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شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

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التوثيق الالكتروني والميكروفيلم

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بالرسالة صفحات لم ترد بالاصل

NEUROPROTECTION IN GLAUCOMA

ESSAY
Submitted for partial fulfillment
of M.Sc. Degree of Ophthalmology

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محضر

أجتماع لجنة الحكم على الرسالة المقدمة من
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في [illegible]

تحت عنوان : باللغة الانجليزية : Neuroprotection in Glaucoma

: باللغة العربية : وقاية العصب البصري في حالات المياه الزرقاء

بناء على موافقة الجامعة بتاريخ / / ١٩ تم تشكيل لجنة الفحص والمناقشة للرسالة المذكورة أعلاه على النحو التالي :-

١) [illegible] من المشرفين
٢) [illegible] متحن داخلي
٣) [illegible] ممتحن خارجي

بعد فحص الرسالة بواسطة كل عضو منفردا وكتابة تقاريره منفردة لكل منهم إنعقدت اللجنة مجتمعة في يوم [illegible] بتاريخ / / ١٩ بقسم [illegible] بكلية الطب – جامعة القاهرة وذلك لمناقشة الطالب في جلسة علنية في موضوع الرسالة والنتائج التي توصل إليها وكذلك الأسس العلمية التي قام عليها البحث .

قرار اللجنة : [illegible] قبول الرسالة

توقيعات أعضاء اللجنة :-

المشرف والمتحن — المتحن الداخلي — المتحن الخارجي

(صام)

Abstract

Glaucoma is one of the leading cause of bilindness all oer the world. It cn be defined as a disease in which one of the pathophysiological consequences of raised intraocular pressure (IOP) is damage to the optic nerve and, subsequently, loss of the restinal ganglion cells. The fact that patients with glaucoma constinue to develop visual field defects long after the therapeutic normalization of their IOP suggests that elevated IOP alone cannot explain the propagation of glaucomatous optic neuropathy. .

However, many of these drugs have been used for the treatment of other conditions, and in order to achieve efficacy in glaucoma, should be used systmeically. This holds the risk of producing adverse reactions affecting other systems. Other drugs are still under clinical studies and are not available commercially.

Keywords: Genglion cell layer- Nerve fiber layer- Optic nerve head- The retina- The Optic nerve - Ocular circuation.

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INTRODUCTION

Glaucoma is the one of the most prevalent causes of global blindness. It can be defined as a disease in which one of the pathophysiological consequences of raised intraocular pressure is damage of the optic nerve, and subsequently loss of the retinal ganglion cells (RGC). Thus the rise of intraocular pressure (IOP) is only the initial insult, that causes impairment of the blood flow, or enhances the increase or release of substances, as glutamate or nitric oxide, with a concentration sufficient to induce RGC death. RGC death is the final common pathway of virtually all diseases of the optic nerve, including glaucomatous optic neuropathy. In recent years, it has been shown that retinal ganglion cells die after a programmed cell death process called apoptosis. There are ample clinical indications for the existence pressure-dependant and pressure independent mechanisms for glaucomatous optic neuropathy. Such mechanisms might operate over a wide range of intraocular pressure, and would contribute to damage both at low or high pressures.

Recent scientific advances include studies of hemodynamic and biochemical factors involved in the development of glaucomatous optic neuropathy. However, treatment of glaucoma continues to be directed at lowering the IOP to decrease the likelihood of disease progression. Interest has been increasing in preventing progression of glaucomatous optic neuropathy using approaches based on the premises that glaucoma is a neurodegenerative disease. Substances that prevent the excitotoxic cascade of glutamate or nitric oxide are, therefore, often found to act as neuroprotective agents.

AIM OF THE STUDY:

This essay deals with the review of the literatures about the pathophysiology of glaucomatous optic neuropathy, early detection of nerve affection as well as the proposed neuroprotective drugs and their therapeutic role in the management of the disease.