TREATMENT OF POST TRAUMATIC INTRACRANIAL HYPERTENSION BY ISOVOLUME HYPERTONIC SOLUTES (HYPERTONIC SALINE 7.5% VERSUS MANNITOL 20%)

Thesis

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List of Contents

List of Abbreviations

ABP ..... Arterial blood pressure

CBF ..... Cerebral blood flow

CPP ..... Cerebral perfusion pressure

CT ..... Computed Tomography

CVR ..... Cerebrovascular resistance

DAI ..... Diffuse axonal injury

GCS ..... Glasgow coma Scale

HR ..... Heart rate

HTS ..... Hypertonic saline

ICH ..... Intracranial hypertension

ICP ..... Intracranial pressure

MAP ..... Mean arterial blood pressure

NE ..... Norepinephrine

$PACO_2$ ..... Arterial carbon dioxide gas tension

PtO_2 ..... Brain tissue oxygen pressure

TBI ..... Traumatic brain injury

List of Tables

List of Figures

List of Figures

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علاج ارتفاع الضغط داخل الجمجمة ما بعد الإصابات الرضحية للرأس بواسطة كميات متساوية من محاليل مفرطة التناضح (محلول الملح مفرط التناضح ٧,٥% مقابل محلول المانيتول ٢٠%)

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فى التخدير

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INTRODUCTION

Traumatic brain injury (TBI) is a major cause of mortality and morbidity in young adults. Cerebral edema and subsequent intracranial hypertension (ICH) is an important factor influencing patient outcome ***(Binder et al., 2005).***

The primary aim of intensive care management of TBI is to prevent and treat secondary ischemic injury using multifaceted neuroprotective strategy to maintain cerebral perfusion to meet the brain's metabolic demands for oxygen and glucose ***(Martin, 2008).***

Intracranial pressure (ICP) monitoring has become as established component of brain monitoring after traumatic brain injury ***(Nau R, 2000).***

Infusion of hyperosmolar solutes is one of the treatment that is currently recognized for ICH after severe head injury ***(White et al., 2006).***

The growing interest in use of hypertonic saline solutions in treatment of post traumatic intracranial hypertension had been challenged the use of mannitol where elevation of ICP > 25mmHg plays major role in worsening of neurologic status through impairment of brain perfusion ***(White et al., 2006).***

AIM OF THE STUDY

Compartive study by increasing osmotic load of boluses of equal volumes of hypertonic saline 7.5% versus mannitol 20%(2ml/kg each dose) in treatment of intracranial hypertension episodes in TBI patients.

Where the ICP is monitored by using camino ICP monitor where microtrancuder tipped can be sited in the brain parenchyma or subdural space.

TRAUMATIC BRAIN INJURY (TBI)

Traumatic brain injury (TBI) constitutes a major health and socioeconomic problem throughout the world ***(Ghajar et al., 2000).***

It is the leading cause of mortality and disability among young individuals in high income countries, and globally the incidence of TBI is rising sharply, mainly due to increasing motor-vehicle use in low income and middle income countries ***(Andrew et al., 2008).***

This has consequences for the type of brain damage currently seen, and contusions (falls in older patients) are becoming more frequent than diffuse injuries (high velocity traffic accidents in younger patients).

Classification of TBI can be isolated, but is associated with extracranial injuries (limb fractures, thoracic or abdominal injuries) in about 35% of cases. Which increases the risk of secondary brain damage due to hypoxia, hypotension, pyrexia, and coagulopathy ***(Jiang et al., 2007).***

Traditionally, TBI has been classified by **mechanism** (closed vs penetrating), by **clinical severity** (Glasgow coma scale [GCS]) ***(Balesteri et al., 2004).***And by assessment of structural damage (neuroimaging; panel) ***(Maas et al., 2005).***

Panel: Approaches to classification of TBI by Mechanism Closed; penetrating; crash; blast. Clinical severity: level of consciousness (Glasgow coma scale) The GCS score comprises the values from three component tests (eye, motor, and verbal scales). Injuries are classified as severe (GCS 3–8), moderate (GCS 9–13), or mild (GCS 14–15) ***(Andrew et al., 2008).***

- Eyes: 1=no response; 2=open in response to pain; 3=open in response to speech; 4=spontaneous
- Motor: 1=no response; 2=extension to painful stimuli; 3=abnormal flexion to painful stimuli; 4=flexion/withdrawal to painful stimuli; 5=localises painful stimuli; 6=obeys commands.
- Verbal: 1=no response; 2=incomprehensible sounds; 3=inappropriate utterances; 4=disoriented, confused; 5=oriented, converses normally.

Clinical severity (injury severity score)

An abbreviated injury scale (range 0–6) is obtained for six body regions. The injury severity score (range 0–75) is the sum of quadratic scores for each of the six body regions.

- Body regions: external (skin); head/neck (includes brain); thorax; abdomen/pelvis; spine; extremities
- Scores: 0=none; 1=minor; 2=moderate; 3=serious; 4=severe; 5=critical; 6=virtually unsurvivable ***(Andrew et al., 2008).***

Structural damage (CT)

- Diffuse injury I: no visible pathology
- Diffuse injury II: cisterns present, midline shift 0–5 mm and/or lesion densities present or no mass lesion >25 mL
- Diffuse injury III (swelling): cisterns compressed or absent with midline shift 0–5 mm or no mass lesion >25 mL
- Diffuse injury IV (shift): midline shift >5 mm, no mass lesion >25 mL.
- Evacuated mass lesion: any lesion surgically evacuated.
- Non-evacuated mass lesion: High or mixed density lesion >25 mL, not surgically evacuated ***(Andrew et al., 2008).***

Prognosis

Classification by expected outcome as calculated from prognostic models as CRASH and IMPACT prognostic models using the coefficient data above and with calculations prognosis could be predicted ***(Andrew et al., 2008).***

Types of brain damage

Primary damage

TBI is a heterogeneous disorder with different forms of presentation. The unifying factor is that brain damage results from external forces, as a consequence of direct impact, rapid acceleration or deceleration, a penetrating object eg. gunshot, or blast waves from an explosion ***(Stein et al., 2002).***

The nature, intensity, direction, and duration of these forces determine the pattern and extent of damage.

On the macroscopic level, damage includes shearing of white matter tracts, focal contusions, haematomas (intracerebral and extracerebral), and diffuse swelling.

At the cellular level, early neurotrauma events (which can occur minutes to hours after initial injury) include microporation of membranes, leaky ion channels, and stearic conformational changes in proteins. At higher shear rates, blood vessels can be torn, causing (micro) haemorrhages ***(Clark et al., 2008).***

Diffuse axonal injury (DAI) is characterised by multiple small lesions in white matter tracts. Patients with DAI are usually in profound coma as a result of the injury, do not manifest high ICP, and often have a poor outcome ***(Lai et al., 2008).***

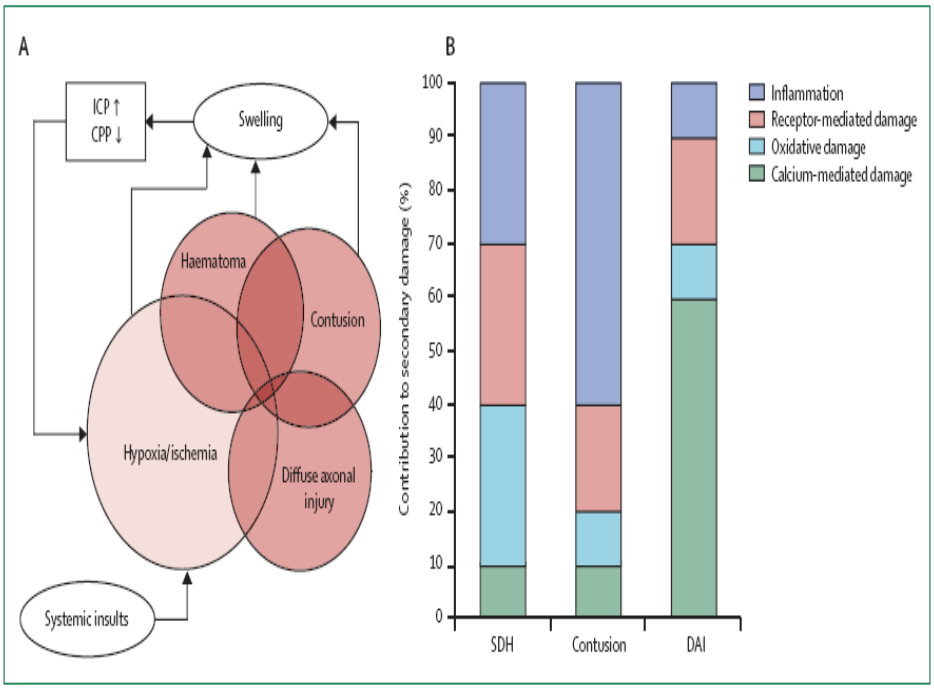

Fig. (1): Components of TBI and importance of different patho-physiological mechanisms; (A) The different components of TBI with ischaemic damage are superimposed on the primary types of injury (haematoma, contusion, and diffuse axonal injury). Systemic insults and brain swelling contribute to ischaemic damage, which might in turn cause more swelling. (B) The relative importance of different pathophysiological mechanisms in various types of TBI. CPP=cerebral perfusion pressure. ICP=intracranial perssure. SDH=acute subdural haematoma. DAI=diffuse axonal injury ***(Graham et al., 2002).***

Secondary damage

Ischaemic brain damage is often superimposed on the primary damage (figure 1), and can be widespread or, more commonly, perilesional. Impaired cerebral perfusion and oxygenation, excitotoxic injury, and focal microvascular occlusion can be contributing factors ***(Harhangi et al., 2008)***

Each type of head injury might initiate different pathophysiological mechanisms, with variable extent and duration. These mechanisms (acting concurrently and often with synergising effects) and the intensity of systemic insults determine the extent of secondary brain damage. Secondary processes develop over hours and days, and include neurotransmitter release, free-radical generation, calcium mediated damage, gene activation, mitochondrial dysfunction, and inflammatory responses ***(Vos et al., 2002).***

Glutamate and other excitatory neurotransmitters exacerbate ion channel leakage, worsen astrocytic swelling, and contribute to brain swelling and raised ICP. Neurotransmitter release continues for many days after TBI in human beings, paralleling the course of high ICP, and, with free-radical and calcium mediated damage, is a major cause of early necrotic cell death ***(Chieregato et al., 2006 and Smits et al., 2007).***

Also TBI could be classified into focal or diffuse according to the neuropathology ***(UK National Institute for Health and Clinical Excellence, 2008).***