CASE REPORTS AND TECHNICAL NOTES

A METASTATIC LESION SIMULATING AN INTRACRANIAL ANEURYSM

Everett F. Hurteau, M.D.*

(Received for publication January 9, 1948)

Considerable attention has been focused, recently, on the diagnosis and treatment of intracranial aneurysms. The question of the indications for arteriography is a much debated one and it is the opinion of some authors1 that: "Arteriography is helpful in making the diagnosis, but is not necessary. It is possible to localize intracranial saccular aneurysms accurately by clinical means alone when they occur in the cavernous portion of the internal carotid artery, giving rise essentially to palsy of the third nerve with evidence of involvement of the first or first and second divisions of the fifth nerve and frequently the fourth, sixth, and motor divisions of the fifth cranial nerve, as well." Others2 have equally emphasized the ease with which aneurysms within the cavernous sinus may be diagnosed and have repeatedly stressed the disadvantages of arteriography. The following case, though pathologically unique, offers rather conclusive evidence that one may be misled by clinical signs and symptoms no matter how typical of aneurysm they may seem. It is offered in support of the opinion3 that: "Cerebral angiography is indicated especially whenever surgical treatment is under consideration and a precise diagnosis is required for such therapy."

CASE REPORT

A 54-year-old female was admitted to the Hospital Sept. 13, 1947, complaining chiefly of intermittent pain in the left maxillary region and behind the left eye. The onset of this pain was abrupt and occurred about Sept. 10, 1947. She had had several teeth extracted without relief. There had been complete amenorrhea for 6 months. The history was otherwise non-contributory.

Examination revealed ptosis of the left upper lid, but no inequality of the pupils. There was an incomplete left 6th nerve paralysis. Hyposalgesia was evident over the distribution of the 1st division of the left 5th nerve. Other cranial nerves were normal. Deep and superficial reflexes were normal. Sensation to pain, touch, vibration and position was normal throughout the extremities and trunk. Routine cerebellar tests were normal. No contributory signs were found on otolaryngological examination.

Lumbar puncture, September 24: Intraspinal pressure, 100 mm. of water; cytological, chemical and serological examination of spinal fluid revealed nothing abnormal. Several later spinal punctures were also normal. Roentgenograms of the skull on September 24 were normal.

On October 13 a mucopurulent postnasal discharge developed and she was seen again at this time by an otolaryngological consultant. X-rays of the paranasal sinuses showed no significant abnormality and it was felt that there was no relevant sinus pathology.

Pain behind the left eye and in the left frontal region continued to be severe. The left ophthalmoplegia progressed and on October 1 there was complete paralysis of all external ocular muscles on the left. The left pupil was dilated and fixed to light. Pupil of the left lid was complete (Fig. 1). There was no impairment of visual fields or acuity. Fundi were normal. Hyposalgesia had now progressed to involve both the 1st and 2nd divisions of the left 5th cranial nerve. The remainder of the neurological examination was negative.

Presumptive Diagnosis: Aneurysm of left internal carotid artery within the cavernous sinus. On a basis of the literature quoted above and the findings and course of this case, it was felt that arteriography was not indicated.

* 175 S. Main Street, Akron 8, Ohio.

498
Carotid compression was started. This did not relieve her pain and after 5 minutes of compression she complained of numbness in the right hand.

Operations. On October 4 the left common carotid artery was ligated. No untoward symptoms developed. On October 16 it was decided that sufficient time had elapsed for the establishment of collateral circulation. Findings at this time were similar to those of October 1 and remained restricted to those due to involvement of the left 3rd, 4th, 5th and 6th cranial nerves. A left transfrontal approach was made and the anterior fossa and parasellar region were explored. No abnormality was found. A silver clip was placed on the left carotid just proximal to the posterior communicating artery. Immediately following this procedure the left internal carotid artery was ligated in the neck.

Course. A right hemiparesis developed which was mild and transient. Postoperative course was otherwise uneventful. On October 29 a right ophthalmoplegia began to develop. Following this her course was gradually downward, and she expired on Nov. 2, 1947.

Autopsy revealed an infiltrating adenocarcinoma of the uterus which had metastasized to the nasopharynx, presumably by way of the paravertebral veins. There were no other metastases. The lesion in the nasopharynx had extended through the supra-orbital fissure and body of the sphenoid bone to completely fill both cavernous sinuses. There was one small intradural nubbin of neoplastic material in the left middle fossa, but the anterior fossae and parasellar region were completely free of intradural extension. There was marked compression of the contents of both cavernous sinuses and some microscopic invasion of the left gasserian ganglion. The brain appeared normal. It seemed probable that death was due to ischemia following compression of the right internal carotid artery, the left having already been ligated.

Comment. Review of postoperative x-rays taken on Oct. 28, 1947 did show erosion of the body of the sphenoid bone but examination of preoperative x-rays of the skull taken on Sept. 24, 1947 and the paranasal sinuses taken on Oct. 13, 1947 failed to show any erosion even in retrospect.

SUMMARY

The case herein presented fulfilled all of the criteria for the clinical diagnosis of unruptured aneurysm of the internal carotid artery within the cavernous sinus. The fallacy of making this diagnosis without arteriography is illustrated.
ON THE SYNDROME OF ARNOLD-CHIARI

REFERENCES


ON THE SYNDROME OF ARNOLD-CHIARI

REPORT OF A CASE

JERZY CHOROBKSI, M.D., AND LUCJAN STĘPIEŃ, M.D.

Service of Neurosurgery, Neurological Clinic, University of Warsaw, Poland

(Received for publication January 10, 1948)

A malformation of the metencephalon, associated with herniation of the spinal cord and of its meninges through a large lumbosacral spina bifida, was first described by Arnold in 1894. Two years later, Chiari published a description of a still more serious developmental anomaly consisting of transposition not only of the abnormal cerebellum, but also of the medulla oblongata into the cervical portion of the vertebral canal. Schwalbe and Gredig (1907) added 4 similar cases and pointed out that, as a rule, both types of malformation, i.e. of the cerebellum and of the medulla oblongata, appear simultaneously, side by side. They emphasized, however, that this is not always so, and that one may be present without the other. Schwalbe and Gredig sub-divided all such cases of malformation of the met- and myelencephalon into several groups, according to the degree of the anomaly, which in extreme instances may consist of displacement of the cerebellar substance through the central canal to the lumbar part of the spinal cord. On the other hand, it may be characterized only by a slight elongation of the vermis cerebelli of an otherwise normal metencephalon. Generally speaking, the difference between the malformation described by Arnold and by Chiari is only that of the degree of the pathological changes that occurred in the hind-brain during embryological life; therefore, it is not surprising that between the extreme cases many intermediary forms are found. This variability in the severity of the anomaly of the hind-brain is due to the difference in the length of time during which the maldevelopment has existed, the severe forms being the result of earlier changes.

Associated with the malformation of the met- or myelencephalon, or with both, is a spinal dysraphism, also differing, from case to case, in its dimensions. Although most frequently found in the lumbosacral region, it can be located in other portions of the spinal column. The disturbance in the development of the vertebral column leading to the absence of fusion of the vertebral laminae, occurs—according to Schwalbe and Gredig—either at the same time as the disturbance in the development of the hind-brain or even earlier. They admitted that the relationship between the two anomalies may be that of a cause to an effect, the spina bifida causing, mechanically, a maldevelopment of the hind-brain. However, it may happen that both anomalies develop independently, at the same time, and due to the same unknown factor.

The spina bifida is frequently complicated by a myelomeningocele. Yet, Schwalbe and Gredig emphasized that the malformation of the hind-brain may be encountered also in the presence of a less pronounced form of spinal and meningeal herniation. In fact, Russell and Donald observed a case of malformation of Arnold and Chiari associated with a meningoele only, and it was clear to them that, in such cases, the deformity of the hind-brain would be of a far less severe order than that seen in cases of myelomeningocele, although it would be of a similar kind. A patient of Penfield and Coburn suffered from the malformation of Arnold and Chiari associated only with an upper thoracic meningocele. No evidence of spina bifida was seen in the case of Aring or in Case I of Adams, Schatzki and Scoville.