KASR ALAINY

Clinical Outcome Of Decompressive Craniectomy Operation For The Management Of Acute Traumatic Brain Injury

Thesis

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By

Ahmed Mohammed Abdulsamie Elsharkawy
(M.B.; B.Ch. Cairo University)

Under supervision of

Prof. Dr. Hazem Abdelbadie Gouda
Professor of neurosurgery,
Faculty of Medicine, Cairo University

Prof. Dr. Mohamed Ahmed Elbeltagy
ASS.Professor of Neurosurgery,
Faculty of Medicine, Cairo University

Dr. Ahmed Mohammed Ali
Lecturer of Neurosurgery,
Faculty of Medicine, Cairo University

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LIST OF ABBREVIATIONS

ABS	: Acute brain swelling
ASDH	: Acute subdural hematoma.
ATLS	: Advanced Trauma Life Support
BTF	: Brain Trauma Foundation
CBF	: Cerebral blood flow
CBV	: Cerebral blood volume
CMR	: Cerebral metabolic rate
CPP	: Cerebral perfusion pressure
CSF	: Cerebrospinal fluid
CT	: Computed tomography
CVR	: Cerebrovascular resistance
DAI	: Diffuse axonal injury
DC	: Decompressive craniotomy
EVD	: External ventricular drain
GCS	: Glasgow coma scale
GOS	: Glasgow outcome scale
HTS	: Hypertonic saline
ICH	: Intracranial hypertension
ICP	: Intracranial pressure
ISS	: Injury severity score
LOC	: Loss of consciousness
MAP	: Mean arterial pressure
PTA	: Duration of post traumatic amnesia
PVI	: Pressure volume index
TBI	: Traumatic Brain Injury
TCDB	: Traumatic Coma Data Bank
V i/c	: Intracranial volume
VS	: Vegetative state

ABSTRACT

In this study we reviewed 20 patients with acute traumatic brain injury and they're all treated from different pathologies with Decompressive craniectomy after failed medical treatment. We concluded that Decompressive craniectomy operation has a novel role in lowering the increasing ICP considering the early surgical intervention and keeping in mind the relatively good GCS.

Key words:

Clinical Outcome Of Decompressive Craniectomy Operation For The Management Of Acute Traumatic Brain Injury

INTRODUCTION

Trauma is the most frequent cause of young adult (under 45 years of age) fatalities worldwide, and Traumatic Brain Injury (TBI) accounts for up to 50% of these deaths **(Jennett, 1998)**.

The incidence of traumatic brain injuries (TBI) is increasing globally, largely due to an increase in motor vehicle use in low- and middle-income countries **(Hawryluk & Bernstein, 2006)**.

Intracranial hematoma formation following head injury is the major injury in which death may have been potentially avoidable and in which many survivors are unnecessarily disabled following head injury due to a delay in the evacuation of the hematoma. The incidence of intra cranial hematoma and the type of hematoma vary widely depending on the different admission policies. The general classification depends on the relationship of the hematoma to the dura and brain, as extradural, subdural and intracerebral hematomas **(Andrew, 2005)**.

Acute subdural hematoma (ASDH) is one of the most common and most morbid traumatic neurosurgical emergencies with reported mortality rates ranging from 36% to 90%. Mortality rates for ASDH are significantly greater in elderly patients when compared with younger patients. Some believe the relationship of age to outcome after ASDH is a continuous function, where morbidity and mortality increase with increasing age **(Westermaier, 2007)**.

Acute subdural hematoma commonly arises from tearing of the bridging veins. They are often located in the temporal and frontal regions, and the morbidity and mortality are related to the extent of the underlying brain damage. The visco-elastic behavior of the bridging veins and their lack of reinforcement by arachnoids trabecula in the subdural space explain why they tear under high rates of acceleration during trauma **(Crooks, 1991)**.

An understanding of the pathophysiological mechanisms of primary and secondary injury is crucial to set optimal protocols of care to enable further advances in the outcome of patients with TBI. Primary injury occurs at the time of impact and is not reversible. Direct damage involves contact energy transfer. In addition to these mechanical forces to the brain itself, primary injury also occurs to the cerebral vasculature leading to vessel shear and disruption. Secondary injury evolves as a cascade of cellular events that are set in motion at the time of the primary impact. Within the first hours after injury a complex array of inflammatory, oxidative stress, metabolic, vascular and mitochondrial mechanisms have been activated and each has progressed to initiate further injury **(Shirley & Geoffrey, 2008)**.

The main factors affecting the prognosis of patients with ASDH include age, sex, injury severity score (ISS), intracranial pressure (ICP), presence of subarachnoid hemorrhage, Glasgow coma scale (GCS), hypotension, hypoxia, and time from injury to operation **(Yi, 2008)**.

Among these factors, only the time from injury to operation (that is, the operative timing) can be well intervened, thus it has

been more emphasized. However, it has been the most controversial factor so far. The mortality and functional survival rate are related to operative timing of patients with ASDH **(HZ, 2009)**.

Sawauchi et al. in 2004 developed rat animal models of traumatic brain swelling associated with ASDH. They found that the effects of TBI act synergistically with ASDH, exacerbating brain edema and increasing intracranial pressure. Moreover, a hypoxemic secondary insult contributes to marked brain swelling and refractory ICP. The pathophysiology of TBI may develop from the complex interaction of the hematoma volume, the severity and distribution of the primary traumatic insult, and the presence of secondary insults **(Sawauchi et al., 2004)**.

Intracranial hypertension (ICH) is a major cause of secondary brain injury and often follows trauma or stroke. Because ICP varies with changes in the volume of the intracranial contents, the traditional approach for treating intracranial hypertension has been to reduce the volume of one or more of the compartments, which include brain parenchyma, cerebrospinal fluid (CSF), and blood volume, either surgically or non-surgically **(Messing & Junger, 2003)**.

An alternate approach is to increase cranial volume by removing the skull and opening the dura. The underlying brain can then swell under the relatively distensible skin. The use of Decompressive craniectomy to control ICP has been advocated for a number of disease processes, including stroke, tumors, and trauma. The rationale for Decompressive craniectomy is to

prevent secondary injury caused by intracranial hypertension **(Holland, 2004)**.

The first DC was presented by Kocher in 1901, Cushing in 1903 and Horsley in 1906, in a period when most surgery was realized through a large "exploratory" bone flap to decompress the brain from the lesion. Unfortunately, the aesthetical results were disastrous and led to limiting the technique. **(Holland, 2004).**

In traumatic pathology the method knew supporters and detractors: Erlich (1904) suggested a Decompressive Craniectomy for all head injuries with persistent coma for more than 24-48 hours- without a specific diagnosis most cases would have died anyway. Rowbotham (1942) recommended DC for all traumatic comas which improved at first and when medical treatment was ineffective for 12 hours. Munro (1952) suggested that if intraoperative the brain was contused and swollen to realize a large craniectomy and opening the dura matter.
(Holland, 2004)

Among adversaries are Mayfield, Lewin, and Moody, whose papers during 1960-1970 notes a high mortality rate for the technique and discourage its utilization. **(Albanese, et al., 2003)**

After the introduction of CT scan, in 1975, Ramshoff, Morantz and other present series of comatose patients with acute subdural hematomas, operated during the first 6-12 hours, with DC realized by necessity. Despite technical and esthetical problems, they reported a survival rate of 40% with 27% return back to a previous life – however the method doesn't meet a general approval. **(Albanese, et al., 2003)**

The desert of rediscovering the DC belongs to Guerra in 1999 who presents in Journal of Neurosurgery his personal results of 20 years of DC using CT scan and ICP monitoring. His good results lead to the acceptance of the technique as second-tier therapy for refractory intracranial pressure. The following 5 years know more than 50 papers dedicated to DC leading to sections of neurosurgical meeting dedicated to the technique and at least 2 prospective double blind multicentric studies in going on its indication in traumatic pathology. **(Albanese, et al., 2003)**

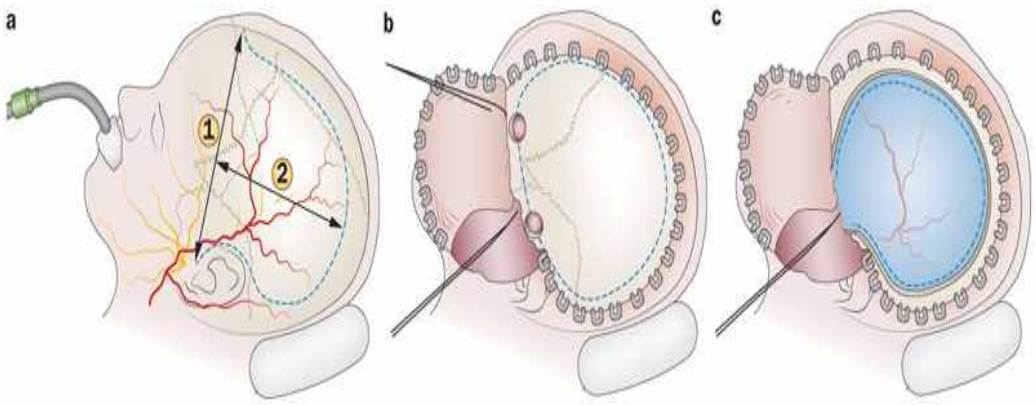

Fig.1. Decompressive Craniectomy operation steps

AIM OF THE WORK

The aim of the study is to review the literature and recent publication regarding the acute traumatic brain injury and the efficacy of the Decompressive Craniectomy procedure on the post-operative clinical state of these patients.

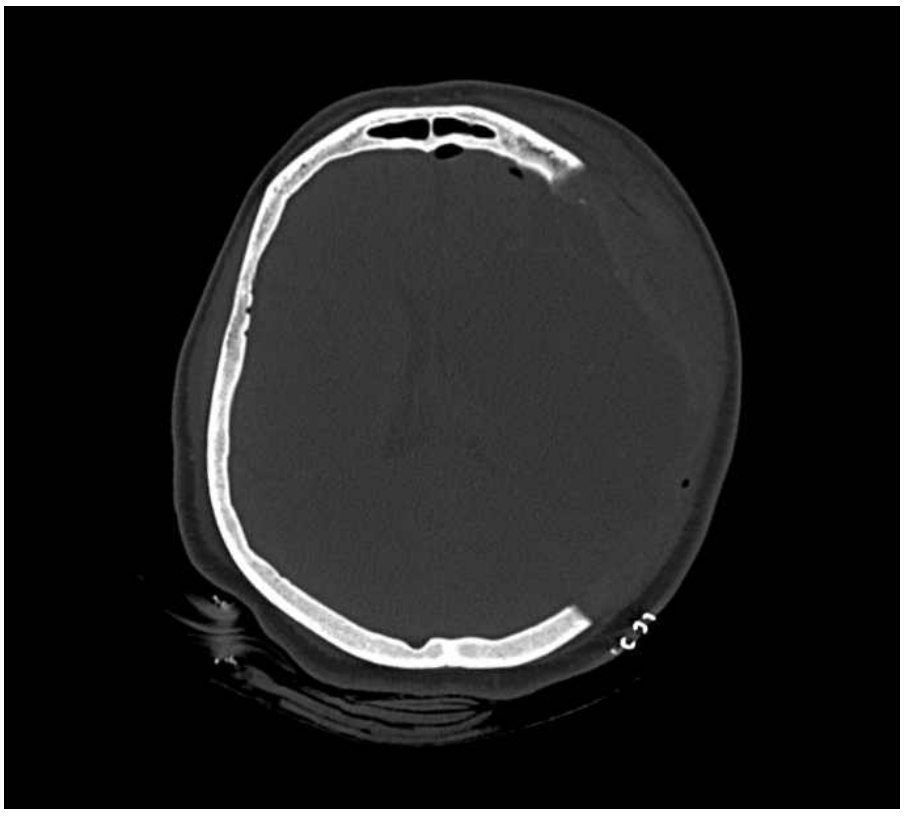

Fig.2. Postoperative CT scan of DC surgery

ANATOMY

The brain and spinal cord are surrounded by three membranes, or meninges: the dura mater, the arachnoid mater, and the pia mater **(Snell, 2001)**.

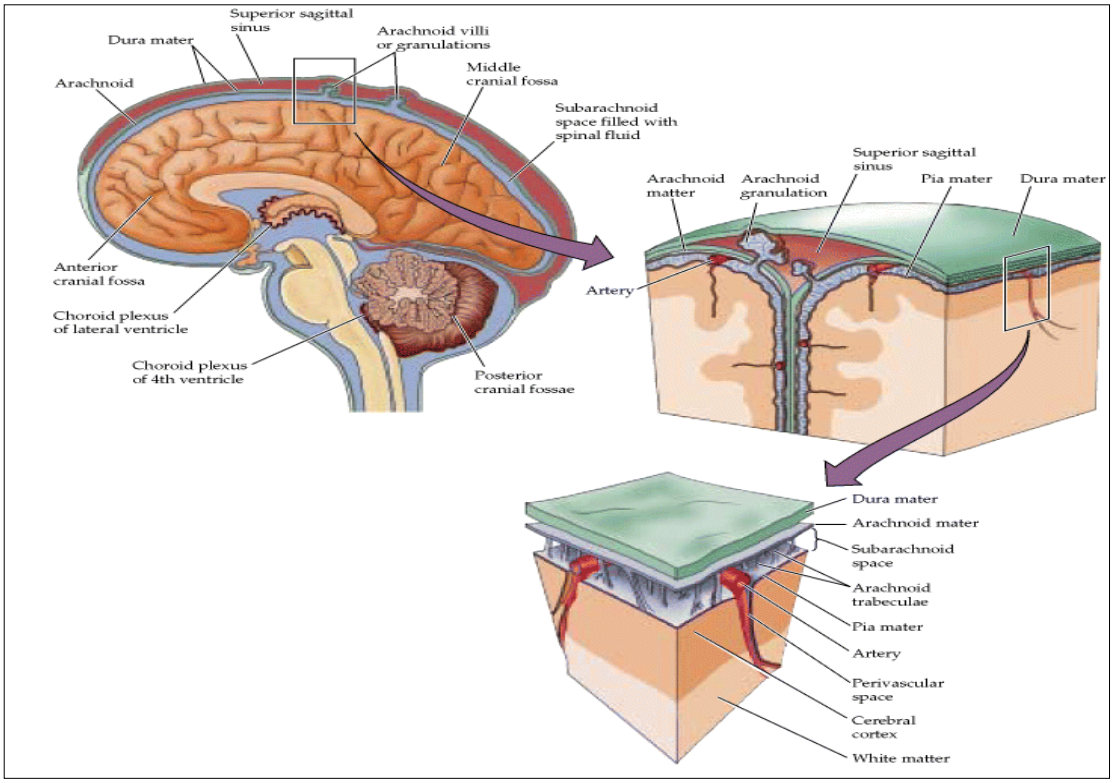

Fig.3. Layers of the brain **(Snell, 2006)**

The meninges comprise the outer dura, inner pia mater and intermediate arachnoid mater. The dura mater is the outer tough fibrous layer of the cerebral membranes. It comprises the endosteum of the skull vault, to which is fused the true (meningeal) layer of dura. These 2 layers are intimately attached to each other, and are separated only by the venous sinuses and where the inner layer invaginates to form the dural reflections **(Ellis, 2004)**.