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Case Report

Post-traumatic ischaemic stroke in a teenager after head trauma: A case report

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ABSTRACT

Background: Lacunar strokes in the pediatric population are very uncommon, as well as trauma-induced strokes. It is extremely rare for a head trauma induced ischaemic stroke to occur in children and young adults.
Case report: We describe a case of a 13-year-old boy who reported acute ischaemic lesions, and in particular a right basal ganglia ischaemic stroke after falling from a height of 10 m, presumably secondary to the stretching-induced occlusion of the recurrent artery of Heubner, with a favorable outcome.
Conclusion: Ischaemic strokes can rarely be subsequent to head trauma in young adults, in relationship with the degree of maturity of the perforating vessels. Although very rare, it is important to avoid the lack of recognition of this condition, thus awareness is necessary.

Introduction

Childhood acute ischaemic stroke is a rare condition, since it has an annual incidence of 2.3/100,000 per year [1], and comprises just 1 %–5 % of all strokes [2]. Risk factors of ischaemic stroke differ greatly between adults and children, being associated with hypertension and atherosclerosis in the former, and with congenital heart diseases, vascular or prothrombotic anomalies, or genetic or metabolic diseases in the latter [3,4].

Furthermore, basal ganglia ischaemic stroke in childhood represents a particularly infrequent event, accounting for only 6 % of all ischaemic strokes [5].

A lacunar stroke in children may be caused by trauma as an extremely rare event [6], and in the literature there is scant description of basal ganglia or internal capsule ischaemic stroke subsequent to head trauma, especially in children over 6 years of age [7–10], in fact ischaemic stroke due to minor head trauma accounts for <2 % of all the cases [11]. Most occurrences of cerebral artery occlusion are related to dissecting aneurysms, vasospasm, embolus, thrombus or congenital vascular anomalies, but ischaemic stroke limited to basal ganglia subsequent to minor head trauma in childhood is a very rare event, and it is attributed to intimal trauma with thromboembolism and transient arterial spasm of the small perforating arteries that originate from the middle cerebral artery, the medial lenticulostriate arteries and the anterior cerebral artery [7,12]. Moreover, since ischaemic strokes in children usually involve the anterior cerebral circulation, a damage to the posterior circulation leading to occipital strokes is a very infrequent event, as well [13].

Here we present a case of a teenager who was referred for a polytrauma and who presented acute ischaemic stroke lesions.

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Case presentation

A 13-years-old boy with no significant past medical issues was brought to our emergency room after an unintentional falling from a height of 10 m, while playing on the rooftop of an apartment building.

The patient reported thoracic, abdominal and skeletal injuries (Injury Severity Score ISS > 15), and a moderate head trauma (GCS 13: E4, V3, M6). The teenager was in a confusional state, his pupils were normal sized, isochoric and normoreactive.

Orotracheal intubation and pleural drainages were required due to chest trauma with hypoxia, hypotension, tachycardia and bilateral pneumothorax. Adequate and timely interventions allowed the SpO2 never to fall below 90 %, while the mean arterial pressure was maintained over 65 mm Hg. Splenic rupture resulted from blunt abdominal trauma required an emergency splenectomy and red blood cells transfusion.

Upon arrival, the first head computed tomography (CT) was read as normal (Fig. 1), with no skull fractures, brain contusions, or intraparenchymal or extra-axial hemorrhages. Furthermore, no injuries to the cervical carotid and vertebral arteries, potentially leading to postinjury ischaemic stroke or cerebral infarction [14], were found. Further immediate imaging showed bilateral pulmonary contusion and pelvic fractures.

The next day a second CT was performed (Fig. 2), and a right basal ganglia ischaemic stroke (approximately 3 cm), was found, in particular affecting the anterior limb of the internal capsule, and the angiogram did not show any signs of arterial dissection or arterial occlusion. Two further small temporo-parietal cortico-subcortical hypodense areas were found. No intracranial hemorrhages did manifest.

Since the ischaemic lesions were localized in unusual sites, inconsistent with a hypoxic-ischaemic brain injury due to a marked decrease in oxygen or blood flow, a Magnetic Resonance (MRI) imaging was performed at 48 h after the trauma (Fig. 3), in order to deepen the study of the extension of the lesions and their underlying mechanisms. Restricted diffusions were diagnosed on diffusion-weighted imaging (DWI) in the bilateral fronto-parietal and in the right temporo-occipital cortico-subcortical areas, involving the hippocampus, the putamen, the head of the caudate nucleus, and the anterior limb of the internal capsule. The MR angiography confirmed the absence of vascular alterations such as dissecting aneurysms, vasospasm, embolism.

A transcranial Doppler ultrasound was used to assess cerebral circulation, and normal mean cerebral blood velocity was demonstrated.

Since the finding of a traumatic lacunar infarct in childhood is a very rare event, a transthoracic echocardiography was performed, but no patent foramen ovale or other anomalies were found.

Laboratory investigations, such as complete blood count, hematocrit level, prothrombin time, activated partial thromboplastin time, erythrocyte sedimentation rate, anticardiolipin, antinuclear antibody, rheumatoid factor, complement levels, lupus anticoagulant assay, homocysteine in plasma or urine, creatine phosphokinase, and coagulation panel, including protein C, protein S, antithrombin III, factor VIII levels, fibrinogen levels, plasminogen levels, factor V leiden, were all normal.

After 6 days upon arrival, a percutaneous tracheostomy was performed at the bedside in the ICU, and on day 8 the analgosedation was suspended: the patient rapidly regained conscience and spontaneous upper and lower limbs movements; reflexes were normal and symmetric. At day 14 the child was decannulated, oral feeding was resumed and he was discharged to an acute care rehabilitation at day 22. During the hospitalization he did not develop any neurological deficit or convulsive episodes. At day 39 the patient was discharged home with full muscular strength, and his cognitive performances were acceptable, apart from a mild cognitive dysfunction and a probable left homonymous hemianopsia, probably related to the parietooccipital stroke.

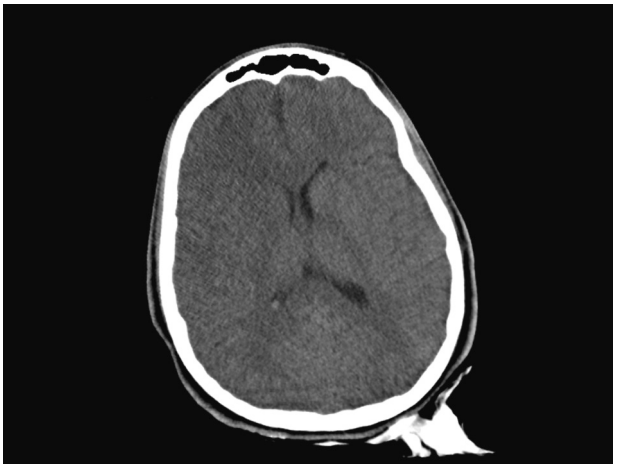

Fig. 1. Head CT scan read as normal.

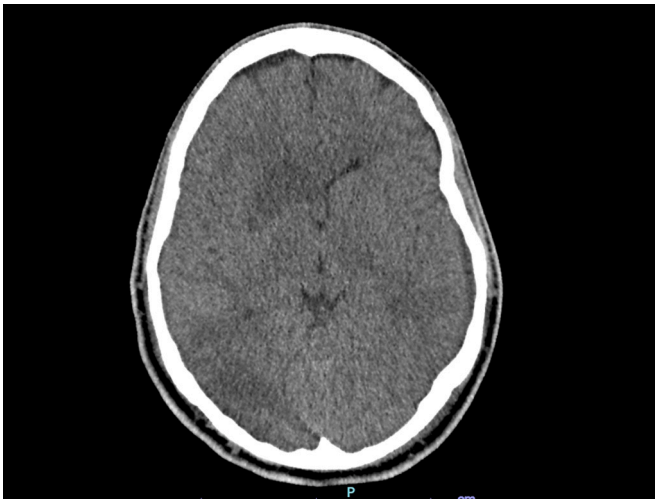

Fig. 2. Head CT scan showing a right basal ganglia ischaemic stroke and two small temporo-parietal cortico-subcortical hypodense areas.

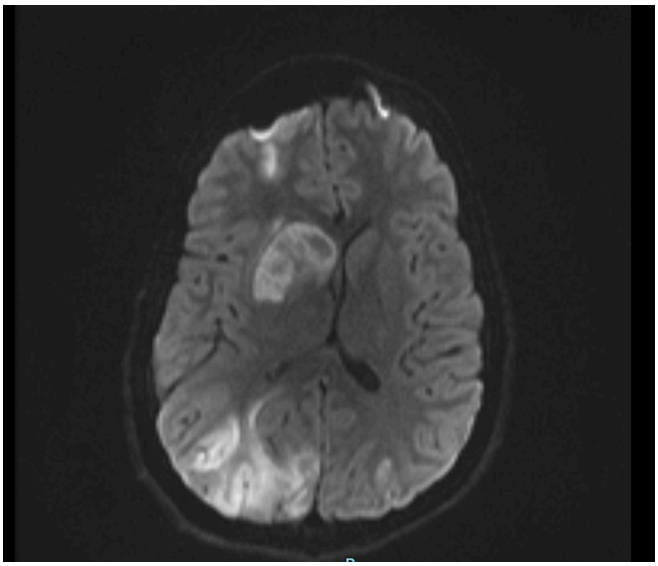

Fig. 3. Diffusion weighted cerebral MRI showing restricted diffusion in the bilateral fronto-parietal and in the right temporo-occipital cortico-subcortical areas consistent with ischaemia.

Discussion

Acute ischaemic events in childhood are quite rare, with an annual incidence of from 2 to 3 to 13/100,000 [1,15], and a basal ganglia ischaemic stroke is particularly infrequent among the pediatric population.

In absence of known predisposing conditions (such as congenital heart diseases, vascular or prothrombotic anomalies, or genetic or metabolic diseases), a minor head trauma is the other most frequent risk factor for lacunar strokes in childhood [7]. It has been proposed that the likelihood of an acute ischaemic stroke in pediatric age following a head trauma might be increased by a previous infection with varicella zoster, with a mechanism of sensitization to vasospasm in small arteries [16], and it is presumable that different genetic factors may increase the risk of a stroke after a minor head trauma, but there is a very limited understanding of them [17].

The pathogenic mechanism leading to the internal capsule ischaemic stroke in children, as a consequence of a mild head trauma, has been proposed to rely on the stretching of the deep perforating arteries; hence, an occlusion or vasospasm of the recurrent artery of Heubner occurs, followed by an acute ischaemia in its supply area [12], which consists in the head of the caudate nucleus, the anterior inferior part of the internal capsule's anterior limb, the anterior globus pallidus and putamen and the anterior thalamus, and some parts of the uninate fasciculus, olfactory region and the hypothalamus [18]. The case we report, as shown in the brain MRI, is compatible

with an acute ischaemia at the recurrent artery of Heubner supply area. In children, the anatomical characteristics of the deep perforating arteries of the carotid system facilitate the occurrence of vasospasm following a mild head trauma, which may result in basal ganglionic infarction [7,19]. In fact, in children the arterial end branches which supply the caudate, putamen and the internal capsule arise at a more acute angle from the middle cerebral artery as compared to adults, and follow a recurrent course before penetrating the anterior perforated substance [20]; furthermore, the incomplete development of the sphenoid bone eases the brain movement during head trauma [11,17]. These characteristics contribute to the flow disruption or intimal trauma in the perforating branches following a head trauma, with subsequent occlusion or vasospasm.

With regard to stroke-related visual impairment, most studies were performed in adults. Visual deficit subsequent of stroke may occur secondary to damage to the posterior circulation, but it is known that in children ischaemic strokes usually involve the anterior cerebral circulation (73 %) [21]. Visual field defects are more common in post-chiasmal ischaemic damage, while retrochiasmal lesions are rare in children, and there is no anatomical or functional reason for the low occurrence of stroke-associated visual deficits in children [13,22].

In our opinion, the pathogenic mechanism leading to parietooccipital ischaemia in the presented case should be sought in the stretching of deep arteries leading to arterial occlusion or vasospasm, similarly to the basal ganglionic infarction already discussed.

The link between head or neck trauma in the pediatric population and the ischaemic event has been assessed in several studies, and it has been found that a recent head trauma is an independent risk factor for ischaemic stroke [20,23,24].

Buonpadre et al. [7] have described a series of 28 pediatric patients (aging from 3 months to 10 years) who presented basal ganglia and internal capsule ischaemic stroke, stating that varicella-zoster infection and mild head trauma are the main risk factors, and they confirmed the good outcome already showed in other studies, and which characterizes the presented case. Remarkably, the majority of traumatic ischaemic strokes in children occur in patients younger than age 6 years and are very rare after that age, while in a 13-years-old patient a greater anatomic maturity of the perforating arteries should be expected, thus underlining the peculiarity of the present report.

In summary, the present case points out that a head trauma may be associated with stroke in teenagers, as already described in case reports regarding younger children, although it appears to be an extremely rare condition, presumably in relationship with the degree of maturity of the perforating vessels. Fortunately, this event is characterized by a favorable outcome, with no or minor neurological residual deficit.

Further research is warranted to better understand the pathogenic mechanisms causing trauma related strokes in children and young adults.

As this is a rare event, as there is often no apparent cause of ischaemic stroke in the pediatric population, it is desirable that the presented case will contribute to a better awareness of head trauma as a risk factor for stroke in children and young adults.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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