شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

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التوثيق الالكتروني والميكروفيلم

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بالرسالة صفحات لم ترد بالاصل

THE ROLE OF VISUAL EVOKED POTENTIALS IN EARLY DETECTION OF VISUAL PATHWAY INVOLVEMENT IN DIABETIC PATIENTS

Thesis

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ABBREVIATIONS

μV	Microvolt
AFCL	Axillary F central latency
Amp	Amplitude
CNS	Central nervous system
CSF	Cerebrospinal fluid
CMAP	Compound muscle action potential
DM	Diabetes mellitus
Hz	Hertz
IOAD	Interocular amplitude difference
IOD	Interocular difference
IOLD	Interocular latency difference
Lat	Latency
mg|dl	milligram per decelliter
mm	millimeter
ms	millisecond
m/s	meter per second
mV	millivolt
NPDR	Non proliferative diabetic retinopathy
PDR	Proliferative diabetic retinopathy
PR-VEP	Pattern reversal visual evoked potential
SNAP	Sensory nerve action potential
VEP	Visual evoked potential

CONTENTS

INTRODUCTION

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Diabetes mellitus comprises an etiologically and clinically heterogeneous group of hyperglycemic disorders. The hyperglycemia is the consequence of a relative or absolute deficiency of insulin in the presence of a relative or absolute excess of glucagons.[1]

Diabetes is associated with a set of late complications involving the eyes, the kidneys, nerves and blood vessels.[2] In all of these tissues the major cause of tissue damage is a vascular disease affecting both microvasculature and macrovasculature.[3] The most common pathologies of microvessels occur in diabetic retina and kidney.[4] Macrovascular diseases occur in large peripheral arteries of the lower limbs, in cerebral vessels and coronary arteries.[3,5]

Diabetic neuropathy

There are various forms of diabetic neuropathy that could be recognized: symmetrical sensorimotor polyneuropathy, autonomic neuropathy and focal neuropathies, which include diabetic proximal neuropathy, mononeuropathies of cranial nerve and peripheral nerves, and truncal neuropathies.[6,7]The first two are considered metabolic in etiology, whereas

mononeuropathy is usually attributed to disease of vasa nervosum.[7]

The cause of diabetic neuropathy

The cause of diabetic neuropathy is unknown. Three hypotheses have received most attention: the vascular hypothesis the axonal hypothesis and the metabolic hypothesis.[7]

Ischemic disease of the arteriole is generally acknowledged as a primary cause of mononeuropathy but microvascular disease is also considered as a primary contributor to other forms of neuropathy. The axonal hypothesis suggests early functional changes, such as slow axonal transport, followed by structural degeneration. [8]

Understanding metabolic abnormalities of diabetic nerve comes from studies in rats. In experimental diabetes the myo-insitol content of nerves and motor conduction velocity decreases in parallel.[7] Persistent hyperglycemia may activate the polyol pathway (aldose reductase) in nerve causing sorbitol accumulation. Sorbitol content of nerves is inversely related to the number of myelinated fibers.[7] The sequence appears to be as follow: hyperglycemia will lead to increased sorbitol and decreased myo-inositol in Schwann cells and axons.

Consequently axolemmal sodium and potassium ions adenosin triphosphatase activity decreases. This will lead to abnormal energy metabolism, nerve dysfunction and structure damage.[7]

CNS involvement in diabetes mellitus has gained much attention in the last few years. Contrary, to some early impressions, the CNS is not spared in diabetes.[9,10] The metabolic dysregulation influences the cerebral blood flow. In diabetic patients alteration in substrate transport and metabolism have been demonstrated at the neurochemichal, electrophysiological and structural levels.[11]

Neuropathological and neuro-imaging studies revealed structural damage in the brain tissue, demyelination, signs of micro and macroangiopathy and cerebral atrophy.[11] The postulated pathogenic mechanisms are glucose related, including abnormalities in polyol metabolism, glycation and vascular changes.[12]

Diabetic ocular lesions

The main ophthalmologic diabetic complications are oculomotor disturbance, optic neuropathy, rubeosis iridis leading to neovascular glaucoma, cataract and diabetic retinopathy, which is particularly frequent.[13]

Diabetic retinopathy

Retinopathy is a common complication of diabetes and is the principale cause of blindness in adult population. Studies have suggested that eventually up to 75% of all diabetic patients will develop retinopathy.[14] The incidence of diabetic retinopathy increases with the duration of diabetes. Other factors which affect the progression of diabetic retinopathy are the age of onset of diabetes, the level of glycemic control and blood pressure.[14]

General architecture of the retina

The retina is the inner most layer of the eye. It consists of the outer pigmented retina which is formed of the pigmented simple cuboidal epithelium and inner sensory retina which responds to light. The sensory retina contains 120 million photo receptor cells called rods and another 6 or 7 million cones, as well as numerous relay neurons.[15]

As seen in cross section by light microscopy, the retina is represented by 10 layers: retinal pigmented epithelium, photoreceptor layer of rods and cones, external limiting membrane, outer nuclear layer, outer plexiform layer, inner nuclear layer, inner plexiform layer, ganglion cell layer, nerve