ARTERIOVENOUS MALFORMATION OF THE HEAD OF THE CAUDATE NUCLEUS

REPORT OF A CASE WITH TOTAL REMOVAL

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Vascular lesions protruding into the lumen of the ventricles, either from the roof or from the structures of the floor, are infrequently mentioned in the literature. Bassett reported a patient (Case 5) who appeared to have had an intraventricular vascular anomaly arising from the corpus callosum and draining through the internal cerebral veins to the great vein of Galen. No air study was performed and ligation of the anterior cerebral artery distal to the anterior communicating artery was the method of treatment employed; no neurological sequelae were noted. Scott, Simril and Seaman described a patient (Case 18) with an arteriovenous malformation in the left hemisphere extending into the left lateral ventricle, fed by the supra-callosal artery, and draining into the vein of Galen. The patient died of "cerebral edema and thrombosis" after surgical extirpation.

The following case is of interest in that an arteriovenous malformation arising from the head of the caudate nucleus and protruding into the left lateral ventricle was successfully removed by a transventricular approach.

CASE REPORT

V.A. #37031. B.G.S., a 38-year-old white male, was admitted to the Veterans Administration Hospital, Houston, Texas on Feb. 8, 1954, 2 weeks after the onset of apparently severe subarachnoid hemorrhage. Past history was noncontributory, except for intermittent, poorly localized headaches of 8 years' duration. In 1951, 3½ years before admission, he had what was then called an "epileptic convulsion" in which he lapsed into unconsciousness for a period of 2 hours, without apparent residua; further details are unavailable concerning this episode. Three weeks before admission the headaches became severe and were associated with numbness and tingling of all extremities. Two weeks before admission he suddenly announced that he was "about to have a stroke" and immediately thereafter became comatose. He was admitted to another hospital, where it was noted that the cerebrospinal fluid was grossly bloody and his B.P. was 200/100. He remained unconscious for approximately 1 week, requiring oxygen and parenteral fluids; congestive heart failure was treated by digitalization. There was marked fluctuation in his blood pressure, with numerous episodes of severe hypertension. Upon regaining consciousness, he was irrational, had difficulty in swallowing and had a minimal right hemiparesis. Vomiting unaccompanied by nausea was frequent. He was transferred to the Veterans Administration Hospital.

Examination. His pulse rate was 82 and B.P. was 100/70. He was fairly alert, being oriented for person, but not for time and place. Both Kernig and Brudzinski signs were positive. There was minimal right-sided weakness. Sensory findings were normal, as were the cranial nerves; there was no papilledema. No bruit was audible.

Laboratory Data. Hemogram was normal. Bleeding time was 2 minutes, clotting time 4½ minutes, and platelet count 352,000. Blood chemistries, consisting of sodium, potassium, chloride, carbon dioxide, urea nitrogen, and fasting sugar all were within normal limits.
Kline was normal. CSF pressure on Feb. 15, 1954 was 140 mm. The fluid was xanthochromic and contained 6 WBC and 4 RBC per c.mm.; protein was 144 mg. per cent; colloidal gold curve was normal; Wassermann test was negative. Roentgenograms of skull and chest revealed no abnormalities. ECG was normal. EEG done on Feb. 9, 1954 showed grossly abnormal and paroxysmal generalized frontal dominant slow bursts with some left-sided lateralization.

Course. The patient was thought to have had a subarachnoid hemorrhage. Cerebral angiography was performed under local anesthesia on Feb. 16, 1954, injecting each common carotid artery and taking multiple serial exposures. Injection of the left side revealed a small cluster of vessels deep in the left hemisphere, presumably in the left lateral ventricle, which drained almost immediately into the left internal cerebral vein, great vein of Galen, and straight sinus. Injection of the right side, without compression on the left, resulted in filling of the arteriovenous malformation. During the procedure the patient suddenly became confused and his blood pressure precipitously rose to 200/100.

On Feb. 23, 1954, under surital anesthesia, a 40 cc. pneumoencephalogram was performed, this being followed by another left carotid angiogram with anteroposterior and lateral single views. These (Figs. 1 and 2) showed the relationship of the vascular lesion to the ventricular system; it was thought that the lesion arose in the head of the caudate nucleus and protruded into the left lateral ventricle.

Operation. On Mar. 9, 1954, a left frontal craniotomy was performed (Fig. 3). A plug of cortical tissue was removed anterior to the coronal suture and the left lateral ventricle was entered. The arteriovenous malformation was seen in the head of the caudate nucleus 2 cm. from the anterior pole of the ventricle, protruding into the ventricular lumen. This portion of the lesion consisted of numerous loops of vessels, which seemed to drain into the left internal cerebral vein via a curved vessel posteromedial to the body of the lesion. No feeding vessels were visible, specifically, none from the choroid plexus. The ventricular system was deformed; there was complete communication between the atria of both ventricular cavities, with the
Fig. 3. Artist's conception of lesion and operative approach. The ventricular defect refers to the intercommunication of both ventricles in the atrial region. Note the visibility of the choroid plexus in the tela choroidea of the third ventricle and its course into the lateral ventricle, all of which were visualized at operation.

Fig. 4. Photomicrograph of operative specimen, showing communicating racemose blood spaces with endothelial lining, a delicate media, and absent adventitia, ×40.
choroid plexus and venous structures in the roof of the third ventricle clearly visible; the complete course of the choroid plexus through the foramina of Monro into the lateral ventricles could thus be seen. There was also a perforation in the remaining septum pellucidum anteriorly. The ependymal lining of the ventricular system showed a greenish-yellow discoloration, probably caused by old hemorrhage.

Under induced hypotension (Arfonad, or RO 2-2222), the lesion was initially attacked by placing silver clips on the visible vessels, with the hope of obstructing the main feeder. This did not prove feasible and because of persistent bleeding, the dissection was carried into the head of the caudate nucleus for approximately 1 cm., clipping the vertically coursing feeding vessels arising from the middle cerebral artery. The lesion was completely excised, clipping the draining vessel last, this being collapsed and devoid of blood at this time.

Two interesting episodes occurred during anesthesia, one prior to institution of hypotension. This consisted of a sudden drop in pulse to 48 with a rise of systolic blood pressure to 170 mm. Hg, associated with ECG evidence of ventricular extrasystoles and partial heart block; the response to intravenous atropine sulfate was immediate, with reversion to normal rhythm. At the close of the procedure, when the blood pressure was normal, there was a drop of the pulse to 28, with bigeminy and trigeminy, and a marked hypertension of 220/170. The blood pressure gradually lowered and the pulse became regular in response to intravenous pronestyl.

Pathological Report. The surgical specimen (S-434-54) revealed large thin-walled blood spaces communicating with one another, compatible with the diagnosis of hemangioma (Fig. 4).

Postoperative Course. This was uneventful, except for a minimal right hemiparesis, more marked in the leg, which rapidly and completely cleared. The patient was ambulatory on the 10th day. His mental confusion, poor memory, tendency to perseverate, and emotional lability, all gradually cleared.

Left-sided angiography was performed on April 7, 1954 (Figs. 5 and 6), revealing the absence of the previously shown arteriovenous malformation. A small dilated vessel (Fig. 6),
just proximal to the clips, may represent a residual portion of the lesion, or a separate small anomaly.

Fifteen months after operation the patient showed no abnormal neurological findings. There was minimal reduction in performance on psychological testing.

COMMENT

The use of combined pneumoencephalography and angiography served to delineate the relationship of the arteriovenous malformation to the ventricular system. It should be noted, however, that less of the lesion was floating free in the left lateral ventricle than would seem apparent from the radiographs. It was unfortunate that a complete study of the ventricular system was not performed (this was thought too risky at the time, only brow-up anteroposterior and lateral views being taken), since it would undoubtedly have revealed the intercommunication of both lateral ventricles, as previously described.

In discussing the theories of origin of the cavum septi pellucidi and the cavum vergae, Wolf and Bamford support Corning’s view that the cavum vergae is probably a portion of the interhemispheric fissure whose lateral boundaries are the medial aspects of the cerebral vesicles ventral to the caudally extending corpus callosum. The defect present in this case, although showing no evidence of either of the above anomalies, may have been caused by a failure of development of the medial portion of the cerebral vesicles, and consequent intercommunication of the portions of the ventricles. The defect would therefore be on a congenital basis, rather than caused by the secondary results of massive intraventricular bleeding.

The marked fluctuations in cardiovascular responses, prior to and during surgery, likewise remain unexplained. Both the severe hypertension and transient heart block of varying degree may well have been related to the site of the pathological lesion, since autonomic changes have been elicited by stimulation in neighboring areas (the “septo-hypothalamic” region of Heath, et al.3,4).

SUMMARY

The successful removal, without significant clinical residua, of an arteriovenous malformation in the head of the left caudate nucleus and left lateral ventricle is reported.

REFERENCES