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SPECIAL ANNUAL ISSUE

Endovascular management of arteriovenous malformations and other intracranial arteriovenous shunts in neonates, infants, and children

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Abstract
Purpose We discuss the management of cerebral arteriovenous shunts in neonates, infants, and children, with emphasis on our experience with pediatric cerebral arteriovenous malformations (AVMs). The management of vein of Galen malformations is discussed in a separate chapter.
Methods An all-inclusive retrospective chart review of the endovascular surgery operative record database at the Hyman Newman Institute for Neurology and Neurosurgery at Roosevelt Hospital in NYC was conducted. All consecutive pediatric patients (newborn to 18 years of age) with intracranial arteriovenous shunts who presented from January 1, 2004 to June 16, 2009 were included.
Results A total of 151 consecutive pediatric patients with intracranial arteriovenous shunts were evaluated from the period of January 1, 2004 to June 16, 2009. This included 56 patients with vein of Galen malformations, 48 cerebral AVMs, 11 patients with pial arteriovenous fistulae, six patients with dural arteriovenous malformations, and 30 patients with mixed intracranial vascular malformations. Forty-four patients underwent a total of 163 endovascular embolizations. The complications rate for endovascular embolizations was 6.7% (11 in 163), 5.5% with temporary complications and 1.2% with permanent complications. The mortality rate for the group of patients (excluding patients with vein of Galen malformations) that underwent endovascular embolizations was 0.0%.
Conclusions Careful clinical observation and timely intervention are important in the management of pediatric patients with intracranial arteriovenous shunts. Trans-arterial endovascular embolization with liquid embolic agents is the treatment of choice for safe stabilization and/or improvement of symptoms in the group of pediatric patients with intracranial arteriovenous malformations.

Keywords Pediatric · Cerebral AVM · Pial arteriovenous malformation · Intracranial arteriovenous shunt

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Introduction

Cerebral arteriovenous shunts (AVS) in children differ from those in the adult population in their presentation, evolution, natural history, and angioarchitecture. It is for these reasons that their management has to be tailored to the specific type of lesions. Present adult-based classifications and arteriovenous malformation (AVM) grading are based in their size, location, and venous drainage [12]. This may forecast surgical outcome but is particularly inappropriate in children since the cerebral

Table 1 Subtypes of cerebral vascular lesions in children

Arteriovenous shunts	Vein of galen aneurysmal malformation
	Mural
	Choroidal
	Vein of Galen aneurysmal dilatation
	Dural
	Sinus malformation
	Dural shunt (isolated, multifocal or hereditary)
	PIAL AVM
	Fistula (isolated, multifocal or hereditary)
	Nidus (isolated, multifocal or hereditary)
Venous malformations	Cavernoma (isolated, multifocal or hereditary)
	Dysplasia
Proliferative diseases	Hemangiomas (isolated or multifocal)
	Proliferative angiopathy
	Moya moya

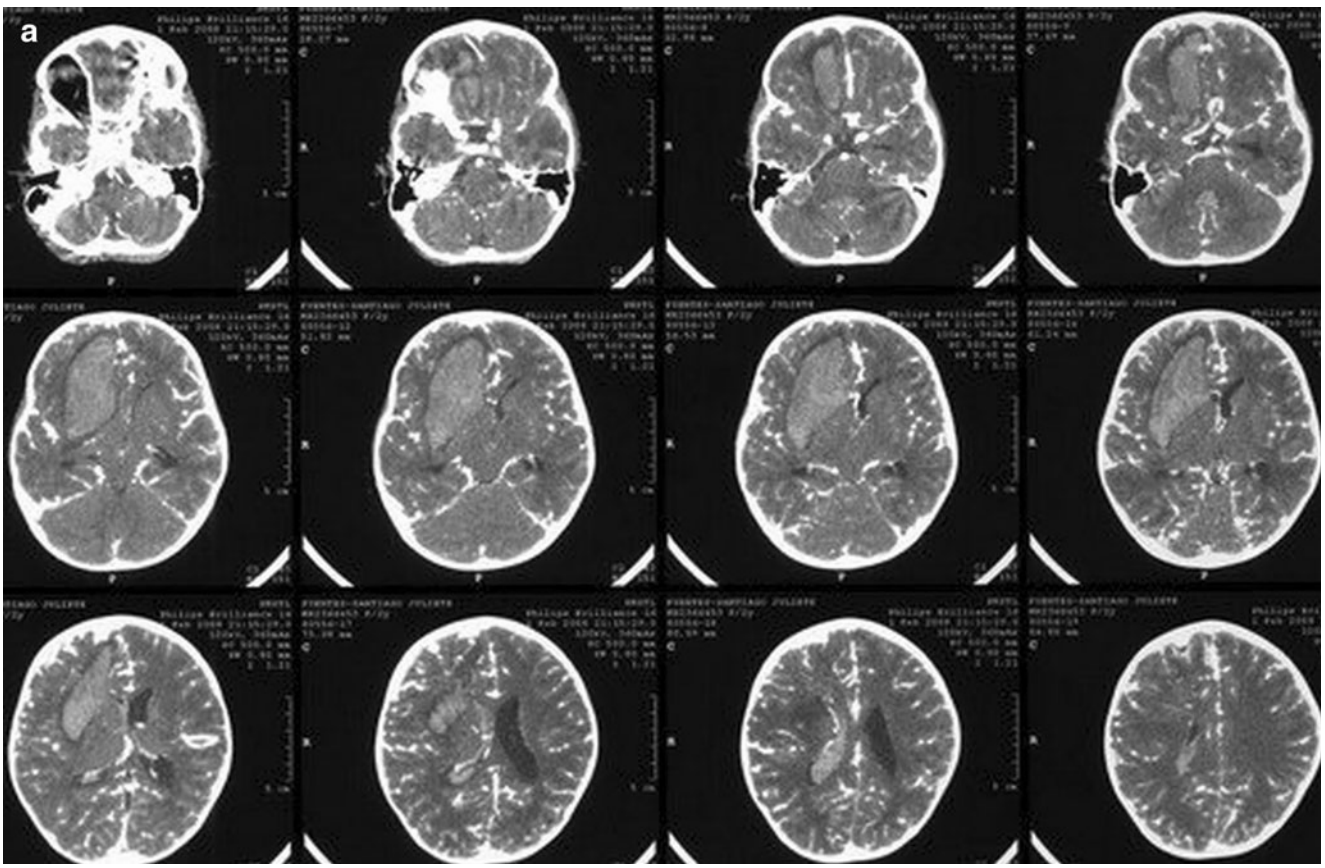

Fig. 1 The head CT with contrast of a 3-year-old girl who presented with a left hemiparesis and lethargy demonstrated an intraparenchymal hematoma in the right frontal lobe (**a**). The femoral cerebral angiogram in AP and lateral projections early arterial and late arterial phase of the right and left carotid arteries injections demonstrated the areas of hypervascularity in the cortical region of the right hemisphere (**b**, **c**, **d**, **e**, **f**, **g**, **h**, **i**). The areas with early venous drainage in the area of hemorrhage were catheterized super-selectively with flow-guided microcatheters. Super-selective endovascular embolizations with *n*-butyl-cyanoacrylate mixtures were performed (**j**, **k**, **l**, **m**, **n**, **o**, **p**, **q**). The patient has not suffered more hemorrhages but a follow-up femoral cerebral angiogram demonstrated progression of the hypervascularity in the right frontal cortex (**r**)

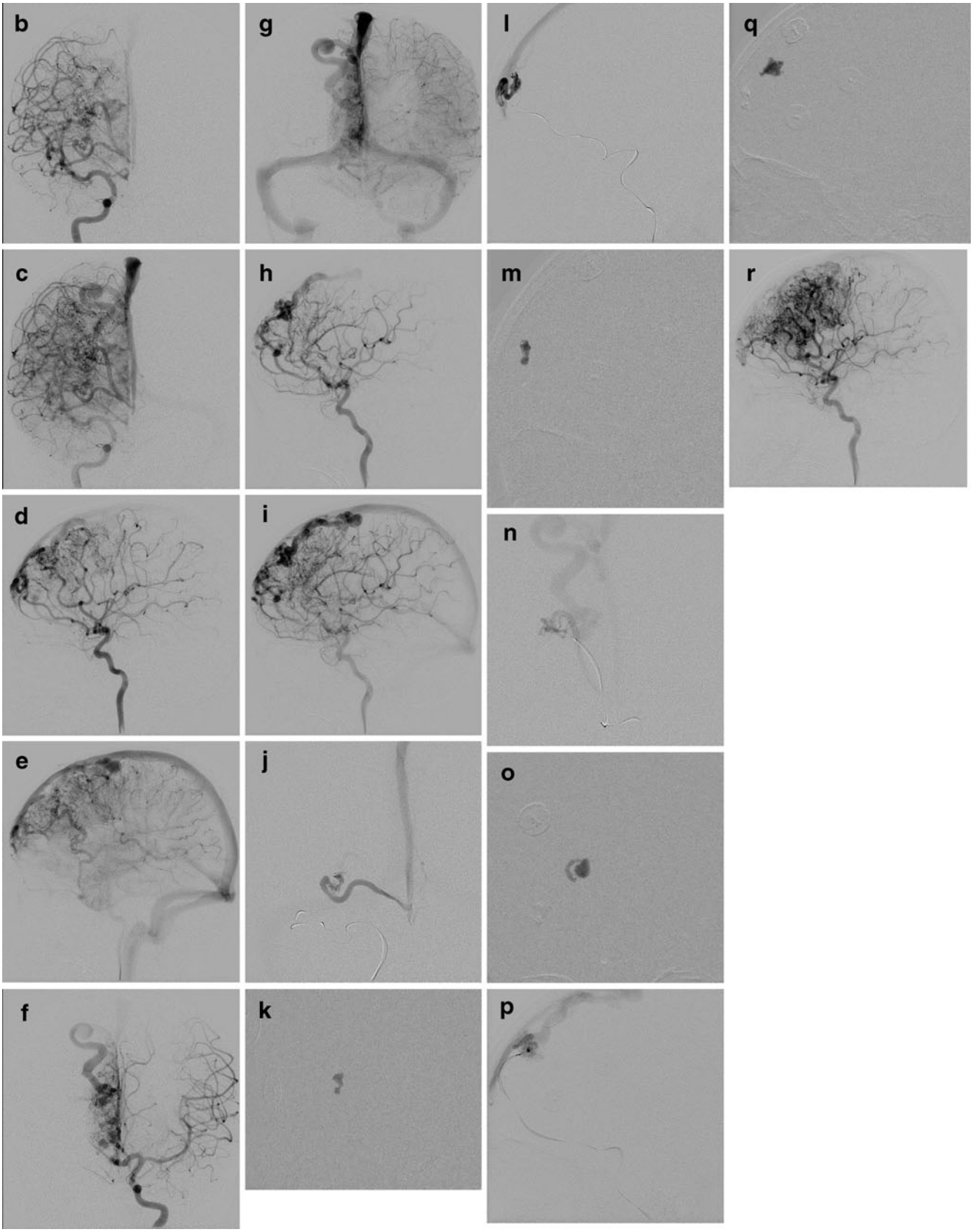

Fig. 1 (continued)

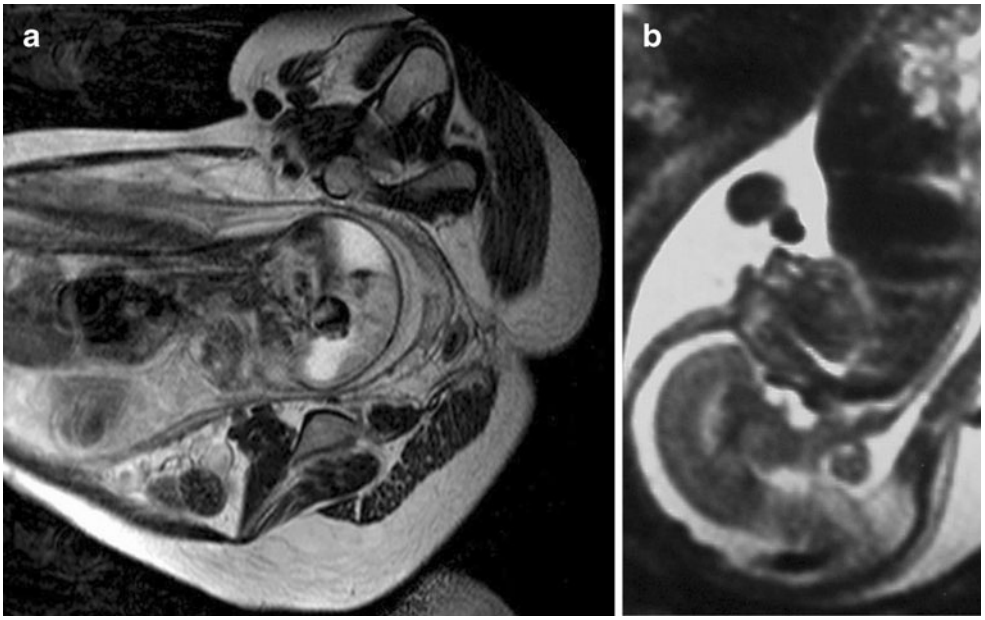

Fig. 2 Intrauterine MRI demonstrating a vein of Galen malformation (**a**) and a dural sinus malformation (**b**)

eloquence is difficult to assess, and changes as the infant or child grows. The timing of intervention and the type of intervention are also different in vascular malformations with different types of arteriovenous shunts. Most lesions have fistulous components, and are frequently multifocal, venous drainage usually affects the entire venous system and is commonly associated with venous anomalies, non-development, or venous/sinus thrombosis; and the potential for recovery and eventually remodeling is different [4]. Although rare, there are few reports, with relatively few cases that demonstrated a better outcome in surgically accessible cerebral AVMs in children (32 patients) when compared to the adult cohort [9].

Anatomic and physiologic characteristics of the neonatal and infant brain and the immaturity of the systemic flexibility (hydrovenous system) create specific symptoms and therapeutic challenges. This type of lesions can be lethal and cause irreversible neurological pathology, whereas a similar lesion in an adult might be asymptomatic.

The vascular lesions in children can be divided in arteriovenous shunts, proliferative diseases, and venous malformations. The arteriovenous shunts can be divided in vein of Galen aneurysmal malformation, dural shunts, and pial arteriovenous shunts (Table 1) [7]. Vein of Galen malformations can be divided into those that are a malformation of the precursor of the vein of Galen, the

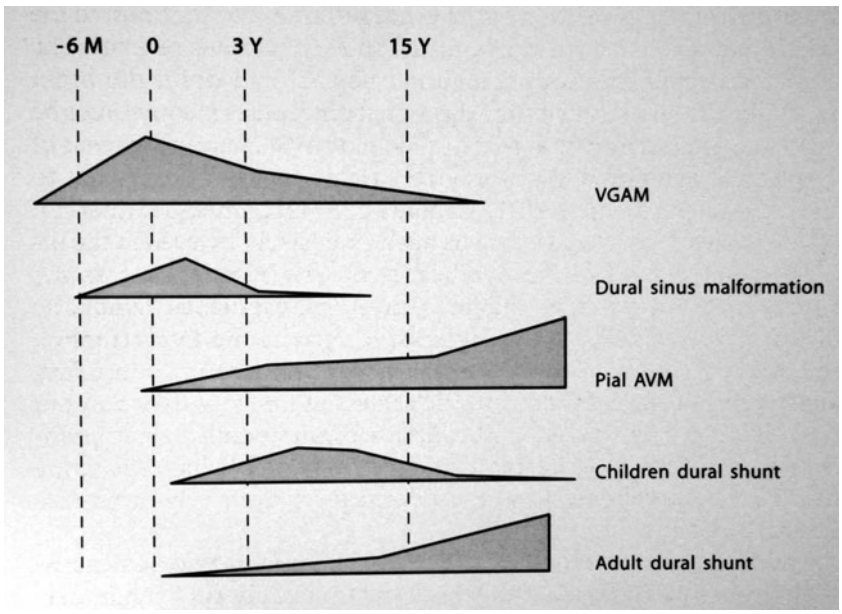

Fig. 3 Age of onset of pediatric intracranial AVS. With permission from [4]

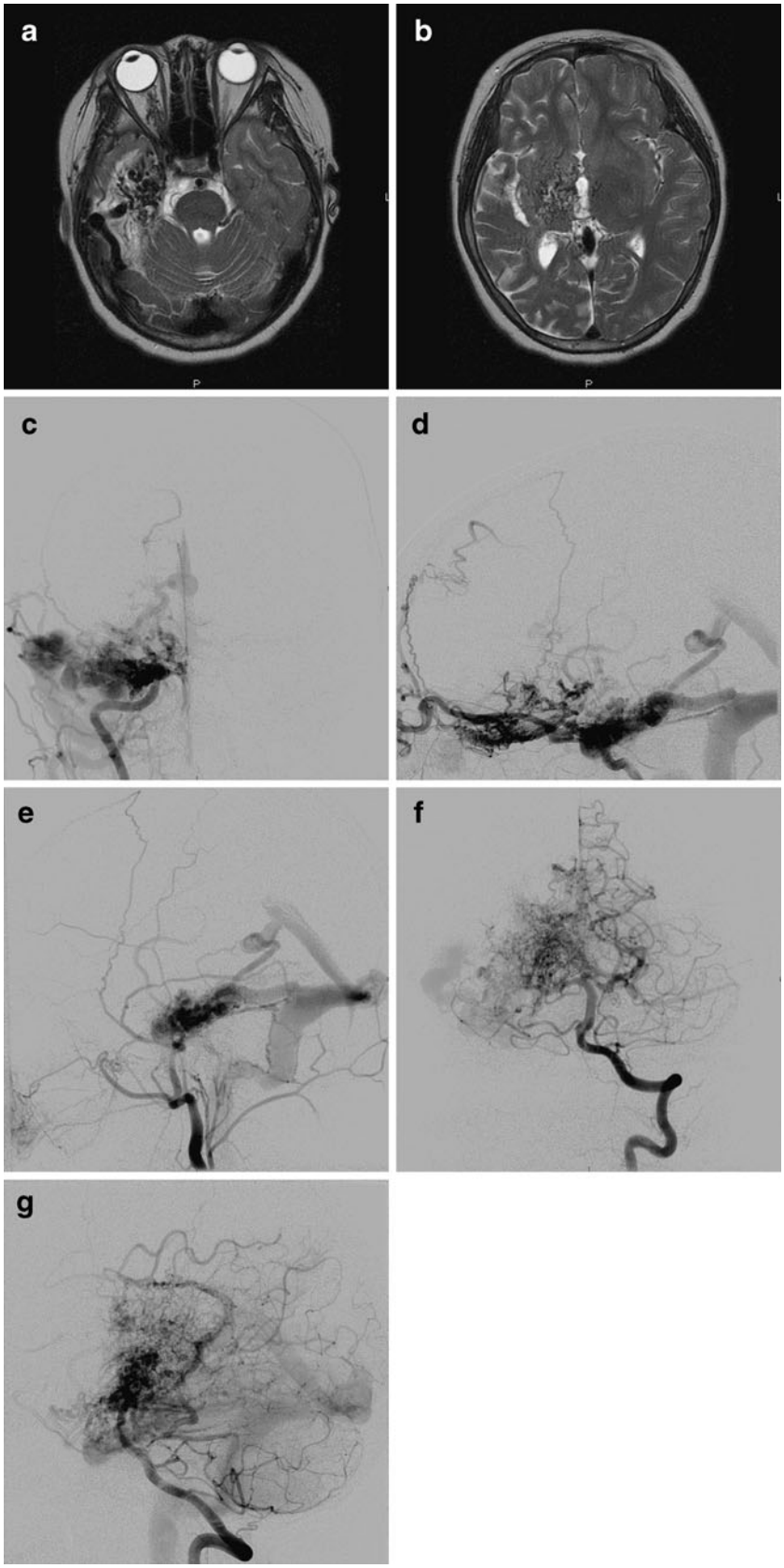

Fig. 4 The patient is a 10-year-old girl who presented with left hemiparesis. Neuroimaging work-up demonstrated evidence of intraventricular hemorrhage as well as an extensive arteriovenous malformation involving the right optic nerve, right temporal lobe, and right basal ganglia regions (**a**, **b**). Femoral cerebral angiogram demonstrated that there is a right optic nerve and right basal ganglia region AVM (**c**, **d**). There is occlusion of the right internal carotid artery at the level above the ophthalmic artery. There is evidence of meningeal supply to the malformation (**e**), and supply to the thalamic component of the malformation from the right anterior and posterior thalamoperforators as well as the posterior choroidal arteries (**f**, **g**). Drainage of the malformation is mainly through the deep venous system

Table 2 Intracranial vascular pathology in our series of 151 consecutive pediatric patients

Type of intracranial shunt	Number of patients	% (151 Patients)
Vein of Galen malformations	56	37
Pial AVM	48	31.7
Pial AVF	11	7.3
Dural	6	4
Mixed complex	30	20

median vein of the prosencephalon and referred to as aneurysmal malformations of the vein of Galen, and those that are deep AVMs draining through a dilated vein of Galen, with some outflow restriction (aneurysmal dilatations of the vein of Galen) [4]. The proliferative diseases correspond to a group of vascular lesions that are hypervascular and although it may appear to have an early draining vein(s), they for the most part without shunts. This pathologic entity may need targeted endovascular intervention only after presenting with intracranial hemorrhage or deteriorating neurological symptomatology (Fig. 1).

The different arteriovenous lesions encountered will depend on the meningeal space from which they develop: dural, pial, subarachnoid, or choroidal. The onset of the pediatric intracranial AVS varies depending on the disease. The galenic malformations are the earliest and most severe expression of the disease, and present in the newborn period with peak by 1 year of age. Dural sinus malformations present slightly later and peak between 1 and 2 years of life. Both of these conditions can be diagnosed during the prenatal stage with intrauterine ultrasound and MRI (Fig. 2). Pial AVMs on the other hand are rarely seen in the first year of life. In children, they may present at around 3 years to approximately 15 years of age. The majority of pial AVMs, however, present in the second and third decade of life. Dural shunts in children are usually seen between 5 and 10 years of age, whereas adult dural shunts are seen more frequently in the third, fourth, or fifth decade of life (Fig. 3).

Table 3 Angioarchitecture of cerebral AVMs in 48 consecutive pediatric patients

	Patients	% (48 Patients)
Location		
Cortical	19	40
Deep	17	35
Posterior fossa	8	17
Corticoventricular	4	8
Venous drainage		
Cortical	6	12.5
Deep	20	41.5
Mixed	22	45.8
Associated aneurysms		
Flow-related	2	4.2
Intranidal	6	12.5
Venous	6	12.5

Several cases of AVS are known to be hereditary. Some have been linked to chromosomal abnormalities. Hemorrhagic hereditary telangiectasia or Osler–Weber–Rendu syndrome, a heterogeneous disorder linked to chromosome 9q33-34, is associated with cerebral AVS and particularly with arteriovenous fistulae [6]. Patients with neurofibromatosis type 1 (chromosome 17q11.2) can also present with arteriovenous fistulae after rupture of weak vessel walls. There is a group of patients with cerebral AVS that also have facial AVS. The cerebrofacial arteriovenous metameric syndromes encompass a spectrum of phenotypic expressions [1]. Examples of this type of lesions have been named Bonnet–Dechaume–Blanc or Wyburn–Mason syndrome (Fig. 4).

In children, the therapeutic challenges cannot be measured by the size of the target, but related to the understanding of the process and the vasculature to anticipate the relationship between the cerebral AVM and the brain in process of maturation. Depending on the age and the type of AVS and status of the venous system, patients may present with congestive heart failure and pulmonary hypertension, hydrodynamic disorders (macrocrania, ventriculomegaly), seizures, mental retardation, melting brain syndrome, hemorrhage, venous ischemia, failure to thrive, and headaches among others. Staged partial treatment may be indicated for specific patients that present with one or several of these symptoms.

Timing of treatment may be crucial. There is a "therapeutic window" to treat children with cerebral AVMs and failure to obtain a normal maturation process may constitute a therapeutic failure [4].

In this report, we detail our experience in the management of cerebral AVS in neonates, infants, and children. In the "Discussion" section, we will review the existing literature in the management of pial AVMs. The management of vein of Galen malformations is discussed in a separate chapter.

Table 4 Management of 48 consecutive pediatric patients with cerebral pial AVMs

Treatment modality	Number of patients	% (48 Patients)
Embolization alone	26	54
Embolization and radiosurgery	12	25
Embolization and surgical resection	6	12.5
Radiosurgery alone	2	4.2
Conservative management	2	4.2

Methods

Patients

An all-inclusive retrospective chart review was performed. We identified all consecutive pediatric patients that presented from age 0–18 years with intracranial arteriovenous shunts that underwent follow-up angiography, first-time endovascular treatment, or staged embolization over the period from January 1, 2004 to June 16, 2009 at the Hyman Newman Institute for Neurology and Neurosurgery at Roosevelt Hospital in NY. The initial presenting symptoms, as well as age of presentation, were recorded. The age of presentation was divided into five groups: prenatal, neonatal (0–30 days), infancy (30 days to 2 years), early childhood (2–12 years), teenage (13–18 years).

Classification and description of arteriovenous shunts

Arteriovenous shunts were categorized as cerebral arteriovenous malformations, pial arteriovenous fistulae, dural arteriovenous malformations, vein of Galen malformations, and mixed/other intracranial vascular malformations. The angioarchitecture of the brain arteriovenous malformations was documented for each patient. Location of the brain arteriovenous malformation (BAVM) was divided into four groups: cortical, corticoventricular, deep (basal ganglia, thalamic, and/or internal capsule), and posterior fossa. The venous drainage of each malformation was registered as cortical/superficial, deep venous drainage into the Galenic system, or a combination. The presence of venous anomalies, stenosis or thrombosis was also documented. The presence of associated aneurysms was evaluated, and if present they were categorized as flow-related, intranidal, or venous.

Treatment modalities and outcome

The type of treatment each patient received was recorded. Treatment modalities included medical management of presenting symptoms, endovascular embolization, stereotactic radiation therapy, microsurgical resection, ventriculo

Table 5 Treatment related complications of pediatric patients who underwent management of cerebral AVMs (163 endovascular embolizations, 17 radiation treatments, and seven surgeries)

	Patients (N=13)	Complication
Endovascular embolization		
Permanent neurologic	1	Ischemic stroke with homonymous quadrant field deficit
	2	Ischemic stroke with residual arm hemiparesis
Temporary neurologic	3	Ischemic stroke on MRI, transient left hemiparesis
	4	Asymptomatic intracerebral hemorrhage
	5	Asymptomatic occlusion of distal right anterior cerebral artery with NBCA
	6	Transient left arm weakness
	7	Midbrain stroke on MRI, transient diplopia and left arm weakness
	8	Intraventricular hemorrhage requiring ventriculostomy
	9	Transient left hand weakness
	10	Asymptomatic middle cerebral artery branch occlusion with NBCA
	11	Right cerebellar stroke with transient diplopia
Microsurgical resection	12	Increased visual field deficit after surgery
	13	Abscess at craniotomy site treated with oral antibiotics
Radiosurgery		None

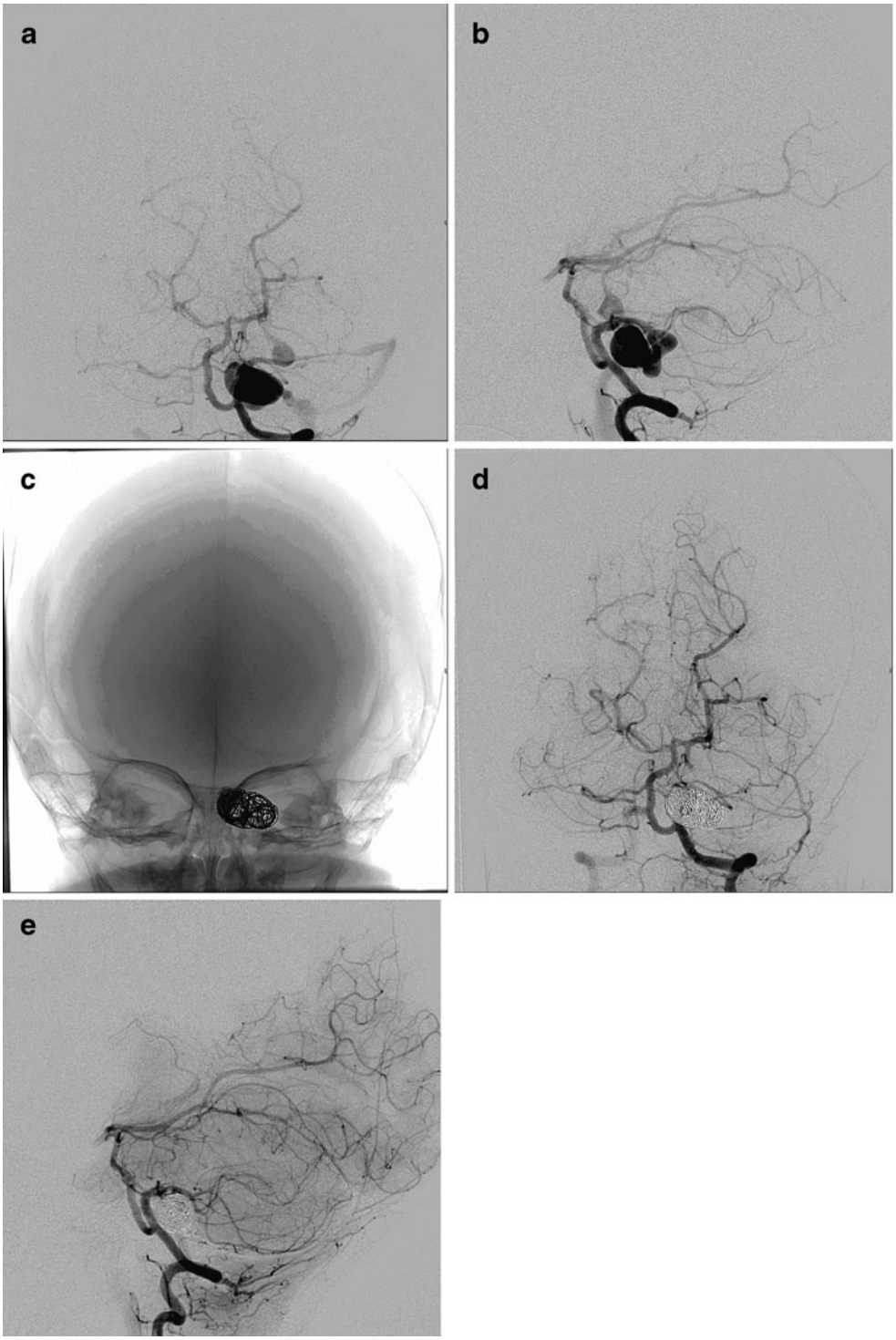

Fig. 5 Patient who presented at 10 months of age with macrocrania and developmental delay. The left vertebral artery injection (**a**, **b**) demonstrates the fistulous type arteriovenous shunting with supply from the left posterior inferior cerebellar artery and draining through the left lateral ponto-mesencephalic vein. Coils were used to prevent the liquid embolic agent from migrating distally (**c**). The left vertebral artery injection after the embolization demonstrated complete obliteration of the arteriovenous malformation (**d**, **e**)

peritoneal shunt, or third ventriculostomy, or a combination of therapeutic modalities. The details of treatment including the number of treatments of each modality, average number of treatments per patient, materials employed, and angiographic cure rate was recorded. Complications of treatment were categorized as temporary, permanent neurologic mild, permanent neurologic severe, and mortalities. Clinical outcome was determined by chart review and was trichotomized as worsening, stabilization, or improvement of presenting symptoms. Patients with documented angiographic cures were also considered improved independent of presenting symptoms.

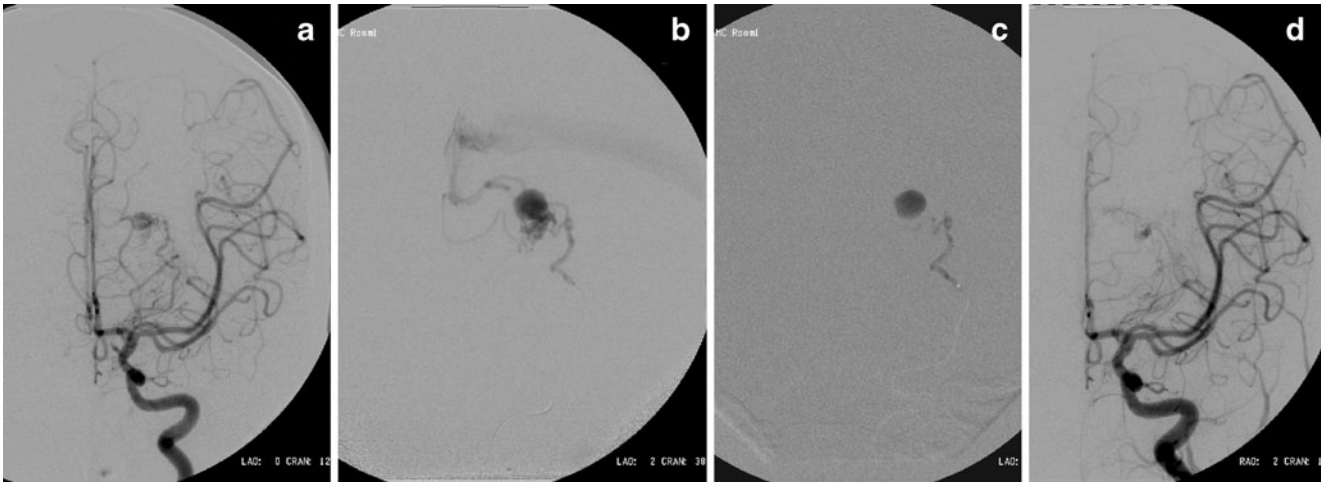

Fig. 6 Patient that presented with a left basal ganglia hemorrhage with intraventricular extension. Femoral cerebral angiogram demonstrated supply to an AVM by the anterior choroidal artery (**a**). There is evidence of a pseudo aneurysm. Super-selective catheterization (**b**) and targeted embolization (**c**) with *n*-butyl-cyanoacrylate mixture was performed. The follow-up femoral cerebral angiogram demonstrated residual AVM but complete obliteration of the angioarchitectural weakness (**d**)

Results

Patient demographics

A total of 151 consecutive pediatric patients with intracranial shunts were evaluated from the period of January 1, 2004 to June 16, 2009. This included 56 vein of Galen malformations (37%), 48 cerebral AVMs (31.7%), 11 patients with pial arteriovenous fistulae (7.3%), six patients with dural arteriovenous malformations (4.0%), and 30 patients with mixed intracranial vascular malformations (20%; Table 2).

Of the 48 BAVM patients, 24 (50%) were girls. Mean age of presentation was 116 months (9.6 years) and the most common period of presentation was early childhood in 26 patients (54.2%), followed by teenage years in 17 patients (35.4%), infant period in nine patients (19%), and prenatally in one patient (2.0%). The most common presenting symptoms were intracranial hemorrhage in 25 patients (52%), seizures in eight patients (17%), progressive hemiparesis/spasticity in six patients (12.5%), headaches in four patients (8.3%), incidental in four patients (8.3%), and because of a cutaneous vascular birthmark in one patient (2.0%).

Brain arteriovenous malformation angioarchitecture

In this series of patients with BAVM, the lesions were most frequently located in the cortex (19 patients, 40%), followed by deep structures (17 patients, 35%), followed by the posterior fossa (eight patients, 17%), and least frequently in the corticoventricular region (four patients, 8%). Venous drainage was cortical/superficial in six patients (12.5%), exclusively deep to the galenic venous system in 20 patients (41.7%), and mixed in 22 patients (45.8%). The presence of associated aneurysms was noted in 14 (29.2%) of 48 patients. Aneurysms were classified as flow-related in two patients (4.2%), intranidal in six patients (12.5%), and venous in six patients (12.5%; Table 3).

Treatment and outcomes

Forty six (95.8%) of the 48 patients underwent treatment during the study period. Forty-four patients underwent a total of 163 endovascular embolizations (average 3.4/patient), six patients underwent microsurgical resection for a total of seven surgeries, and 14 patients underwent a total of 17 radiosurgery treatments. Twenty-six patients (54.2%) underwent endovascular embolization as the only mode of treatment; two patients (4.2%) underwent radiosurgery as the only mode of treatment. Eighteen (37.5%) patients received multi-modality treatment, 12 (25.0%) with embolization and radiation and six (12.5%) with embolization and microsurgical resection (Table 4).

n-Butyl cyanoacrylate (NBCA, Cordis Neurovascular, Miami, FL) was the most commonly used embolic agent in 32 patients (72.7%) as the only agent, followed by ethylene vinyl alcohol copolymer (ONYX, EV3, Irvine, CA) as the only embolic agent in four patients (9.1%). Combination therapy with ONYX and NBCA was utilized in eight patients (18.2%).

The angiographic cure rate was 20.8% in all patients and 21.7% when excluding the two patients who were managed

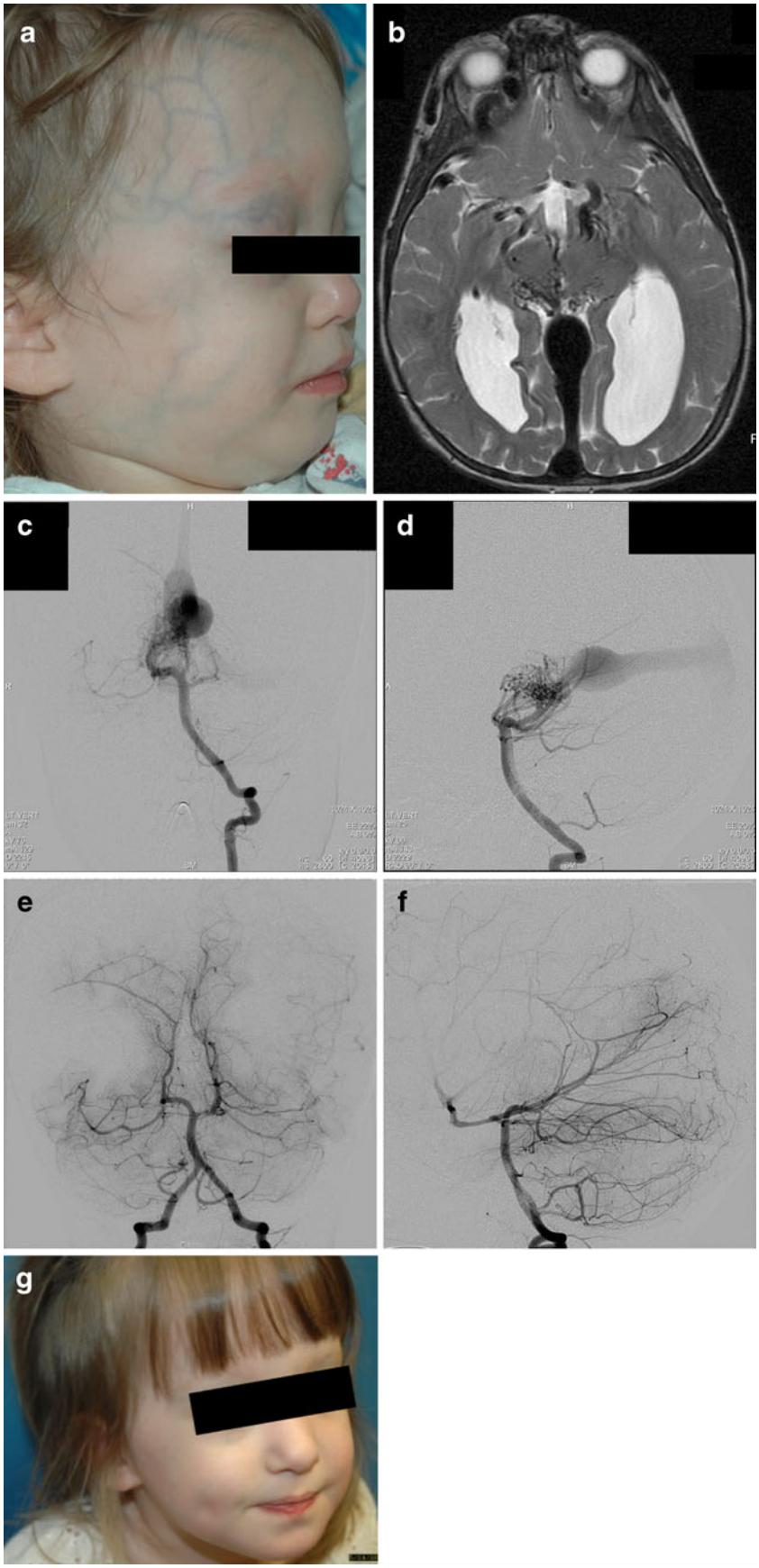

Fig. 7 The patient is a 2-year-old girl that presented with macrocrania and prominent facial veins (**a**). The MRI demonstrated a tectal AVM with a dilated vein of Galen (**b**). Femoral cerebral angiogram demonstrated supply to the AVM from posterior thalamoperforators and posterior choroidal arteries with drainage through the dilated vein of Galen (**c**, **d**). After multiple sessions of targeted endovascular embolization, there was complete obliteration of the AVM (**e**, **f**) with improvement of clinical symptoms (**g**). Photographs taken with consent of the parents

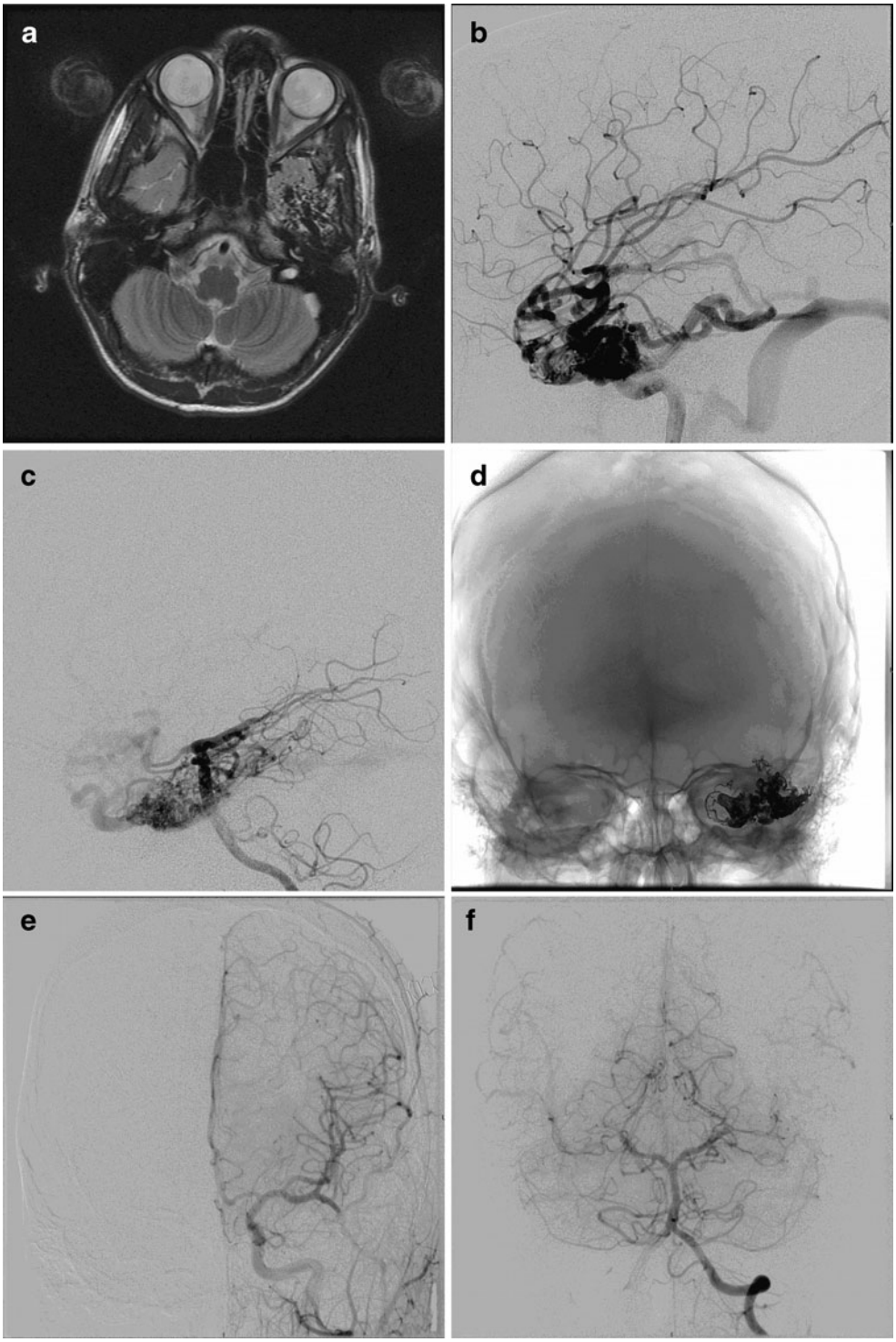

Fig. 8 Patient that presented at 14 years of age with an arteriovenous malformation of the left temporal lobe (dominant hemisphere) during work-up for intractable headaches not controlled on medical therapy (**a**). The left internal carotid artery injection mid arterial phase (**b**) demonstrates opacification of the AVM with supply from branches off the middle cerebral artery and cortical as well as deep venous drainage. The left vertebral artery, mid arterial phase (**c**), demonstrated supply from the temporal branches off the posterior cerebral artery. The patient underwent endovascular embolization with ONYX (**d**) followed by surgical resection and complete obliteration (**e**, **f**). Acknowledgement for Dr David Langer for the surgical resection of the AVM

conservatively without intervention. Clinical symptoms improved in 13 (27.1%) patients, were stable in 28 (58.3%) patients, and worsened in seven (14.6%) of patients.

The complications rate for endovascular embolizations was 6.7% (11 in 163) with 5.5% temporary complications and 1.2% permanent complications. The mortality rate for the group of patients that underwent endovascular embolizations was 0.0%. The complication rate for the group that underwent microsurgical resection was 28.5%, all permanent complications with no deaths. Patients did not have complications from radiosurgery in this series (Table 5).

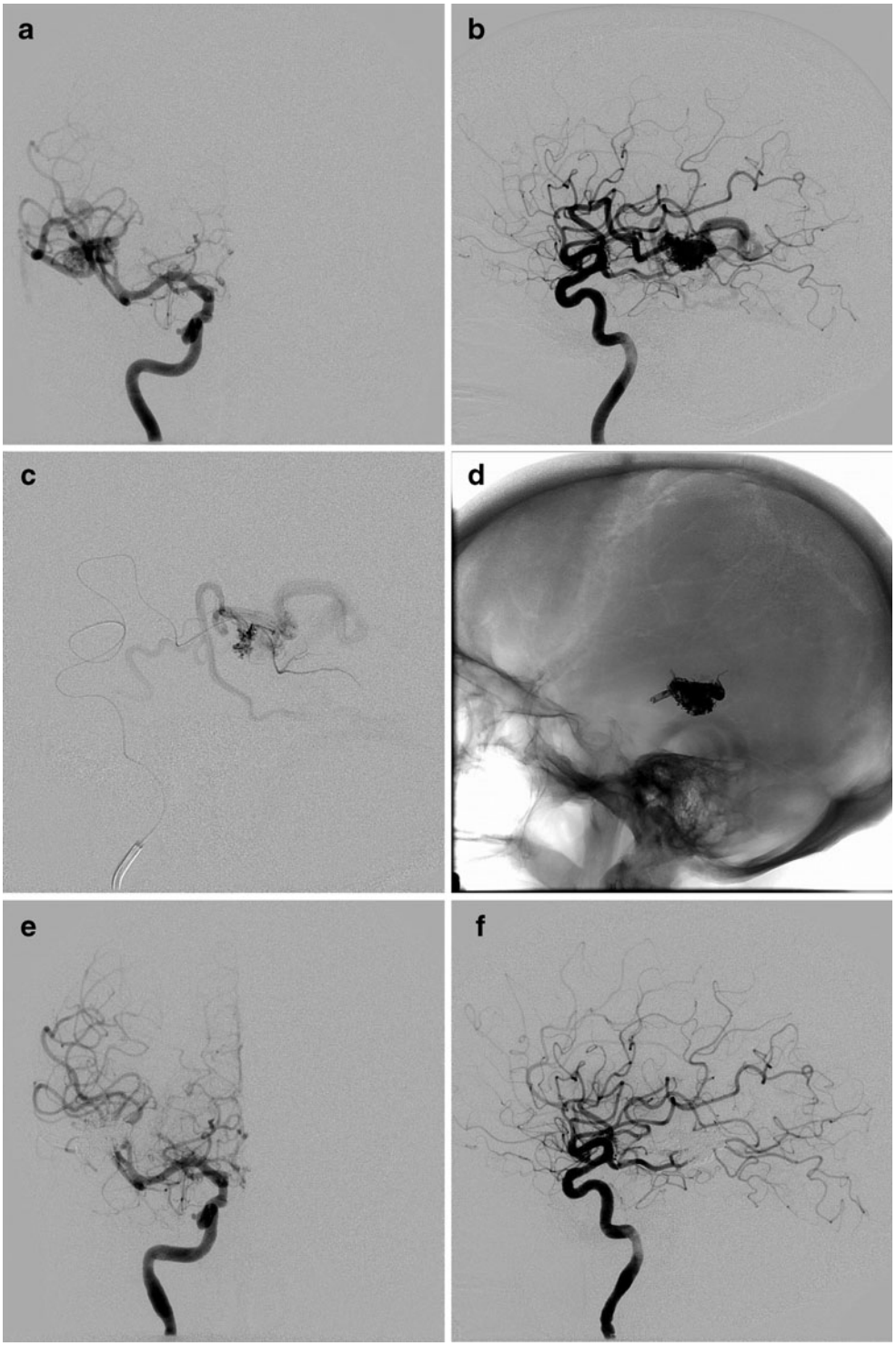

Fig. 9 Patient that presented with headaches and seizures underwent neuroimaging that demonstrated a right temporal AVM. She underwent a femoral cerebral angiogram that demonstrated supply from right middle cerebral artery branches and cortical venous drainage (**a**, **b**). The AVM nidus was catheterized super-selectively (**c**) and embolization was performed with 4 mL of ONYX (**d**). The follow-up angiogram demonstrated complete obliteration of the AVM (**e**, **f**)

Discussion

Pediatric patients with intracranial arteriovenous shunts were mostly of the vein of Galen malformation type in our patient cohort. This group of patients will be published at a later time; and are discussed by the John Hopkins Medical Institute group in a separate report of this issue.

A comprehensive review of the literature demonstrates the differences in the pathophysiology, symptomatology, and treatment of the intracranial shunts in the pediatric population. There are two primary types of isolated cerebral AVMs in children, those in which the angioarchitecture is a fistulous type, single or multiple large direct arteriovenous communications connecting into one large dilated venous

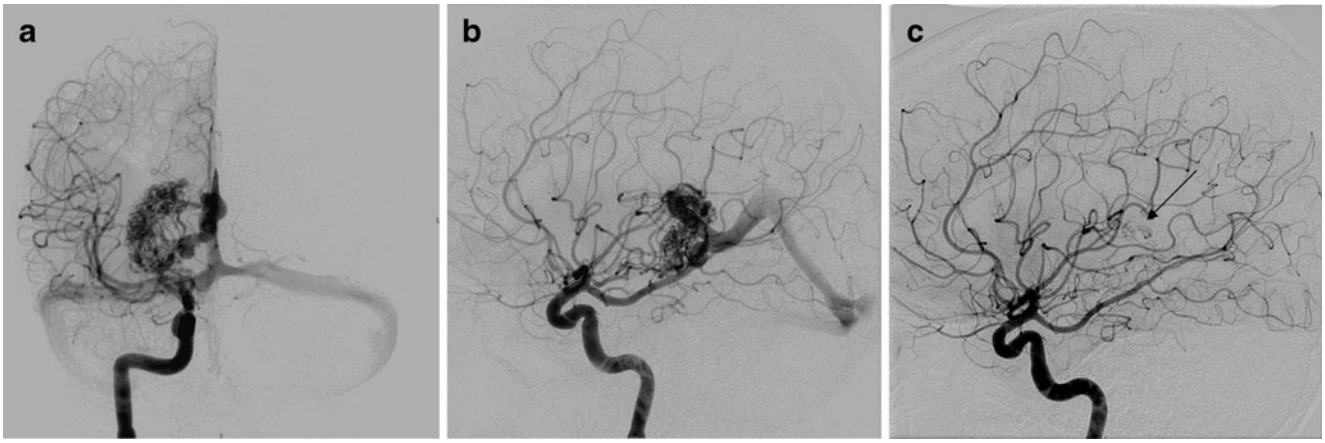

Fig. 10 Patient that presented at 14 years of age with a right thalamic hemorrhage. Neuroimaging demonstrated an arteriovenous malformation with supply from the right anterior choroidal and posterior choroidal arteries and drainage through the deep venous system (**a**, **b**). She underwent targeted super-selective embolization followed by radiosurgery. The follow-up femoral cerebral angiogram 3 years later demonstrated a small area of faint arteriovenous shunting with supply from the anterior choroidal artery (**c**)

sac, and those with a true nidus with an arteriolar type network (Fig. 5). This communication may be at the same or different sites. Angioarchitectural differences may influence the different management options.

Pial arteriovenous malformations with nidus

The brain arteriovenous malformations, pial arteriovenous malformations, cerebral arteriovenous malformations, or non-galenic cerebral arteriovenous malformations, refer to the same entity of arteriovenous communications in the subpial compartment of the central nervous tissue. The angioarchitecture of this lesion is one of nidus versus the arteriovenous fistulae, which can be described as a different group of lesions.

Cerebral AVMs are subpial in location. They may be deep or superficial. Isolated lesions can have a small nidus, called micro AVMs, or a large nidus, called macro AVMs. The feeding vessels to the malformation are usually markedly dilated whereas the veins may be apparently normal in micro AVMs and significantly dilated with ectatic changes in those that have outflow restriction. They may have a typical cortical ventricular pyramidal shape with the tip of the pyramid abutting against the ventricle and they may be buried in the white matter. Cerebral AVMs do not contain brain tissue or nerve fibers in the nidus, a point of differentiation with proliferative angiopathy. The venous drainage pattern of an AVM will influence the surrounding brain area that may eventually suffer hemodynamic consequences, and will play a role in clinical manifestations [4]. Cerebral vascular malformations are randomly distributed in the central nervous system. The apparent frequency variation is related to the proportion of original mass of tissue in which they developed.

Multifocal lesions in children are twice as frequent as that of adults (17.2% vs. 9%) [4, 11, 13]. It is believed that some AVMs in multifocal pediatric cases thrombose spontaneously. The fact that an AVM becomes evident in children indicates an earlier disruption of the equilibrium of the vascular system created by a given trigger.

Proliferative angiopathy lesions may resemble AVMs on neuroimaging. These lesions may resemble a moya moya pattern, but the capillary ectasia, rapid venous filling, and type of dural angiogenesis are different. These patients usually do not present with acute neurological deficit or hemorrhage. They commonly present with seizures, headaches, and progressive neurological deficits [4].

All AVMs undergo changes in the angioarchitecture as time passes. Understanding these changes and their correlation with the natural history are important at the time of planning a treatment strategy [4]. Changes in the angioarchitecture include: venous angiopathy, dural sinus high flow, venous ischemia and thrombosis, venous hemorrhage, venous enlargement, arterial angiopathy, and spontaneous thrombosis of the AVM.

While complete elimination of the pediatric cerebral AVM is an acceptable goal, it should be considered in the context of the anticipated natural history of the AVM in a particular child versus the risk of treatment [4]. The multiple approaches include complete exclusion, partial, staged, and palliative treatment (Fig. 6). These results can be obtained by endovascular embolization, surgical excision [2], radiosurgery [8, 10], or a combination (Figs. 7 and 8). Trans-arterial endovascular embolization with liquid embolic agents has been our preferred treatment approach based on cure rate, as well as the morbidity and mortality of the lesions and the treatment. The primary embolic agent has been NBCA (Cordis Neurovascular, Miami, FL), ideally

with flow-guided microcatheters, under flow control, and with systemic hypotension. More recently, we have used the ONYX embolic material (EV3, Irvine, CA), which will be playing a more important role in the future, with a potential significantly higher rate of cure [3] (Fig. 9). In association to the liquid embolic agents, on selective cases, flow-guided coils (Berenstein Liquid Coils, Boston Scientific, Freemont, CA) were used to reorient vascular supply, to protect normal territories, or to prevent the permeation of the embolic agent to normal tissues [5]. Treatment of the AVM needs to be accompanied by treatment of other systemic manifestations (heart failure, seizures, extracranial AVMs, etc.).

The most common pathology in our cohort was vein of Galen malformations. The most common age of presentation of a pediatric cerebral AVM was early childhood. These AVMs were most commonly cortical in location with combined venous drainage (cortical and deep). In our group of pediatric patients with cerebral AVMs, angiographic obliteration of the lesion was accomplished in 21.7% of the patients. There is a group of patients with "near angiographic cure" in whom there is evidence of arteriovenous shunting after treatment but the risk of treatment for complete obliteration might outweigh the benefit. These patients need a longer follow-up but until now they have not presented with bleeding or other complications from the small residual (Fig. 10).

Our group of patients presented mostly with intracranial hemorrhage. Stabilization or relief of symptoms was accomplished in 85.4% of the patients. In small malformations with a single pedicle, including the fistula-type lesions, the results of embolization are far superior than in those that involve a larger surface of brain. These outcomes were accomplished with a low morbidity of endovascular embolization procedures (1.2% of permanent complications) and without mortality. The complication rate was high in the group of patients that underwent surgery, but it was out of a small sample of seven patients with a total of two complications. The treatment approach, including staging, partial, or palliative treatment, was determined based on a number of factors that include the age of the patient, the clinical presentation, and the angioarchitecture of the AVM. All these factors are based on the existing literature as well as our previous personal experience.

In conclusion, pediatric intracranial arteriovenous shunts are dynamic lesions mainly ruled by the venous drainage pattern. Careful clinical observation and timely intervention are important for good results. Trans-arterial endovascular embolization with liquid embolic agents is the treatment of choice for safe stabilization and/or improvement of symptoms in the group of pediatric patients with intracranial arteriovenous malformations. The best outcome in this patient population is obtained when management is performed by an interdisciplinary team with extensive experience in the treatment of neonates, infants, and children with intracranial arteriovenous shunts.

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