CASE REPORT

Indonesian Journal of Neurosurgery (*IJN*) 2019, Volume 2, Number 3: 60-63
P-ISSN.2089-1180, E-ISSN.2302-2914

Post-traumatic cerebral infarction associated with a depressed skull fracture in children: a case report

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ABSTRACT

Introduction: Post-traumatic cerebral infarction (PTCI) is one of the most severe yet very rare complications after traumatic brain injury in children. Various mechanisms have been suggested, including direct vascular compression, dissection, embolization, cerebral vasospasm, vascular injury, and systemic hypoperfusion. PTCI secondary to direct vascular injury by a skull fracture in children has never been reported, although it is mechanically possible. We reported a case of PTCI secondary to direct vascular injury by a skull fracture in a child.

Case presentation: A 5-years old boy had decreased consciousness and left-sided hemiplegia on admission after experiencing a motor vehicle accident. On examination, he had a GCS score of 11, left-sided hemiplegia, and lacerated wound on his right temporoparietal region with no exposed bone. Head CT scan revealed closed depressed fracture on the right temporoparietal with a large cerebral infarction below the fracture segment. Emergency craniotomy and duramater repair were performed. Improved consciousness was achieved two days after the operation; however, the hemiplegia did not improve. Digital Subtraction Angiography (DSA) after surgery showed disruption of the M3 segment of the right middle cerebral artery. The patient was consulted to do routine physiotherapy. At five months follow up, he came with an improvement in motor strength on his left extremities.

Conclusion: PTCI secondary to direct vascular injury by a skull fracture is very rare in children, but still mechanically possible. Head CT scan and DSA is beneficial for a proper diagnosis. Physiotherapy is vital in children with PTCI due to brain neuroplasticity capabilities that differ from adults.

Keywords: post-traumatic cerebral infarction, pediatric head injury, skull fracture

Cite This Article: Lazuardy, M.A., Sutiono, A.B., Adam, A., Arifin, M.Z. 2019. Post-traumatic cerebral infarction associated with a depressed skull fracture in children: a case report. *Indonesian Journal of Neurosurgery* 2(3): 60-63. DOI: 10.15562/ijn.v2i3.54

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Received: 2019-03-02
Accepted: 2019-06-02
Published: 2019-12-01

INTRODUCTION

Post-traumatic cerebral infarction (PTCI) is one of the most severe secondary insults after Traumatic Brain Injury (TBI), with a prevalence ranging from 1.9% to 10.4%.[1,2] PTCI in children is an exceedingly rare sequela.[3,4] Various mechanisms have been suggested in the pathogenesis of PTCI, including direct vascular compression by mass effect, dissection, embolization, cerebral vasospasm, systemic hypoperfusion, and vascular injury.[1,2,5,6] Cerebral infarct secondary to direct vascular injury by a skull fracture is very rare in adults,[1] and no case has been reported in children. Compared with adults, who often show long-lasting cognitive and motor impairment,[1,2,7] children with cerebral infarction often show surprisingly good functional outcomes.[8] We report one case of a child with PTCI secondary to a depressed skull fracture, including its possible pathogenesis, clinical manifestation, diagnosis, management, and outcome, with a brief literature review.

CASE PRESENTATION

A 5-year-old boy was admitted to the Neurosurgery Emergency Unit of Hasan Sadikin Hospital after experiencing a motorcycle accident two days before. He was lethargic with normal vital signs. The GCS score on admission was 11 (E3M5V3). Neurological examination revealed symmetrical pupils with normal light reflex and left-sided hemiplegia. A lacerated wound size of 10 x 7 cm was found on the right temporoparietal with no exposed bone. Laboratory parameters demonstrated no abnormality. Head CT scan was performed immediately, and right temporoparietal depressed fracture with massive soft tissue swelling was confirmed. A large low-density area was found on the temporoparietal region of the brain below the fracture segment, indicating the existence of a cerebral infarct. There was also a dispersed small cerebral contusion and brain swelling with midline shift < 5 mm to the left side (**Figure 1 Upper**).

Emergency elevation craniotomy was performed, and the interlocking segment of bone fracture was

found (size 10 x 8 cm). The duramater below the fracture was not intact, with a laceration size of 8 x 6 cm and accompanied by cerebral prolapse (**Figure 2**). We performed duraplasty with fascia and reconstruction of the fracture segment. During the operation, his vital signs were stable, and ECG monitoring showed no cardiac abnormalities.

The patient was extubated and hospitalized in the neurosurgery ward after the operation. Two days later, he was fully conscious (GCS score 15), but still could not move his upper and lower extremities (both motoric strengths was 0; scale 0-5). No cognitive impairment was observed. Control head CT scan was obtained five days later and showed a good position of the previously depressed segment with still a large area of right Middle Cerebral Artery (MCA) infarction. Brain swelling has decreased compared to the previous CT scan, with no midline shift (**Figure 1 Lower**). On the next day, we performed Digital Subtraction Angiography (DSA) to evaluate the vascularization of the infarct area. The DSA result showed an occlusion of the M3 segment of the right MCA with no blood flow to the distal area (**Figure 3**). We decided to consult the patient to the Medical Rehabilitation Department for physiotherapy. He was sent home one week later with same motoric strength and was programmed to do routine physiotherapy in a local hospital near his house. A month later, the patient came to our outpatient clinic with improved motor strength on his left lower extremities (motoric strength 3) but still could not move his upper extremities (motoric strength 0). Five months later, the patient was finally able to walk by himself to the clinic, with improved motoric strength on left upper and lower extremities (motoric strength 2 and 4, respectively).

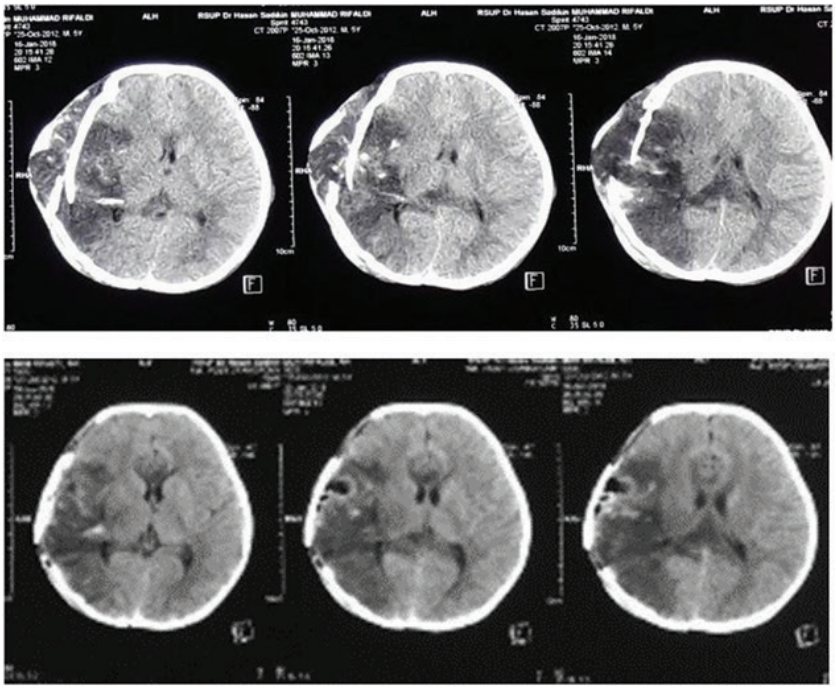

Figure 1. Preoperative computed tomography (CT) showing right temporoparietal depressed fracture with a low-density area in the territory of middle cerebral arteries with dispersed small contusion **(upper)**, CT obtained one week after emergency reconstructive craniotomy **(lower)**.

DISCUSSION

The secondary effects of TBI might be more critical than the primary injuries. Brain edema, intracranial hemorrhage, vasospasm, and posttraumatic hydrocephalus are relatively common secondary brain injuries.[1] PTCI is another potential secondary insult after TBI and is one of the most severe complications, with a rare prevalence ranging from 1.9% to 10.4%.[1,2]

In a study conducted by Dong-Hyeon Bae *et al.*, post-traumatic infarcts were seen most frequently in the area of the Posterior Cerebral Artery (PCA) and followed by the MCA.[1] In our patient, infarct was seen on the MCA territory, which was confirmed by the finding of a low-density mass at the right temporoparietal area on head CT scan. Various mechanisms have been suggested, including direct vascular compression by mass effect, dissection, embolization, cerebral vasospasm, vascular injury, and systemic hypoperfusion.[1,2,5,6] The MCA territory infarction is commonly caused by shifting or stretching of the MCA due to direct compression from extra-axial hematoma or indirectly by increased intracranial pressure (ICP), severe brain edema, or herniation.[1] It can also occur secondary to post-traumatic intimal dissection or thromboembolic events from the injured MCA, particularly on the M1 segment.[1,5,9]

The case of cerebral infarction due to the direct lesion of the intracranial vessel has been reported to be more scarce. It is a very rare case in adults,[1] and no case has been reported in children. A possible cause of infarct that is mechanically possible is direct MCA injury by inward skull fracture.[1,3,10] The inwardly depressed skull fracture that resulted from the high impact force may lead to direct damage or compression of the underlying neurovascular structures.[10] It was the most likely cause of MCA territory infarct in our case, which was supported by the early head CT scan finding (**Figure 1 Upper**) that showed a contusion along cerebral parenchyma, projecting from the depressed fracture segment to the location where the M3 segment was supposed to be. DSA was performed to confirm any vascular abnormalities and its flow. The DSA result showed the M3 segment disruption as a cause of the cerebral infarct and possibly damaged due to its location

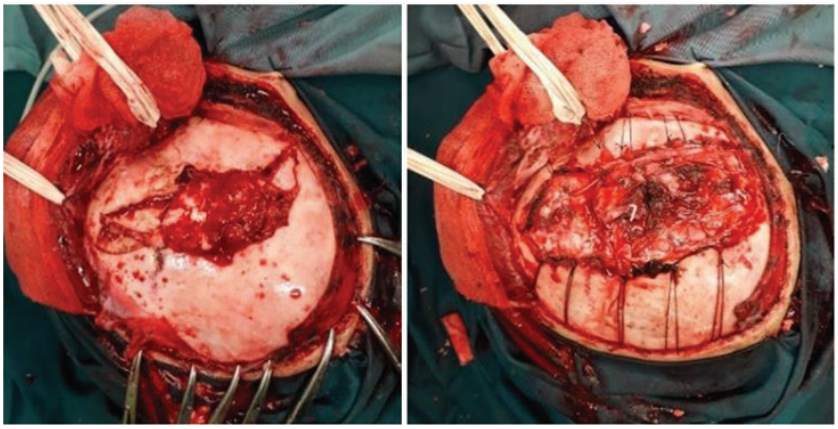

Figure 2. Intraoperative finding during operation showed interlocking segmented bone fracture with lacerated duramater and cerebral prolapse.

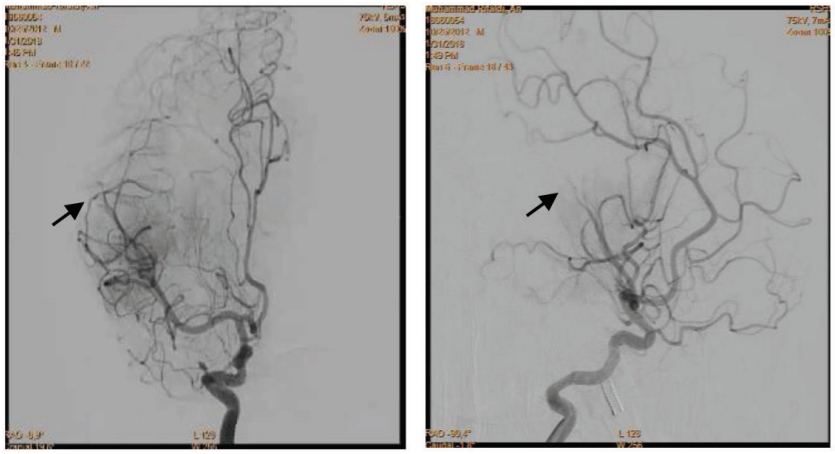

Figure 3. DSA results on eighth-day post-operation, showing disruption of the M3 segment of right MCA (black arrow).

that was closer to the surface of the brain. The force of the impact at the time of the accident should be strong and quick enough to deeply crack and push the bone, according to the biomechanical basis of depressed skull fracture in the literature.[11]

In most patients, PTCI developed early in the hospital course. In 16 (76.2%) patients, the infarction developed within two weeks after the injury.[1] Distinct from the cerebral infarction caused by the rupturing of the intima, the cerebral infarction caused by the local injury on the vessel wall occurs within 24 hours of injury in most cases, which is speculated due to the lag period from the vessel injury to the occurrence of secondary infarction due to the formation of thrombosis.[9] Unfortunately, the diagnosis of infarct in our patient was established two days after the accident due to delay in referral and CT scan, and could not represent the exact pathogenesis.

In 1942, DeVeer and Browder reported a case of coma and hemiparesis resulting from craniocerebral trauma. The autopsy revealed a dissecting aneurysm involving the MCA, resulting in MCA infarct.[7] In our patient, the infarct was not the only cause that lead to decreased consciousness and neurological deficits. Other possible causes of decreased consciousness were the initial impact of head injury, cerebral contusion, and mainly secondary brain swelling. Based on the literature, the larger the infarcted area, the greater the risk of developing fatal brain swelling.[2] However, no significant brain swelling existed in our patient, despite a large area of infarct. The possible cause was the slight decompressive effect of skull and duramater after fracture, as shown in **Figure 1 Upper**.

Based on the head CT scan and DSA result, the right M3 segment was disrupted in our patient. Symptoms of right-sided territory infarcts of this segment should be contralateral motor and sensory loss preferential for upper extremities, given the distribution of the sensorimotor homunculus in the motor and sensory strips. Hemiplegia on upper extremities in our patient was caused by this condition, but not with lower extremities, as its sensorimotor area should be located in the parasagittal side of the parietal lobe.[11] The most possible cause of lower extremities hemiplegia was brain swelling that compressed the responsible area. Direct injury of the fracture segment to the motor cortex should be considered as a possible cause of hemiplegia.

Decompressive craniectomy has been frequently performed to control malignant brain edema by reducing ICP and resistance to cerebrospinal fluid outflow with decreased compression of cerebral vessels in cerebral infarction after trauma.[1] In our case, we only performed surgery for fracture segment elevation and dura repair with duraplasty, followed by routine physiotherapy. The consideration was that the CT finding did not show any significant midline shift.

The occurrence of TBI is very common in children, but PTCI is an exceedingly rare sequela.[3,4] Prognosis depends on the severity of infarct and provided fatal in 33% of the cases. It results in significant permanent neurological deficits in most of the survivors.[7] Compared with adults, who often show long-lasting cognitive and motor impairment[1,2,7], children with cerebral infarct often show surprisingly good functional outcomes.[8] In our patient, significant improvement of motoric strength was achieved on the fifth-month postoperative following routine physiotherapy. We believed that neuroplasticity capabilities in children that differ from adults contributed to this condition.[8] We also conclude that physiotherapy is vital in the recovery of patients after PTCI, especially children.

Published by DiscoverSys | *IJN* 2019; 2(3): 60-63 | doi: 10.15562/ijn.v2i3.54

CONCLUSION

PTCI secondary to direct vascular injury by a skull fracture is very rare in children, but still mechanically possible. Head CT scan and DSA is beneficial for a proper diagnosis. Physiotherapy is vital in children with PTCI due to their brain neuroplasticity capabilities that differ from adults.

ACKNOWLEDGEMENTS

The authors would like to thank Akhmad Imron MD, Chief of Neurosurgery Department of Universitas Padjadjaran, Hasan Sadikin Hospital, Bandung, Indonesia, for supporting the authors to write this case report.

CONFLICT OF INTEREST

The authors declare no conflict of interest regarding the publication of this case report.

FUNDING

No specific grant was provided for this article.

AUTHORS' CONTRIBUTION

All authors took part in the design of the study, literature review and writing the manuscript.

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