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Luis Rafael Moscote-Salazar, Willem Guillermo Calderon-Miranda, Andres M. Rubiano, Amit Agrawal
COLOMBIA, MEXICO, INDIA

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Unilateral traumatic hemorrhage of the basal ganglion and bihemispheric cerebral infarction

Luis Rafael Moscote-Salazar¹, Willem Guillermo Calderon-Miranda², Andres M. Rubiano³, Amit Agrawal⁴

¹Neurosurgery, Critical Care Unit, University of Cartagena, Red Latino, Latin American Trauma & Intensive Neuro-Care Organization, Bogota, COLOMBIA
²Universidad Nacional Autonoma de Mexico, Mexico City, MEXICO
³Neurosurgery, Director of Neurotrauma Research, El Bosque University, Bogotá, COLOMBIA
⁴Department of Neurosurgery, Narayna Medical College Hospital, Chinthareddypalem, Nellore, Andhra Pradesh, INDIA

Abstract: Among the various injuries caused by the cerebral traumatic lesion are traumatic brain contusions. Hemorrhagic contusions of the basal ganglia are unusual. Different injuries such as cranial fractures, epidural hemorrhage, subdural hematoma, subarachnoid hemorrhage among others may be associated with brain contusions. In some cases traumatic brain injury arises. We present a case of a patient with unilateral cerebral contusion associated with bihemispheric cerebral infarction.

Key words: Basal ganglia hematoma, bilateral, neurotrauma

Introduction

Cerebral ischaemia is an important contributor to morbidity and mortality in patients with traumatic brain injury. Cerebral infarction in patients with cranial lesions is mainly related to cerebral herniation, compressive effects of intracranial hematomas, cerebral edema, vasospasm, direct vascular injury, fatty embolism and cortical lesions due to cranial fractures. We present the case of a patient with bihemispheric infarction associated with gangliobasal contusion as an illustration of the heterogeneity of the traumatic brain injury.

Case report

A 30-year-old male patient is taken to our emergency department after to present cranial trauma following a vehicular accident. Upon admission, the hemodynamically stable patient, Glasgow coma scale 4, was performed Tac brain showing the Unilateral traumatic hemorrhage of the basal ganglion and bihemispheric Cerebral Infarction. The patient was transferred to an intensive care unit receiving conservative management. No cerebral angiography was performed. The patient presented a drowsy evolution and died at 48 hours.
Cerebral trauma is associated with posttraumatic cerebral infarction in up to 2% of cases. There are several hypotheses that may explain the pathophysiology of cerebral ischemia. Participation has been proposed mechanisms of intravascular thrombosis as an etiology for cerebral infarctions in traumatic brain injury. The above situations may occur in the context of dissections, thrombi or vasoospasmo. In a multivariate prospective analysis, the presence of low systolic blood pressure was one of the statistically significant risk factors for the development of posttraumatic brain injury infarction. Mirvis et al reported the prevalence of postrauma cerebral infarction at 1.9% and Tomberg et al reported a prevalence of 3.3%. The Tian et al study which included the severity of the trauma moderate and severe injuries increasing the prevalence in 11.9% (42/353). Hirata et al reported the case of a patient with cerebral infarction and multiple lesions associated with shock, in this way arise the episodes of severe hypotension as causality of cerebral ischemic lesions. (6).

Patterns of brain injury in post-traumatic infarcts include cortical injury in borderline areas, arterial territories, multiple focal cerebral infarcts. On the other hand, the development of decompressive craniectomies has increasingly increased the presentation of infarcts associated with the procedure, especially in the areas where post-surgical herniation occurs, a compression of veins and arteries occurs in the dural margins of the duraplasty. It has been hypothesized that a rapid reduction of intracranial pressure by surgical compression causes a shearing of the tissues that finally end in infarct the zone. This type of lesion should be carefully studied in future studies.

Discussion

Cerebral trauma is associated with posttraumatic cerebral infarction in up to 2% of cases. There are several hypotheses that may explain the pathophysiology of cerebral ischemia. Participation has been proposed mechanisms of intravascular thrombosis as an etiology for cerebral infarctions in traumatic brain injury. The above situations may occur in the context of dissections, thrombi or vasoospasmo. In a multivariate prospective analysis, the presence of low systolic blood pressure was one of the statistically significant risk factors for the development of posttraumatic brain injury infarction. Mirvis et al reported the prevalence of postrauma cerebral infarction at 1.9% and Tomberg et al reported a prevalence of 3.3%. The Tian et al study which included the severity of the trauma moderate and severe injuries increasing the prevalence in 11.9% (42/353). Hirata et al reported the case of a patient with cerebral infarction and multiple lesions associated with shock, in this way arise the episodes of severe hypotension as causality of cerebral ischemic lesions. (6).

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The presence of lesions associated with cerebral infarctions, such as contusions and hematomas may occur, in our case the presence of a gangliobasal hemorrhage was evidenced. Our case illustrates the heterogeneity of the traumatic brain injury and the importance of the individualization of each patient.

Correspondence
Luis Rafael Moscote-Salazar
Email: mineurocirujano@aol.com

References