Posterior Arterial Ischemic Stroke in Childhood
Clinical Features and Neuroimaging Characteristics

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Background and Purpose—Literature on the clinical manifestation and neuroradiological findings in pediatric patients with posterior circulation arterial ischemic stroke is scarce. This study aims to describe epidemiological features, clinical characteristics, and neuroimaging data on pediatric posterior circulation arterial ischemic stroke in Switzerland using the population-based Swiss Neuropediatric Stroke Registry.

Methods—Children aged from 1 month to 16 years presenting with an isolated posterior circulation arterial ischemic stroke between 2000 and 2016 were included. Epidemiology, clinical manifestation, stroke cause, and neuroradiological features were summarized using descriptive statistics. Stroke severity was assessed using the pediatric National Institutes of Health Stroke Scale. Correlation analysis was performed using the Spearman correlation coefficient.

Results—Forty-three children with posterior circulation arterial ischemic stroke were included (27 boys [62.8%], median age 7.9 years, interquartile range, 5 to 11.7 years). The incidence of posterior circulation arterial ischemic stroke is Switzerland was 0.183/100 000 and represented 16% of all childhood arterial ischemic strokes. Most patients presented with nonspecific neurological complaints, such as headache (58.1%) and nausea/vomiting (46.5%). The most frequent clinical manifestations were ataxia (58.1%) and motor/sensory hemisyndrome (53.5%/51.2%). Unilateral focal cerebral arteriopathy was the most common cause (11 children, 25.6%). Most infarcts were located in the cerebellum (46.5%) and thalamus (39.5%). A shorter diagnostic delay correlated with more severe stroke symptoms at presentation (rho= −0.365, *P*=0.016).

Conclusions—Pediatric posterior circulation arterial ischemic stroke was caused by focal cerebral arteriopathy in one quarter of the patients in our cohort. The frequently reported nonspecific clinical symptoms, especially when associated with mild neurological findings, risk delaying the diagnosis of stroke. A high index of suspicion and increased awareness are required for timely diagnosis and treatment initiation. **(*Stroke*. 2019;50:2329-2335. DOI: 10.1161/STROKEAHA.119.025154.)**

Key Words: ataxia ■ nausea ■ neuroimaging ■ pediatrics ■ stroke

Arterial ischemic stroke (AIS) occurs in ≈1.7 to 3 per 100 000 children per year and is an important cause of childhood morbidity.[1–4] Childhood AIS mainly occurs in the anterior circulation,[5] whereas only 15% to 22% of cases occur in the posterior circulation.[6–8] The most common causes of posterior circulation AIS (PCAIS) in children are extracranial vasculopathy and focal cerebral arteriopathy (FCA).[6,9] Epidemiological information on PCAIS in children is scarce and comes from hospital-based studies.[6,8] No population-based information on the incidence of pediatric PCAIS has been reported to date.

Pediatric PCAIS often presents with nonspecific symptoms, such as dizziness, nausea, vomiting, and headache—symptoms that are most often associated with benign and more common disorders in the pediatric population.[10] Therefore, PCAIS can be easily missed or diagnosed late, especially in young children. At a time when recanalization treatment is increasingly used in the pediatric population, timely recognition of pediatric PCAIS is crucial to enable initiation of the appropriate treatment.[11–14] Neuroimaging—especially magnetic resonance imaging (MRI)—plays a key role in the diagnosis of childhood PCAIS and in its acute and long-term management. Large infarctions may cause cerebral edema in the posterior fossa, leading to coma and death through obstructive hydrocephalus, tonsillar herniation, and brain stem compression, occurring most often within 72 hours after estimated stroke onset.[15] Furthermore, neuroimaging is important to rule out stroke mimics such as migraine, seizures, intracranial infections, vascular malformations, tumors, or reversible posterior leukoencephalopathy syndrome among others.[16]

This study aims to describe epidemiological data, clinical characteristics, and detailed neuroradiological features of a large, population-based pediatric cohort presenting with PCAIS in Switzerland.

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Methods

Study Population and Data Collection

The authors declare that all supporting data are available within the article and in the online-only Data Supplement. For the present study, all patients aged from 1 month to 16 years with a PCAIS registered in the Swiss Neuropaediatric Stroke Registry (SNPSR) between January 2000 and December 2016 were included. The SNPSR is a population-based registry prospectively collecting data on childhood stroke (children <16 years with AIS and cerebral sinus venous thrombosis) in Switzerland. All patients participating in the SNPSR or their legal guardians are asked to provide written informed consent. Furthermore, the SNPSR is approved by the cantonal ethics boards and by the Swiss Ministry of Health, allowing for basic data collection on all Swiss pediatric stroke patients. Details of the SNPSR have been published previously.[4,17]

AIS was defined as a focal neurological deficit of acute onset. Confirmation of the diagnosis by MRI was needed, showing infarction determined by localization of a lesion that was consistent with the neurological signs and symptoms. PCAIS was defined as symptomatic AIS in the vertebrobasilar territory. Patients with PCAIS recognized retrospectively from imaging studies in the context of acute AIS of the anterior circulation were excluded. Some patients included in this study have also participated in earlier studies on the SNPSR.[12,18,19]

Clinical data were extracted from the SNPSR. Stroke cause was classified according to the Childhood AIS Standardized Classification And Diagnostic Evaluation (CASCADE) system.[20] Diagnostic delay was assessed using the predefined categories of the SNPSR (≤6 hours, >6 hours–≤12 hours, >12 hours–≤24 hours, >24 hours). Stroke severity was assessed using the pediatric National Institutes of Health Stroke Scale (pedNIHSS). For patients diagnosed before 2011, the pedNIHSS was obtained retrospectively.[21,22] Age at stroke onset, sex, and signs and symptoms at initial presentation were noted.

Neuroimaging Features

MRI was initiated by the local care team and reviewed centrally. MRI was performed on 1.5- or 3.0-Tesla machines as the initial imaging modality. Sequences performed included diffusion-weighted imaging, apparent diffusion coefficient images, axial T2-weighted images, 3-dimensional time-of-flight magnetic resonance angiography (MRA) of the intracranial circulation, and contrast-enhanced MRA of the neck vessels. The presence of hemorrhagic transformation was evaluated on gradient-echo images (T2*) or susceptibility-weighted imaging, or—when parenchymal hematoma was present—on T2-weighted images. Vascular abnormalities on MRA were classified as absent, vessel lumen irregularity, stenosis, occlusion, or dissection.

Based on the New England Medical Center Posterior Circulation Topographical Classification System,[23] infarct location was classified as either proximal, middle, or distal according to the vascular supply of the infarcted regions (Figure I in the online-only Data Supplement). The proximal segment includes the vascular territories of the intracranial vertebral arteries (VAs) and the posterior inferior cerebellar arteries; the middle segment—that of the basilar artery (BA) including the anterior inferior cerebellar arteries, the penetrating branches and up to (but excluding) the superior cerebellar arteries; the distal segment includes the vascular territories supplied by the superior cerebellar arteries, distal BA (basilar tip), and the posterior cerebral arteries.

One neuroradiologist (Dr Slavova) and 3 neuropediatricians (Drs Bigi, Steinlin, and Grunt), blinded to the clinical information, reviewed the imaging studies to identify the area of ischemia and the affected vessels. For 5 children, no images were available for review, and posterior stroke classification was made based on external reports (case nos. 18, 19, 20, 39, 40).

Statistical Analysis

Variables were summarized using descriptive statistics. The yearly incidence and the 95% CIs of AIS and PCAIS in Swiss children <16 years was calculated from the figures published by the Swiss Federal Statistical Office (http://www.bfs.admin.ch). Group comparisons were made using nonparametric statistics (Mann-Whitney *U* Test), and correlations were calculated using the Spearman correlation coefficient. A 2-sided *P* value of <0.05 was considered statistically significant. Analysis was performed using SPSS (Version 25, IBM Corp, Armonk, New York).

Results

Study Population and Epidemiology of AIS and PCAIS in Switzerland

During the study period, 266 children <16 years were diagnosed with AIS (100 females [38%]; median age 5 years 9 months, interquartile range, 1.9–11.6). Forty-eight pediatric patients with posterior circulation events were identified, and the anatomic location of the infarct was identified from imaging at presentation. Five patients in whom anterior circulation ischemia was also evident were excluded from our study.

Figure 1 shows the annual incidence of AIS and PCAIS over the study period. The overall incidence of AIS in Swiss children <16 years was 1.13 per 100 000 inhabitants (95% CI, 0.97–1.29), whereas the incidence of PCAIS was 0.18 per 100 000 inhabitants (95% CI, 0.13–0.24). The incidence of PCAIS remained stable over the study period.

The baseline characteristics, cause, and treatment of the remaining 43 patients are summarized in Table 1. Twenty-seven patients (62.8%) were male. The median pedNIHSS was 3 (interquartile range, 2–6) and did not differ significantly between girls and boys. The most common stroke cause was unilateral FCA (25.6%), followed by aortic/cervical arteriopathy (20.9%). Most of the patients (69.8%) were treated with aspirin monotherapy. Recanalization treatment was performed in 2 children (4.6%): both patients suffered from thrombosis in the BA, presenting with severe clinical symptoms (tetraparesis, aphasia) and a pedNIHSS of 21 and 25, respectively.

Neuroimaging Findings

The neuroimaging findings of the 43 study patients are illustrated in the Table I in the online-only Data Supplement. Since some patients had >1 infarction in >1 vascular territory within the posterior circulation, 46 distinct posterior infarct locations (confined to the vascular territories of the VA, posterior inferior cerebellar arteries, BA, anterior inferior cerebellar arteries, superior cerebellar arteries, and posterior cerebral arteries) were identified. Seventeen patients (39.5%) had infarctions in multiple locations within the posterior circulation. Most PCAISs were located in the distal segment (28 events, 65.2%), 14 (32.6%) were in the middle segment, and 13 (30.2%) were confined to the proximal segment. The most commonly affected vascular territory was that of the posterior cerebral artery (n=19, 29.7%) followed by the posterior inferior cerebellar artery (n=12, 18.8%). BA infarctions accounted for 26.5% (11/43 patients) of the PCAIS cases. Least common were infarctions in the vascular territory of the medullary VA branches (in only 2 patients, 3.1%). MRA was available for 36 of 43 patients (83.7%) and was abnormal in 20 patients (55.6%). Five patients (13.9%) had multiple abnormalities, that is, of >1 vessel within the posterior circulation. Computed tomography angiography was available in 8 out of 36 patients (22%, case nos. 23, 26, 28, 32, 34, 42, acquired before MRA) and revealed no additional information in comparison with MRA.

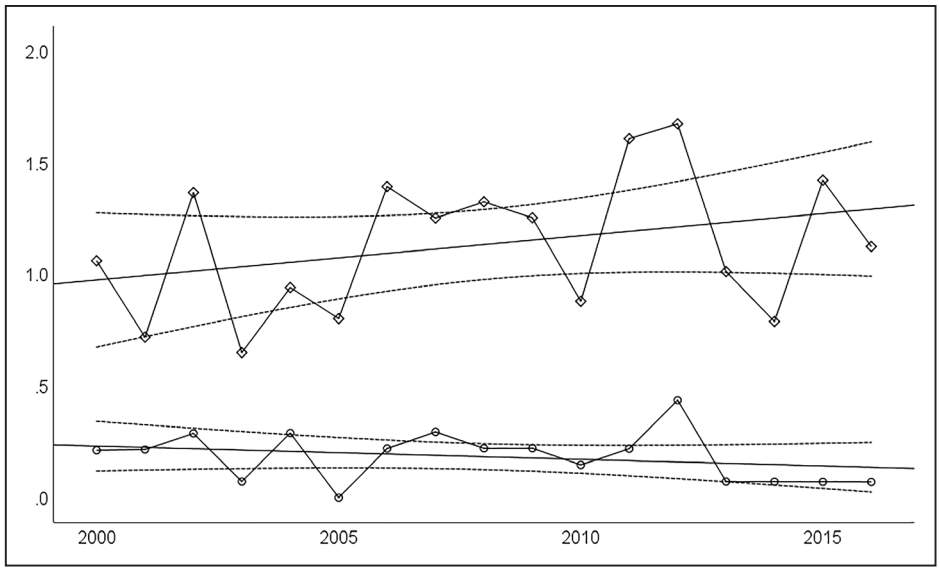

Figure 1. Incidence of arterial ischemic stroke (AIS) and posterior circulation AIS (PCAIS) in Swiss children <16 y. The lower line (open circles) represents the yearly incidence of PCAIS (cases per 100 000 inhabitants <16 y) registered in the Swiss Neuropaediatric Stroke Registry (SNPSR) from 2000 to 2016. The upper line (diamonds) represents the yearly incidence of pediatric AIS cases registered in the SNPSR in the same period. The solid line represents the regression line with the 95% CI (dashed lines).

Vascular pathological findings at diagnosis included stenosis in 17 children (39.5%), occlusion in 11 children (25.6%), dissection of the VA in 3 children (7.0%), vessel wall irregularity in 1 child (2.3%), and 1 child (2.3%) had Moyamoya disease with no evidence of infarctions in the anterior circulation. In 16 children (37.2%), no underlying vascular abnormality was identified on imaging despite evident infarctions,

The most common anatomic location of PCAIS was the cerebellum (n=25, 36.2%), followed by the thalamus (n=19, 27.5%) and the pons (n=11, 15.9%). None of the patients in our study population with posterior cerebral artery occlusion showed temporal lobe infarction. Hemorrhagic transformation was already present in 3 children (7.0%) on initial neuroimaging. Two of them were diagnosed within the first 6 hours of stroke onset; the third patient had a diagnostic delay of >48 hours. Two cases are illustrated in Figures 2 and 3.

Clinical Manifestation and Association With Infarct Localization

Clinical manifestations and infarct localization are summarized in Table 2. Most patients presented with nonspecific neurological symptoms, such as headache (58.1%), nausea/vomiting (46.5%), or dizziness (32.6%). The main clinical findings were ataxia (58.1%), motor and sensory hemisyndrome (53.5% and 51.2%, respectively), altered consciousness (34.9%), and cranial nerve palsy (32.6%). Infarct topography on neuroimaging correlated with symptoms at presentation. Of the 20 (47%) patients with infarction of the cerebellum, 15 (75%) had ataxia. Of the 17 (40%) patients with AIS in the thalamus, 13 (76%) presented with a motor or sensory hemisyndrome. Eleven (26%) patients had an infarction in the pons, of whom 5 (45%) presented with facial palsy or oculomotor involvement. Hemi-anopsia was the presenting symptom in 4 (57%) of the 7 patients with occipital stroke.

Diagnostic Delay and Stroke Severity

In 12 (27.9%) children, PCAIS was diagnosed within 6 hours from symptom onset, whereas more than half of the patients received their PCAIS diagnosis >12 hours after symptom onset (Table 1). Shorter diagnostic delay correlated significantly with initial pedNIHSS (rho=−0.365, P=0.016). Age at presentation showed no correlation with initial stroke severity (pedNIHSS) or diagnostic delay.

Discussion

This study assessed the clinical and neuroimaging features presented by pediatric PCAIS patients in a population-based cohort. The main findings of the study were (1) The incidence of PCAIS is 0.18/100 000 Swiss children and adolescents aged <16 years; (2) arteriopathy, mainly unilateral FCA, was the most common cause of PCAIS; (3) children with PCAIS frequently present with nonspecific symptoms, such as headaches and nausea/vomiting; (4) infarct topography on neuroimaging correlates with symptoms at presentation, and PCAIS most commonly involves the distal segments, in line with previous reports; (5) more than half of the PCAIS patients experienced a diagnostic delay of >12 hours; and (6) initial stroke severity correlates with time to stroke diagnosis.

Epidemiology and Demographics

In the present study, we found a frequency of PCAIS of 16% when compared with the frequency of overall AIS, and a yearly incidence of 0.18/100 000 Swiss inhabitants aged <16 years. To our knowledge, this is the first population-based incidence calculated for PCAIS. Previous reports on epidemiology of PCAIS are either based on extrapolations from hospital-based studies, and therefore at risk for referral bias, or include patients in whom anterior circulation stroke was also evident.[6,8] This could explain the higher incidence of PCAIS reported in the study by Carey et al[8] (crude incidence

Table 1. Baseline Characteristics, Cause, and Treatment of the Cohort (N=43)

Demographic Characteristics	
Age, y, median (IQR)	7. 9 (5–11.7)
Male, N (%)	27 (62.8)
pedNIHSS, median (IQR)	3 (2–6)
Diagnostic delay, h	
≤6, n (%)	12 (27.9)
>6–≤12h, n (%)	5 (11.6)
>12–≤24, n (%)	12 (27.9)
>24, n (%)	14 (32.6)
Stroke cause, N (%)	
Small vessel arteriopathy	4 (9.3)
Unilateral focal cerebral arteriopathy	11 (25.6)
Bilateral cerebral arteriopathy	4 (9.3)
Aortic/cervical arteriopathy	9 (20.9)
Cardioembolic	5 (11.6)
Other	9 (20.9)
Multifactorial	1 (2.3)
Treatment, N (%)	
Aspirin monotherapy	30 (69.8)
LMWH monotherapy	2 (4.7)
Aspirin and LMWH	8 (18.6)
IV lysis	1 (2.3)
Mechanical thrombectomy	1 (2.3)
None	1 (2.3)

IQR indicates interquartile range; IV, intravenous; LMWH, low–molecular weight heparin; and pedNIHSS, pediatric National Institutes of Health Stroke Scale.

of PCAIS of 0.38/100 000 children per year) and the frequency of 37% for PCAIS in the study by Mackay et al.[6] Mackay et al[6] also concluded that the high number of cases reported in their study may be attributable to better detection of PCAIS with the advantage of MRI. Nevertheless, in our study, MRI was performed in every child, and we did not find more cases of PCAIS compared with previous studies where MRI was not necessarily used. However, more epidemiological studies are required to more accurately estimate the frequency and incidence of pediatric PCAIS.

The incidence of PCAIS in children and adults is similar. The literature on adult posterior circulation stroke reports that PCAIS occurs in 15% to 28% of all adults with AIS.[8,23,24] Considering the different risk factors and causes of children and adults,[18] this is an interesting finding.

The male predominance reported in overall childhood AIS is also seen in children with PCAIS. The frequency of 62.8% male PCAIS stroke patients in our study is in line with previous ones, which reported frequencies of PCAIS in male pediatric patients of 72% and 76%, respectively.[6,9] This increased frequency is reported to be due to increased head and neck risk factors, such as trauma, dissection, and cervical spine abnormalities in boys.[6,7,25] Ganesan et al[7] reported that the greatest proportion of males with PCAIS occurred among children with AIS and a history of trauma (75%, P=0.008). Furthermore, patients with PCAIS are older compared with AIS patients overall (7.9 versus 5.8 years). This finding is in line with a study by Uohara et al,[9] reporting a median age of 9.2 years for PCAIS patients and a median age of 7.7 years for overall AIS. An age- and localization-related variation in the prevalence of risk factors, such as arterial dissection due to trauma, in older male patients might be one explanation for this age difference.[6,9] Another explanation might be that cardioembolic stroke frequently occurs in young children and affects the anterior circulation more than the posterior circulation.

Cause of PCAIS

Arteriopathy is a well-known cause for pediatric AIS.[26–28] In our study, FCA was the most common cause of PCAIS. Uohara et al[9] compared causes of anterior and posterior AIS, classified by CASCADE.[20] Although cardioembolic events were identified as the most frequent cause in anterior AIS, extracranial (cervico-aortic) vasculopathy was the most common stroke subtype of PCAIS (accounting for 43.5%).[9] The predominance of extracranial vasculopathy in the study by Uohara et al[9] was explained by a potential referral bias in a single-center study at a tertiary care center. Arteriopathy was also the most common stroke cause in our study. However, we noted a slightly higher frequency of FCA (25.6%) than aortic/cervical arteriopathy (20.9%), mainly cervical artery

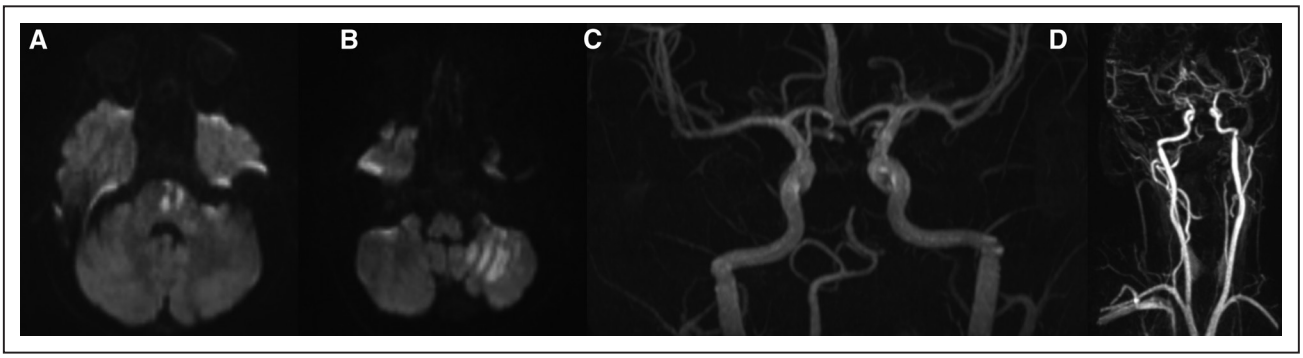

Figure 2. Images from a 7-y-old boy with a history of varicella infection 1 mo before stroke onset (case number 34). Diffusion-weighted imaging (**A** and **B**) revealed pontine and left cerebellar infarctions. Time-of-flight magnetic resonance angiography (MRA) maximum intensity projection (MIP) reconstruction imaging (**C**) showed absence of flow through the distal basilar artery and the left vertebral artery, indicating occlusion of the basilar artery and of the left vertebral artery in its intracranial segment (V4). Contrast-enhanced MRA MIP reconstruction image (**D**) confirmed the findings.

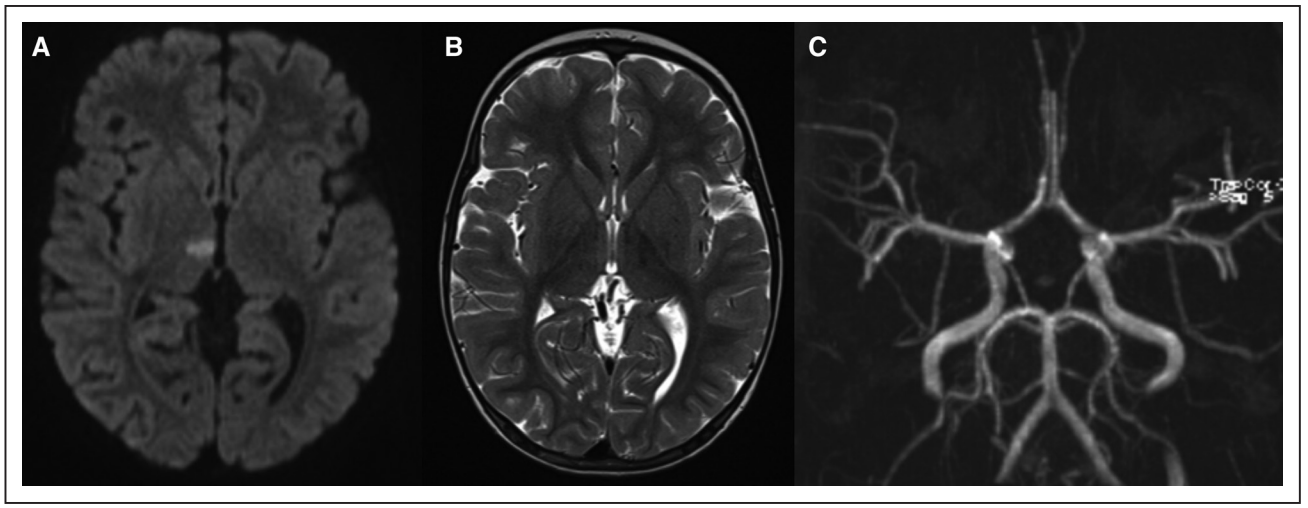

Figure 3. Images from a 2-y-old girl with congenital heart disease (case number 25). Diffusion-weighted imaging (**A**) revealed an acute right medial thalamic infarction, not yet demarcated, with a normal T2-weighted image (**B**). Time-of-flight magnetic resonance angiography (MRA) maximum intensity projection reconstruction (**C**) showed no vessel occlusion, especially patent basilar tip and posterior cerebral artery on the right. The absence of vessel irregularity, stenosis or occlusion on MRA, and the clinical history suggested a thromboembolic event.

dissection. To detect a cervical dissection reliably, dedicated T1-weighted fat-saturated imaging of the neck vessels would have been valuable. This sequence was unavailable for most of our patients because MRI protocols differed between centers. Therefore, the lower percentage of cervical artery dissections in our PCAIS population might be attributable to under-recognition of this condition based on our imaging data. We could have missed some dissection cases in the absence of secondary dissection signs, such as lumen narrowing, intima flap, or occlusion on MRA. Furthermore, it is important to realize that FCA is a purely morphological and descriptive means of classification and refers to unifocal stenosis or occlusion according to the CASCADE classification. It does not distinguish between intrinsic vessel wall disease versus partial or complete apparent stenosis or occlusion from a thrombus; that is, the important distinction between inflammatory versus undetermined cause remains unsolved. As recanalization techniques are increasingly used in pediatric patients, early determination of inflammatory cause is becoming more pressing. Being aware of the high frequency of (potentially

Table 2. Clinical Manifestation and Association With Infarct Localization (N=43)

Infarct Localization	Median Age, y	Median pedNIHSS	Symptoms				Signs								
			Headache	Nausea/ Vomiting	Dizziness	Seizure	Altered Consciousness	Ataxia	Hemi-Mot	Hemi-Sens	Cranial Nerves	Facial Palsy	Oculomotor	Hemi-Anopsia	Dys-Arthria
Cerebellar (n=20)	7.3	3	14	14	9	0	10	15	10	10	7	5	7	1	9
Isolated cerebellar (n=10)	7.8	3	6	8	4	0	4	7	2	2	2	0	3	0	3
Thalamic (n=17)	9.8	5	10	4	4	2	5	8	12	13	4	5	5	2	7
Isolated thalamic (n=10)	8.2	4.5	4	2	2	2	2	3	7	6	1	2	2	0	3
Pons (n=11)	7.5	4	8	6	4	0	4	7	8	7	4	5	5	1	5
Isolated pons (n=4)	10.3	3	2	1	1	0	1	2	3	2	2	2	2	0	1
Mesencephalon, all isolated (n=1)	4.2	1	1	1	0	0	1	1	0	0	0	0	0	0	0
Medulla oblongata, all isolated (n=3)	8.9	6	1	1	1	1	1	2	2	2	2	2	0	0	0
Parietal (n=2, none isolated)	8.6	3.5	0	0	0	0	0	1	0	1	1	0	0	2	1
Occipital (n=7)	8.9	2	4	3	1	0	1	3	1	3	2	0	0	4	1
Isolated occipital (n=2)	9.2	1.5	2	1	0	0	0	0	0	0	1	0	0	2	0

Hemi-mot indicates hemimotor syndrome; Hemi-sens, hemisensory syndrome; and pedNIHSS, pediatric National Institutes of Health Stroke Scale.

inflammatory) FCA in the PCAIS population is particularly important, as the use of recanalization techniques in patients with an underlying FCA of presumed inflammatory origin remains contentious.

Neuroimaging and Clinical Manifestations of PCAIS

Brain MRI should be considered the gold standard for pediatric stroke diagnosis and is superior to computed tomography[5,6] because it precisely detects ischemic lesions, which computed tomography cannot do. Particularly in the case of PCAIS, there is a risk of missing the infarcted area if computed tomography alone is performed. Furthermore, as children often present with nonspecific neurological complaints, MRI can provide additional information about nonvascular disorders that could mimic posterior stroke, like tumors, postictal conditions (Todd paresis), migraine, metabolic changes, intracranial infection, demyelinating diseases, and parainfectious conditions.[16] In our study, we used the advantages of MRI to correlate stroke location and symptoms at presentation focusing on PCAIS only without overlap of symptoms attributed to the anterior circulation. Focal neurological symptoms were correlated with the topography of infarct location. Using neuroimaging, we determined both anatomic location of stroke and segmental vascular topography. Together with the findings from MRA, this enabled us to better identify stroke cause because abnormalities of the cerebral vasculature like arterial dissection, FCA of childhood, Moyamoya syndrome, etc are known to be predisposing factors for childhood AIS.[29] To our knowledge, this is the first study to assess both anatomic vascular pathology on MRA and topographical location of the ischemic changes in pediatric PCAIS based on the knowledge of vascular territories of the posterior circulation,[6,23,30] in combination with symptoms at presentation. Previous studies have established the correlation between vascular topography of the infarct and the MRA findings, including in patients with coexistent stroke in the anterior circulation.[6] The focus was on outcome in correlation with the segmental distribution or specified anatomic location in cases of isolated VA dissection or recurrence only.[9] Our study provided data on anatomic location as well as vascular topography in isolated PCAIS cases. This may help to recognize PCAIS more quickly in the clinical setting, with a particular focus on the underlying cause, and enable timely initiation of appropriate treatment.

Delay in Pediatric Stroke Diagnosis

A greater awareness of AIS in the pediatric population would lead to more timely diagnosis.[11] In the present study, a statistically significant correlation between severity of stroke at initial presentation manifestation and diagnostic delay was found; that is, children presenting with more severe symptoms were also likely to be diagnosed sooner. This results in a more timely diagnosis of children with severe stroke symptoms. However, more than half of the children with PCAIS were diagnosed >12 hours after symptom onset—outside the accepted time window for recanalization treatment—even in the vertebrobasilar territory. Furthermore, early recognition of PCAIS is crucial. Particularly in patients with FCA—a frequent cause of PCAIS—stuttering symptoms and propagating infarctions are a risk for development of space-occupying cerebral edema in the posterior fossa. These conditions lead to coma and death through obstructive hydrocephalus, tonsillar herniation, and brain stem compression, most often within 72 hours after estimated stroke onset.[15] A high index of suspicion and increased awareness are essential for a timely diagnosis of pediatric PCAIS. In summary, early diagnosis of stroke in the posterior circulation is particularly important for 2 main reasons: First, recanalization treatment can be considered in selected cases, for example, if PCAIS is due to a cardioembolic event. Second, complications due to large or propagating infarctions can be minimized.

A major strength of our study is the population-based nature of the data collection. By focusing exclusively on PCAIS, we were able to collect information on a more homogeneous cohort with regard to the vascular territory. The limitations include the retrospective analysis of the data and the lack of standardized neuroimaging protocols leading to a risk of underdiagnosis of certain causes, such as cervical dissection.

Conclusions

PCAIS accounts for a small but important proportion of overall AIS, with an incidence of 0.18/100 000 Swiss children and adolescents aged <16 years. Patients frequently present with nonspecific symptoms and signs; stroke severity at onset is often mild, and this leads to a delay in diagnosis. The high frequency of underlying FCA in our study has implications for the acute management of pediatric PCAIS patients. Systematic MRI protocols with sequences sensitive enough to detect arterial dissection are important.

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Disclosures

None.

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