Pediatric Intracranial Aneurysms

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KEYWORDS

• Intracranial aneurysm • SAH • Endovascular treatment
• Pediatric aneurysm

Intracranial aneurysms in children are rare; 0.5% to 4.6% of intracranial aneurysms occur in patients aged 18 years or younger.[1–7] In a cooperative study reported in 1966, only 41 of 6368 (0.6%) ruptured aneurysms were found in patients younger than 19 years.[8] Aneurysms occurring in very young children and infants are exceedingly rare. In adults, aneurysms are believed to form as a result of multiple risk factors (eg, family history, age older than 50 years, smoking, cocaine use, and hypertension) present over the course of an individual's life span. In childhood, most of these aneurysmal risk factors do not exist, and for this reason, the pathogenesis is believed to be different.[9–11] Some investigators have proposed that a vasculopathy predisposes regions of the cerebral vasculature to aneurysm formation.[12,13] In multiple case series, primarily since the 1970s, pediatric aneurysms have been reported to exhibit features that differ from those in adults, such as male predominance, a higher incidence in locations such as the posterior circulation and internal carotid bifurcation, and greater numbers of giant aneurysms.[2,14–17]

Discrepancies exist in the clinical description of pediatric aneurysms, likely related to the small numbers reported in most case series. Since the comprehensive review by Huang, other case series (including from the authors' institution) have been published.[18–20] Data from these larger series have confirmed some earlier findings and have contradicted others. It is likely that considerable heterogeneity exists with respect to the pathology, diagnosis, and treatment of these lesions.

The past 20 years have witnessed a gradual shift from exclusively surgical approaches toward endovascular treatment and multimodality therapeutic plans.[12,13,21] The increasing number of options available for the treatment of complex aneurysms suggest that treatment should be guided by the best available evidence and executed by centers with expertise in each of these therapeutic tools.

CLINICAL PRESENTATION

As in adults, children with intracranial aneurysms can present with subarachnoid hemorrhage (SAH), headache, direct compressive effects, focal neurologic deficits, or seizures. If SAH is present, nearly 60% of patients will have a cerebral aneurysm.[22] Fusiform aneurysms tend to present with nonhemorrhagic deficits.[19] Many patients (30%–85%) with SAH confirmed by radiographic imaging or lumbar puncture typically present good clinical function defined by a Hunt and Hess grade between 1 and 3. Patients with poor clinical function have a Hunt and Hess grade of 4 to 5, occurring 15% to 42% of the time (**Table 1**).[12,17,18,24,25] The reason for the better clinical grade at presentation is unclear but may be because of several factors, such as fewer comorbidities, clouding the initial diagnosis, and a greater tendency to refer cerebrovascular cases to tertiary centers. Specific biologic features such as the activity of

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Table 1
Summary of published reports describing pediatric intracranial aneurysms

Year	Authors	Cases (n)	Boys (%)[a]	Girls (%)[a]	SAH (%)	Good Grade (%)	Poor Grade (%)	Giant (%)	ICA Terminus (%)	Posterior Circulation (%)	Surgical or Endovascular Treatment (%)	Good Outcome (%)	Death (%)	Years of Follow-Up
1939	McDonald and Korb[40]	61	60	34	87	[a]	[a]	[a]	16	23	[a]	[a]	[a]	[a]
1940–64	Case reports	9	78	22	100	22	78	11	22	22	33	33	66	[a]
1963	Stehbens[41]	3	33	66	33	0	100	[a]	[a]	[a]	[a]	[a]	100	[a]
1965–1990	Case reports	35	[a]	[a]	[a]	[a]	[a]	[a]	[a]	[a]	67	25	75	[a]
1965	Matson[23]	13	92	8	92	[a]	[a]	[a]	8	15	92	62	23	0–12
1966	Locksley et al[8]	41	73	27	100	[a]	[a]	[a]	15	17	[a]	[a]	[a]	[a]
1971	Patel and Richardson[5]	58	55	45	100	69	31	0	34	5	64	52	31	1–22
1973	Sedzimir and Robinson[7]	50	56	44	100	[a]	[a]	[a]	36	4	50	70	28	2–15
1975	Amacher and Drake[42]	16	69	31	88	44	44	44	13	31	69	56	38	[a]
1977	Almeida et al[43]	11	55	45	91	82	18	0	55	9	91	64	27	0–15
1978	Batnitzky and Muller[44]	12	67	31	83	[a]	[a]	25	25	25	[a]	[a]	25	[a]
1980	Gerosa et al[2]	15	67	33	80	87	13	20	33	0	100	67	13	2–22
1981	Heiskanen and Vikki[16]	32	53	47	100	[a]	[a]	[a]	50	6	100	75	6	0.5–11
1981	Amacher et al[15]	26	62	38	65	96	4	[a]	[a]	[a]	96	92	4	[a]

1982	Storrs et al[45]	29	45	55	76	38	62	31	31	31	72	45	34	[a]
1983	Schauseil-Zipf et al[46]	15	67	33	60	20	[a]	[a]	[a]	7	80	13	33	1–17
1983	Ostergaard and Voldby[4]	43	58	42	77	72	28	5	44	7	81	53	30	0.25–14
1985	Humphreys et al[47]	35	[a]	[a]	74	11	63	29	26	20	66	40	40	[a]
1986	Pasqualin et al[48]	31	[a]	[a]	94	[a]	[a]	3	29	3	61	52	3	[a]
1988	Roche et al[6]	43	70	30	81	[a]	[a]	7	26	16	95	79	12	[a]
1989	Meyer et al[3]	24	71	25	54	50	4	54	8	46	100	92	4	1–7
1991–2002	Case reports	47	[a]	[a]	[a]	[a]	[a]	[a]	[a]	[a]	70	63	26	0–19
1991	Herman et al[49]	16	56	44	63	38	6	19	6	19	94	75	6	0.67–6
2001	Proust et al[17]	22	73	27	95	59	36	14	36	9	100	64	23	[a]
2004	Huang et al[50]	19	68	32	58	42	16	37	11	42	84	95	5	0.1–9
2005	Lasjaunias et al[12]	59	59	41	50	30	18		4	27	67	52	10	0–6
2005	Agid et al[24]	33	16	17	9	[a]	[a]	30	22	30	70	64	15	12
2005	Krishna et al[25]	22	64	36	91	68	32	13.6	20	24	77	82	10	0.1–2.5
2006	Sanai et al[20]	32	44	56	22	85	15	40	13	28	90	78	0	6
2008	Vaid et al[51]	36	52	48	92	58	42	21	18	30	75	78	11	0.1–3.5
2009	Hetts et al[19]	77	52	48	32	[a]	[a]	11	[a]	22	77	[a]	1	[a]
Total		965	60	37	74	51	34	21	24	19	79	62	24	

[a] Numbers do not sum to 100% in all cases, reflecting discrepancies in the original reports.

the nitric oxide synthase pathway and the robustness of leptomeningeal arterial collaterals may also play a role.[12,26]

Matson[23] originally noted that pediatric aneurysms occur slightly more commonly in men. This finding has been affirmed in multiple case series with male/female ratios ranging from 1:1 to 11:1.[1,17,23,27] In the larger case series, however, the male/female ratio is closer to 1.5:1 (see **Table 1**).[1,4–8,12,19] One possible explanation for this difference is that 14% to 39% of pediatric aneurysms are a result of trauma, which is substantially more common among men.[28,29] In situations in which behavior is less likely to influence the clinical presentation, Buis and colleagues[27] noted that the male/female ratio of aneurysms was 1.1:1 in children younger than 1 year.

DIAGNOSIS

With the increasing availability of multidetector computed tomographic (CT) scanners, magnetic resonance imaging (MRI), and 3-dimensional image processing, the identification of a cerebral aneurysm as a cause of SAH can occur not only in a tertiary referral center but also at a community hospital. Furthermore, the technical quality of catheter angiography has improved over the last decade with advances such as 3-dimensional rotational angiography and angio-CT. These technological improvements have allowed more detailed studies to be obtained in affected patients.

For a child with a suspected SAH, the diagnostic sequence begins with a noncontrast CT scan of the head. If this study is normal but the clinical story is consistent with an SAH, then a lumbar puncture is required. The presence of blood or xanthochromia should prompt a contrast-enhanced imaging study of the intracerebral vasculature. At this point, the diagnostic decisions may vary depending upon the age of the child, the clinical status, and availability of invasive imaging and interventional treatments. In adults, a CT angiogram (CTA) or MR angiogram (MRA) is usually performed after the noncontrast CT scan to quickly identify the source of the SAH. At the authors' institution, children older than 14 years receive a CTA immediately after the identification of subarachnoid blood on noncontrast CT. Some institutions, including the authors', proceed directly to catheter angiography after a noncontrast head CT, particularly because the false-negative rate of CTA exceeds 10% in some studies.[30] The choice of the imaging modality is made on a case-by-case basis with an effort to reduce radiation exposure while obtaining the necessary data needed to plan subsequent interventions and postoperative management. Although Hetts and colleagues'[19] and other studies have reported no delayed sequelae of radiation exposure to patients undergoing endovascular therapy, short follow-up times limit the ability to draw any definitive conclusions.[31]

For those young individuals with unruptured aneurysms that initially present with generalized headaches or even seizures, a postcontrast CT scan may miss the underlying pathology. It is notable that non-SAH headaches and seizures have been reported to be the presenting symptoms of pediatric aneurysm up to 40% of the time.[18,19] Consequently, MRI or MRA is a valuable tool for examining these individuals and following them in the years to come if conservative management is the chosen treatment strategy, as well as for surveillance for development of new or enlarging aneurysms that may require treatment at a later time.

ANEURYSM FEATURES

The overall location and size of aneurysms in children differ from those found in the adult population.[12,19,32,33] Aneurysms of the internal carotid artery (ICA) typically occur at similar frequencies in both populations. However, there is a greater preponderance of aneurysms at the ICA terminus in the pediatric patient compared with the adult (**Table 2**). Aneurysms of the middle cerebral artery (MCA) appear to occur in similar distributions between the 2 subgroups. Approximately 18% of identified aneurysms are found along the MCA in both adults and children. The distribution of aneurysms in the posterior and anterior circulation, however, appear to be different in the subgroups. In adults, anterior cerebral artery (ACA) (including anterior communicating artery) aneurysms occur approximately 34% of the time, whereas in children recent reviews have encountered ACA aneurysms only about 5% to 10% of the time. In contrast, children appear to be more prone to aneurysms of the posterior circulation. Aneurysms in this region appear approximately 25% of the time in children, whereas in the adult population they are less common ($\sim$8%).

In the most recent series from the authors' institution, 40 females (52%) and 37 (48%) males who ranged in age (at diagnosis) from 3 months to 18 years (mean, 12 $\pm$ 5 years) were included. This gender distribution differs from other reports in the literature in which an overall male predominance is noted. There were a total of 103 aneurysms, 11% of which were greater than 25 mm in diameter and were defined as giant aneurysms.

Table 2
Adult and pediatric aneurysms by location

Location	Pediatric Aneurysm (%)[a]	Adult Aneurysm (%)[b]
ICA terminus	39–51 (24[c])	38.1 (4[c])
Anterior cerebral artery (ACA)	5.4–10	34.6
Middle cerebral artery (MCA)	13.5–21	18.4
Posterior circulation	22–27	8.6

[a] *Data from* Refs.[12,19,32]
[b] *Data from* Locksley HB. Natural history of subarachnoid hemorrhage, intracranial aneurysms and arteriovenous malformations. Based on 6368 cases in the cooperative study. J Neurosurg 1966;25(2):219–39.
[c] ICA terminus data from **Table 1**.

About 22% of these aneurysms were found to be in the posterior circulation, which is in agreement with the other series in which a posterior circulation location occurred in 4% to 27% of patients.[7,19] In contrast, aneurysms in adults are seen in the posterior circulation only 8% of the time (see **Table 2**) and giant aneurysms are even rare.[12,18,19,24,32,34]

Three large case series have subdivided aneurysms into 4 categories: fusiform, saccular, infectious, and traumatic.[12,19,34] These studies have identified that the rate of hemorrhagic presentation differs considerably among aneurysm subtypes. Hemorrhage was more likely a result from saccular aneurysm rupture (**Fig. 1**) than from fusiform or infectious aneurysms (**Fig. 2**, **Table 3**). Furthermore, traumatic aneurysms, as expected, were seen closer to the skull base in the anterior and posterior circulations than in the distal MCA or ACA (**Fig. 3**).

TREATMENT

General Principles

It is clear that a multidisciplinary team consisting of stroke neurologists, cerebrovascular neurosurgeons, and neurointerventional radiologists is best able to treat complex intracranial aneurysms and achieve optimal results.[35] Treatment options include observation, endovascular therapy, or surgical clipping. Endovascular treatment includes the use of detachable coils with or without stents delivered through microcatheters after transfemoral selective catheterization of the involved intracranial arteries. Surgical treatment includes direct clipping, clip reconstructions, and aneurysm trapping with or without bypass procedures.

Aneurysm morphology and etiology have therapeutic implications. In the series by Lasjaunias and colleagues[12] and Hetts and colleagues,[19] saccular aneurysms were the most likely to rupture (>75%

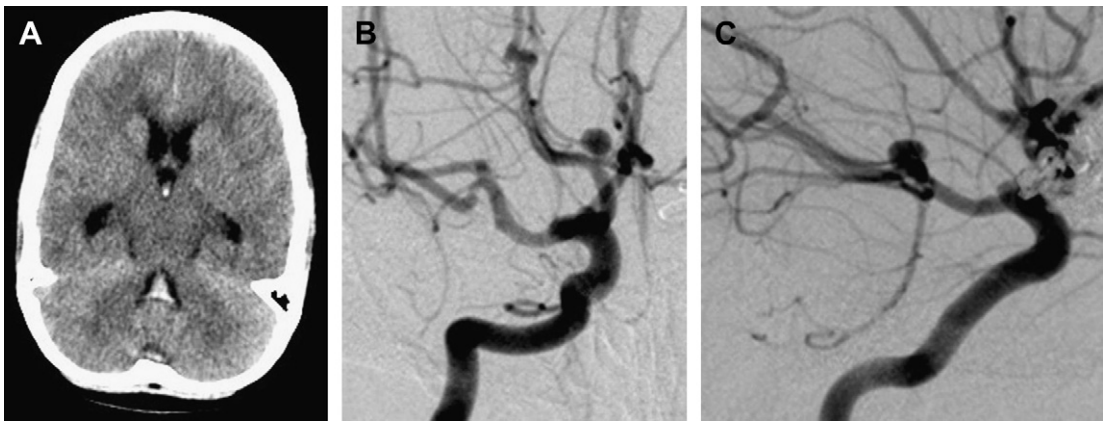

Fig. 1. Saccular aneurysm. A 17-year-old girl with Majewsky type II osteodysplastic dwarfism presented with headache. Subarachnoid and intraventricular hemorrhage was demonstrated on nonenhanced CT (*A*). Initial diagnostic catheter angiography demonstrated 10 saccular intracranial aneurysms, the majority and largest of which were associated with the left anterior circulation. The patient underwent a left pterional craniotomy with clipping of multiple aneurysms. A saccular right P1/P2 posterior cerebral artery (PCA) aneurysm, filling via a near-fetal PCA on anteroposterior (AP) (*B*) and lateral (*C*) right ICA angiograms, underwent endovascular coiling during the same admission.

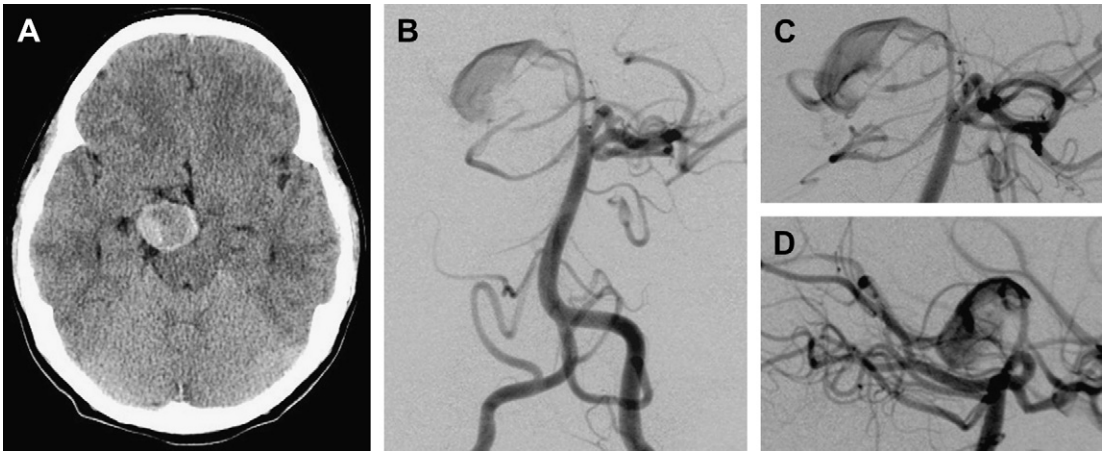

Fig. 2. Fusiform aneurysm. A 13-year-old otherwise healthy boy presented with 2 months of headache and right cranial nerve III palsy. A nonenhanced CT scan (*A*) demonstrates a hyperdense mass in the right suprasellar cistern with mass effect on the midbrain and interpeduncular fossa. Catheter angiography of the left vertebral artery in the AP (*B*), magnified Water (*C*), and magnified reverse Schuller (*D*) projections confirmed the presence of a giant partially thrombosed fusiform in the right P1/P2 posterior cerebral artery aneurysm. This lesion subsequently underwent endovascular coiling, with resolution of the patient's oculomotor palsy.

and >35%, respectively). Infectious aneurysms in both series ruptured at a relatively low rate (17% and 13%, **Fig. 4**). The behavior of fusiform aneurysms is unclear because these 2 studies report disparities in their rupture rates, 8% in the series by Hetts and colleagues and 40% in the series by Lasjaunias and colleagues. Some of these discrepancies may relate to the nature of the aneurysms themselves, as Lasjaunias and colleagues proposed that dissecting aneurysms be put in the fusiform group, whereas the authors proposed that only if a dissection can be demonstrated

Table 3
Recent studies demonstrating aneurysm location and features

	Lasjaunias et al[12]	Agid and Terbrugge[34]	Hetts et al[19]
Patient (n)	59	33	77
Number of Aneurysms	75	37	103
Age	7.6 y (8 d–15 y)	10.2 y (1 d–17 y)	12 y (3 mo–18 y)
Sex (% male)	59%	48%	48%
Morphology			
Fusiform	56%	19%	31%
Saccular	27%	46%	46%
Infectious	14%	8%	12%
Traumatic	3%	14%	14%
Giant (>25 mm)	1.3%	—	11%
Multiple	15%	—	16%
ICA Aneurysm	39%	40%	51%
MCA Aneurysms	21%	13.5%	17%
Anterior cerebral artery Aneurysms	9%	5.4%	10%
Posterior Circulation	27%	24%	22%
Hemorrhage	54%	30%	32%
Cerebral Infarction	3.1%–12.5%	12.1%	7.8%
Mortality	10.4%	15%	1.3%

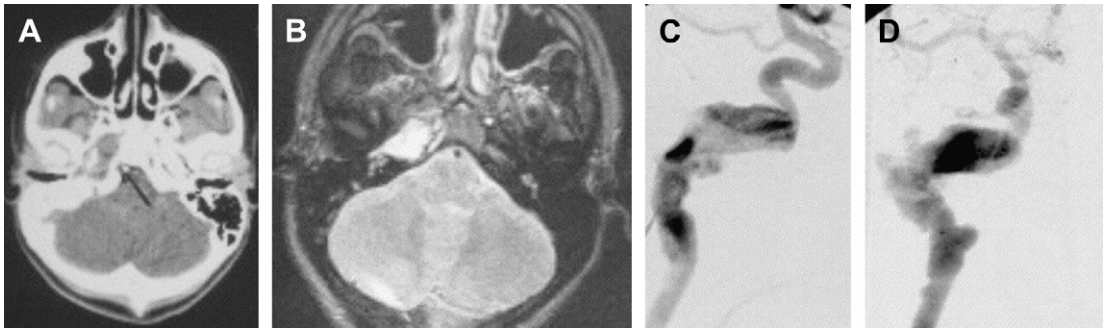

Fig. 3. Traumatic aneurysm. A 15-year-old girl presented with right-sided pulsatile tinnitus and a vascular mass behind the right tympanic membrane 3 years after a motor vehicle accident in which skull base fractures were sustained. A nonenhanced CT (*A*) and T2-weighted MRI (*B*) demonstrate a mass enlarging at the right petrous carotid canal. Catheter angiography of the right ICA in the lateral (*C*) and AP (*D*) projections confirms a fusiform dissecting traumatic aneurysm of the high cervical and petrous segments of the ICA. This lesion was treated with endovascular parent artery occlusion, with resolution of tinnitus.

(pathologically, from angiographic imaging with a dissection flap seen or from MRI with thrombus identified between layers of the artery wall) can an aneurysm be termed dissecting, whether it is fusiform or not.

Endovascular Treatment

Agid and colleagues[24] examined 33 patients younger than 18 years at their institution over a 12-year period (between 1992 and 2004), and they

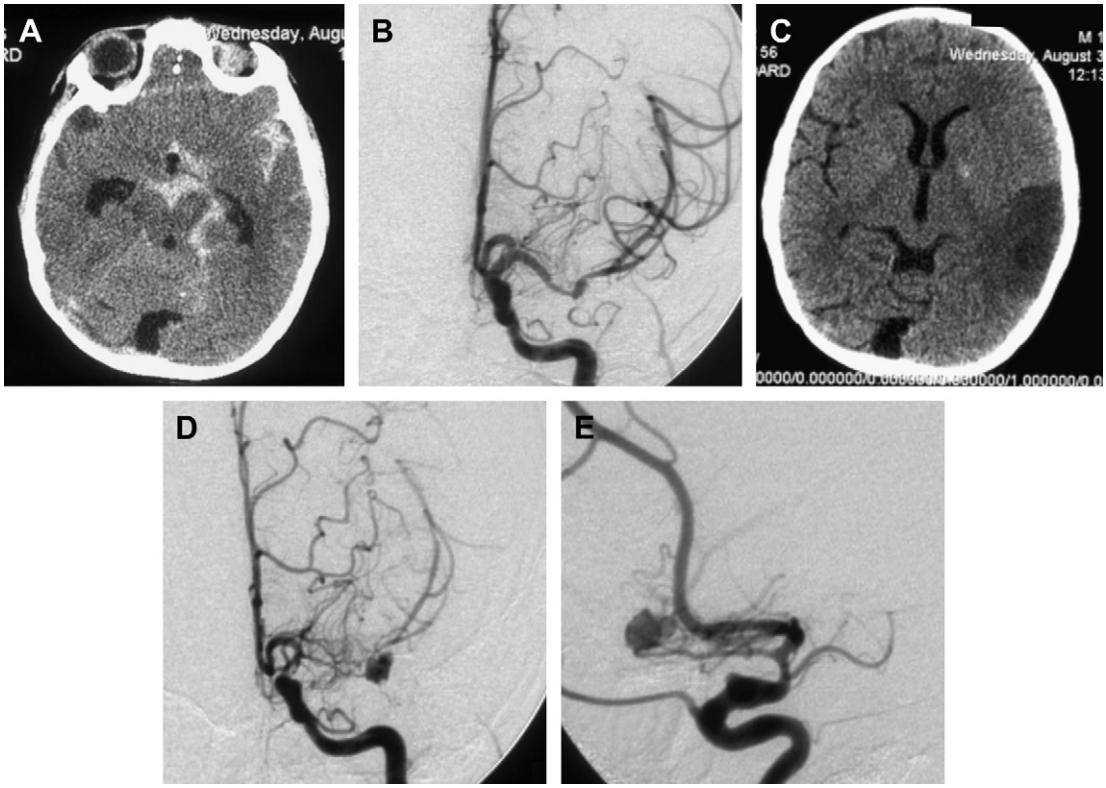

Fig. 4. Infectious aneurysm. A 12-year-old boy with trisomy 21 and enterococcus endocarditis and bacteremia presented with SAH (*A*). The initial angiogram showed evidence of a vasculopathy involving the left MCA (*B*). One week after the SAH, a left MCA distribution infarction was clearly visible (*C*). A follow-up angiogram done at that time showed evolution of the aneurysm with an increase in size and irregularity of the dome (AP [*D*], lateral [*E*]). The aneurysm was treated with superficial temporal artery to MCA bypass and trapping.

retrospectively reviewed surgically treated patients with endovascularly treated patients. Of the 37 aneurysms identified, 10 were treated surgically (27%), 13 were treated endovascularly (35.1%), and 14 were managed conservatively (37.8%). They reported an increasing trend toward endovascular treatment of their patients since 1997.

Various considerations must guide the best treatment option.[19,20] In the pediatric patient treated with endovascular coiling, sufficient attention must be addressed to the life expectancy of the child, the need for continued follow-up, and the possible need for retreatment. In the authors' series, a total of 103 aneurysms were identified in 77 patients over a 27-year period. Among the 77 patients, 19 underwent primary aneurysm coiling, 19 underwent primary surgical clipping, 11 were treated with carotid occulusion, 10 were treated with trapping and/or vascular bypass, and 18 with long-segment vascular dysplasia involving multiple territories were managed conservatively.

In the patients treated with coil embolization, discussions must be performed regarding the potential need to re-treat the coiled aneurysm. During an average 2-year follow-up period, 4 of 19 (21%) patients treated by selective coil embolization required retreatment of the index aneurysm. This recanalization rate is similar to the initial adult experience with aneurysm coiling using bare platinum detachable coils; more recently developed bioactive coils have typically shown closer to a 10% recanalization rate[36] in adults, although bioactive coils have not been studied extensively in children. No patients receiving coiling in the authors' pediatric cohort had rerupture of the treated aneurysm during the follow-up period. In this same group, 2 of 19 patients (10%) crossed over to surgical treatment.[19] Given the cumulative nature of aneurysm recanalization and evidence of delayed recanalization in children (such as an ICA terminus aneurysm with partial recanalization 6 years after initial coiling), it is possible that longer follow-up would reveal a higher proportion of treatment failure.

In the conservatively managed group, most of whom had long-segment fusiform vascular dysplasia that was not amenable to simple clipping or coiling, none presented with SAH over an average follow-up period of 41-months (6 months to 10 years). These patients underwent serial MRI studies to follow aneurysm growth. Two patients showed minimal aneurysm enlargement. One patient had significant enlargement and underwent primary stent coiling of a paraclinoid saccular component of a fusiform aneurysm 4 years into the observation period. No other patients required treatment. None of these patients had SAH during the follow-up period.

Surgical Treatment

Since 1981, there has been an evolution in microneurosurgical and endovascular techniques used at the authors' hospital. Separating the University of California San Francisco (UCSF) cohort into discrete time periods underscores the changes that have occurred in aneurysm management. Between 1981 and 1985, 100% of cases were treated surgically (clipping or bypass/trapping). From 1986 to 1990, 18% of cases were treated surgically, 73% were treated endovascularly (all being parent artery occlusions), and 9% were observed. Endovascular coiling of pediatric aneurysms at UCSF began in 1991; during the next 5 years, 33% of cases were treated surgically, 47% were treated endovascularly (57% by coiling and 43% by parent artery occlusion), and 20% were observed. From 1996 to 2000, 27% of cases were treated surgically, 33% were treated endovascularly (all coiling), and 40% were observed. Since 2001, 45% of cases were treated surgically, 45% were treated endovascularly (14% parent artery occlusion and 86% coiling or stent coiling), and 10% were observed.[19]

From a technical viewpoint, the surgical approaches and tools are similar to those used in the adult setting. Specific issues related to small children include the careful monitoring of blood loss and adequate volume replacement, the use of immobilization techniques that reduce the likelihood of pin-related complications, and the age-appropriate neuromonitoring techniques.

Outcome

Current treatment-related morbidity and mortality rates are low in both endovascular and surgical groups compared with rates in the earlier medical literature. Complication rates have generally decreased with time. The overall infarction rate was higher after surgical treatment (4 of 29 or 14%) than after endovascular treatment (2 of 30 or 7%).[19] Selection bias may play a role in these complication rates, because more complex aneurysms were likely referred for surgical treatment.

In the authors' experience, microsurgical therapy for pediatric aneurysms seems to have higher rates of complete obliteration and lower rates of recurrence, indicating an advantage over endovascular therapy in the areas of efficacy and durability.[20] The issue of treatment durability is particularly an issue with children, because in them life expectancy is measured in decades. Overall, clinical factors such as aneurysm

configuration, surgical risks, and patient variables such as age and comorbidities should be discussed by the entire cerebrovascular team to achieve a treatment consensus. Finally, parental concerns should be addressed with serious discussions regarding durability of treatment, delayed complications, angiographic surveillance, and the potential for additional treatment later in life.

Posttreatment Management

SAH is generally considered better tolerated by children than by adults, although the mechanism for this better tolerance is uncertain. Although it has been reported that approximately 30% of SAH have radiographic evidence of vasospasm, most of these radiographic findings seem to lack any clinical significance. Several series have reported the low rate of clinically significant vasospasm in cases of ruptured pediatric intracranial aneurysms.[12,18,37] The authors' series confirmed this observation: 4 patients had imaging-proved vasospasm and only 1 required therapy to reverse an ischemic neurologic deficit. The basis for better tolerance of vasospasm in children, a leading cause of morbidity in adult patients with SAH, is unclear. Although it has been proposed that the leptomeningeal collateral supply to watershed areas may be more abundant in the pediatric patient, thereby making distal ischemic infarcts less frequent, proving this theory is challenging.

PROPOSED FOLLOW-UP GUIDELINES

Regardless of aneurysm treatment or observation, children with intracranial aneurysms require follow-up imaging and clinical surveillance, given their expected long life spans during which additional aneurysms could arise or treated aneurysms could recur. As suggested previously,[12] aneurysmal disease is both an acute and chronic condition and requires longitudinal management, with emphasis on the ongoing disease entity itself as opposed to just the mode of treatment when an aneurysm becomes acutely symptomatic. The fact that 6 patients in the authors' series developed de novo aneurysms or significant enlargement of previously untreated aneurysms between 6 months and 12 years after initial presentation underscores the need for careful follow-up.

Although some of these new aneurysms may have formed due to increased wall stresses on inherently dysplastic vessels in the setting of increased flow through remaining vessels after occlusion of a carotid or vertebral artery, other aneurysms grew despite preservation of parent artery flow. New and enlarging aneurysms developed in patients initially treated surgically and endovascularly, as has been reported previously by other investigators.[10,38,39] These findings suggest that a brain aneurysm in a child should be considered as a potentially chronic progressive condition. Vigilant long-term follow-up with appropriate clinical and minimally invasive imaging surveillance seems warranted and prudent. Consequently, the following is proposed:

- For endovascularly coiled aneurysms, an initial diagnostic and therapeutic catheter angiogram is done with a follow-up catheter angiogram in 6 months. If there is no evidence of recanalization at 6 months then an annual MRA is done, eventually decreasing to once every 5 years.
- For surgically clipped aneurysms, initial preoperative and postoperative catheter angiograms are done. If no residual aneurysm after clipping is noted then an annual MRA is done, eventually decreasing to once every 5 years.

SUMMARY

Intracranial aneurysms in children differ from those in adults in location, morphology, etiology, natural history, and, hence, management. Several series have been published detailing the experience of various institutions with pediatric intracranial aneurysms,[12,18,37] including the authors' institution.[19,20] Childhood aneurysms have a higher rate of posterior circulation, ICA terminus, fusiform, and giant aneurysms as compared with adult patients with aneurysms. Furthermore, improved short-term outcomes compared with those reported before surgical- and endovascular-treatment series are likely a reflection of continued improvements during the past 3 decades in endovascular and microsurgical techniques for aneurysm treatment and of improved early diagnosis of aneurysms and optimization of intensive care for pediatric patients.

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