POST - TRAUMATIC BRAIN ABSCESS

Thesis

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BY

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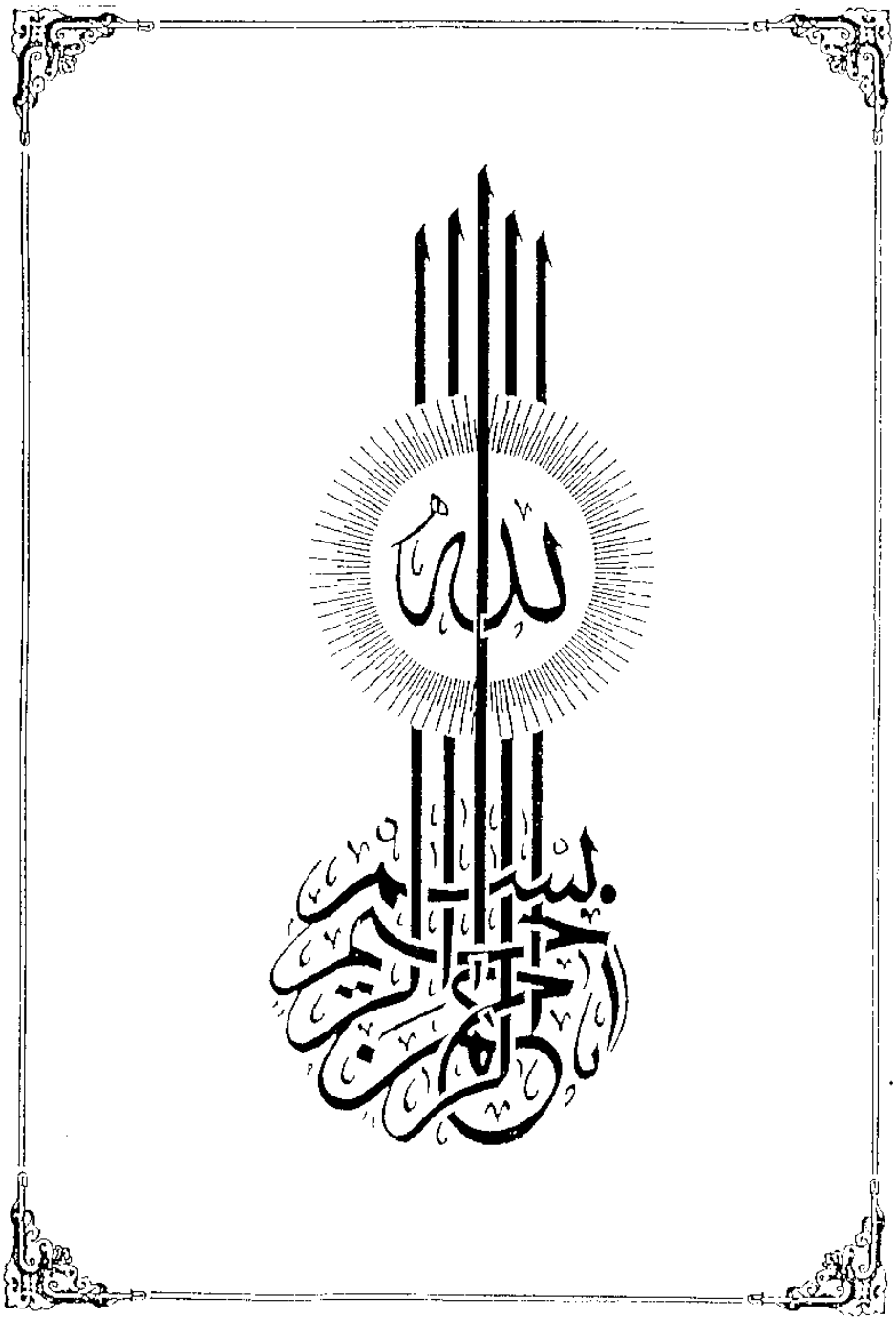
بسم الله الرحمن الرحيم

" قالوا سبحانك لا علم لنا الا ما علمتنا
انك أنت العليم الحكيم "

صدق الله العظيم



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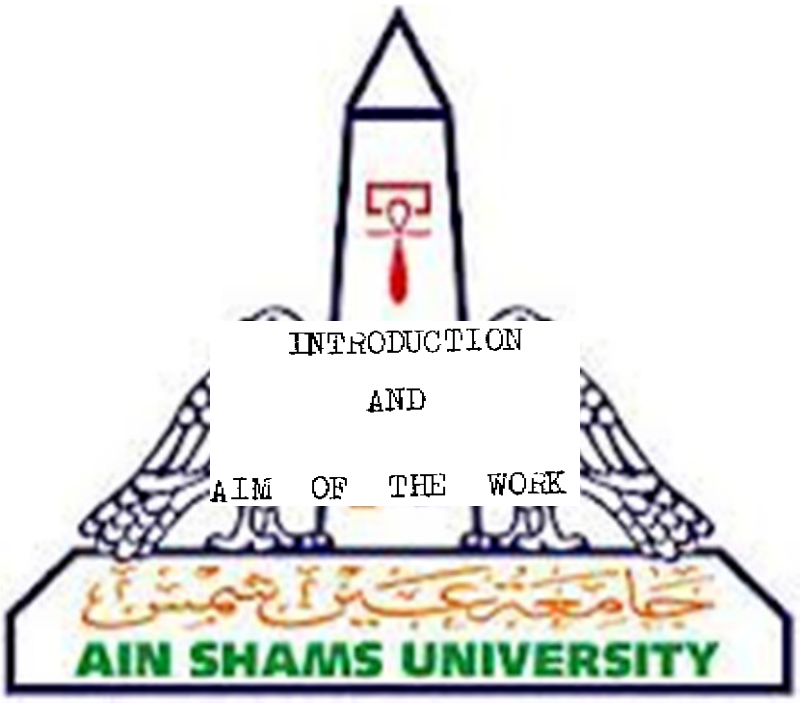

INTRODUCTION

Post - traumatic brain abscess is not infrequently encountered in neurosurgical practice. It is one of the most serious complications of head injuries.

In this country, the published reports concerning brain abscess in general emphasize the high incidence of post - traumatic abscess.

(Al - Sharif et al 1974, Sorour et al 1977 and Salama et al 1980).

Many authors showed that the number of cases of post - traumatic brain abscess increased in the last years in relation to the other varieties e.g. otogenic brain abscess (Martin 1973),

AIM OF THE WORK

We proposed to study cases of post - traumatic brain abscess that were admitted to the neurosurgical unit Ain - Shams University during the past five years. Our aim is to identily the factors that paved the way for the development of this serious complication, the methods of diagnosis and treatment, hoping to conclude the possible measures of prophylaxis and methods of early diagnosis and to select the best method of treatment.

REVIEW OF LITERATURES

Factors leading to the development of post-traumatic brain abscess and means of avoiding its occurance:-

Post-traumatic brain abscess seems to be increasing in the last years as compared to other types of brain abscess. It may complicate depressed or compound fracture of the skull, penterating injuries with missile and other foreign bodies and even closed head injuries. Many authors attributed its occurance to delayed (after 24 hrs) or inadequate management of head injuries. Vinken & Bruyn 1975 reported that the combination most likely to cause the condition is infection, necrotic brain tissue, blood and foreign body. They stated that an operation for head injury e.g. skull fracture or penterating injury should be performed within 24 hours. Shaving and thorough sterilisation of the head, meticulous debridement of the scalp wound, periosteum, dura mater and if necessary brain tissue is vital, with removal of bone fragments, hair, clothing and metal foreign bodies unless metal removal is likely to increase the damage, the torn dura is well closed and intracranial hematoma - if present - is evacuated. The same authors reported that brain abscess readily forms about the bone fragments, hair or clothing, this may be due to the organic nature of these foreign bodies which might help growth of organisms, or due to the self

sterilisation of the metal shell fragments by the heat generated from its terrific speed, also the knife blade may be scrapped clean by the bone as it penterates the skull. But it is note-worthy that cases of brain abscess developing around retained knife or missile after many years of the injury were reported by Cairns & Donald 1934, Drew & Fager 1954 and Horner et al 1964. Jennet & Miller 1972 emphasized the importance of thorough examination of any head injury and doing skull x ray when needed, many of their patients with established abscess had never been unconscious and their injury was of trivial nature.

Brain abscess may also occur after evacuation of traumatic intracranial hematoma due to contamination. This possibility increases with inadequate antibiotic cover.

It is note-worthy that the prophylactic use of adequate antibiotic cover especially when infection is suspected or formal debridement has to be delayed for some particular reason, usually associated injury is thought to reduce the incidence of post - traumatic brain abscess. Salama et al 1980 stressed that a strict correlation was present between the organisms found in the infected head injuries and those found in the abscess cavities resulting as a sequalae to these injuries, hence the importance

of taking a swab from the head wound for culture & sensetivity tests and giving the proper antibiotic in adequate doses in prophylaxis of post-traumatic brain abscess .

If infection supervenes early detection of brain abscess in the stage of cerebritis by careful clinical examination and using the recent diagnostic aids such as CAT and isotope brain scanning together with vigorous antibiotic treatment indicated by culture and sensetivity tests of a swab from the original head wound if possible, followed by careful observasion of the patients may prevent abscess formation. (Crocker et al 1974, Brewer et al 1975, &Connor et al 1977)

Pathology:-

Generally the pathological changes of brain abscess are often referred to as stages for descriptive purposes only. However the process is smoothly continuous, one stage merging imperceptibly with the next. Rarely the abscess develops in the grey matter as it commonly occurs in the white matter. The great vascularity of the grey matter gives it sort of resistanee. Initially there is an expanding area of septic encephalitis showing the characteristic changes associated with acute inflammation. Thus, the white matter is swollen with oedema and there is vasodilatation, diapedesis, rupture and thrombosis of small vessels and petechial haemorrhages. The changes are more advanced at the center which becomes softened partly as a result of the in tense oedema and partly from tissue necrosis then it liquefies. Vascular occlusion around the periphery of this area results in acceleration of extension of this necrotic area (Putnam & Alexarder 1938). The total extent of the oedema may occupy much of a lobe though its purulent core may be very small. The cavity wall is poorly defined and irregular. Small areas of necrosis subsequently coalesce. Histological examination shows necrosis bordered by hyperemic tissues infiltrated with polymorphs and veins heavily cuffed, beyond this there is

a zone of microglial activity and oedema in diminishing intensity. As time passes the abscess cavity expands, its wall becomes smooth and can be identified by nacked eye. It is propably firmer than the surrounding brain. This stage can be correlated histologically with the development of a lining granulation tissue. The fibroblasts being derived from the proliferating capillaries bordering the necrotic zone or by migration from nearby meninges. There is vigorous activity by microglia beyond this proliferation of astrocytes. The granulation tissue matures to form an inner fibrous wall to the abscess cavity which may be very tough. It is surrounded by ensheathing astrocytes which fade into the surrounding brain in an irregular deposition. The oedema of the surrounding brain steadily diminishes during the maturation of the abscess wall. The wall of the abscess is usually thicker and firm on its cortical aspect and thinnest on its deeper aspect. This may be due to the richer vascular hyperplasia available from the cortical vessels. This is particularly noticable in post - traumatic abscess. This has important consequences, as the pus increases in amount the cavity will more easily distend in a deep direction towards the ventricle, and as the wall is thin it may rupture into the ventricle (Falconer et al 1943).

The stages seen in the human brain were also observed in experimentally produced abscess by Russel et al 1941 and Falconer et al 1943. It is widely accepted that profound oedema is commonly associated with brain abscess and it is a principal factor in development of raised intracranial tension and herniation. Herniation is the commonest cause of death as shown by post-mortum studies (Tutton 1953 and Northfield 1973).

Pathogenesis:-

The skull is the first line of defence against invasion of the intracranial cavity by micro-organisms from the adjacent focus of infection. Skull fractures and penterating head trauma give a way to infection. The dura mater is the second line of defence against infection, on account of its close and tough texture and its good vascularity as emphasised by Rowbothan & little 1965. The dura may completely resist infection so that an extradural abscess is formed and the outer surface becomes covered with granulation tissue. Penteration of the dura occurs due to penterating head injuries or skull fractures in which the dura is torn. In closed skull fractures or in head injuries that do not tear the dura, its penteration most probably takes place along the course of the small