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Paediatric Stroke: Diagnostic Strategies and Therapeutic Dilemmas

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Abstract

Background. Paediatric arterial ischemic stroke is a potentially disabling neurological condition that remains underrecognized due to its rarity and atypical clinical presentation. Diagnosis in children is often delayed due to heterogeneous clinical presentation and differences in stroke mechanisms compared with adults. Despite advances in neuroimaging and acute reperfusion therapies, evidence-based paediatric-specific diagnostic and treatment strategies remain limited.

Aim. The aim of this review was to summarise current evidence regarding the epidemiology, pathophysiology, diagnostic approach, and management of acute ischemic stroke in the paediatric population.

Material and methods. A narrative review of the literature was conducted using structured searches of PubMed, Google Scholar, and relevant clinical guideline repositories. Articles published between 2015 and 2025 were considered. The search included original research articles, systematic reviews, meta-analyses, and international clinical guidelines. The review focused on studies addressing risk factors, clinical presentation, neuroimaging techniques, acute treatment, and secondary prevention of paediatric ischemic stroke.

Results. Paediatric stroke differs significantly from adult stroke in terms of etiology, clinical features, and outcomes. The most frequent causes include cerebral arteriopathies, congenital heart disease, infections, and haematological disorders, including sickle cell disease. Magnetic resonance imaging remains the diagnostic modality of choice, with diffusion-weighted imaging, arterial spin labelling, and vessel wall imaging improving diagnostic accuracy. Intravenous thrombolysis and mechanical thrombectomy appear feasible and relatively safe in selected paediatric patients; however, high-quality evidence supporting their efficacy is still limited.

Conclusion. Paediatric ischemic stroke remains a significant diagnostic and therapeutic challenge. Delayed recognition, heterogeneous underlying mechanisms, and a lack of robust paediatric-specific clinical trials continue to limit optimal management. Further studies are needed to establish evidence-based guidelines, optimize imaging protocols, and refine acute and long-term treatment strategies to improve outcomes in this vulnerable population.

Key words: paediatric stroke, ischemic stroke, neuroimaging, thrombolysis, mechanical thrombectomy, acute treatment

1. Introduction

Stroke is a leading cause of death and disability worldwide and concerns all age groups. It is commonly thought that stroke is a disease which occurs in the elderly people, but it is important to highlight the fact that it also affects infants and children resulting in lifelong neurological disability [1]. More than half children after stroke would have permanent neurological deficits, especially hemiparesis, epilepsy and motor deficits [2]. The World Health Organization defines stroke as 'rapidly developing clinical signs of focal or global disturbance of cerebral function, with symptoms lasting more than 24 hours or leading to death, with no apparent cause other than vascular origin' [3]. It is crucial to recognise alarming symptoms which include sudden unilateral weakness, numbness, visual loss, speech difficulties, vertigo, headache [4]. In contrast paediatric stroke have various clinical presentations which cause delay in diagnosis or misdiagnosis [5] . Paediatric stroke is typically divided into neonatal stroke diagnosed between birth and 28 days of life and childhood stroke diagnosed between 29 days and 18 years old [6]. Numerous studies show that the median time from onset of symptoms to diagnosis of Acute Ischemic Stroke (AIS) is more than 20 hours [1]. Multiple significant physiologic differences

in the paediatric and adult brain lead to different clinical presentations of stroke. Studies show that by the age of five, the brain uses up to 200% more glucose, which makes them more susceptible to focal neurological damage during episodes of ischemia [5]. Paediatric, as well as adult strokes, are a medical emergency and should be treated promptly to maximize beneficial patient outcomes. Clinical presentations include focal and diffuse neurological deficit, and non-specific systemic symptoms. The most frequent are seizures, hemiparesis, altered level of consciousness including coma, apnoea, headache, nausea, vomiting and fever [7]. It is essential to underline, there are many conditions that imitate stroke which can hinder diagnosis e.g. migraine with aura, epileptic seizure, facial nerve paresis, hypoglycaemia [8].

2. Research Materials and Methods

The review was conducted using Pubmed and Google Scholar databases searching key words: 'paediatric stroke', 'ischemic stroke', 'neuroimaging', 'thrombolysis', 'mechanical thrombectomy', 'acute treatment'. The review was written on the basis of 32 publications from 2015–2025. Inclusion criteria were: peer-reviewed original research articles, systematic reviews, meta-analyses and international clinical guidelines published in English. Exclusion criteria included single case reports, animal studies, conference abstracts without full text, and non-English publications.

3. Epidemiology

Stroke among the adult population is not uncommon, one in four people will suffer a stroke in their lifetime, furthermore strokes are the second most common cause of death and one of the most important causes of disability [6,9]. Last statistics from 2023 show that each year, more than 795,000 people in the United States have a stroke, and the mortality rate is about 19% [10]. Approximately 85% of strokes are ischemic strokes, caused by reduced blood perfusion usually resulting from artery obstruction [9]. The remaining 15% are haemorrhagic strokes caused by rupture of cerebral arteries, among which we can distinguish intracerebral and subarachnoid strokes [11]. Cerebellar stroke accounts for approximately 3% of all strokes. Its most common causes include embolism, atherosclerosis, and vertebral artery dissection. Symptoms of cerebellar stroke are nonspecific and include nausea, vomiting, and headache. It is estimated

that modifiable risk factors are responsible for about 90% of strokes [12]. These include smoking, diabetes, hyperlipidaemia, physical inactivity and excessive alcohol use [13]. The most significant risk factor to both ischaemic and haemorrhagic stroke is hypertension. Atrial fibrillation is a specific risk factor for ischemic stroke [14]. Among the paediatric population, stroke is classified as a rare incident, with an estimated incidence of 2-13 cases per 100,000 per year. Additionally, infants under 2 years of age and boys have a higher prevalence of stroke [15]. The mortality rate of strokes among children is roughly 10-25% [5]. Although the incidence of ischemic and haemorrhagic stroke are comparable, haemorrhagic stroke has a significantly higher mortality rate [16]. The most common risk factors for ischemic stroke in children population are arteriopathies including aortic dissection, Moyamoya disease, vasculitis, congenital heart diseases, infections, venous thrombosis of the cerebral sinuses and sickle cell disease.In contrast, major risk factors for haemorrhagic stroke include arteriovenous malformations, aneurysms, thrombocytopenia, coagulopathies including haemophilia A and liver failure [17]. Genetic disorders are an important risk factor for pediatric stroke, the most common are listed in Table 1.

Table 1. Genetics disorders associated with pediatric stroke [5,15].

	Description	Vacular complications
Marfan syndrome	**Autosomal dominant inheritance** characterized by ocular abnormalities, tall slender habitus with skeletal deformities, cardiovascular involvement including aortic dilatation, aneurysms and mitral-valve prolapse.	Arterial dissections
Ehlers-Danlos Syndrome-type IV	**Autosomal dominant inheritance** characterized by hyperextensible joints, easy bruising skin and vascular lesion	Aneurysm carotid-cavernous fistulae dissections
Pseudoxanthoma Elasticum	**Autosomal recessive inheritance** characterized by progressive skin changes	Intracranial aneurysm dissections moyamoya -like vasculopathy small vessel disease
Progeria	**Autosomal dominant inheritacne** premature aging characterized by alopecia lipoatrophy, atherosclerosis, joint contractures. Coronary artery disease, skeletal abnormalities such as mandibuloacral dysplasia	Cardiovascular disease stroke- involving large intracranial arteries vertebral & carotid arteries
Mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes (MELAS)	**Maternal inheritance** characterized by developmental delay, recurrent vomiting, headaches, deafness, muscle weakness. diabetes mellitus, renal disease, short stature, cardiomyopathy	Recurrent stroke-like episodes involving non-vascular territories
Fabry Disease	**X-linked inheritance** characterized by dysmorphic features, anhidrosis, angiokeratomas, acroparesthesia, ophthalmologic complications	Small vessel ischemic disease, intracranial arterial dolichoectasia intracerebral hemorrhage cardiogenic embolism
Von Hippel-Lindau Disease	**Autosomal dominant inheritance** characterized by pheochromocytomas, renal cell carcinoma, pancreatic cystadenomas, hemangioblastomas	Intracranial hemorrhage aneurysm
Sturge-Weber Syndrome	**Somatic mosaic R183Q mutation** characterized by port-wine stain, glaucoma, mental retardation, seizures, contralateral hemiparesis & hemiatrophy	Intracranial hemorrhage

4. Diagnosis

Stroke in children may lead to long-term morbidity. Rapid and efficient diagnosing process in acute ischemic stroke in children is critical for the future complications and patients life quality. A major challenge is the high number of conditions that can mimic stroke, such as postictal paralysis, migraine, hypoglycaemia, or alternating hemiplegia [18]. A detailed medical history is crucial and should include head and neck trauma, recent infections (especially varicella within the last 12 months), unexplained fever, exposure to toxic substances or drugs, developmental disorders, hematologic diseases, and a family history of neurological and

vascular disorders [19]. The primary diagnostic tool is neuroimaging, with magnetic resonance imaging (MRI) being the method of choice, as it outperforms computed tomography (CT) in detecting acute ischemia and allows differentiation from other causes of symptoms [20]. Although CT can detect intracranial haemorrhage, it should only be performed when other methods are not available. Radiation exposure and lack of sensitivity in early acute ischemic stroke make this method a second-line choice [21]. MRI is the most sensitive method to confirm stroke and should be obtained as soon as possible. MRI protocol consists of axial diffusion weighted imaging (DWI), T2-weighted or fluid-attenuated inversion recovery (FLAIR) sequences, susceptibility weighted imaging (SWI) or gradient recalled echo (GRE) for detection of blood. DWI is a sequence highly sensitive for ischemia and is included in rapid stroke MRI protocol [22]. Arterial spin labelling (ASL) is increasingly used to assess cerebral perfusion, as it does not require contrast administration and is particularly useful in children [23].

Table 2. Comparison of Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) in the Diagnosis of Pediatric Stroke [20,21].

MRI	VS	CT
High; better differentation of stroke mimics	SENTIVITY FOR ACUTE ISCHEMIA	Low; limited sensitivity, especially in early stroke; less specific
Long-term	ABILITY TO DETECT HEMORRHAGE	Short-term; immediate impact
No; safe for repeated use	RADIATION EXPOASURE	Yes
variable avaibility; longer scan time increasing accessibility	AVAILABILITY	widely available; faster to perform
first line; diagnostic tool	ROLE IN DIAGNOSTIC PROTOCOLE	second line; used if MRI unavailable

A complete stroke diagnosis should include cardiological, haematological, metabolic, and inflammatory tests, as well as cerebral vascular imaging [24]. Routine, extensive testing for thrombophilia is of limited value and should be reserved for cases with a family history or recurrent thrombosis [25]. Modern techniques, such as vascular wall imaging (VWI), can help

differentiate between arteriopathy and detect inflammatory processes, although their availability and clinical usefulness are still being researched [26]. Genetic testing is also playing an increasingly important role. Identifying a genetic etiology can guide family counselling, help estimate the risk of recurrence, and improve prognostic accuracy. In some cases, genetic findings can substantially alter clinical management. For instance, a diagnosis of adenosine deaminase 2 deficiency (DADA2) may lead to stopping antiplatelet therapy and starting treatment with TNF inhibitors [27]. Despite ongoing advances, significant gaps remain in our knowledge regarding the optimal scope of diagnostic testing in children with stroke.

5. Treatment

In the acute phase, management primarily focuses on rapid stabilization, targeted diagnostic evaluation, and urgent interventions aimed at preserving cerebral perfusion and minimizing secondary brain injury. Current treatment strategies emphasize a multidisciplinary approach involving paediatric neurologists, intensivists, haematologists, and interventional specialists [28]. Although therapeutic frameworks for adults, such as intravenous thrombolysis and mechanical thrombectomy, inform parts of paediatric care, their application in children requires careful consideration. Factors to consider include age-specific safety profiles, dosing, vascular anatomy, and the distinct spectrum of stroke etiologies in younger patients [29]. The latest guidelines on stroke in children state that recombinant tissue plasminogen activator (rTPA) can be used in children aged 2 years and older with persistent impairing neurological deficits (e.g., NIH Paediatric Stroke Scale score $\geq$ 6 at the time of intervention) and radiographically confirmed occlusion of a large cerebral artery, within 4.5 hours of the known onset of symptoms [18]. Currently, it is recommended that tPA be administered at an adult dose of 0.9 mg/kg intravenously, with the first 10% of the dose given as a bolus and the remainder over 1 hour. [28]. A 2020 study by Amlie-Lefond et al. retrospectively collected data on children treated with intravenous rTPA for arterial ischemic stroke at 16 former Thrombolysis in Paediatric Stroke Study (TIPS) [30]. It was shown that the overall risk of symptomatic intracerebral haemorrhage after intravenous rTPA administered within 4.5 hours of symptom onset was only 2.1%. For comparison, the risk in adults was 6.4%. The 2020 Save Childs study provided multicenter evidence for the use of mechanical thrombectomy in children with large vessel occlusion in ischemic stroke [31]. In recent decades, no new insights have been developed regarding standards of care when thrombolysis or endovascular treatment are not possible, and

most suggested protocols are based on traditional anticoagulant therapies. Failure to initiate anticoagulant therapy to prevent stroke is associated with a 1.5–2.5-fold increased risk of recurrence after the first episode [18]. Long-term treatment is usually based on low molecular weight heparins or acetylsalicylic acid, with no clear data suggesting which of these two therapies is better in children and no safety data supporting either approach [32]. Long-term preventive therapy should be considered in patients with genetically determined thrombophilia or in children with stroke following congenital heart defects. In other cases, the duration of treatment may depend on the underlying etiology. The preferred time frame usually ranges from 2 years to lifelong [18].

6. Conclusion

Paediatric ischemic strokes remain a significant diagnostic and therapeutic challenge, primarily due to their mechanisms of development, which differ from those in adults, and their frequent atypical clinical presentations. In the paediatric population, non-atherosclerotic intracranial arteriopathies, thromboembolic complications associated with congenital heart defects, and haematological diseases such as sickle cell anemia or coagulopathies play a key etiopathogenetic role. At the same time, strokes in children are often asymptomatic or atypical, which may delay diagnosis and prolong the time to treatment. Despite significant advances in diagnostics, including the development of neuroimaging techniques such as VWI and ASL MRI, there are still significant gaps in knowledge regarding both, the optimal diagnostic strategy and the treatment of acute stroke. Although there is a growing number of data indicating the feasibility and safety of intravenous thrombolysis and mechanical thrombectomy in children, there is still a lack of high-quality evidence confirming their effectiveness. The optimal dose and selection of thrombolytic drugs, as well as the criteria for eligibility for surgical treatment methods, have not been defined. However the diagnosis and treatment of stroke in children has improved significantly, there is a need for research to develop standardized, evidence-based guidelines for management. Understanding pathophysiological differences, developing advanced imaging techniques, and evaluating new therapeutic and rehabilitation methods are key to further improving the prognosis in this patient population.

Disclosure

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