ANATOMICAL STUDY OF AN ARTERIOVENOUS MALFORMATION DRAINED BY THE SYSTEM OF GALEN

J. GAGNON, M.D., AND G. BOILEAU, M.D.

Department of Pathology, Ste Justine Hospital, Montreal, Canada

(Received for publication June 23, 1958)

Arteriovenous malformations in the region of the pineal gland reported in the literature are rare. French and Peyton in 1954 reviewed the literature and added 5 personal cases to 8 others reported previously. It seems that the first case of arteriovenous malformation drained by the great vein of Galen and accompanied by internal hydrocephalus was reported by Jaeger et al. in 1937. From 1940 until 1953, cases were reported by Russell and Nevin, Alpers and Forster, Oscherwitz and Davidoff, Boldrey and Miller, Olivecrona and Riives, and Gillingham. In 1955, Rosenberg published an autopsied case. In 1956, a clinical and therapeutic study of 2 cases was made by Nayrac et al.

Thus far, we know of 16 cases and it is believed that this anatomical study might be useful in the understanding of the anomaly, since most of the authors have stressed the clinical, angiographic or surgical aspect of the question.

MATERIAL AND METHODS

As a routine procedure at Ste Justine Hospital all the brains of children are injected arterially at autopsy so they can be worked on more easily. This is achieved through the right internal carotid artery and the right vertebral artery. The brain is first washed with an isotonic solution containing gum acacia, sodium chloride and distilled water. Then it is fixed with the following isotonic solution: gum acacia, cobalt sulfate, formaldehyde, calcium chloride and distilled water. About 500 cc. of each solution are used (Koenig et al. method slightly modified).

The brain was first cut in four frontal sections and it was only after these slices were made that the huge pouch was discovered. In order to obtain antero-posterior sections, all the pieces were included in a gelatine block (12 gm. of gelatine mixed with a 1 per cent solution of phenol), then the block was hardened and preserved in a 10 per cent formaldehyde solution at 5°C.

It is interesting to note that the arteries, and the arteries only, appeared thrombosed; an artefact that has permitted an easy recognition of the calibers and course of these vessels.

REPORT OF A CASE

Ste Justine Hospital, No. 403919. The patient was a 2-day-old white male, first born, full term and delivered by forceps, who was admitted on Nov. 19, 1957. Cyanosis, noted since birth, increased gradually until death, accompanied by respiratory difficulties.
Examination at the time of admission revealed normal temperature, cyanosis and tachypnea. The weight was 3.5 kg. Moro's reflex, and sucking and grasping reflexes were absent. The fontanelle was slightly tense although the child was dehydrated.

Three hours after admission, the respiratory rate was more rapid and tirage was noted. A clinical diagnosis of anoxia neonatorum and cerebral hemorrhage was made. Despite treatment with oxygen, vitamin K and respiratory stimulants, the condition progressed rapidly downhill. Exitus occurred 10 hours after admission.

Autopsy (No. 236-57) was performed 17 hours post mortem. Meningeal hemorrhage, a huge ductus arteriosus, dilatation of the right cardiac ventricle, and hepatic and pulmonary congestion were found.

A few weeks later the fixed brain was examined. Its weight was 410 gm. General congestion was noted together with subarachnoid suffusions on the left hemisphere. All the arteries were thrombosed and a huge arteriovenous aneurysm in the great cerebral vein of Galen and the arteries originating from the circle of Willis was found.

ANATOMICAL STUDY OF THE ARTERIOVENOUS MALFORMATION

The anatomical findings that are discussed here consist essentially in an intricate arterial network encircling the cerebral peduncles. This arterial network is drained in an enormous venous sack. Such anomaly concerns the posterior cerebral and the anterior cerebellar arteries. After a capricious and asymmetric course around the cerebral peduncles the vessels become anastomosed behind the middle line at the level of the lamina quadrigemina. They form there a network from which arise nine vessels which bring the arterial blood into the venous sack. The latter is the anterior continuation of the straight sinus (Fig. 1).
Venous Sack (Fig. 2). On sagittal medial section, the straight sinus is very evident and enormously dilated. It is continued forwards by a pouch, the anterior extremity of which reaches the anterior extremity of the corpus callosum. Thus is formed a venous channel extended posteroanteriorly and inferosuperiorly from the confluens sinuum to a frontal plane at the level of the optic chiasm.

This venous sack measures 6 cm. in length and 1 ½ cm. in diameter, and is situated slightly to the right of the medial sagittal plane. It is under the corpus callosum and posteroanteriorly overrides successively: the tentorium cerebelli, the cerebral peduncles, the pineal body and the habenulae, the tela choroidea and the choroid plexuses of the third ventricle. Its round anterior extremity begins approximately at the level of the interventricular foramen. Its superior aspect begins approximately at 1 cm. from the head of the corpus callosum and follows the concave line of the latter, which is pressed upwards and diminished in thickness up to 1 mm. The splenium of the corpus callosum bulges in slightly and constricts the venous sack. This sack is continued backwards by the straight sinus in the falx cerebri. This voluminous venous pouch shifts the subjacent structures forwards and downwards, especially the cerebral peduncles and the thalami. The third ventricle is greatly distended and the massa intermedia is reduced to a string, 1 mm. in diameter. The choroid plexuses of the third ventricle are not very apparent and are in direct continuation, through the interventricular foramen, with those of the lateral ventricles. This shift is so marked that the pineal body

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**Fig. 2.** Diagram of a medial sagittal section showing the right side. A of S. = aqueduct of Sylvius, C.C. = corpus callosum, C.S. = confluens sinuum, I.V. = interventricular foramen, M.I. = massa intermedia, O.C. = optic chiasm, P.B. = pineal body, S.C.C. = splenium of corpus callosum, S.O.R. = supra-optic recess, S.S. = straight sinus, IV V. = fourth ventricle.
is displaced forwards 1½ cm. from the splenium of the corpus callosum and
the lamina terminalis is at a frontal plane with the anterior extremity of the
corpus callosum. On the inferior internal surface of the venous pouch, be-

hind the cerebral peduncles at the level of the lamina quadrigemina, one can
see nine arterial openings which are spread over an area 1 cm. in width and

1 cm. in length. In a coronal section the lateral ventricles are moderately
dilated and the venous sack fills the cavity of the septum pellucidum. In
frontal section this cavity is roughly triangular, its base being superior and
slightly oblique so that the right superior angle is 1 cm. higher than the
left superior angle (Fig. 3). The dilatation of the lateral and third ventricles
is probably the result of the pressure exerted by the venous sack on the thin
dorsal wall of the aqueduct of Sylvius, although the latter is anatomically
patent.

Arterial Vessels (Fig. 4). The vessels that are connected with the venous
pouch at the level of the lamina quadrigemina have their origin in a vascular
ring which encircles the cerebral peduncles. These encircling vessels arise
mainly from the anterior cerebellar artery and the posterior cerebral artery,
although there are some anastomoses with the middle cerebral artery. The
arteries which form this vascular ring in the cisterna ambiens are, in medio-
lateral order, (1) the anterior cerebellar arteries; (2) the long circumferential
artery of the midbrain, and (3) the posterior medial choroidal arteries. All
these vessels from each side of the cerebral peduncle unite over the lamina
quadrigemina. They form there a network from which arise the small vessels
that supply the arterial blood to the venous pouch. In this network there
are many laterolateral anastomoses which close the posterior segment of the vascular ring. On the left side of the cerebral peduncles the three arteries, beside being anastomosed with those of the opposite side, become fused into a common vessel which is 4 mm. in diameter and irrigates the venous pouch directly. Consequently, it is felt that the circulation resulting from the high

caliber of this vessel is prevalent on the left side. This shift of the arterial circulation to the left is also caused by the fact that the left middle cerebral artery gives off a big branch which anastomoses with one of the vessels forming this great trunk of the left side. From this vascular ring, collateral arteries, some of which are the lateral choroidal arteries, radiate peripherally.

SURGICAL INDICATIONS

Among the reported cases, a few patients have been operated upon. But it seems that most of the arteriovenous aneurysms that are drained by the
system of Galen are beyond every therapeutic resource. This is well stated by Nayrac and collaborators in their clinical and therapeutical studies.

Technically speaking, these aneurysms are very difficult to reach. In the surgically treated patients, a clip was put on the afferent arteries, in one or more operative steps. French and Peyton have treated 4 patients quite successfully. Nayrac et al. have succeeded in two resections, although Leppo et al. have expressed the opinion that they were not actual arteriovenous aneurysms "englobing the deep system of the vein of Galen." In the same paper, it is stated that "the bilateral and midline a.v.a. seem to be the only inoperable."

If we consider our case and if we try to find a solution, we are left with the procedure of clipping nine vessels which are located on different frontal planes, near the midline. Moreover, it is believed that the pre-operative angiography in this case would have resulted in a much confused picture because of the small size of the brain and the multiple anastomoses.

Another procedure would have been to clip the basilar artery, leaving the irrigation of the brain to the anastomoses from the middle cerebral arteries, thus reducing the blood flow; but this procedure would be a very hazardous one.

Hence, in our case the lesion would seem to have been practically inoperable and would confirm Leppo’s statement.

REFERENCES


