VASCULAR MALFORMATION OF THE LEFT THALAMUS
REPORT OF A CASE OF SUCCESSFUL TREATMENT, WITH A NOTE ON
OCCLUSION OF THE LEFT INTERNAL CEREBRAL VEIN

BRUCE L. RALSTON, M.D.,* AND CHRISTOS A. PAPATHEODOROU, M.D.
Department of Neurological Surgery, The Mount Sinai Hospital, New York, New York
(Received for publication December 3, 1958)

Although arteriovenous malformations may occur anywhere within the central nervous system, they have only rarely been described within the thalamus. It is unusual that such a lesion may be so diagnosed and fortuitously located that it may be removed without severe residual deficit.

CASE REPORT

The patient,† a 41-year-old right-handed school-teacher and housewife, had had a subarachnoid hemorrhage without localizing signs at the age of 16 years. She was well until 5 days before her admission, at which time, while at home, she suddenly experienced severe headache, nausea and vomiting, and became comatose. Lumbar puncture revealed bloody spinal fluid. She was then hospitalized at a local hospital. Neurological examination disclosed a right hemiparesis and some difficulty with speech. She continued to improve until 4 days later when there was an increase in the severity of the symptoms and of the blood in the spinal fluid. She was then transferred to the Mount Sinai Hospital.

Examination. She was a noncommunicative, aphasic patient with right-sided weakness, including the face. Right hyperreflexia and Babinski’s sign were present. The fundi were normal. There was vertical nystagmus, with paralysis of upward gaze. An electroencephalogram showed severe diffuse slowing, accentuated in the left frontotemporal region. Her state of alertness improved but the signs persisted. It was now possible to ascertain that there was no apparent sensory disturbance within the limits of testing and that visual fields were full to confrontation.

Thirteen days after admission, a left carotid angiogram was carried out. It showed, in the anteroposterior projection (Fig. 1), a small vascular malformation lying lateral and slightly dorsal to the internal cerebral vein, to which it shunted. The latter structure was not shifted across the midline. The malformation seemed to derive most of its blood supply from ganglionic vessels arising inferiorly. The lateral angiogram (Figs. 2 and 3) also showed the malformation and its shunt to the dilated internal cerebral vein. The latter structure was displaced upwards, indicating a mass in the thalamic region. The anomaly could be seen to project superiority to the striatothalamic vein and it was judged that at least part of it might be in proximity to the left lateral ventricle. For this reason a pneumoencephalogram was carried out. The anteroposterior erect films disclosed marked elevation of the thalamic shadow (Fig. 4), with some irregularity of its roof, possibly indicative of the malformation. The lateral view (Fig. 5) showed a marked general increase of the left thalamic outline with local accentuation anterodorsally, in the area under suspicion. There was no filling of the posterior part of the third ventricle.

The impression was that this was an arteriovenous anomaly of the left thalamus which was superficial and had ruptured into the thalamus and the left lateral ventricle. It was felt that the lesion might be safely approached through the hydrocephalic ventricle, and removed, because of its small size.

Operation. Under hypothermia and hypotension, 6 weeks after admission, the left su-

* Present address: 55 East 92nd St., New York 28, N.Y.
† From the service of Dr. Sidney Gross.
Fig. 1. Preoperative angiogram. Anteroposterior projection. Note small vascular malformation located lateral and dorsal to internal cerebral vein, which fills in arterial phase.

perior intermediate frontal and central regions were exposed. There was no evidence of subarachnoid bleeding. A core of cerebral tissue, 3 cm. in size, was resected in the region of the middle frontal gyrus, just anterior to the motor strip. The dilated left lateral ventricle was opened. The normal-appearing choroid plexus could be seen rising backwards over the thalamus. The septal veins were 2-3 mm. in diameter and similar large veins ran from the striothalamic sulcus through the velum interpositum. These were not arterialized. On the surface of the thalamus, about 1.5 cm. behind the foramen of Monro, hairpin-like loops of arterial vessels, extending at right angles from the thalamic surface for about 2 mm., were seen. These were coagulated with bipolar forceps to a depth of about 0.5 cm. into the thalamic substance. Bleeding was easily controlled. The large crossing veins were also coagulated to make exposure possible.

Postoperative course was uneventful. The organic mental syndrome cleared completely. The right hemiparesis improved rapidly. The weakness of upward gaze persisted. The difficulty with speech resolved somewhat more slowly. At the time of discharge, 4 weeks after operation, the only abnormal findings were limitation of upward gaze and slight difficulty in naming objects. Over the subsequent year, there has been further improvement in speech.

A left carotid angiogram was performed just prior to discharge (Figs. 6 and 7). It showed elimination of the vascular malformation and its associated arteriovenous shunt. Of considerable interest (Fig. 8) was the finding that the left internal cerebral vein, dilated prior to surgery (Fig. 3), now no longer filled, although the great vein of Galen and the vein of Rosenthal were well seen.
Fig. 2. Preoperative angiogram. Lateral projection. Early arterial phase. Malformation visualized just above sylvian group of vessels. Internal cerebral vein can be made out faintly.

Fig. 3. Preoperative angiogram. Lateral projection. Late arterial phase. Malformation still faintly visible. Dilated internal cerebral vein shunting to straight sinus. Internal cerebral vein is displaced dorsally.
Fig. 4. (left). Preoperative pneumoencephalogram. Anteroposterior, erect. Marked elevation of left thalamic shadow with some irregular serrations on its surface.

Fig. 5 (right). Preoperative pneumoencephalogram. Lateral projection. Left lateral ventricle. Considerable increase in thalamic size, especially anterodorsally.

Fig. 6. Postoperative angiogram. Anteroposterior projection. Absence of vascular malformation and venous shunt.
Fig. 7. Postoperative angiogram. Lateral projection. Absence of vascular malformation and venous shunt.

Fig. 8. Postoperative angiogram. Lateral projection. Venous phase. Complete absence of filling of left internal cerebral vein with good filling of great vein of Galen and vein of Rosenthal.
DISCUSSION

This case is presented to show that a vascular malformation located in an apparently inaccessible area of the brain may be rendered operable by a combination of circumstances: in this instance its small size, proximity to the lateral ventricle, and the presence of hydrocephalus.

The occlusion of the left internal cerebral vein is attributed to propagated thrombosis from the site of coagulation of the striatothalamic vein. The fact that this can occur in the dominant hemisphere without overt clinical manifestations, is of considerable interest.

SUMMARY

A case is reported of successful elimination of an arteriovenous malformation of the thalamus of the dominant hemisphere. The fact that occlusion of the left internal cerebral vein may occur without significant clinical residua, is discussed.