CASE REPORTS

HEMANGIOMA OF THE SPINAL CANAL AND PREGNANCY

ROBERT L. LAM, M.D., GEORGE E. ROULHAC, M.D., AND HERBERT J. ERWIN, M.D.

Department of Neuropsychiatry and Division of Neurological Surgery, Washington University School of Medicine and Homer G. Phillips Hospital, St. Louis, Missouri

(Received for publication April 10, 1951)

Vascular tumors of the spine and spinal cord are not uncommon, but they have rarely been noted as a complication related to pregnancy. Glaser reported a case of angiosarcoma of the cord in which progressive paraplegia developed during the last half of pregnancy. Following delivery there was a brief interval of improvement prior to gradual decline and death. Balado and Morea found an extradural angiolipoma at autopsy on a woman whose seventh pregnancy had been characterized by paraplegia with improvement after delivery. Permanent paralysis developed during her eighth pregnancy, culminating in death. Temporary improvement of spinal cord signs following delivery was noted in 2 cases of hemangioma reported by Delmas-Marsalet and Guthkelch. The former lesion was proved at operation; the latter was found at autopsy. The clinical manifestations in a case of hemangioma recently observed by us conform closely to the previous descriptions.

CASE REPORT

A well-circumscribed extradural hemangioma of the upper dorsal segments is reported. The initial clinical manifestations appeared in the last trimester of the ninth pregnancy of a 36-year-old woman; there was subsequent remission of symptoms and signs during the immediate postpartum period. Cessation of that improvement led to operation and complete removal of the tumor with a remarkable recovery.

#49-42612. W.M.S., a 36-year-old female, para viii, gravida ix, was admitted to the Obstetrical Service of the Homer G. Phillips City Hospital, Nov. 13, 1949, during the last month of her pregnancy. Gestation had been unremarkable until 1 month before admission, when she noted the gradual onset of weakness, paresthesias, coldness, and impaired sensation in both lower extremities, more severe on the right. Subsequently, she complained of dull thoracic back ache and a girdle sensation about her abdomen. There developed increased urinary frequency. There was no history of similar complications associated with any of her previous pregnancies. A primary syphilitic lesion had been found in 1933, with a positive serological test. Intensive antiluetic therapy had been given, resulting in persistently negative serology from 1939 to her recent hospitalization.

Examination. She was a well-developed, well-nourished young colored female in approximately the 34th week of a normal pregnancy. There was spastic paraplegia, with considerably greater involvement on the right. All tendon reflexes in the lower extremities were increased. The right knee and ankle jerks were more active than those on the left. Bilateral ankle clonus was present, but was more sustained and active on the right. Patellar clonus was unsustained bilaterally. Marked extensor toe signs were elicited on the right. Those on the left were equivocal. Abdominal reflexes were absent. Sensory examination showed a strongly suggestive level at L-1, involving particularly pin prick and temperature appreciation. Deep sensation
was not significantly diminished. Both lower extremities were cold to palpation. Three days after lumbar puncture, examination demonstrated a progression of the sensory level to the 3rd thoracic dermatome, and mild but definite decrease of position, motion and vibratory sense in the lower extremities.

CFS pressure was 240 mm. water. Fluid was clear and colorless and contained no cells; total protein was 91 mg. per cent with positive Pandy; Wassermann and colloidal gold tests were negative. Manometric studies gave the impression of an incomplete block. Roentgenograms of the thoracic, lumbar, and sacral spine were negative. The interpedicular distances were normal. Blood and urine were normal.

Course. On Nov. 29, 1949, the patient gave birth to a normal 6½ pound infant after an uncomplicated 7-hour delivery. The postpartum recovery was entirely satisfactory. Regression in the sensory level rapidly followed. There was considerable return of strength in the right lower extremity. She was discharged on Dec. 10, 1949, returning home for convalescence. However, she did not continue to improve and was hospitalized on Jan. 28, 1950 for reevaluation. Shortly before, she had noted incontinence of urine.

Examination on Readmission. The sensory level was again at D-3, without sacral sparing and dissociation of sensation noted during her previous hospitalization. A sweating level corresponded to the sensory loss. Motor signs were more prominent, with a paraplegia in extension. Again, complete spine studies by x-ray showed no evidence of bony changes. Lumbar puncture disclosed an initial pressure of 150 mm. water, and complete block on manometrics. Subsequent myelography indicated a failure of pantopaque to flow beyond the T-3 level.

Operation. With a tentative diagnosis of a slowly growing benign extramedullary tumor, probably a vascular meningioma, dorsal laminectomy was performed by one of us (GER) on Feb. 23, 1950.

Surgical removal of the lamina of the 2nd, 3rd and 4th thoracic vertebrae disclosed a highly vascular epidural space containing a dark-red tumor mass measuring 3×1½ cm. The lesion was discrete, entirely extradural, and partially encompassed the dorsal surface of the dural sac, extending more to the left than to the right. The mass was removed in toto.

Histopathological Diagnosis: Hemangioma (Fig. 1).

Fig. 1. Extradural hemangioma of the spinal canal from case W.M.S. Hematoxylin and eosin stain, ×108.

The postoperative course was entirely uncomplicated and during the latter part of the first week of recovery, motor and sensory function definitely improved. Subsequent return of
neurological function was rapid and the patient left the hospital April 28, 1950, ambulatory with the aid of a cane, and without any evidence of sensory loss. Several weeks later, completely normal activity was possible without any mechanical aids. Deep tendon reflexes were normal, and pathological toe signs were absent.

**DISCUSSION**

In the case we have just presented, as in cases previously reported, symptoms of an intraspinal vascular lesion developed during the last half of pregnancy, and improved temporarily following delivery.

In explaining the influence that pregnancy may have upon hemangiomas of the spinal canal, two possibilities must be considered: accelerated neoplasia and vascular changes due to obstruction or to endocrine influences of pregnancy. The relationship between the highly malignant chorioepithelioma and hydatidiform mole is well established. On the other hand, the direct effect of pregnancy upon behavior of other neoplastic lesions, especially tumors of the nervous system, is not clear. In neurofibromatosis increased pigmentation may occur during pregnancy, with later diminution during the puerperium. The same observations have been noted in epulides. Isolated reports suggest that acoustic neurinoma and meningioma occasionally manifest themselves during pregnancy. On the other hand, the opinion that pregnancy has little direct effect upon tumor growth has been expressed from other clinical evidence and experimental work. It is even suggested that in most cases pregnancy may afford a protective mechanism to neoplasia itself (Emge). At this time, there seems to be little evidence to indicate that tumors of the nervous system occur with greater frequency in pregnancy.

The stage of maximum enlargement of the uterus seems to bear a definite relationship to the appearance of symptoms in hemangioma of the spinal cord; and its emptying at delivery with definite improvement. The indictment of vascular changes in or about the tumor as a result of interference with the venous drainage from the spinal canal and its contents may explain the peculiar increase and decrease of symptoms seen in this lesion during pregnancy. In an unrelated study McLennan has recently investigated femoral vein and antecubital vein pressures in pregnancy. He reports that the femoral vein pressures rise slowly during the early part of the 2nd trimester, and exceedingly rapidly once the 3rd trimester is reached, to attain a peak at term. The postpartum state is characterized by a sudden fall to subnormal levels. No significant alterations were seen in the venous pressure in the upper extremity. The abnormal venous pressure is considered to be the consequence of obstruction to venous return by the pregnant uterus. Thus, it is reasonable to assume that the same factor may exercise a marked influence in the venous outflow from the spinal and vertebral vessels to the inferior vena caval system. The fact that in this case the symptoms were temporarily ameliorated during the postpartum period of subnormal or normal femoral vein pressure is very suggestive that venous engorgement in or about the tumor is an important contributory factor.

Vascular phenomena secondary to estrogenic influences may possibly contribute to the symptoms of hemangioma with pregnancy. Vascular spiders and palmar erythema have been seen to appear during pregnancy and to fade rapidly following delivery. The high incidence of vascular spiders has been well known since reported by Corbett in 1914. Palmar erythema has been noted more recently, in 1939 by Feldman. Bean et al., studying over a thousand cases, have confirmed the previous observations and have advanced the hypothesis that the cutaneous vascular changes
were due to high levels of estrogens circulating in the blood for long periods of the pregnancy.

**SUMMARY**

(1) Spinal cord symptoms and signs in a case of extradural hemangioma became manifest during the last month of pregnancy, improved rapidly during parturition, and completely disappeared after surgical removal.

(2) Exacerbations and remissions in symptoms are considered in relation to mechanical obstruction to venous drainage as a result of an enlarged pregnant uterus, and to possible estrogenic factors giving rise to increased vascularization.

We are indebted to Dr. J. O. Blache, Pathologist, Homer G. Phillips Hospital, for preparing and interpreting the histological sections in this case, and to Dr. Henry G. Schwartz, Professor of Neurological Surgery, and Dr. James L. O’Leary, Professor of Neurology, for their helpful criticisms in preparation of the text.

**REFERENCES**


**SUBDURAL HYGROMA COMPLICATING MENINGOCOCCIC MENINGITIS**

Stanley H. Steinberg, M.D.,* and James Peter Murphy, M.D.†

Washington, D.C.

Received for publication April 11, 1951

To the list of complications of meningitis must be added subdural hygroma (hydroma or effusion in the subdural space), which has become prominent only recently as a complication of acute bacterial meningitis.3,7 The majority of cases of meningitis in which subdural hygromas have been found have been due to *Haemophilus influenzae*. However, cases in which *D. pneumoniae*, *Salmonella sp.*, paracolon bacillus, *Ps. aeruginosa*, and *N. meningitidis* were the etiological agents have also been reported.7 The relative paucity of information in the literature in regard to subdural hygroma as a complication of meningitis, and the fact that the patient

* 4214 16th St., N.W., Washington 11, D. C.
† 1904 R St., N.W., Washington 9, D. C.