral angiography in spontaneous subarachnoid hemorrhage, it seems apropos that attention be given this possible syndrome for certain lumbar intradural tumors.

The first case of meningeal hemorrhage caused by a tumor of the cauda equina was reported by André-Thomas et al. in 1930. The only other case in the literature was chronicled by Abbott in 1939. It is of passing historical interest, as pointed out by André-Thomas, that Vigneras was in error in his interpretation that “the existence of a yellow fluid on spinal puncture” in 3 of his cases indicated meningeal hemorrhage caused by medullary tumors of the spinal cord. André-Thomas likewise directed similar criticism to Elsberg’s report of bleeding tendencies in spinal cord tumors, for Elsberg also thought the xanthochromic spinal fluid was of hemorrhagic origin. So it stands that André-Thomas recorded the first experience of a true recurrent spinal hemorrhage associated with a verified tumor of the cauda equina. The fluid withdrawn in his case was “the color of port-wine... withdrawn easily and abundantly... when centrifuged the fluid remained colored showing an extensive hemolysis.” Cisternal puncture was performed on André-Thomas’ patient on the day following a lumbar puncture, and the “cerebrospinal fluid withdrawn was slightly colored but infinitely less bloody than that from the subarachnoid lumbar spaces.” Abbott’s case

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represents the extreme frequency with which the symptomatology of such a spinal cord tumor may recur. His patient bled spontaneously on more than 25 occasions. His experiences further illustrate the practical clinical considerations that may be the more evident explanations for these patients' complaints. The primary objective neurologic evidence is likely to be directed to the cerebrum as the site of origin of the spontaneous bleeding.

CASE REPORTS

There are 5 cases on which this study is based. Three of these are from the author's records. Four of the patients were operated upon by other neurosurgeons.

1st Attack, April 1923. Severe pain, acute in onset, more severe at night; included "the whole pelvis and thighs." Necessitated several injections of morphia. Patient had had some pain in sacral region and down the posterior thighs since 1918.
2nd Attack, March 1927. "Had to be hospitalized twice for three weeks for pain," of the same character and in the same areas of the attack of 1923. Improved spontaneously and "on dismissal returned to his job."
3rd Attack, April 1929. This necessitated hospitalization. Again severe "pain in the sacrum, perineal pain radiating into the thighs and up to the shoulders." Recovery was after a short interval and he returned to work. "Some sphincter dysfunction persisted. Permanent stiffness of his spine."
4th Attack, April 1930. "Sacro-lumbar pains radiating to the lower limbs." For this attack he was hospitalized and on May 2, 1930 there were motor, sensory, reflex and sphincter disturbances adequate to suspect a lumbar or sacral neoplasm. Lumbar punctures (May 2, 6, 10) showed "still quite bloody cerebrospinal fluid." Pressure and manometrics were not reported.
Myelography, May 12, 1930. Lipiodol. Obstruction "at upper edge of the second lumbar vertebra."
Laminectomy by Dr. de Martel, May 24, 1930. Intradural tumor "somewhat to the right of the midline, completely extracted from the lumbar 1–2 level."
Histology. Neuroglioma.
Results. "Incomplete paralysis of extensors of the right foot (improving) and diminution of knee reflex."

Comment. It is to be noted in Case 1 that André-Thomas recorded the positive statement that on the day of his examination of the patient "he does not complain of headaches." He did state, however, that his patient suffered radicular irritations remote from the site of the spinal root compression by the tumor. With bloody spinal fluid obtained from the cisterna magna it is quite likely that his patient had had adequate meningeal irritation to produce a cephalalgia of some degree, if not that particular day, at least in previous attacks. The other 4 patients' records indicate headaches as dominant complaints.

In Woodhall's patient (Case 4 of this report), headache "developed a week later than the appearance of severe pain in both legs," and was accompanied by nausea and vomiting. This patient had had headaches as a
dominant symptom in the first episode of spinal bleeding 4 years previously. He recalled that on his first experience he had had "a double vision with that attack of violent sciatica" and stated the diplopia developed with a severe headache. This supposition of the presence of increased intracranial pressure with the first attack was enhanced by the parents' description of "the development of cross-eyedness." It is further noted from the author's records of this case that "on the three occasions" when the patient's attending physician withdrew "bloody spinal fluid" on lumbar punctures the headaches and pains in the child's legs "were definitely relieved." The strabismus disappeared after the third spinal puncture. When this patient was seen in his second attack the headaches and projectile vomiting were as distressing symptoms as his spinal rigidities and spontaneous radicular contractures of the lower extremities.

In Abbott's1 patient (Case 2 of this report), as well as in Cases 3 and 5, headache was as outstanding a symptom as the lumbar spinal root complaints. It would appear from Woodhall's records in Case 4, as well as in André-Thomas' in Case 1, that there comes a time in the expansion of these cauda equina tumors when continued spinal nerve root compression dominates the clinical picture. The symptoms produced by the spontaneous hemorrhage may then be subjectively minimized. But it is hoped that alertness to this possible syndrome of cauda equina tumors may result in earlier diagnosis and treatment before the development of objective neurological symptoms.

**Case 2.** Male, aged 16. Reported by Dr. Kenneth H. Abbott1 in September 1939.


2nd Attack, September 1937. From then until November 1938 some "25 more attacks of acute low back pain and acute headaches." During 3 of these attacks spinal puncture yielded bloody fluid and the supernatent fluid was xanthochromic. No clinical or manometric evidence of spinal cord or low spinal nerve root compression.


Myelography, Jan. 19, 1939. Obstruction at D12 level.

Laminectomy by Dr. R. B. Raney, Jan. 21, 1939. Encapsulated tumor, enveloped by nerves of cauda equina from the tip of the conus medullaris.

Histology. Ependymoma.


**Comment.** The histology of these growths that are prone to initiate periodic spinal hemorrhage is of interest. In Cases 2, 4 and 5 the tumors were typical ependymomas. The chronicity of this type of glioma5,4 is well estab-
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lished and hence the long histories of recurrent symptoms are understandable. The hemorrhagic tendencies of tumors of the cauda equina have heretofore escaped the cognizance this condition demands. The hypertrophy of the pial vessels associated with these tumors is partially attributed to a mechanical obstruction. With violent physical efforts, because of the thin walls of these vessels, a rupture is conceivable. Spontaneous hemorrhage without physical influences, however, escapes logical interpretation. In André-Thomas’ case the histologic reproduction is printed on a poor grade of paper but the depiction could well be that of an ependymoma. His histological diagnosis, even though the tumor “was traversed by a spinal nerve root,” was “vascular neuroglioma.” In view of the fact that this was reported as late as 1930, it is unlikely that the tumor was a neurofibroma. Thus it is possible that 4 of the 5 tumors associated with proven episodes of repeated spontaneous spinal bleeding may be of the same cellular type. Case 3 is the only instance of the tumor being definitely not of ependymal origin. This lesion was reported by a most able pathologist as a “neurofibroma of unusual vascularity.” So in this proposed syndrome, one might, with added experiences, not only learn that a spinal cord tumor is the explanation for the spontaneous appearance of headaches and sciatic pain, but might even venture to predict the histologic character of the growth with a fair degree of accuracy.


1st Attack, November 1933. Sudden attack of pain in left side of face, behind eyeballs; pain in both inguinal regions radiating into testicles. Pain severe for 6 hours. No residuals after 24 hours. No treatment.

2nd Attack, January 1936. Onset of severe vertex headache. The following day pain in low back and inguinal regions. Treated symptomatically. Returned to work after 10 days with minor headache and low back pain.

3rd Attack, March 1936. Abrupt onset of severe headache and discomfort in low back area. After 24 hours pain radiated into sacrum and down the left leg posteriorly. Patient remarked that “sudden movement or coughing caused a simultaneous exaggeration” of his vertex headache and left sciatica. Hospitalized. Spinal fluid: pressure 200 mm. water; fluid bloody; contained 216 WBC, with 53 per cent lymphocytes; Pandy ++ +++. No objective neurologic handicaps. Fluid cleared as did his headaches and sciatica after the third spinal puncture. Each puncture was performed on consecutive days. Patient had a fourth spinal fluid study 6 months later in an asymptomatic period. Queckenstedt test, omitted purposely initially, was at this time normal, as was complete microscopic and chemical examination of the fluid. It was the neurologist’s opinion that a spinal cord tumor could be excluded at that time.

4th Attack, Nov. 23, 1936. Sudden onset of violent sacral pain with an associated severe headache. After a week, because of the increasing intensity of symptoms, he was hospitalized. Spinal fluid grossly bloody; Pandy ++; pressure 180 mm. water. Symptoms subsided after the one puncture.

Myelography. Dr. J. B. Ayer: “No block on spinal puncture. Lipiodol. Obstruction, L1 level.”
Laminectomy by Dr. William J. Mixter, April 9, 1937. Lumbodorsal level. Intact total extirpation of neurofibroma.

Results 13 years after operation. No recurrence of symptoms. No neurological objective symptomatology.

Comment. As illustrated in all of these cases, it has been contrast radiologic studies that have established the diagnosis. If one reviews one's experiences in spontaneous subarachnoid hemorrhages one finds that lumbar-sacral radicular complaints are no rarity in the conscious patient. This is logically explained on the basis of low spinal nerve root irritation from the hemolyzed blood. One theoretical answer for the lumbar lower extremity pain has been that these patients may have had some pre-existing minor mechanical nerve root compression. This lumbar-sacral nerve root pain is common following cerebellar craniotomies and is often relieved by the lumbar drainage of bloody spinal fluid. In the abrupt spinal bleeding in cases of tumor of the cauda equina the nerve root pain is much more severe and offers much greater therapeutic competition than any other of the painful spinal root situations. One cannot rely on manometric evaluations for the diagnosis of these tumors in the lumbar region that precipitate ruptures of the blood vessels. Queckenstedt's test at the time of the impulsive bleeding may be omitted because of the disturbing measurable increase in intracranial pressure as recorded on the attached spinal manometer cylinder. These manometric studies even in the hemorrhagic phase may be normal. Spinal fluid protein determinations in the acute condition are unreliable because of the volume of blood mixed with the spinal fluid. Spinal fluid studies, in the interval of freedom from lower extremity pains and headaches, can be perfectly normal both physically and chemically. Thus the early recognition of this syndrome hinges on contrast myelography. Even though some spinal cord implications were objectively evidenced at some period in the clinical course of 2 of the 5 cases, the conclusive lead to the final diagnosis in all instances was obtained by myelography.

Case 4. #42-687. Male, aged 12 years. First seen Sept. 24, 1942. Referred by Dr. J. T. McCall.

1st Attack, 1938. Acute onset of headache, nausea and vomiting. Pain in low back, radiating into buttocks. Spinal puncture reported as "bloody and the pressure increased." Body plaster cast for a week. Two subsequent spinal punctures at 2-day intervals revealed changes in the fluid similar to initial diagnostic puncture. Cerebral and low back sciatic symptoms relieved following each spinal tap. No residual physical handicaps.

2nd Attack, Sept. 19, 1942. Abrupt onset of pain in left leg, and low back. Followed by stiffness of neck and headache. Low back spasticity and opisthotonos obviated spinal puncture under novocain local anesthesia, and 3rd lumbar puncture was performed under pentothal narcosis. Pressure: 430 mm. water. Queckenstedt omitted as being contra-indicated. Grossly bloody fluid. Symptoms disappeared remarkably following puncture.

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Histology. Ependymoma (Fig. 1).

Results 8 years after operation. No recurrence of symptoms. No motor, sensory, or sphincter residuals.

Comment. If one could add to the dominant clinical characterizations of these spinal cord tumors, it would be the statement that these patients exhibit no objective neurologic evidences in the early phases of the recurrent spinal hemorrhages implicating lumbar spinal cord levels. The symp-

![Image](image_url)

Fig. 1. Case 4. Photomicrograph of ependymoma (×180).

tomatology, in the early manifestation of this disease, is such as to suggest a cerebral origin for the bleeding. The high spinal fluid pressure, the intensity of the headaches and even the appearance of optic nerve swellings (Case 3) logically center attention on a cerebral origin for these patients’ complaints. The intensity of the radicular distress in the period of hemorrhage appears to be a clue to the diagnosis. This sciatic pain, dominantly unilateral or bilateral, is of such a severe character as to produce malignant irreducible opisthotonos, and intense muscular irritations which involve one or both lumbar lower extremity muscle groups. This pain may be paroxysmal. It may be spontaneous or initiated on sensory irritation of the skin of the lower extremities or buttocks. Its violence may be of such a character as to be in the category of the pain of child-birth. This spinal nerve root reaction strikes without warning, without any necessary physical antecedents and may persist for many days. Apparently the pain can be allayed by spinal fluid drainage. The headaches from which these patients may suffer are in the same category as the throes from spontaneous subarachnoid leakage of a ruptured cerebral blood vessel.
The objective evidences of profound Kernig and Brudzinski reactions serve as misleading neurologic findings. The vomiting characteristic of increased intracranial pressure is explained on the finding of an increased manometric spinal fluid pressure or the presence of "choked disks." Both of these objective evidences serve to further direct the examiner's attention away from the spinal cord pathology. The development of cranial nerve alterations results from the arachnoidal reaction to the irritative blood or its by-products within the subarachnoid absorptive spaces. These serve as further diagnostic misleads. At any rate, the sudden appearance of severe lower extremity radicular pain associated with deflagrating headaches should call to one's diagnostic mind the possibility of the existence of a low-placed spinal cord tumor that has initiated a spontaneous spinal hemorrhage.

Case 5. #46-222. Female, aged 18 years. Referred by Dr. S. D. Brown, Royston, Georgia, Feb. 24, 1946.

1st Attack, August 1944. Abru pt onset of intense lumbar, gluteal, thigh pain with severe cervico-occipital distress. Duration 10 days. No subjective motor, sensory or sphincter handicaps.

2nd Attack, November 1944. Again lumbosacral radiculitis pain and rigorous cephalalgia. Less severe and of shorter duration than initial upset. Again no motor, sensory or sphincter dysfunctions.


4th Attack, May 21, 1946. Acute bilateral lumbago-sciatica. No headache. Spinal fluid bloody: pressure 270 mm. water. No obstruction to bilateral jugular compression. Injection of 2 cc. lipiodol: total obstruction to oil on fluoroscopy (Fig. 2).


Histology. Ependymoma (Fig. 3).

Results 5 years after operation. No neurologic objective residuals. No objective or subjective neurologic or physical symptomatology.

SUMMARY

It seems definite from a detailed study of 5 cases of cauda equina tumors associated with spinal subarachnoid hemorrhages that the symptomatology of these growths in their early development is entirely subjective. There is no intimation in the beginning of this disease of any motor, sensory or
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Fig. 3. Case 5. Photomicrograph of ependymoma (×100).

### TABLE 1
Summary of 5 cases of spinal cord tumor with spontaneous subarachnoid hemorrhage

<table>
<thead>
<tr>
<th>Case</th>
<th>Maximal C.S.F. Pressure Recorded</th>
<th>Character C.S.F. Fluid</th>
<th>Maximal Objective No. of C.S.F. Manometry</th>
<th>Objective Spinal Cord Symptons</th>
<th>Myelography</th>
<th>Location of Tumor</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4 in 12 years reported</td>
<td>Blood: 3 separate occasions</td>
<td>Not reported</td>
<td>No block</td>
<td>Lipiodol</td>
<td>L2, 3</td>
<td>Neuroglioma</td>
</tr>
<tr>
<td>2</td>
<td>25+ in 1 1/2 years 300 mm.</td>
<td>Blood: 4 separate occasions</td>
<td>No block</td>
<td>No Lipiodol L1, 2, 3, 4</td>
<td>Ependymoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>4 in 4 years 200 mm.</td>
<td>Blood: 4 separate occasions</td>
<td>No block</td>
<td>No Lipiodol D12, L1</td>
<td>Neurofibroma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>2 in 4 years 400 mm.</td>
<td>Blood: 4 separate occasions</td>
<td>No block</td>
<td>Lipiodol Hypo-reflexia</td>
<td>Ependymoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>4 in 2 years 320 mm.</td>
<td>Blood: 2 separate occasions</td>
<td>No block</td>
<td>No Lipiodol L2</td>
<td>Ependymoma</td>
<td></td>
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sphincteric spinal cord dysfunctions. The diagnosis should be suspected on the appearance of one or more bouts of intractable lower extremity pain and an accompanying profound cephalalgia. The development of spontaneous subarachnoid bleeding in these patients as disclosed on spinal puncture should indicate the diagnosis. This bloody spinal fluid should prompt early myelographic contrast studies before compressive or destructive spinal nerve root symptoms are in objective evidence. A syndrome is defined by Webster as "a group of signs and symptoms that occur together and characterize a disease."

REFERENCES