Cognitive and Neurological Complications of Meningitis at Abbassia Fever Hospital

Thesis

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List of Abbreviations

Abb. Meaning

ADA	Adenosin deaminase
AFB	Acid fast bacilli
BBB	Blood-Brain barrier
CBC	Complete blood count
CDC	Centers for Disease Control and Prevention
CTD	Connective tissue disease
CN	Cranial Nerve
CNS	. Central nervous system
CRP	C-reactive protein
CSF	Cerebrospinal fluid
CT	Computed tomography
DCL	Disturbed conscious level
DIC	Disseminated intravascular coagulopathy
EEG	Electroencephalogram
ESR	Erythrocyte sedimentation rate
GCS	Glasgow coma score
GOS	Glasgow outcome scale
HIV	Human immunodeficiency virus
HSV	Herpes simplex virus
ICP	Intracranial pressure
117	Introvencya

JCV.....John Cunningham virus

MMSEMini Mental State Examination

MRIMagnetic resonance imaging

PMNPolymorphonuclear leukocytes

RT-PCR.....Reverse transcriptase polymerase chain reaction

SIADH.....Syndrome of inappropriate antidiuretic hormone secretion

SLE.....Systemic lupus erythematosus

TB.....Tuberculous

TNF- α.....Tumor necrosis factor alpha (TNF-α)

WHO.....World health organization

WMS......Wechsler Memory Scale

Introduction

cute infections of the nervous system are among the most important problems in medicine because early recognition, efficient decision-making, and rapid institution of therapy can be lifesaving. These distinct clinical syndromes include acute bacterial meningitis, viral meningitis, encephalitis and other focal infections. Each of these clinical syndromes may present with a nonspecific prodrome of fever and headache, this prodrome in a previously healthy individual may initially be thought a benign illness (Longo et al., 2012).

Acute meningitis is clinically defined as a syndrome characterized by the onset of meningeal symptoms over the course of hours to up to several days. Headache is a prominent early symptom, often followed later by a state of abnormal consciousness, or coma (**Tunkel et al., 2010**).

Despite advances in medical care, the introduction of cranial computed tomography (CT) and improvements in intensive care support, the mortality from bacterial meningitis remains high. The global spread of multidrug-resistant bacteria has further complicated the treatment of patients with bacterial meningitis (van de Beek et al., 2010).

Worldwide, it has been estimated that 1-2 million cases of bacterial meningitis occur annually. The problem is more significant in resource-poor countries including those in some regions of Sub-Saharan Africa, Southeast Asia, and Latin America (Franco-Paredes et al., 2008).



Tuberculous meningitis is a life-threatening form of tuberculosis and is the most common form of central nervous system (CNS) tuberculosis (Girgis et al., 1998).

There is little information about the severity and distribution of the different types of sequelae. Additionally, the financial burden that families incur in caring for disabled individuals is commonly not calculated or is underestimated. Data are especially poor from lowincome countries, where the risks of infection are highest and care is least accessible (Edmond et al., 2010).

Bacterial meningitis is associated with high mortality and risk of neuropsychological sequelae (Ramakrishnan et al., 2009).

After bacterial meningitis, patients are at high risk of cognitive impairment (Hoogman et al., 2007).

Research on cognitive outcome in bacterial meningitis is scarce and often with methodological shortcomings (Weisfelt et al., 2006).

Mortality and serious long-term sequelae still occur in about 50% of patients with tuberculous meningitis (Anderson et al., 2010).

Patients suffering from viral meningitis may experience cognitive impairment following the acute course of the disease (**Damsgaard et al.**, 2011).

Fungal meningitis is a serious disease caused by a fungal infection of the CNS mostly in individuals with immune system deficiencies (Liu et al., 2012).



Outcome is frequently assessed by the Glasgow outcome scale (GOS), which is a well validated scale with good interobserver agreement (Weisfelt et al., 2006).

Inpatient hospital audit and peer review has improved markedly over the past decade, but post-discharge care has not received the same attention (Edmond et al., 2010).

${f A}$ IM OF THE ${f W}$ ORK

The aim of this work was to study and clarify the different cognitive and neurological complications in patients diagnosed with meningitis and presented to Abbassia fever hospital.

Introduction To Meningitis

Anatomy of the meninges:

The meninges are an important connective tissue envelope investing the brain. Their function is to provide a protective coating to the brain and also they participate in the formation of blood—brain barrier. Understanding their anatomy is fundamental to understanding the location and spread of pathologies in relation to the layers (Patel and Kirmi, 2009).

The cranial meninges also form the supporting framework for arteries, veins, and venous sinuses; and enclose a fluid-filled cavity, the subarachnoid space. The meninges are composed of three membranous connective tissue layers (**Fig 1**):

- Dura mater (dura): tough, thick external fibrous layer.
- Arachnoid mater (arachnoid): thin intermediate layer.
- Pia mater (pia): delicate internal vascular layer.

The arachnoid and pia are continuous membranes that make up the leptomeninx. The arachnoid is separated from the pia by the subarachnoid space, which contains cerebrospinal fluid (CSF). This is a clear liquid similar in constitution to blood; it provides nutrients but has less protein and a different ion concentration. CSF is formed predominantly by the choroid plexuses within the four ventricles of the brain. CSF leaves the ventricular system of the brain and enters the subarachnoid space, where it cushions and nourishes the brain (Fig 2) (Keith et al., 2010).

Dura mater

The dura mater consists of two layers, an external periosteal layer and an internal meningeal layer. The two dural layers are indistinguishable except where they separate to enclose the dural venous sinuses (**Tank**, **2005**).

Dura mater is thick, dense and fibrous. It is predominantly acellular, and consists mainly of densely packed fascicles of collagen fibres arranged in laminae. The fascicles run in different directions in adjacent laminae, producing a lattice-like appearance which is particularly obvious in the tentorium cerebelli and around the defects or perforations that sometimes occur in the anterior portion of the falx cerebri. The meningeal layer of the dura is reflected inwards to form four septa, namely the falx cerebri, falx cerebelli, diaphragma sellae and tentorium cerebelli that partially divide the cranial cavity into compartments (**Standring, 2008**).

In the skull, the outer layer of the dura mater is closely applied to the inner table of the skull and corresponds to the periosteum. It does not only have connective and supportive functions but also may play a role in the formation of the cranial bones. This outer portion of the dura mater contains the meningeal arteries and veins, which form grooves in the inner table of the skull (Sze, 1993).

Spinal dura mater forms a loose sheath round the spinal cord, extending from the foramen magnum to the second sacral vertebra. Thereafter it encloses the filum terminale and fuses with the periosteum of the coccyx (**Fig 3**). It is an extension of the inner layer of cerebral dura mater and is separated from the periosteum of the vertebrae and ligaments within the