DIAGNOSIS AND MANAGEMENT OF CHRONIC NON NEOPLASTIC INTRACRANIAL SPACE OCCUPYING LESIONS

THESIS

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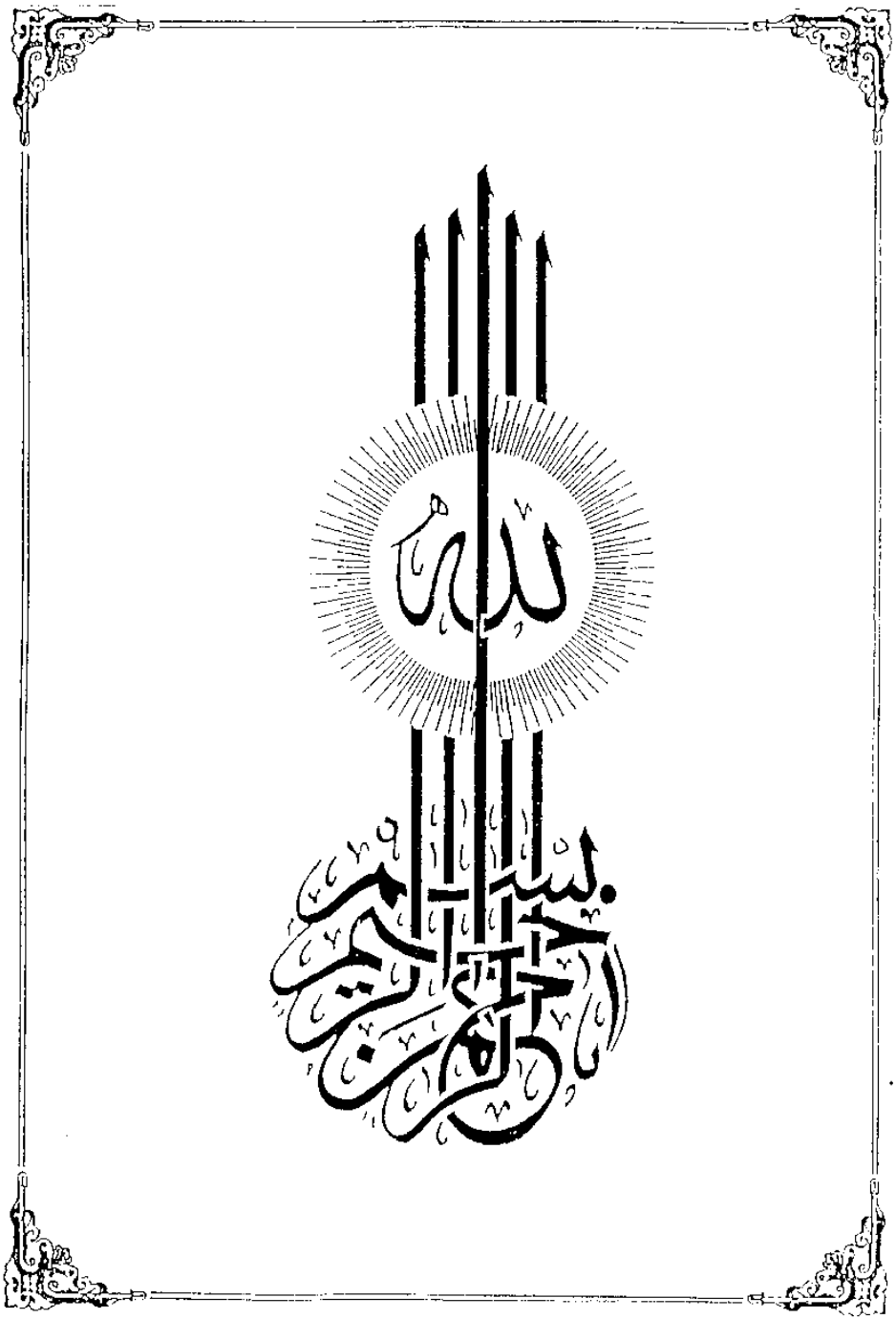

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AIM OF THE WORK

Neoplasms acount for majority of intracranial space occupying lesions. However, some lesions of non neoplastic nature are known to form a mass lesion in the brain thus resulting in a similar clinical manifestations. Investigations may be of help in detection of the pathological nature of the space occupying lesion wheather neoplastic or otherwise. The diagnosis of the nature of intracranial space occupying lesion is important in planning the management particularly the surgical procedure. The introduction of CAT scan into the field of diagnosis, as well as advances in serological tests has been of significant help in this field.

The aim of this work is to review various non neoplastic intracranial space occupying lesions with a study of their pathological nature clinical features methods of investigations and treatment. This work also include a study of 60 cases of intracranial non neoplastic space occupying lesions that were treated at Ain-Shams University: the department of Neuro-surgery in the past 7 years.

INTRODUCTION

Chronic non neoplastic intracranial space occupying lesions can be classified into infective and non infective groups.

THE INFECTIVE GROUP INCLUDES :-

- Pyogenic brain abscess.
- Tuberculoma
- Amebiasis
- Schistosomiasis
- Actinomycosis
- Blastomycosis
- Cladosporiosis
- Histoplasmosis
- Paracoccidioidomycosis
- cysticercosis
- Hydatid disease
- Paragonimiasis
- *Aspergillosis.*
- Candidiasis
- Cryptococcosis
- Nocardiosis.

THE NON INFECTIVE GROUP INCLUDES :-

- Dermoid and epidemoid cysts.
- Chronic Subdural hematoma
- Colloid cyst.
- Arachnoid cyst.

Review of the literature

PATHOLOGY & CLINICAL PICTURE

PYOGENIC BRAIN ABSCESS

Brain abscess is a purluent infection of the brain parenchyma and is, invariably, Tissue destructive existing as a mass lesion (Youmans, 1982)

As regard pathogenic mechanism responsible for brain abscess, Frontal nasal sinutis and middle ear disease or mastoditis . head most lists (Youmans,1982).The abscess is adjacent to the contiguous bony focus and often is attached to the dura mater at the site of the infection's spread from bone through meninges and into the parenchyma. Such a " stalk" may be noted at time of excision (Beller, 1973).

With otogenic sources, the temporal lobe and the cerebellum are the sites of abscess in a ratio of 3:2, (Show, 1975).

Metastatic spread may occur with general sepsis from a purluent intra Thoracic source,from pyogenic dental processes,furncules, distant osteomyelitis,endocarditis,or infected prothetic device,(Krayenbuhl, 1967).

Cyanotic congerital heart disease or other processes associated with a lesser or greater circulation shunt is a third mechanism,(Bhatra et al;1976).

Penetrating Trauma including craniotomy may give rise to abscess formation,(Morgan et al 1973).

Finally the origin may be unknown; an unexplained group of abscess remains in almost all series (Youmans 1982).

The thick walked abscesses are usually secondary to penetrating Trauma and retained foreign bodies such as bone, or to contiguous middle ear or mastoid disease;less often are the Thick walled lesion due to frontal sinusitis (Morgan et al,1073).

Abscesses secondary to cyanotic heart disease and those hematogenously spread from a purluent Thoracic process or more remote focus frequently thin walled (Bhatra et al; 1976);

Surrourding edema is a common characteristic of all forms of brain abscess except the most chronic very Thick-walled forms (Carey et al.,1972).

Solitary lesion are not infrequently multiloculated, and multiple lesion may occur, especially when hematogenous spread from a distant site occur.(Yoshikawa et al;1974).

Clinical evolution of an abscess is frequently described as occuring in three stages; The initial stage characterized by Transitory malaise,fever, headache & seizures.The latent stage when such symptoms having largely disappeared and the patient seems relatively well.The terminal in which a full clinical picture develops which will depend upon the size and number of lesion (if multiple) .The specific brain sites involved, and the neighbourhood anatomical disturbances involving ventricles, Venous siruses and cisterns (Northfield, 1973).

TUBERCULOMA

Although the decreasing incidence ofcerebral Tuberculosis represents the general trend throughout the world , some Countries in eastern Europe, the Middle East, and South America reported an incidence of 8% or more (Arseni, 1958), 20% (Ramamurthi et al.,1961).

Multiple foci may be present, Multiple foci were present in 15% of Arseni' case (Arseni, 1958),and in 33% of those of Asenjo et al,(1951).

Hematogenous spread from Tuberculous lesions of other parts of the body accounts for the origin of most cerebral Tuberculosis. Wilson, 1940,stated that if there was no evidence of Tuberculosis in other parts of the body the search should be taker as incomplete because Tuberculosis are never the primary lesion of a Tuberculous infectior.(Wilson, 1940).

Tuberculoma may occur at any age but about one half of the patients are less than 20 years of age and the cerebellum is frequently affected, particularly in the young,(Northfield, 1963).

The Tuberculomas usually appear as nodular or irregular a vascular masses localized beneath the cerebral or cerebellar cortex,although some are more superficial and adherent to the dura.They vary greatly in size, some are very small,while others are much larger.Cerebral edema is nearly always present around

the lesion and is quite sever in some cases.(Youmans,1982).

Higazi (1963),states that the hard mass lying in jelly-like softened brain renders the naked eye appearances characteristic.

Histologically, the tuberculomas give a typical picture of granulomatous lesions with necrotic areas and Langhans cells. The Turbercles have caseous centers and more rarely form real tuberculous abscess (Youman, 1982).

The symptoms and signs of a Tuberculoma are those of an exparding lesion,in accordance with its anatomical situation and in some cases rendered complex by the multiplicity of lesions. The great majority of patients develop sever papilledema and in about one half consecutive optic atropy causes permanent blindnessor sever loss of vision (Descuns et al,1954).

General symptoms such as asthenia,weakness and perispiration occur in 56% of cases,and fever in 40% of cases,(Chediack and Carrizo,1961).

PARASITIC & FUNGAL DISEASES

The parasitic and fungal diseases of the central nervous system are of increasing importance.It is estimated that they,in conjunction with the protozoan diseases,may infect as many as four to six hundred people at any given time,(Bruyn,1978).Of those infected,an estimated three millions die annually.International Travel has changed the traditional epidemiological pattern of diseases once thought to be exclusively restricted to endemic areas.

Parasitic & fungal diseases manifest themselves primarily in the pulmonary & gastero-irtestinal system.Involvement of the central nervous system oy any of them without systemic involvement else where is uncommon. Conversely,the onset of neurological dysfunction in patient with known parasitic or fungal diseases should suggest the possibility of dissemination to the central nervous system,(Bruyn,1978).

Fungi are commor in the environmert,but relatively few are pathogenic.An importart characteristic of those that are pathogenic is their dimorphism,or ability to assume two different forms in vivo under different environmental cordition,thus enhancing the pathogenicity of the organism.Mycotic diseases generally are rot transmitted from one person to another,nor are they contingious from animal to man.Epidemics commonly arise from an environmental source,and the mechanism of inoculation is ofter inhalation.A few of the entities such as candida albican and actinomyces Israeli, which are normal flora of mouth or gastro-intestinal tract are endogenous and capable of producing antoinfection, (Youmans, 1982).

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CYSTICERCOSIS

It is a parasitic disease that is usually produced by infestation with the larval stage of Taenia worm. Man serves as both the definitive and intermediate hosts for the adult tapeworm and for its larvae.When eggs containing an embryonal form of the organism (onchosphere) reach the stomach, the onchospheres are released during digestion.They,then penetrate the mucosa of the bowel and carried via the mesenteric circulation to various tissue sites, where they develop into the larval form of the organism(Obrador, 1948).

Five types of cerebral cysticercosis have been described on the basic of their anatomical area of involvement as well as histopathological features.They are the racerose (meningobasal), the cystic (Parenchymal),the mixed (Cerebromeningeal),the interventricular,and the spinal (Ahuja, 1975).

Cystic form of the disease produces a clinical syndrome congruous with the presence of mass lesion but symptoms and signs may begin within months of infection or may not occur for several years (Latovitzki, 1976).

HYDATID DISEASE

It is caused by the larval or hydatid stage of the dog tape worm Taenia Echinococcus. Man serves as an intermediate host.When the egg is ingested by man.The egg capsule is digested releasing hexacanth onchosphere in the jejunum where it penetrates the intestinal mucosa and passes into the portal circulation.65% become entrapped in the small intrahepatic venules,20% pass to lung,2-3% to central nervous system, 4-5% to bone,and other sites 7-9%(Ayres, 1963).

Following distribution to these various sites,the embryo begins the transformation into the vesicular structure that will become the cyst. After several moths the cystwall differinate into an internal granular layer and outer cuticular laminated non nuclear layer.The host reacts by forming an odventitial fibrous capsule.The cavity of the cyst contains hydatid fluid with daughter cysts,broad capsules,and scolices.(Ayres,1963).

Cerebral cyst is usually single,supratentorial and tend to occur in the distribution of middle cerebral arteries.These cyst can attain large sizes because they encounter relatively little resistence.Multiple cysts occur either by embolization of multiple larvae or by spontaneous cys rupture.Iatrogenic spread can occur during investigative procedures or at operation (kaya, 1975).

The localization of cysts is relatively evenly divided betweer frontal,temporal,and parietal lobes.Less than 3% are seen in the cerebellum.Intra ventricular cysts have also been