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Vein of Galen vascular malformations in infants: clinical, radiological and therapeutic aspect

A. Borthne[2], M. Carteret[1], J. Baraton[1], J.-V. Courtel[1], F. Brunelle[1]

[1] Department of Pediatric Radiology, University Paris V, Hôpital Necker-Enfants-Malades, 149, rue de Sèvres, F-75743 Paris Cedex 15, France
[2] Department of Pediatric Radiology, Ullevå l University Hospital, N-0407 Oslo, Norway

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Abstract. A series of 14 vein of Galen vascular malformations diagnosed in the pediatric populations and treated at the Hospital for Sick Children-Necker, Paris, between 1988 and 1994 is presented. Five of the patients were diagnosed in the neonatal period, of whom 4 presented with life-threatening, intractable cardiac decompensation and high-flow arteriovenous fistulae. Embolization was performed on vital indications in 4 patients during the first week after birth. One embolization failed with fatal outcome. Of the 3 who were embolized, 2 succumbed within 1 week and 1 survived with marked improvement of cardiac symptoms. The older children presented with hydrocephalus and neurologic symptoms. The 10 patients older than 1 year were embolized. These procedures were successful in 90 %, with hemodynamic stabilization and improvement of clinical symptoms. In this group the mortality rate was 10 %. The total mortality rate was 29 %. Hydrocephalus was secondary to a compression of the Sylvian aqueduct in 44 % of cases. Five patients had ventricular drainage before embolization followed by a staged elective embolization. Transarterial embolizations were performed in 11 patients, whereas 2 patients were embolized via the transvenous route.

Key words: Vein of Galen – Embolization – Vascular malformation – Children

Introduction

Vein of Galen aneurysmal malformation (VGM) is a rare congenital intracranial vascular malformation predominantly diagnosed at the pediatric age (1 % of all and 30 % of pediatric vascular malformations) [1]. Depending on the degree of arteriovenous shunting, the condition is often associated with life-threatening congestive heart failure in the neonate. Later in childhood hydrocephalus may develop either as a result of Sylvius aqueduct compression or because of venous hypertension.

The main feeding arteries of the malformation are derivates of choroidal arteries before the mature vascularization of the brain fully established [2]. The arteriovenous fistula occurs in early embryogenesis between the choroidal plexus and the forerunner of the galenic vein, the median prosencephalic vein, which opens directly into the falcine sinus. The connections between the afferent arteries and the venous aneurysm may consist of either direct fistulae to the venous pouch (type I), indirect arteriolovenous fistulae through an intervening tangle of vessels (nidus) between the arteries and the vein (type II), or a mixture of direct arteriovenous and arteriolovenous fistulas (type III; Figs. 1, 2).

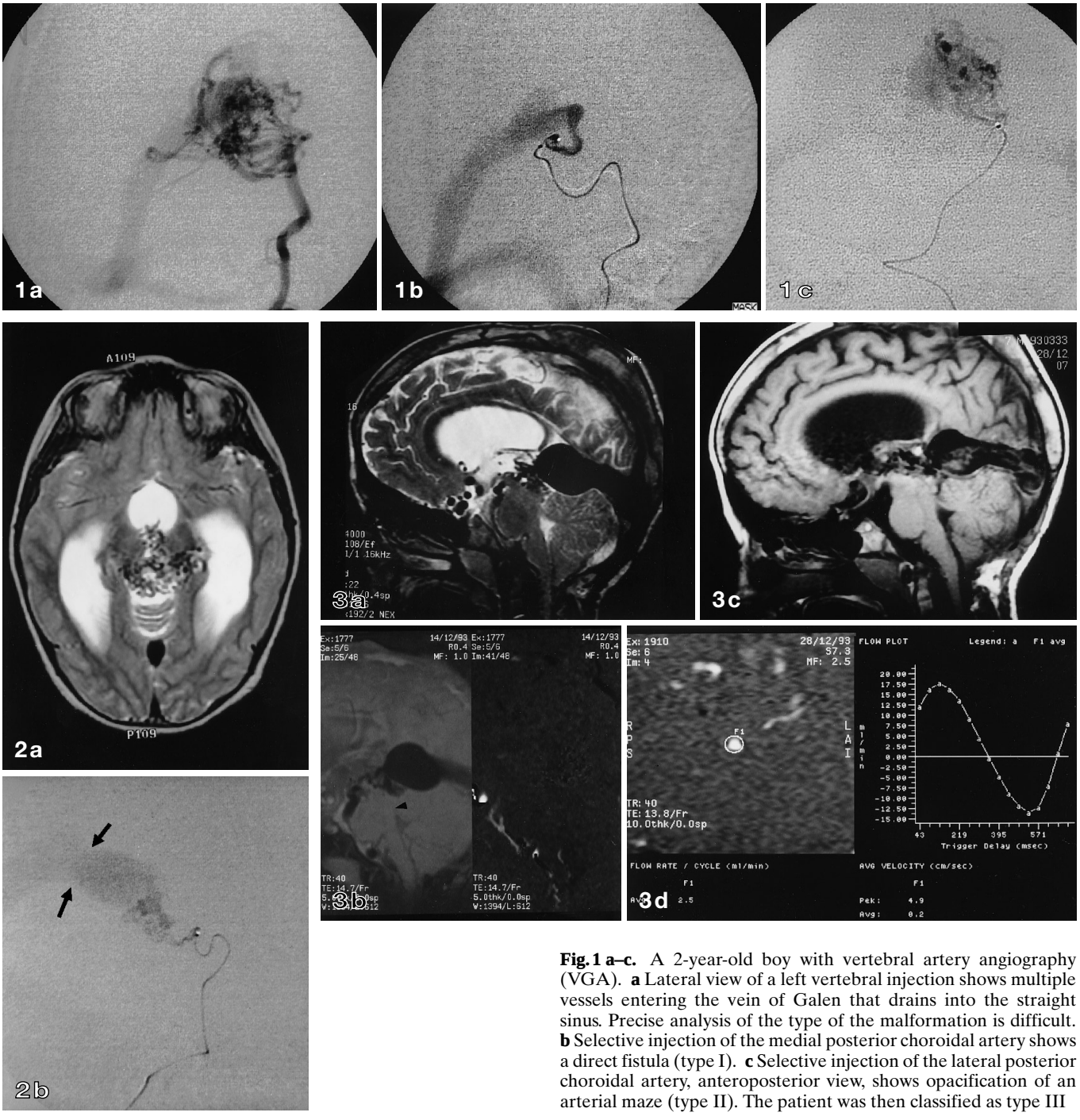
The neonates presenting with high-flow congestive heart failure have very high morbidity and mortality rates. The results of neurosurgery are disappointing and the condition is called the Gordian knot of cerebrovascular surgery. Interventional endovascular staged embolization either by the transarterial, transvenous, or transtorcular approach has improved the outcome and is regarded as the primary treatment. We present our series of 14 children treated between 1988 and 1994 to emphasize the relevance of modern endovascular therapy for the management of this condition.

Materials and methods

Fourteen consecutive children with vein of Galen malformations were treated at the Hospital for Sick Children, Necker, Paris, between 1988 and 1994. Four patients were referred to our institution from abroad which partly explains the short follow-up.

The clinical and radiographic files were reviewed. The age at diagnosis and the gender distribution are

Correspondence to: F. Brunelle



summarized in Table 1. Five of the children were neonates (35.7%), 5 were infants between 1 month and 1 year, and 4 (28.6%) were older than 1 year, ranging from 13 months to 5.5 years with a mean age of 3 years, 3 months.

The initial diagnosis was made with ultrasound, CT, or MRI. The diagnosis was made prenatally (38 weeks) in 1 patient. Twelve of the children (86%) were examined either with CT (9 patients) or MR scans (7 patients), and 4 were examined with both CT and MR before angiography. Two children were studied with cine phase contrast MRI (cine PCF) to assess quantitatively the cerebrospinal fluid flow through the Sylvian aque-

Fig. 1 a–c. A 2-year-old boy with vertebral artery angiography (VGA). **a** Lateral view of a left vertebral injection shows multiple vessels entering the vein of Galen that drains into the straight sinus. Precise analysis of the type of the malformation is difficult. **b** Selective injection of the medial posterior choroidal artery shows a direct fistula (type I). **c** Selective injection of the lateral posterior choroidal artery, anteroposterior view, shows opacification of an arterial maze (type II). The patient was then classified as type III

Fig. 2 a, b. A 4-year-old girl with VGA and hydrocephalus. **a** Axial T2-weighted MRI shows hydrocephalus and multiple transmesencephalic vessels (type II). **b** Selective injection of one of the transmesencephalic vessels shows the arterial maze interposed between the artery and the drainaing falcine sinus (lateral view; *arrows*)

Fig. 3 a–d. A 3-year-old boy with VGA and hydrocephalus. **a** Sagittal T2-weighted MRI shows compression of the tectum and Sylvius aqueduct. **b** Sagittal phase contrast image shows absence of flow in the aqueduct *(arrowhead)*. **c** After partial embolization, vein of Galen is smaller and a thin lumen is seen in the aqueduct of Sylvius. **d** Axial phase contrast image shows a normal systolodiastolic pattern of cerebrospinal (CSF) in the aqueduct

Table 1. Age and gender distribution

Age group	*N*	Gender		%
		M	F	
0–1 month	5	5	–	35.7
> 1 month–1 year	5	3	2	35.7
> 1 year	4	2	2	28.6
Total	14	10	4	

Table 2. Age of onset and dominant symptomatology. CHF congestive heart failure

Dominant symptom	Age at presentation		
	0–1 month	> 1 month–1 year	> 1 year
Systemic CHF	5	1	–
Macrocephaly/ Hydrocephalus	–	4	3
Seizures	–	–	1
Total	5	5	4

Table 3. Age at presentation

Type of malformation	*N*
Type I (Houdart a)	2
Type II (Houdart b)	8
Type III (a + b)	4

duct. One patient was studied pre- and post embolization (Fig. 3). Nine patients presented with hydrocephalus and 5 of them where shunted with a ventriculoperitoneal shunt before embolization.

All patients underwent diagnostic angiography, and all were embolized, except 1 patient in whom embolization failed. All the angiograms were done by the transfemoral approach. A 3- or 4-F femoral sheath catheter and a 3-F flow-guided Balt set was used for the endovascular procedures. Low osmolar contrast medium (Hexabrix 320, Guerbet, Aulnay-sous-Bois, France) was used in all cases both for screening and for superselective studies of the cerebral vessels. Each malformation was detailed according to the Yasargil classification with the modifications according to Houdart: Type I is a direct arterial fistula to the vein of Galen. Type II is made of multiple arteriolovenous fistulae with a plexiform tangle of more than three feeding arteries reaching the single draining venous pouch. Type III is a mixture of types I and II, where enlarged direct afferents are seen together with the arterial plexiform feeding arteries. Two of the embolizations were performed via the transvenous route, and the others were transarterial. Systemic heparinization was achieved with an intravenous bolus injection of 50 UI/kg of heparin, followed by an intravenous bolus injection of 50 UI/kg of heparin every 2 h. Acrylic glue (N-butyl cyanoacrylate and isobutyl-2-cyanoacrylate) mixed with lipiodol was utilized in 11 of 13 (85%) transarterial embolizations, whereas nylon filaments and metalic coils were used in the transvenous embolizations. Staged embolizations were performed in 7 patients. A total of 46 vessels were embolized implying a mean of four feeding arteries per patient. One patient had stereotactic radiosurgery on the remaining nidus.

Clinical presentation

The clinical presentations are listed in Tables 2 and 4.

Neonates

Congestive heart failure (CHF) was the dominant symptom in the neonates. Three neonates were severely ill with rapidly intractable CHF, precordial murmur, and need of ventilatory support. One neonate had marked hydrocephalus and one had minor cardiac symptoms.

Infants

Hydrocephalus was the major presenting feature in infants between the age of 1 month and 1 year (80%). The single infant without hydrocephalus had moderate cardiac failure responding to medical treatment. Three infants of this age group had cardiac failure, 3 had scalp bruit, and 3 had dilated facial veins. Some degree of cerebral atrophy was diagnosed in 3 and 1 had minor developmental delay.

Children

All the children older than 1 year had hydrocephalus and none had cardiac failure. Two children presented with dilated facial veins, 2 had visual disturbances, 1 had epileptic seizures, and 1 had retarded development. Cerebral atrophy was diagnosed in 1 child and subcortical calcifications in 1 child.

Hydrocephalus

The hydrocephalus was explained by a direct mechanical compression of the cerebral aqueduct or posterior part of the third ventricle in 4 of 9 (44%) of the cases judged from MR examinations. In 2 patients the compression of the aqueduct was confirmed by cine PCF which demonstrated that no flow or minimal flow of cerebrospinal fluid (CSF) was present. In 1 patient examined before and after treatment, reestablishment of normal CSF flow after embolization was demonstrated (Fig. 3). Ventriculoperitoneal shunts were implanted in 5 patients before embolization. In 1 child with major hydrocephalus and neurologic disturbances the surgical procedure was complicated by bilateral subdural hematomas, shunt malfunction, and infection.

Table 4. Classification of vein of Galen malformations in our series

N	Age (years)	Clinical findings	Angiographic type
1	1 day (premature 30 weeks)	CHF, oxygen-/ventilatory support	Direct fistulae (anterior cerebral, posterior choroidal) to VG (diameter 2.5 cm); vascular steal; type I
2	1 day	CHF, oxygen-/ventilatory support; dilated facial veins; scalp bruit; seizures	Mixture of direct (anterior cerebral) and indirect fistulae (posterior choroidal and transmesencephalic) to VG (diameter 2.5 cm); vascular steal; type III
3	1 day	CHF, oxygen-/ventilatory support; scalp bruit; neonatal hemorrhage	Mixture of direct (anterior cerebral) and indirect fistulae (posterior choroidal predominantly left side) to VG; vascular steal; type III
4	3 years	Seizures, macrocephaly, subcortical calcifications; dilated facial veins	Indirect fistulae (posterior and anterior choroidal) to VG (diameter 4 cm); obstruction of the cerebral aqueduct; type II
5	Prenatal (36 weeks)	CHF, minor symptoms	Indirect fistulae (anterior cerebral, posterior choroidal) to VG (diameter 1 cm); type II
6	4 years	Macrocephaly/hydrocephalus; dilated facial veins	Mixture of direct (anterior cerebral) and indirect fistulae (posterior choroidal) to VG (diameter 3.5 cm); straight sinus obstruction; obstruction of the cerebral aqueduct; Type III
7	6 months	CHF; scalp bruit; hydrocephalus; atrophy; seizures	Indirect fistulae (anterior cerebral, posterior choroidal and transmesencephalic to VG (diameter 3 cm); type II
8	7 months	Macrocephaly/hydrocephalus; atrophy	Indirect fistulae (anterior and posterior choroidal) to VG (diameter 2.5 cm); type II
9	4 days CHF	Hydrocephalus	Indirect fistulae (thalamic perforants and posterior choroidal) to VG (diameter 2 cm); type II
10	13 months	Macrocephaly/hydrocephalus; atrophy	Indirect fistulae (posterior and anterior choroidal) to VG (diameter 2.5 cm); obstruction of the posterior part of the third ventricle; type II
11	6 months	Macrocephaly/hydrocephalus; scalp bruit	Direct fistulae (posterior choroidal) to VG (diameter 2 cm); straight sinus obstruction; type I
12	7 months	Macrocephaly/hydrocephalus; atrophy	Indirect fistulae (posterior and anterior choroidal, transmesencephalic) to VG (diameter 2.5 cm); type II
13	9 months	Hydrocephalus; dilated facial veins	Indirect fistulae (posterior choroidal to VG (diameter 2.8 cm); type II
14	5 years	Hydrocephalus; nystagmus; strabism; papillary oedema	Mixture of direct (posterior choroidal, right side) and indirect fistulae (posterior choroidal and transmesencephalic) to VG (diameter 3 cm); obstruction of the posterior part of the third ventricle; type III

Vascular anatomy

The angio-architecture is presented in Tables 3 and 4. The posterior choroidal arteries, both the medial and lateral branches, were involved in all patients. Supply from the pericallosal arteries of the anterior cerebral arteries occurred in 9 patients (64 %), from transmesencephalic arteries in 5 (36 %), anterior choroidal arteries in 2 (14 %), and thalamostriate arteries in 1 (7 %). Cerebral vascular steal, judged from angiograms, was seen in 3 patients (21 %) with CHF. The diameters of the vein of Galen aneurysms, measured by the CT and MR scans, varied between 1 and 4 cm with a mean diameter of 2.6 cm. The straight sinus was absent and the falcine sinus present in 11 of 14 (79 %) cases. Venous hypertension, documented either by narrowing of the draining veins or sinuses, venous dilatation, or indirectly by collateral venous drainage were present in 7 patients (50 %).

Treatment and results

Embolizations were performed within the first week of life in 4 neonates who all suffered from life-threatening CHF. One embolization failed because of technical reasons and the patient died. Two patients were treated transarterially. Both succumbed shortly after the procedure despite embolizations: Approximately 25–30 % of the arterial feeders were occluded in 1 patient and the main fistula was occluded in the other; one of the two died from pulmonary embolism. One child who was embolized transvenously survived with improvement of the cardiac- and hemodynamic state. The mortality rate in this group is 75 %.

Ten patients were embolized electively after the age of 4 months (Fig. 4). One patient died: a 6-month-old presenting with moderate cardiac failure, marked hydrocephalus, cerebral atrophy, and developmental delay. The type-II malformation nourished by the anterior and posterior choroidal arteries was treated with transvenous embolization of the 3-cm Galenic venous pouch. However, the nylon filaments were insufficient to create thrombosis of the aneurysm, and the patient succumbed 1 week after embolization from cardiac insufficiency. The mortality rate of the patients treated after 1 year of age is 10 %. The total mortality rate is 29 %.

Discussion

The clinical presentation of the vein of Galen malformations is frequently more dramatic in neonates, with a natural history of intractable CHF leading to death in

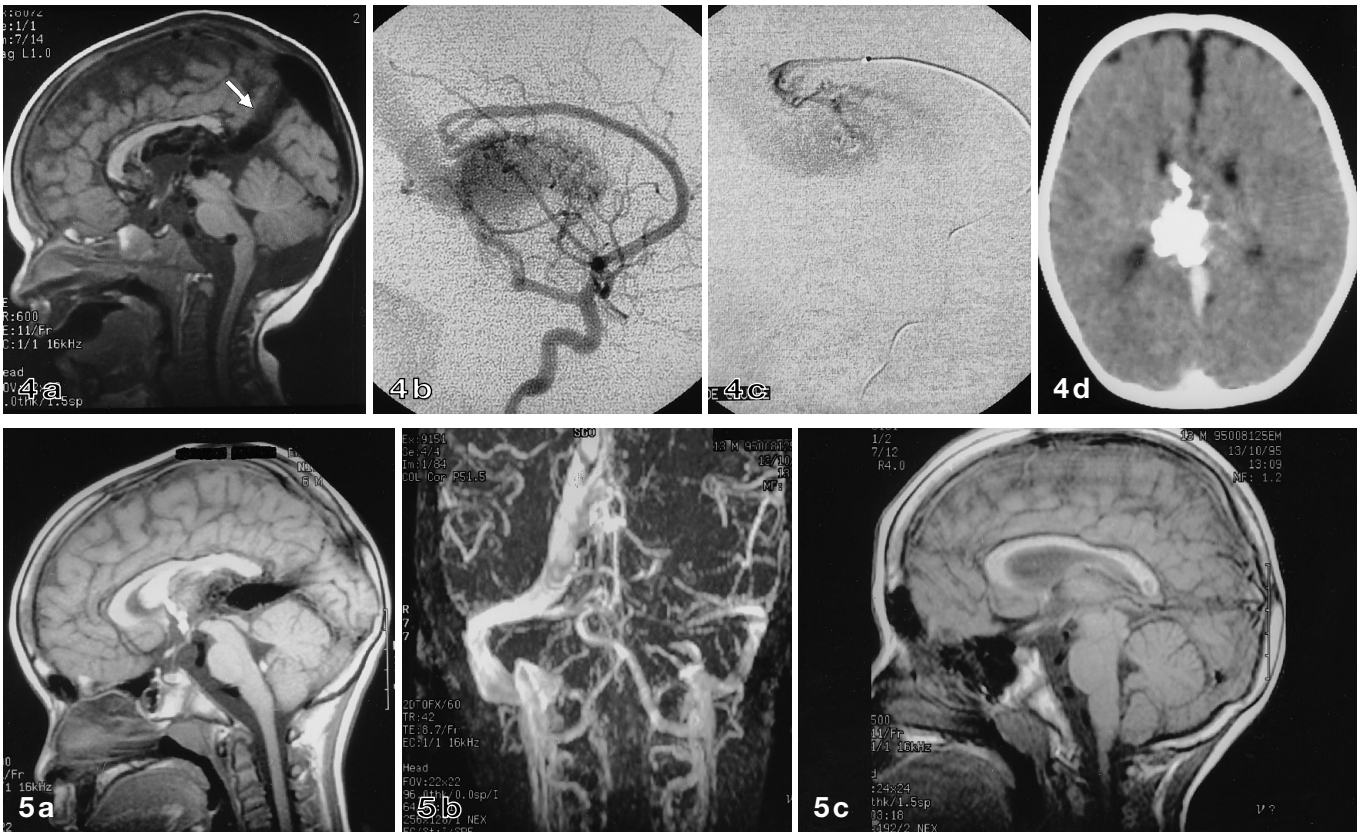


Fig. 4 a–d. A 6-month-old baby boy with an antenatal diagnosis of VGA. No cardiac insufficiency at birth. **a** Sagittal T1-weighted MRI shows multiple arterial and vascular structures in the region of the third ventricle. The straight sinus is absent. The vein of Galen drains into a falcine sinus *(arrows)*. **b** Sagittal view of a right carotid artery injection. The malformation is fed by the anterior cerebral artery *(ACA)* and by some posterior choroidal arteries. **c** Selective catheterization and embolization of the distal portion of the ACA; selective injection shows type-II malformation. **d** Same patient. Control CT scan after embolization. The radiopaque glue is visible in the region of the VGA

Fig. 5 a–c. A 7-year-old boy with intracranial hemorrhage and VGA. **a** Sagittal T1-weighted MRI shows hemorrhage in the region of the roof of the third ventricle. Note presence of a clot in the fourth ventricle. The arteriovenous malformation drains into a falcine sinus. **b** Magnetic resonance angiography shows occlusion of (anteroposterior view) the left lateral sinus. **c** Same patient 1 year after embolization and stereotactic radiosurgery. No more arteriovenous malformation is seen. Patient has no sequellae

untreated patients. The symptoms appear shortly after birth, due to the redistribution of the blood volume. The systemic vascular resistances increase abruptly when the placental circulation is interrupted and the infant enters a colder environment [3]. However, the vascular resistance of the cerebral malformation remains low. Up to 50–60% of the cardiac output may pass through the malformation in severe cases. Although the symptoms rapidly escalate from the first day of life, it has been shown by color-flow imaging and Doppler US that the shunting may be important, even antenatally [4–6]. Serious cerebral ischemic damage may already have evolved. A complete evaluation of the brain with CT or MRI is therefore imperative before selecting the patients for endovascular treatment [6–8]. Because of the poor outcome of these patients, embolization has been considered contraindicated in neonates with demonstrated cerebral tissue damage or uncontrollable systemic failure [6, 7]. However, the recently published results of endovascular treatment of neonates with intractable CHF encourage a more positive and aggressive treatment [1, 2, 9, 10]. Several questions remain unanswered: Who, how, and when should be treated? The outcome of surgical treatment is very bad [11]. This is explained by the complexity of the lesion. Despite encouraging results of microsurgical treatment of the malformation in older children [12], direct surgery is regarded to be inferior to endovascular embolization, and it seems unlikely that improved surgical technique alone will alter the outcome substantially [11].

When treatment has been decided upon, one has to determine what kind of strategy to choose, depending on the angio-architecture: (a) endarterial embolization with acrylic glue, detachable balloons, coils, or solid particles; or (b) endovenous or transtorcular embolization with coils or detachable balloons [13]. In some instances combined transarterial and transvenous strategy is preferred. All the superselective embolizations in our study, except two, were performed transarterially, with 3-F Balt microcatheters. One procedure failed, as it was impossible to advance the tip of the catheter far enough into the arterial feeder close to the galenic vein. We preferred to embolize with acrylic material (isobutyl-2-cyanoacrylate and later N-butyl cyanoacrylate). The newer polymers have improved characteristics and

decreased risk of complications such as catheter gluing and pulmonary embolism [10]. The use of large PVC particles (750–1000 m) is potentially dangerous in cases of high-flow direct fistulae because of increased risk of embolic complications [13]. The classification by Yasargil is useful to select the type of embolic material to be used [14].

Of the 5 neonates in our study, 4 presented with intractable cardiac insufficiency due to major cerebral arteriovenous shunting with large fistulae. The 4 patients with CHF, of whom 3 were sent to our hospital from abroad, were treated within the first week of life. One embolization failed after two consecutive attempts and this patient died 1 week later. Of the 3 patients who were embolized, 2 were treated endarterially and 1 transvenously with coils. Both patients with endarterial embolizations died within a few days after treatment despite apparently successful occlusion of the major fistulas. One of the patients died from disseminated pulmonary embolism. The one who was embolized with coils did well with improvement of cardiac symptoms and partial thrombosis of the Galenic vein. We experienced, in other words, a mortality rate of 2 of 3 (66%) of those who were embolized during the neonatal period. Including the patient whom we failed to embolize, the mortality rises to 3 of 4 (75%). These unpromising figures illustrate the difficulties of treating these very sick neonates, and also the complexity of these malformations. It also puts focus on the necessity of a complete clinicoradiologic examination before selecting the patients who are candidates for embolization. However, although it is difficult to compare the results of different series, other series present encouraging results of neonatal endovascular treatment. Lylak et al. [9] managed to successfully control the severe CHF by emergency embolization in 8 of 11 neonates (73%). Long-term follow-up revealed good outcome in 5 (45%), neurologic deterioration in 2 patients, and death of 1 at 13 months of age related to cerebral progressive occlusive venopathy [9]. Ciricillo et al. [4] treated 8 neonates with severe, high-output CHF by combined transarterial and transvenous embolic therapy. Six patients survived, but brain infarctions occurred in 5 of 6 infants treated with solid particles [4]. Lasjaunias et al. [7] presented 9 neonates with CHF; 3 underwent embolization. One of these subsequently died from intracranial hemorrhage [7]. In a recent series of 11 patients with vertebral artery angiography (VGA) malformations, embolizations with acrylic glue were performed by Friedman et al. [10] in 5 neonates with CHF. All the patients survived, 3 of them with neurologic symptoms [10].

The prognosis is better in older children (Fig. 5) [1, 3, 6, 7, 9, 10, 16]. This reflects the less dramatic vascular shunting in the malformations of these children. The neurologic symptoms and hydrocephalus dominate in older children, whereas the cardiac insufficiancy often can be controlled by medical treatment. We embolized 10 patients after the age of 4 months. One child with marked hydrocephalus and cardiac failure died 1 week after transvenous treatment. Autopsy findings did confirm that the Galenic vein was not thrombosed. All the other children survived, with stabilization of the hemodynamic and clinical status. The mortality rate, 1 of 10 (10%), in this age group correlates well with other reports [6, 7, 16].

Even with successful primary treatment, there is no guarantee of good long-term outcome. A major reason for this is probably the persistently elevated cerebral venous pressure which may lead to a progressive occlusive venopathy with progressive ischemic brain damage, subcortical calcifications, and neurologic deterioration [6, 7, 9, 17]. It is possible to decrease the venous pressure by inducing thrombosis of the Galenic vein. This is one of the reasons why a transvenous occlusion is regarded by some authors as the rational treatment of these high-flow fistulous malformations [1, 17–19]. A rapid thrombosis may, however, lead to rupture of the weak subependymal veins and fatal cerebral hemorrhage due to the rapidly developing intravascular hypertension [9, 19]. To avoid this unwanted sudden effect, the flow should first be reduced by arterial embolization and the venous occlusion should be carried out subsequently [1, 17, 19].

The elevated venous pressure is regarded to be a cardinal trait of these malformations and the main cause for the development of hydrocephalus [7, 17, 20]. The venous hypertension is transmitted via the dural sinuses to the cortical and medullary veins of the brain with subsequent difficulty for the CSF to enter from the subarachnoid space into the venous compartment. According to this hypothesis the hydrocephalus is secondary to the increased intracranial CSF volume. Consequently, the ventricular drainage does not solve the fundamental problems produced by the venous hypertension. It is claimed that the drainage may in fact deteriorate the clinical and local status: The water stagnation in the white matter may become aggravated, and the venous aneurysm may enlarge due to the shrinkage of the ventricles [17].

The other hypothesis explains the development of the hydrocephalus by the compressive mass effect on the cerebral aqueduct [11]. Our MR findings show that the aqueduct or posterior part of the third ventricle were compressed in 4 of 9 (44%) of the patients with hydrocephalus. This was confirmed by cine PCF studies in 2 patients, which demonstrated that no or minimal flow of CSF was present. After embolization, restoration of normal flow was observed. When aqueductal stenosis is present, the ventricular drainage is a rational procedure in order to reduce the grave prognostic consequences of long-term hydrocephalus [21]. Five of the children in our series were drained before embolization. These procedures contributed to clinical stabilization of the patients and feasibility to perform a staged endovascular treatment.

Conclusion

The endovascular and transvenous embolization are the best therapeutic modalities for treating infants with vein of Galen malformations. Both the grave natural history of neonates with CHF and the bad results of neurosur-

gery in infancy favor this interventional radiologic procedure. Our experience of prompt embolization of the malformations on vital indications during the first week of life has been disappointing because of a high mortality rate. However, the results are very encouraging when treating children after the age of 4 months. In cases of marked hydrocephalus, the ventricular drainage has been efficient to permit a staged elective embolization later.

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