

Hemorrhagic Stroke: About a Pediatric Case

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Abstract

Stroke is a rare but serious condition with high mortality and morbidity. We report the case of a 10-year-old child with no known pathological history who was transferred to the pediatric department for hemorrhagic stroke. He had been hospitalized 6 days earlier in a peripheral center for a tonic-clonic seizure accompanied by loss of consciousness and fever. Clinical examination revealed meningeal syndrome, facial paralysis, right hemiparesis, ataxia with disturbed balance, aphasia, and a peripapillary hemorrhage on the fundus. A cerebral computed tomography (CT) scan showed a hemorrhagic stroke with ventricular flooding. A lumbar puncture was performed and a hematic cerebrospinal fluid (CSF) with 12 leukocytes and 8000 red blood cells was obtained. The evolution was marked by the extinction of the infectious and meningeal syndromes, the regression of the pyramidal syndrome and the persistence of the dysarthria justifying a neuropsychological and speech therapy follow-up. Several challenges (diagnostic, therapeutic) exist in the management of children with stroke. Prospective studies with a larger sample are needed to fill the observed gaps.

Keywords

Hemorrhagic Stroke, Pediatrics, Intracerebral Hemorrhage, Subarachnoid Hemorrhage, Malaria, Mali

1. Introduction

Stroke occurs when normal blood flow to the brain is interrupted by either occlusion or rupture of blood vessels [1]. Pediatric stroke is a relatively rare and poorly documented event [2]. The reported incidence of pediatric stroke ranges

from 1.2 to 13 cases per 100,000 children under the age of 18 years [2] [3] [4]. There are two types of stroke: ischemic stroke or cerebral infarction, caused by the formation of a thrombus that clogs an artery, and hemorrhagic stroke due to the rupture of a cerebral artery or aneurysm, which will cause a cerebral or meningeal hemorrhage [3]. Nearly half of pediatric strokes are hemorrhagic [5] [6]. Generally, the term “hemorrhagic stroke” includes spontaneous intraparenchymal hemorrhage and nontraumatic subarachnoid hemorrhage [5]. Congenital heart disease, arrhythmias, infections (sepsis, meningitis, and encephalitis), and chronic anemia (sickle cell anemia and β -thalassemia) are the main risk factors associated with stroke occurrence [6] [7]. The majority of the signs of stroke is nonspecific and can be easily attributed to other causes [2]. The clinical presentation of stroke varies according to the age of the child. The younger the child is, the more atypical the symptoms may be. Hemorrhagic strokes most often present abruptly as severe headache rapidly associated with impaired consciousness. Vomiting, neck pain and neurological deficits may also occur. In younger children, convulsions can be revealing [1] [2] [8]. The diagnosis is clinical and radiological, affirmed by brain imaging primarily by magnetic resonance, diffusion and perfusion. In pediatrics, the etiologies of hemorrhagic stroke are dominated by vascular anomalies (arteriovenous malformations, aneurysms, cavernomas) and non-malformative causes (infectious, hematological, neoplastic, and toxic) [3] [4]. The etiological investigation includes a blood count and a minimal hemostasis workup (hemogram, prothrombin level, activated partial thromboplastin time, fibrinogen) performed urgently in all cases to rule out hemophilia and thrombocytopenic purpura [4]. Pediatric stroke is still associated with high morbidity, mortality and recurrence rates. Its management is not always easy because there are little data to support the effectiveness of interventions [2] [4] [5]. Current treatment of stroke is based on extrapolation from the adult literature and expert opinion, as there are no evidence-based guidelines, except in sickle cell disease [6] [7]. The purpose of this article is to report a case of hemorrhagic stroke in a child and to review the literature on pediatric stroke to provide information for optimal diagnosis and treatment.

2. Observation

This was a 10-year-old boy with no known history who was referred to the pediatric emergency and intensive care unit for hemorrhagic stroke. The patient had been transferred from a secondary health center where he had been hospitalized for six days for tonic-clinical convulsions in a febrile context, loss of consciousness, and a meningeal syndrome (neck stiffness, positive Brudzinski and Kernig signs).

The diagnosis of meningitis and neuromalaria was evoked and the patient received a bi-antibiotic therapy (ceftriaxone and gentamicin) and an artemisinin-based antimalarial treatment for 6 days.

In the pediatric emergency room, the general condition was preserved, the

temperature was normal (36.5°C), the blood pressure was 140/90mmHg and the cardiopulmonary examination was normal.

The neurological examination revealed a facial paralysis with labial deviation, a pyramidal syndrome (presence of Babinski's sign) associated with right hemiparesis, a cerebellar syndrome characterized by a disorder of balance and coordination of movements and Broca's aphasia. A cerebral CT scan revealed a left parietal hematoma of 53 × 37 mm surrounded by large edema responsible for a moderate subfascial involvement with ventricular flooding in favor of a hemorrhagic stroke. The cerebral angiography scan did not reveal any signs in favor of a vascular malformation.

The lumbar puncture had brought back a uniformly haematic CSF. Cyto-bacteriological examination of the CSF revealed 12 leukocytes and 8000 red blood cells. The thick blood drop had found 1100 trophozoites. The blood count showed a discrete normochromic microcytic anemia (hemoglobin = 10 g/dl) and a thrombocytosis of 490,000/mm³.

The haemostasis work-up revealed a prothrombin rate (PT) of 64% (Norms: 70 - 100), an activated partial thromboplastin rate (APTT) of 21 seconds (Norms: 28 - 39); INR = 1.60 (Norms: <2) and a fibrinogen level of 1.82 g/l (Norms: 2 to 4 g/l) Hemoglobin electrophoresis showed a normal profile (AA2). During his stay in the intensive care unit, the initial management consisted in collaboration with the neurosurgeons and neurologists, in the continuation of the probabilistic antibiotic therapy, in the control and in the strict and regular monitoring of the vital parameters (glycemia, cardiorespiratory function and neurological signs). After two months of follow-up, the evolution was marked by a regression of the neurological signs without their complete extinction (**Figure 1**).

3. Discussion

Hemorrhagic stroke is a rare but serious pathological entity in pediatrics. Failure to recognize it exposes surviving children to long-term neuropsychological sequelae [1] [6]. Symptoms of childhood stroke can sometimes be misleading and misinterpreted by clinicians and patients. Approximately half of children presenting with stroke have a previously identified risk factor [5] [7]. The clinical presentation depends on the patient's age, setting (inpatient or emergency department) and stroke subtype [8] [9]. Variable risk factors, atypical clinical presentation, and pathogenesis combine to create diagnostic uncertainty and management dilemmas [2] [10]. As a result, pediatric stroke is underappreciated and there may be delays in its diagnosis and management [10]. In young children, seizures are usually revelatory of stroke as was the case in this observation [1] [2] [7] [10].

In our epidemiological context, these signs will first evoke neuromalaria or meningitis, especially if they evolve in a febrile context. Sudden focal neurological deficits are the clinical features of stroke in 85% of affected children [2]. According to the literature, the most common signs of pediatric stroke are hemiparesis or

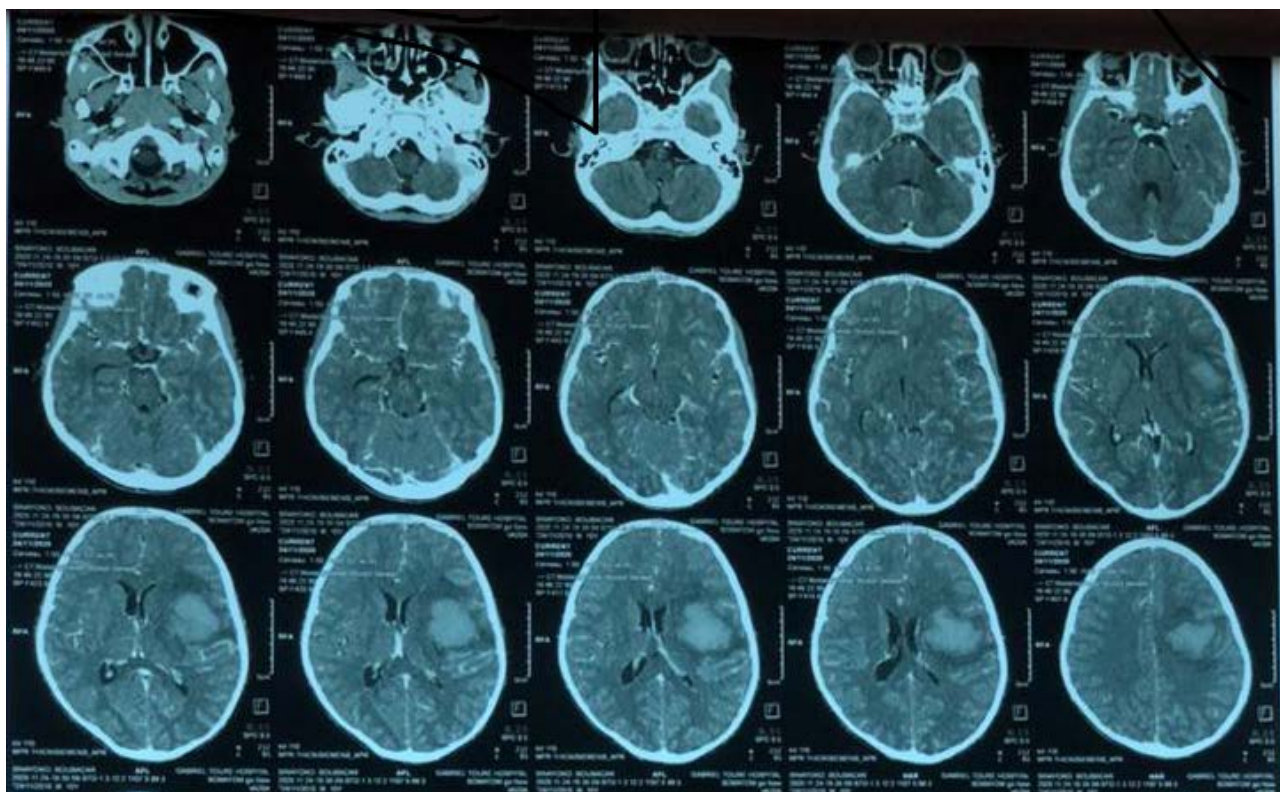


Figure 1. CT appearance in favor of hemorrhagic stroke with ventricular flooding.

hemifacial paralysis (67% to 90%), speech or language disorders (20% to 50%), vision disorders (10% to 15%), headache (20% to 50%), consciousness disorders (17% to 38%), and ataxia (8% to 10%) [1] [2] [7] [10]. Hemorrhagic strokes are more likely to cause vomiting [1] [2]. Bulbar dysfunction and dysarthria indicate brainstem involvement, whereas aphasia (or dysarthria) suggests basal ganglia, thalamus or cerebral hemisphere involvement [2]. The reference examination is cerebral MRI. It shows early the affected areas, whether it is ischemia or a hemorrhage [1] [2] [8] [10]. Angiomagnetic resonance imaging (MRI) allows visualization of the extent of brain damage, especially in the first few hours [1] [3]. MRI allows to search for the cause of the stroke on the cerebral vessels, but also on the cerebral vascularization itself and to consider its treatment, by privileging, when possible, the endo-vascular route (Interventional Neuroradiology) [1] [10] [11].

Cerebral CT, a first-line examination, retains its place in the emergency. Performed without injection of contrast medium, it allows eliminating a cerebral hemorrhage which is translated by a homogeneous spontaneous hyperdensity, well limited, associated with a mass effect [11]. CT allows a very reliable diagnosis (90% - 95% in the first 24 h) of subarachnoid hemorrhage (SAH), intracerebral or intraventricular hemorrhage, hydrocephalus, possible aneurysmal localization, intra-aneurysmal calcifications of giant aneurysms. Its sensitivity decreases thereafter due to blood resorption in the CSF, reaching 50% at 1 week and 30% at 2 weeks [1] [2]. The literature suggests a lumbar puncture in case of

suspected hemorrhagic stroke. A hemorrhagic fluid and especially xanthochromia confirm SAH [2] [3]. The risk of involvement in case of intracerebral hematoma imposes to do it after CT scan, in any case during the 72 hours after an acute accident.

According to several authors, the diagnostic delay for hemorrhagic stroke is shorter than for ischemic stroke [1] [2] [3] [12] and [13]. In this observation, the diagnosis was delayed not only because of the ambiguity of the clinical picture but also because of the late performance of cerebral CT. Delays in diagnosis and management can lead to parental anxiety and refusal of treatment.

Unlike adults, the causes of pediatric stroke are diverse and include vascular, infectious, hematologic, neoplastic, and toxic etiologies [1] [5]. Arteriovenous malformations are much more common than aneurysms, cavernomas and other non-malformative pathologies [1] [2]. Treatment of hemorrhagic stroke is based on treatment of the cause [1]. The prognosis depends primarily on the time to diagnosis and management. Hemorrhagic stroke has a significantly higher mortality rate than ischemic stroke. The literature reports that approximately 10 to 25% of children with stroke will die and up to 25% of children will have a recurrence [3] [14]. Certain factors are predictive of higher mortality: age less than three years; intracranial bleeding due to hematological disorders or aneurysmal pathology; subtentorial location; and admission GCS score less than or equal to 7 [3] [11] and [14]. In all cases, the child should benefit from regular clinical and radiological follow-up with multidisciplinary collaboration.

4. Conclusion

Hemorrhagic stroke is a medical emergency and requires early diagnosis and prompt treatment to avoid late sequelae and improve quality of life. Increased training of practitioners in the early detection of signs and symptoms may contribute to earlier diagnosis and reduced sequelae. Many challenges exist in the recognition and management of stroke, hence the need to equip our facilities with MRI and prospective multicenter and multidisciplinary studies focused on the epidemiological, diagnostic, and therapeutic aspects of hemorrhagic stroke.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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