Traumatic Brain Injury In Intensive Care

Essay
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List of Abbreviations

ABP	:	Arterial blood pressure
ADH	:	Antidiuretic hormone
$AJDO_2$	:	Arterio venous oxygen difference
bTBI	:	Blast traumatic brain injury
CA	:	Cerebrovascular autoregulation
CBF	:	Cerebral blood flow
CCO	:	Cytochrome oxydase
cEEG	:	Continous electroencephalo graphy
CHI	:	Closed head injury
$CMRO_2$	:	Cerebral metabolic rate for oxygen
CPP	:	Cerebral perfusion pressure
CSF	:	Cerebro spinal fluid
CSWS	:	Cerebral salt wasting syndrome
CT	:	Computed tomography
DI	:	Diabetes insipidus
ECF	:	Extra cellular fluid
FV	:	Flow velosity
GCS	:	Glasgow coma score
ICAM	:	Intercellular adhesion molecules
ICP	:	Intra cranial pressure
ICU	:	Intensive care unit
IJV	:	Internal jagular vein

List of Abbreviations (Cont.)

LPR	:	Lactate pyruvate ratio
MAP	:	Mean arterial pressure
MD	:	Microdialysis
MRI	:	Magnetic resonant imaging
NIR	:	Near infrared
$pBrO_2$	:	Brain tissue oxygyn tention
PEEP	:	Positive end expiratory pressure
PET	:	Positron emission tomography
PI	:	Pulsatility index
PRx	:	Pressure reactivity indx
PtO_2	:	Tissue oxygen pressure
SAH	:	Sub arachenoid hemorrhage
SIADH	:	Inappropriate ADH secretion
SjO_2	:	Jagular venous oxygen saturation
SRS	:	Spectroscopy
TBI	:	Traumatic brain injury
TCD	:	Transcranial doppler
TDF	:	Thermal diffusion flowmetry
VCAM	:	Vascular adhesion molecules

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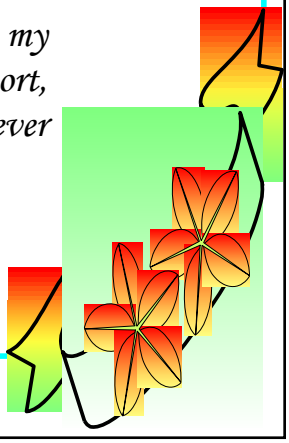

الملخص العربي

إن إصابات المخ التصادمية من أعظم أسباب الإعاقة والوفاة بالنسبة لحوادث التصادم. وهذا حقيقيا بالنسبة لإصابات المخ التصادمية الشديدة و التي تكون مصاحبة بنسبة عالية غير محببة من الإعاقة المرضية والوفاة أيضاً. ويمكن تقسيم باثوفسيولجية إصابات المخ التصادمية إلى أحداث أولية وثانوية: الحدث الأولى غير متوقع و لا يمكن تجنبه أما الحدث الثانى يمكن تجنبه وعلاجه لدرجة معينة، وبالتالي فإن تجنب الحدث الثانى من الممكن أن يؤدى إلى التحسن الملاحظ فى نتائج مرض إصابات المخ التصادمية.

وتربط إصابة المخ التصادمية الضغط الميكانيكي على أنسجة المخ بعدم التوازن بين رذاذ الضغط الدماغي والأيض الغذائي، تكون الإرتشاح الخلوى السام، عمليات الالتهابات، وعملية الموت الخلوى المبرمج.

ويعرض فهم التسلسل المتعدد الأطراف للإصابة خيارات علاجية تتضمن معالجة ضغط المخ التدفقى، التنفس الصناعي، العلاج الحركي لتحسين نسبة الأكسجين بالدم وتقليل الضغط المخى الداخلى والتدخل الدوائى لتقليل الضغط المخى الداخلى والتسمم الخلوى. و لكن عدم توقع الباثوفسيولوجيا الخاصة بكل فرد تتطلب ملاحظة المخ المصاب لتفصيل العلاج حسب الحالة الخاصة بكل مريض.

و وسائل الملاحظات المتعارف عليها فى وحدات الرعاية المركزة للأعصاب تتيح للأطباء والتمريض معالجة المرضى كرد فعل فقط عند تدهور الحالة

العصبية أو الفسيولوجية للمريض. ووجد أن وسائل الملاحظات الحالية مثل ضغط الأكسجين المخى، درجة حرارة المخ، الضغط المخى الداخلى، رذاذ الضغط الدماغي والأيض الغذائي المخى بالإضافة إلى الموجات الصوتية عبر المخ و رسم المخ المستمر ومؤشرات القلب والأوعية الدموية تعطى لأطباء وحدات رعاية الأعصاب رؤية حالية بالنسبة وفسيولوجية مناطق المخ المعرضة لخطر نقص الدم والإصابة وتسرع من إمكانية التدخل الفعال لعكس الحالات المرضية على أسس فردية. ووجد أن دمج ملاحظة ضغط المخ الداخلى ورذاذ الضغط الدماغى مكملا بضغط الأكسجين بالدم والنسبة بين اللاكتات والبيروفات من الممكن أن تفرق مناطق المخ التى تتعرض للخطر والتى يمكن أن تستجيب لتعديل ضغط الدم الرئيسى وذاذ الضغط الدماغى.

Introduction

Traumatic brain injury (TBI) is defined as a functionally significant disruption of brain function manifested as immediately apparent cognitive or physical impairments that result from blunt or penetrating trauma or rapid acceleration or deceleration forces. This definition excludes lacerations or contusions of the face, eye, or scalp, and fractures of facial bones alone. Additionally, injury to the brain resulting from birth trauma, hypoxic-ischemic (anoxic), inflammatory, toxic, or metabolic encephalopathies, primary ischemic or hemorrhagic strokes, seizure disorders, intracranial surgery, and cerebral neoplasms also are outside the definition of TBI ***(David and Thomas ,2008).***

TBI has a dramatic impact on the nation's health: it accounts for 15 – 20% of deaths in people aged 5–35 yr old, and is responsible for 1% of all adult deaths ***(Jennett and MacMillan, 1981).*** Approximately 1.4 million people in the UK suffer a head injury every year resulting in nearly 150 000 hospital admissions***(Hodgkinson et al., 1994)*** . Of these, approximately 3500 patients require admission to ICU. The overall mortality in severe TBI, defined as a post-

resuscitation Glasgow Coma Score (GCS) ≤8, is 23% ***(Hyam et al., 2006).***

In addition to the high mortality, approximately 60% of survivors have significant ongoing deficits including cognitive competency, major activity, leisure and recreation ***(Dikmen et al., 2003).*** This has a devastating financial, emotional, and social impact on survivors left with lifelong disability ***(Helmy et al., 2007).***

The principal mechanisms of TBI are classified as (a) focal brain damage due to contact injury types resulting in contusion, laceration, and intracranial haemorrhage or (b) diffuse brain damage due to acceleration/deceleration injury types resulting in diffuse axonal injury or brain swelling ***(Nortje and Menon, 2004).***

Outcome from head injury is determined by two substantially different mechanisms/stages: (a) the primary insult (primary damage, mechanical damage) occurring at the moment of impact. In treatment terms, this type of injury is exclusively sensitive to preventive but not therapeutic measures. (b) The secondary insult (secondary damage, delayed non-mechanical damage) represents consecutive pathological processes initiated at the moment of injury with delayed clinical presentation. Cerebral ischaemia

and intracranial hypertension refer to secondary insults and, in treatment terms, these types of injury are sensitive to therapeutic interventions ***(Werner and Engelhard , 2007).***

Aim of the work

- Understanding the pathophysiology after TBI for adequate and patient oriented treatment.
- Importance of monitoring the injured brain to enable the detection of harmful physiological events before they cause irreversible damage to the brain.

Pathogenesis of Traumatic Brain Injury (TBI)

Classically, TBI is thought to have at least two phases. The first or initial injury occurs as a direct result of the primary traumatic event. A second injury phase occurs from multiple neuropathologic processes that can continue for days to weeks after the initial insult ***(Geoffrey and Scott ,2008).***

Primary injury

Primary injury is immediate and not amenable to treatment. If severe, death can occur almost instantaneously. Typically, the damage that occurs from this primary phase is often complete by the time medical care can be instituted ***(Bledsoe and Li, 2005).***

There are two classical types of head injury: closed head injury (CHI) and penetrating TBI. In CHI, direct impact of neuronal tissue against the bony vault, and shearing of neurovascular structures from rotational or rebounding forces, results in cell damage at the cell body and axon level. In the United States, most CHI is caused by motor vehicle accidents.Other causes are falls, sporting event injuries, and assault. Motor vehicle accidents, which are high-speed