

Exceptional Complication of a Benin Cranial Trauma in a 3-Year-Old Child

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Abstract

Internal carotid artery (ICA) dissection with associated stroke is rare event in children. The usual mechanism is either direct trauma or sudden neck movements. We describe the case of a 3-year-old patient admitted with right hemiplegia following a stroke. The diagnosis was confirmed by ANGIO-MRI. During treatment with anticoagulants and antiplatelet agents, the patient showed significant improvement in the right-sided hemiplegia. Minor head trauma is a possible pathological mechanism for ICA dissection in children. However, the scenario is extremely rare.

Keywords

Child, Head Trauma, Internal Carotid Artery Dissection, Stroke

1. Introduction

Dissection of the internal carotid artery (ICA) with associated stroke is a rare event in children [1]. It is defined by the presence of a mural hematoma in the wall of an intracranial artery [2]. The mechanism is either direct trauma or due to sudden movements of the neck, this trauma is responsible for a mechanical breach of the carotid artery with the formation of a sub-intimal hematoma causing significant hemodynamic stenosis. Symptoms are heterogeneous and depend on the affected vascular territory [3]. Neuro-imaging plays an essential role in establishing both diagnosis and complications [4]. Treatment consists of the administration of platelet aggregation inhibitors and anticoagulants [5]. We report the case of a child hospitalized in the department of pediatrics of Mohammed VI University Hospital in Oujda, Morocco for a minimal trauma revealing in him an ischemic vascular accident. Our objective is to show that minimal cranial trauma

can be carotid dissection mechanism.

2. Observation

We describe the case of a 3-year-old male with no significant pathological history (no stay in neonatology, good cranial development, good psychomotor development) admitted for head trauma. On the day of admission, the child sustained a minor head injury as a result of falling down the stairs. The clinical examination on admission found a conscious child, afebrile, hemodynamically stable (heart rate was 86 bpm, blood pressure was 125/60 mmHg) and respiratory stable (respiratory rate was 18 cpm, arterial oxygen saturation 98%). Weight: 15 kg, height: 105 cm. The neurological examination revealed a pyramidal syndrome with left hemiplegia, present Babinski sign and sharp ROT, without consciousness disorder and with preserved sensitivity. The cardiovascular examination was normal, the peripheral pulses were present and symmetrical, there were no murmurs or additional noises, and the rest of the clinical examination was without particularity.

The child received an axial CT scan without contrast injection showing cortico-hypodense subcortical and central nuclei opposite the homolateral sylvian artery spontaneously hyperdense in favor of a recent ischemic attack of the left sylvian (**Figure 1**). An MRI of the brain and supra-aortic trunk was performed, which showed a carotid dissection with a mural hematoma and a cerebral image related to an ischemic stroke (**Figure 2, Figure 3**). His biological workup was normal. Therapeutically, the patient was given 0.1 IU/12H enoxaparin for 10 days and then 3 mg/kg acetylsalicylic acid for 03 months without any side effects

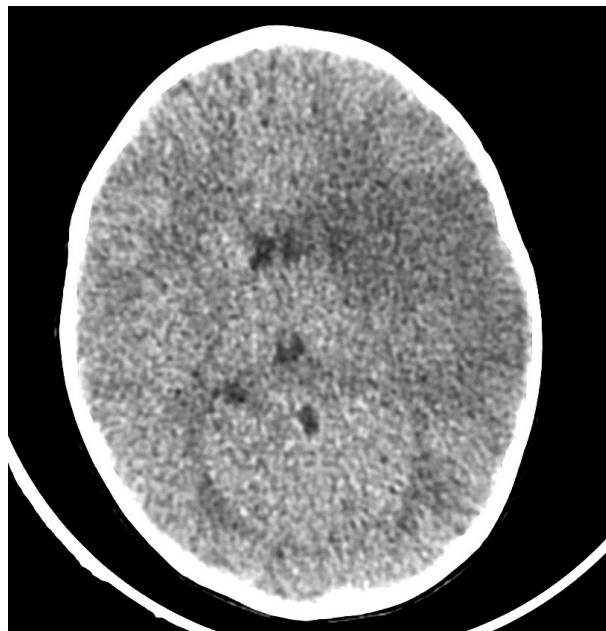


Figure 1. Axial CT scan without contrast injection showing cortico-hypodense subcortical and central nuclei opposite the homolateral sylvian artery spontaneously hyperdense in favor of a recent ischemic attack of the left sylvian.



Figure 2. MRI of the supra-aortic trunk showing a carotid dissection with a mural hematoma.

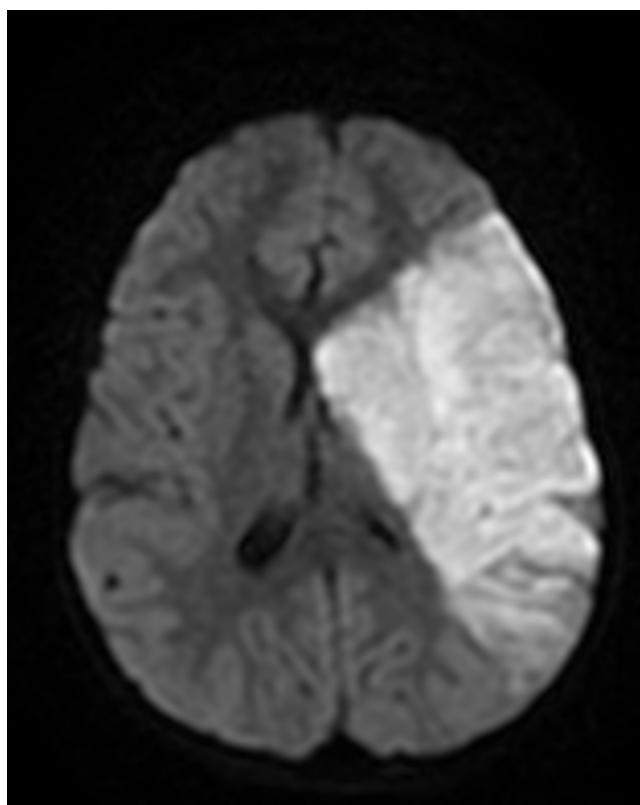


Figure 3. MRI of the brain showing hypersignal diffusion in the territory of the left sylvian artery in favour of an ischemic stroke.

observed in motorized physiotherapy.

The clinical evolution was marked by the persistence of a slight residual neurological deficit; hemiparesis type, which was treated by motor physiotherapy during one year with good clinical evolution.

3. Discussion

Ischemic stroke is an important cause of mortality and morbidity in children. Male predominance and high prevalence of extracranial location was observed [4]. Risk factors include hypertension, vasculopathy, cardiac disease, metabolic disorder, infection and coagulopathy [6]. Etiologies include trauma, or are spontaneous (hyperextension, cervical manipulations...), constitutional wall abnormalities, infection, and transient vasculitis [2]. Posttraumatic cervical artery dissection in children has been previously described in 4 children in a series of 12 patients, and in a 4-month-old child in another study. The 3-year-old child described in this report had an internal carotid artery dissection with a stroke after a fall down a flight of stairs with posttraumatic vomiting without any notion of initial loss of consciousness at his first presentation to the emergency department, and he subsequently presented a right hemiplegia. This is consistent with other studies [1] [4]. But, there were other symptoms that were not present in our case, such as headaches, neck pain and loss of consciousness. Interpretation of symptoms is difficult in children and the fact that symptoms of post-traumatic vascular injuries are not specific often leads to poor diagnosis [5]. Thus the dissection event is often followed by a latent period of few minutes to few days or even longer [3]. In our case, the initial C-brain CT scan was in favor of a left sylvian ischemic stroke. CT is not sensitive to detect arterial dissections but it can show fractures of the base of the skull [5]. It was the ANGIO-MRI that made the diagnosis possible. The ANGIO-MRI is very useful for the evaluation of neurovascular diseases in children and to diagnose cervical artery dissections and cerebral infarctions [7]. It also allows us to date dissection-related thrombosis which can help in the decision to start or not anticoagulants, but it may overestimate the degree of stenosis and the extent of longitudinal extension. In our case, the patient received 10 days of anticoagulant (enoxaparin) and 03 months of antiagregant, (Acetylsalicylic acid). The treatment should be administered early to prevent ischemic injury and improve long-term outcomes [3]. Treatment consists of preventing thromboembolic events by administration of antiagregants and anticoagulants. The AHA group and the Cardiovascular Council recommend treatment with anticoagulants (LMWH or UFH or warfarin) for a period of 03 to 06 months. The American College of Chest Physicians, recommends treatment with anticoagulants for a period of 06 weeks, but when a large brain territory is affected, anticoagulant may be a relative contraindication. A Cochrane meta-analysis of 36 observational studies found no statistically significant differences between antiagregators and anticoagulants in stroke treatment. Therefore, the guidelines indicate that antiagregant agents can be replaced by anticoagulants. In intracranial

artery dissection, an Anticoagulant is not recommended because of the risk of subarachnoid hemorrhage [5]. Our patient retained a mild neurological deficit after 03 months of treatment. In other studies, the results ranged from complete recovery or only a mild residual neurological deficit to severe hemiparesis or hemiplegia, or aphasia or seizure [3].

4. Conclusion

Internal dissection of the carotid artery followed by ischemic stroke is rare in children. Causes include direct trauma to the neck, blunt trauma to the soft palate, and traumatic hyperextension and/or hyper-rotation in child abuse. In our case, treatment with anticoagulants and platelet antiaggregators was effective and was not associated with any adverse effects with a good progression of neurological deficit.

Conflicts of Interest

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

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