CASE REPORT – OPEN ACCESS

International Journal of Surgery Case Reports 79 (2021) 358–361

Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.casereports.com

Ischemic stroke following operated head trauma in children: Discussion of a rare clinical case

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ARTICLE INFO

Article history:
Received 22 December 2020
Received in revised form
30 December 2020
Accepted 30 December 2020
Available online 9 January 2021

Keywords:
Brain trauma
Stroke
Pediatrics
Neurosurgery

ABSTRACT

INTRODUCTION: Traumatic brain injury (TBI) is the leading cause of morbidity and mortality in children. TBI in children are responsible for a range of clinical symptoms and signs that are comparable to those in adults, but present several differences in both physiopathology and management. Many postoperative complications may occur, ischemic stroke among others, which is generally related to an injury of an intracranial artery. Out of this case, it may be more difficult to find a suitable explanation to this complication.

CASE PRESENTATION: We report the case of a child aged years old, who was collided by a car causing a polytrauma with head and chest injury. On body scan, she had a fracture of the sixth left rib, and a frontal cranio-cerebral wound. The patient was operated for debridement of the wound, and tight closure of the injured dura mater. Initial postoperative course was uneventful, but 5 days after first surgery patient presented an acute onset of a right hemiplegia followed by an alteration of her state of consciousness, and a left anisocoria. Follow up CT scan showed a stroke of the whole left carotid territory. The patient was re-operated through a left decompressive craniectomy. Following the second surgery, she showed an improvement of her level of consciousness and a normalization of the size of her pupils, but aphasia and a right hemiplegia persisted. 2 weeks after surgery, the patient had a progressive necrosis of the surgical scar, followed by an exposure of the underlying cerebral cortex. Despite of intensive local care, a plastic surgery to recover the wound and antibiotics, the patient presented a meningitis, followed by a septic shock and death.

CLINICAL DISCUSSION AND CONCLUSIONS: Ischemic stroke is probably the most harsh and unpredictable complication that may occur after TBI, mainly in children. Only rigorous surgical approach followed by stringent post-operative care may prevent such outcomes.

1. Introduction

Strokes are defined by the brutal onset of clinical and neurological manifestations, together with radiological evidence of ischemia or infarct in a given arterial territory [1]. Arterial ischemic stroke is a rare, but increasingly recognized disorder in children related to different aetiologies: cardiac and haematological diseases, coagulopathies, vascular malformations (Moya Moya), venous infarcts, metabolic disorders, etc [2]. Post-traumatic strokes are mainly of haemorrhagic nature [2]. Post-traumatic ischemic strokes are understudied features, mainly reported within operated patients in the context of an injury of cerebral vessels [3]. Published reports remain limited to case reports and small case series, especially in children [4].

In this paper, we report a rare case of a child who presented, 5 days after being operated for a traumatic brain injury (TBI), an ischemic stroke in the territory of the carotid artery, and discuss the physiopathology of this uncommon complication.

This work has been reported in line with the SCARE 2020 criteria [5].

2. Case description

We report the case of a 3 years-old girl, with no pathologic background, raised within a middle income social environment. She was victim of a car crash causing a polytrauma with head and chest injury. On initial physical exam, the patient had a GCS at 13/15, and a deep fronto-temporal wound made of 2 contiguous branches: the first, measuring 4 cm in length, was frontal and perpendicular to the eyebrow; the second, measuring as well 4 cm in length, was irradiating to preauricular area. These wounds were not degloved,

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https://doi.org/10.1016/j.ijscr.2020.12.094

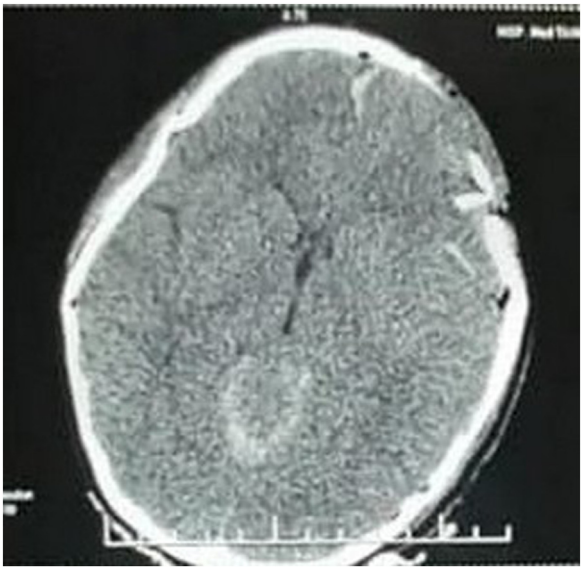

Fig. 1. Axial section of a brain CT scan showing a depressed frontal fracture with subarachnoid haemorrhage, and a subdural haemorrhage of the falx cerebri.

but deep enough to make the cranial bone visible. The patient had a body scan (cerebro-thoraco-abdomino-pelvic scan) that showed a depressed frontal fracture, associated to subarachnoid haemorrhage, and a subdural haemorrhage of the falx cerebri (Fig. 1). In addition, she had a fracture of the sixth left rib. Biological statement showed no blood coagulation disorders. The patient underwent emergent surgery, which was performed by an experimented consultant neurosurgeon. Peroperative, she had a debridement of the wound, a craniectomy of the depressed skull bone, haemostasis of the underlying cortex, and tight closure of the injured dura mater. During the surgery, the patient received prophylactic antibiotics based on Amoxicillin (600 mg). Initial postoperative course was uneventful, and the patient recovered a perfect state of consciousness with no motor or sensory deficit (GCS 15/15). She had no further antibiotics as she did not present any clinical or biological features that may suggest postoperative neuro-meningeal infection. 5 days after surgery, while she was still in reanimation unit, the patient presented the acute onset of a right hemiplegia, followed by an alteration of her state of consciousness. Physical examination found a comatose girl with a GCS of 6/15 and a left anisocoria. Control CT scan showed a stroke of the whole left carotid territory (Fig. 2). Yet again, there were no biological abnormalities on the new assessment. The patient was re-operated through a left decompressive craniectomy (Fig. 3). Postoperative, she was re-transferred to the department of reanimation. The anisocoria gradually regressed, and the patient showed a progressive improvement of her state of consciousness, but aphasia and a right hemiplegia persisted. However, in parallel, the patient presented a progressive necrosis of the surgical scar, followed by an exposure of the underlying cerebral cortex. Despite intensive local care, a plastic surgery to recover the wound and antibiotics, the patient presented 2 weeks after surgery with meningitis, followed by septic shock and death.

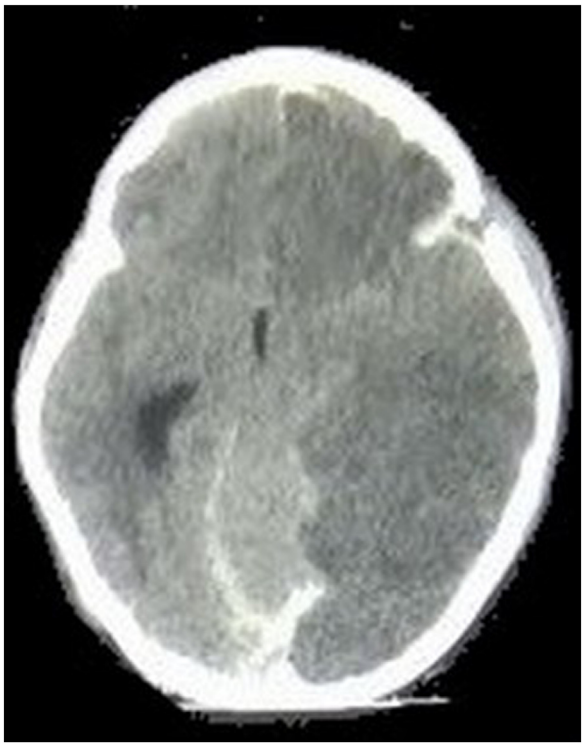

Fig. 2. Axial section of a brain CT scan showing a stroke of the whole left carotid territory.

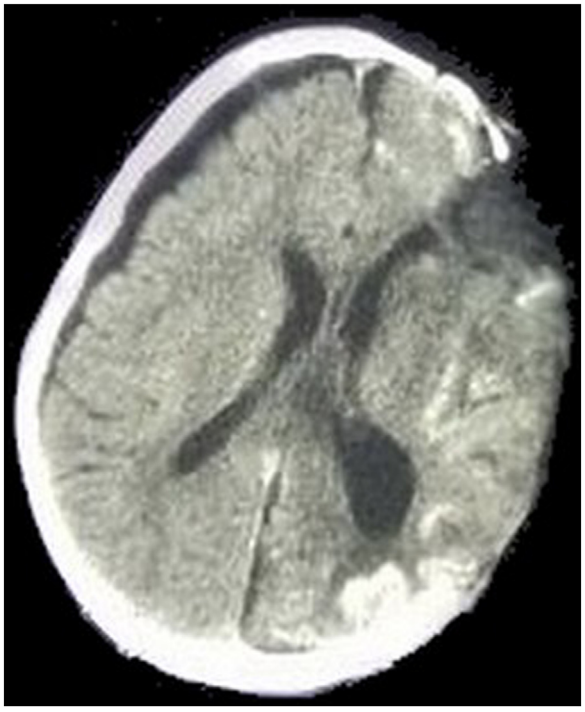

Fig. 3. Axial section of a brain CT scan performed after decompressive craniectomy.

3. Discussion

Stroke has become an increasingly recognized cause of morbidity and mortality in children. The annual incidence of ischemic strokes in children aged below 14 varies between 2 and 8 per 100,000 [4,6,7]. The associated mortality approaches 25 % [7–9]. More than 50 % of survivors have neurologic disabilities, epilepsy, cognitive, or psychiatric impairments [10,11].

The aetiologies of childhood ischemic strokes remains misunderstood in several points, owing to the low incidence of the disease in this age group. The classic risk factors in adults, such as diabetes, smoking, hypertension and obesity among others, are infrequent in children [12]. On the other hand, some instances of paediatric

ischemic stroke, such as congenital cardiopathies, sickle cell disease, and arterial dissection, the aetiology is easily understood. Surgical interventions over the central nervous system are also a cause for strokes, related to surgical damage of intracerebral vessels (veins, major or perforating arteries).

Some reports proved that head and neck injuries would be risk factors for ischemic stroke in childhood. This risk would remain up to 3 months after the traumatic event, but the median time from trauma to the stroke was 0.5 days [13]. Some authors tried to discuss the various pathophysiological theories behind these postoperative complications. The main pathologic mechanisms of stroke after head and neck trauma would be related to stretching or tearing of the carotid or vertebral arteries from sudden and forced hyperextension and contralateral rotation of the head, a direct blow to the neck, or laceration by adjacent fractures. Resulting dissection, arteriovenous fistula, or pseudoaneuryms ultimately interrupts blood flow or causes artery-to-artery thromboembolic events to the brain [14]. Current trauma guidelines suggest that patients with significant head trauma who meet clinical criteria should be considered for screening vascular imaging. However, there is still no defined criteria that may indicate such investigations, and obviously, it is not in always possible to perform an angio CT scan in all paediatric TBI.

Out of the previously elucidated mechanism, the pathogenesis of ischemic stroke after TBI would be related to additional risk factor, such as prior central nervous system infection, causing intimal lesions and thrombosis, congenital abnormalities of the osteo-ligamentar cervico-occipital area that can lead to a susceptibility to posterior circulation stroke [15,16].

But in our case, none of these discussed theories is able to explain the stroke in our patient. She had a TBI without association to a neck trauma. She suffered from no previous pathologies which could be risk factors to the onset of the stroke. Moreover, peroperative, only a limited manipulation of the superficial cortex was performed in order to insure hemostasis, very far from all vessels whose manipulation may lead to an infraction. Thromboembolic mechanisms are unlikely able to explain the stroke, as biological examinations did not show any abnormalities.

The only pathological presumption that remain detrimental in our context is related to haemodynamic mechanisms. This finding is intuitively explained by prominent collateral circulation in the posterior circulation, making the carotid territory more susceptible to suffer from lack of oxygen supply [17]. Peroperative bleeding would have worsened the situation. Nevertheless, the brutal and the delayed onset of the symptomatology runs counter this hypothesis.

4. Conclusions

This case report confirms the fact that exact pathophysiology of ischemic stroke after traumatic brain injury continues to be a mystery in various points. Rigorous surgical approach, as well as adopted pre and postoperative care including the prevention of thromboemblic complications and hypovolemia, are the necessaries to prevent this harmful complication.

Declaration of Competing Interest

The authors do not declare any conficlt of interest.

Funding

This research did not receive any funding.

Ethical approval

This study is exempt from ethical approval in our institution.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author's contribution

Ghassen Gader wrote the manuscript.
Mouna Rkhami made the bibliographic research.
Mohamed Badri and Ihsèn Zammel corrected the manuscript.

Registration of research studies

Not Applicable.

Guarantor

Ghassen Gader is the guarantor for this paper.

Provenance and peer review

Not commissioned, externally peer-reviewed.

Acknowledgement

The authors did not receive any contributions from any individuals and organizations for this manuscript.

References

[1] T. Bernard, N. Goldenberg, Pediatric arterial ischemic stroke, Pediatr. Clin. North Am. 55 (2) (2008) 323–338.
[2] E. Filho, W. Carvalho, Stroke in children, J. Pediatr. 85 (6) (2009) 469–479.
[3] N. Khan, K. Moore, J. Basma, D. Hersh, A. Choudhri, B. Vaughn, Ischemic stroke following elective craniotomy in children, J. Neurosurg. Pediatr. 23 (3) (2019) 355–362.
[4] A. Numis, C. Fox, Arterial ischemic stroke in children: risk factors and etiologies, Curr. Neurol. Neurosci. Rep. 14 (1) (2014) 10–13.
[5] R. Agha, T. Franchi, C. Sohrabi, G. Mathew, The SCARE 2020 criteria: updating consensus Surgical CAse REport (SCARE) Guidelines, Int. J. Surg. 84 (2020) 226–230.
[6] A. Agrawal, J. Timothy, L. Pandit, M. Manju, Post-traumatic epilepsy: an overview, Clin. Neurol. Neurosurg. 108 (2006) 433–439.
[7] L. Jordan, Assessment and treatment of stroke in children, Curr. Treat Options. Neurol. 10 (2008) 399–409.
[8] N. Khan, B. Fraser, V. Nguyen, K. Moore, S. Boop, B. Vaughn, Pediatric abusive head trauma and stroke, J. Neurosurg. Pediatr. 20 (2) (2017) 183–190.
[9] C. Fox, S. Johnston, S. Sidney, H. Fullerton, High critical care usage due to pediatric stroke: results of a population-based study, Neurology 79 (2012) 420–427.
[10] M. Steinlin, K. Roellin, G. Schroth, Long-term follow-up after stroke in childhood, Eur. J. Pediatr. 163 (2004) 245–250.
[11] M. Gardner, N. Hills, S. Sidney, The 5-year direct medical cost of neonatal and childhood stroke in a population-based cohort, Neurology 74 (2010) 372–378.
[12] R. Sträter, H. Vielhaber, R. Kassenböhmer, Genetic risk factors of thrombophilia in ischaemic childhood stroke of cardiac origin. A prospective ESPED survey, Eur. J. Pediatr. 158 (1999) 122–125.
[13] N. Hills, S. Johnston, S. Sidney, Recent trauma and acute infection as risk factors for childhood arterial ischemic stroke, Ann. Neurol. 72 (2012) 850–858.

[14] C. Griessenauer, J. Fleming, B. Richards, L. Cava, J. Curé, D. Younan, Timing and mechanism of ischemic stroke due to extracranial blunt traumatic cerebrovascular injury, J. Neurosurg. 118 (2) (2013) 397–404.
[15] F. Yang, H. Wang, J. Zhang, H. Liang, Clinical features and risk factors of cerebral infarction after mild head trauma under 18 months of age, Pediatr. Neurol. 48 (2013) 220–226.
[16] K. Saito, M. Hirano, T. Taoka, Artery-to-artery embolism with a mobile mural thrombus due to rotational vertebral artery occlusion, J. Neuroimaging 20 (2010) 284–286.
[17] D. Stein, S. Boswell, C. Sliker, F. Lui, T. Scalea, Blunt cerebrovascular injuries: does treatment always matter? J. Traumatol. 66 (2009) 132–144.