

Bruises in Head Trauma

Chaabi Safia*, A Erragh, A Nsiri and R Alharrar

Department of Critical Care of Surgical Emergency, CHU Ibn Rochd Casablanca, Morocco

*Corresponding Author: Chaabi Safia, Department of Critical Care of Surgical Emergency, CHU Ibn Rochd Casablanca, Morocco.

Received: October 31, 2023; Published: January 08, 2024

Abstract

Brain contusions can be dynamic and expansive, leading to delayed neurological deterioration, with cerebral edema being the main cause of death in this case.

Our work aims to study the epidemiological, clinical, paraclinical, therapeutic and evolving data on post-traumatic brain contusions in order to define the main problems posed by these head trauma patients.

The physiopathology of TBI describes two mechanisms: TBI with direct impact on the skull or TBI without direct impact where the head is subjected to a deceleration/acceleration effect.

We distinguish between primary lesions, immediately present after the trauma and which are inevitable, and secondary lesions which develop in a delayed manner over a few hours and which must be detected and treated.

Brain contusion is an imprecise term encompassing all brain lesions linked to the transmission of the shock wave. They become complicated by ICH and progress towards the onset or worsening of a coma, which justifies their treatment in a center specialized in neurotraumatology regardless of the initial clinical state.

Cerebral edema complicates most parenchymal lesions; it can be localized or diffuse.

Traumatic brain contusions are a major cause of mortality in developed countries. It is the most important structural lesion of the brain, it is complex and dynamic and results from a primary lesion.

Keywords: Brain Contusion; Cerebral Edema; Head Trauma; TBI

Introduction

TBI head injuries constitute a major public health problem in Morocco due to their frequency, their severity, and especially their serious after-effects.

It is said to be serious when the Glasgow score is less than or equal to Eight. This severity is increased by the occurrence of secondary cerebral attacks of systemic origin (ACSOS), the consequences of which are well known.

Brain contusions can be dynamic and expansive, leading to delayed neurological deterioration, with cerebral edema being the main cause of death in this case.

Aim of the Study

Our work aims to study the epidemiological, clinical, paraclinical, therapeutic and evolving data on post-traumatic brain contusions in order to define the main problems posed by these head trauma patients.

Materials and Methods

This is a retrospective study carried out in the surgical emergency intensive care unit at the Ibn Rochd university hospital center in Casablanca.

This is a descriptive study spread over 6 years (2015 - 2020) which concerns 302 patients admitted for head trauma.

The data analysis focused on several points:

- The incidence of contusion in head trauma patients
- Scanner results
- Epidemiological data of the patient with a contusion
- Patient assessment
- Patient treatment
- Patient progress.

All patients over 15 years old and admitted to the surgical emergency intensive care unit with a brain contusion following head trauma during the study period were included in the study.

Excluded were patients under 15 years old, admitted for another reason, admitted to another department, arriving deceased, leaving against medical advice and transferred to other structures.

Results

The number of head trauma patients is 302 patients over 6 years, or an average of 50 cases per year.

Public road accidents represent the leading cause of TBI with 64% or 192 cases, followed by falls 13%, assaults 10%, other causes 10%, and work accidents 4%.

Among the 192 AVP cases, 121 were motorcyclists or 63%, followed by pedestrians 25%, and cars 12%.

Brain CT scan identified lesions in 189 patients or 63%.

Contusions presented the greatest proportion of lesions in 136 patients, i.e. 45%, followed by HSD 37% and HED 30%.

The average age of our patients with contusions is 37 years +/- 12 years with a male predominance of 79%.

In our study 57% of patients had no history.

Regarding clinical data

We noted an initial loss of consciousness in 89 patients, or 65% of bruises.

The majority were admitted with an unstable hemodynamic state, with a GCS between 9 and 12 in 65% and 8 to 3 in 35%.

Concerning ACSOS the most common is fever followed by anemia, however the same patient could present several ACSOS at the same time.

The deficit signs were dominated by ICH noted in 102 cases or 75% of contusions, focal signs were found in 83 patients, convulsions in 21 patients.

74% of patients had symmetrical and reactive pupils, 15% were in anisocoria, 7% in mydriasis, and 3% in miosis.

The injury assessment carried out systematically detected trauma to the limbs and spine in 41 patients, chest trauma in 34 patients, facial trauma in 25 patients.

We observed complications in 27 patients, including 10 nosocomial infections, 7 ARDS, 5 refractory HTIC, 3 pressure sores and 2 thromboembolic complications.

Brain CT was performed in 60% of patients within less than 24 hours.

A skull fracture was noted in 80% of cases.

Temporoparietal and temporal sites were the most frequent with 29 and 23% of cases.

In 78% of patients, the volume of the contusion was less than 15 ml.

Cerebral edema is the most common lesion associated with contusions with 29%, followed by HED and SDH.

Concerning therapy

Out of 136 cases of contusions, 50 patients were operated on for embedding, 30 patients had a craniotomy for cerebral edema, and 22 patients operated on for extra dural hematoma.

For conditioning, an electroscope, pulse saturation, BP, central line, gastric tube and urinary catheter are placed in all patients in supine position at 30 degrees.

All patients in our study benefited from intubation upon admission.

Artificial ventilation in the face of signs of respiratory and/or neurological distress.

The duration of intubation was 10 days +/- 4 days. All patients received sedation using hypnovel and fentanyl for 48 hours. 35% required a blood transfusion. Protection against stress ulcer based on PPI was systematic in all patients.

Curative antibiotic therapy was used to treat infectious complications (meningitis, pneumonia, urinary tract infection, etc.). Antibiotic prophylaxis was prescribed for patients who presented with a craniocerebral wound, an open fracture, with soiled skin wounds or inhalation.

Tracheotomy was performed in 20% of our patients around day 14% for ventilatory weaning.

Osmotherapy based on mannitol is used at a dose of 0.5 g/kg over 30 minutes in cases of ICH after failure of symptomatic treatment and in the case of cerebral involvement while awaiting decompressive craniectomy. It was administered to 12 patients with brain contusions, or 9%.

11% of patients required the use of catecholamines.

Methylprednisolone was used in 115 post-extubation patients to prevent laryngeal edema.

The introduction of feeding via nasogastric tube was gradual from day 1, reaching the basic caloric ration in a few days. All patients benefited from nursing and motor and respiratory physiotherapy.

The average length of hospitalization for patients with brain contusion was 28 days with extremes ranging from 14 to 66 days.

A favorable outcome was noted in 241 patients, i.e. 80% compared to 20% who died. 145 patients left the home service, 96 patients were transferred to another service.

Among the 136 patients with contusion, 89 were discharged, 27 were transferred, and 20 died.

Neurological sequelae such as headaches and motor deficits were noted in 25% and 6% of patients respectively, 5% remained in a vegetative state.

Of the 20 deaths noted, septic shock due to nosocomial pneumonia was the most common cause found in 15 patients. 2 patients died following HED, 3 patients had unilateral mydriasis on admission.

After our statistical analysis, we concluded the following poor prognosis factors:

- Age > 60 years
- GCS < 8
- Thickness > 15 ml
- Association of contusion with other brain lesions such as HED and cerebral edema
- Polytrauma.

Discussion

Head trauma is not a trauma like any other. It is a trauma to the body by a mechanical event which affects the head. The dissipation of the physical energy involved during the trauma will cause more or less severe lesions, which are ultimately either tolerated by the body and in this case the patient survives with or without after-effects, or not tolerated and in this case the patient dies.

It is also a trauma for the person, because the potential alteration of mental capacities can give rise to more or less significant personality modifications, sometimes incompatible with previous social, professional or family life.

The incidence is 281 TC/100,000 inhabitants/year with a male predominance whatever the age. AVP represents 60% followed by falls at 32%.

The physiopathology of TBI describes two mechanisms: TBI with direct impact on the skull or TBI without direct impact where the head is subjected to a deceleration/acceleration effect.

We distinguish between primary lesions, immediately present after the trauma and which are inevitable, and secondary lesions which develop in a delayed manner over a few hours and which must be detected and treated.

Among the primary lesions we note scalp wounds, bone lesions, intracranial extracerebral lesions, and brain lesions including the brain contusions studied in our article.

Secondary lesions are represented by extradural, acute subdural, chronic subdural, intraparenchymal hematomas and cerebral edema.

Brain CT is not systematic and is not indicated in all TBI according to the Masters classification, however it is the key examination to clarify the lesion assessment and justify emergency neurosurgical intervention. It must be requested after careful examination and hemodynamic and respiratory stabilization. The reference classification is the Traumatic Coma Data Bank (TCDB), which allows a better assessment of the risk of ICH and the long-term prognosis.

Brain contusion is an imprecise term encompassing all brain lesions linked to the transmission of the shock wave. They become complicated by ICH and progress towards the onset or worsening of a coma, which justifies their treatment in a center specialized in neurotraumatology regardless of the initial clinical state.

Cerebral edema complicates most parenchymal lesions; it can be localized or diffuse. In our study it was encountered in 39 patients [1-4].

Conclusion

Brain contusion is a superficial cortical lesion combining cellular and vascular lesions. They are often barely visible on the initial scanner and more significant after 48 hours.

Traumatic brain contusions are a major cause of mortality in developed countries. It is the most important structural lesion of the brain, it is complex and dynamic and results from a primary lesion.

Bibliography

- 1. The Ministry of Equipment, Transport and Logistics collects statistics on road traffic injuries for 2013.
- 2. Capizzi A., et al. "Traumatic Brain Injury: An Overview of Epidemiology, Pathophysiology, and Medical Management". *Medical Clinics of North America* 104.2 (2020): 231-238.
- 3. Paczynski RP. "Osmotherapy. Basic concepts and controversies". Critical Care Clinics 13.1 (1997): 105-129.
- 4. Lescot T., *et al.* "Opposed effects of hypertonic saline on contusions and noncontused brain tissue in patients with traumatic brain injury". *Critical Care Medicine* 34.12 (2006): 3029-3033.

Volume 16 Issue 1 January 2024 ©All rights reserved by Chaabi Safia., *et al*.