شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

شبكة المعلومـــات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

﴿ قـسـم ﴾

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأفلام قد اعدت دون آية تغيرات

يجب أن

تحفظ هذه الأفلام بعيداً عن الغبار

في درجة حرارة من 15 – 20 مئوية ورطوبة نسبية من 20-40 %

To be kept away from dust in dry cool place of
15 – 25c and relative humidity 20-40 %

بعض الوثائق الأصلية تالفة

بالرسالة صفحات

لم ترد بالأصـــل

Color Doppler Study of the Ophthalmic Artery and the Central Retinal Artery in Patients with Primary Open-Angle Glaucoma

Thesis

Submitted to The Faculty of Medicine

Alexandria University

In partial fulfillment of the requirements

Of the degree of

Master Of Ophthalmology

By

Tamer Hamdy Ibrahim Massoud

MBBCH 1997

Alexandria

FACULTY OF MEDICINE

ALEXANDRIA UNIVERSITY

2002

SUPERVISORS

Prof. Dr. Abel-Kader Al-Saeed Zidan
Professor and Chairman of Ophthalmology Department
Faculty of Medicine
Alexandria University

Dr. Hesham Fouad El-Goweini
Assistant Professor of Ophthalmology
Faculty of Medicine
Alexandria University

Dr. Mohamed Ihab Samy Reda
Assistant Professor of Diagnostic Radiology
Faculty of Medicine
Alexandria University

Co-Worker

Dr. Ahmed El-Sayed Shama
Assisstant Professor of Ophthalmolgy
Faculty of Medicine
Alexandria University

To My Family

Acknowledgments

I would like to express my deepest gratitude and sincere appreciation to ***Prof. Dr. Abdel-Kader Al-Saeed Zidan***, Professor and Chairman of Ophthalmology Department, Faculty of Medicine Alexandria University, for his kind and valuable supervision and for his constructive advice and encouragement throughout the whole work.

I am grateful to ***Dr. Hesham Fouad El-Goweini,*** Assistant Professor Ophthalmology, Faculty of Medicine, Alexandria University, for his great interest, generous help and support throughout the progress of this work.

I am deeply indebted to ***Dr. Mohamed Ihab Samy Reda***, Assistant Professor of Diagnostic Radiology, Faculty of Medicine, Alexandria University, for his great help, patience and experienced guidance through the period of the work.

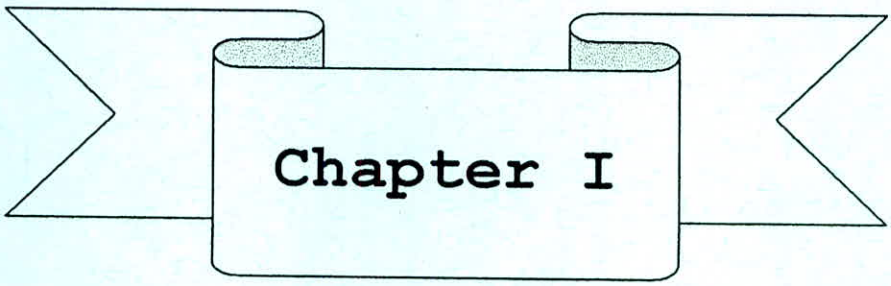
I am also thankful to ***Dr. Ahmed El-Sayed Shama***, Assisstant Professor of Ophthalmology, Faculty of Medicine, Alexandria University, for his continuous assistance and precious advice throughout the work.

Finally, I would like to extend my gratitude to all the staff members and colleagues in the Ophthalmology department for their help and encouragement.

ABBREVIATIONS

IOP	Intraocular pressure
ONH	Optic nerve Head
POAG	Primary open angle glaucoma
NTG	Normal tension Glaucoma
GON	Glaucomatous optic neuropathy
NFL	Nerve fiber layer
CRA	Central retinal artery
OA	Ophthalmic artery
PCA	Posterior ciliary arteries
SPCA	Short posterior ciliary arteries
CDI	Color Doppler Imaging
PSV	Peak systolic velocity
EDV	End diastolic velocity
MV	Mean velocity
RI	Resistivity index
PI	Pulsatility index
MD	Mean deviation
PSD	Pattern standard deviation
C/D	Cup-disc ratio

CONTENTS



INTRODUCTION

Primary Open Angle Glaucoma

Glaucoma can be defined broadly as a group of pathological entities with different pathophysiological mechanisms characterized by ocular damage, related - in part -to intraocular pressure (IOP) higher than the tissues of the eye can tolerate. However, pressure independent mechanisms, such as vascular and structural alterations of the optic nerve head (ONH) may contribute in some cases to glaucomatous damage of the optic nerve. [1]

Primary open angle glaucoma (POAG) is a chronic, slowly progressive, multifactorial, usually bilateral, though not necessarily symmetrical, anterior optic neuropathy characterized by atrophy and cupping of the ONH, resulting in a distinctive pattern of visual field defects, with or without elevated IOP, in the presence of widely open angle of the anterior chamber, and in the absence of other causes of damage to the nerve fiber bundles. [2, 3]

In many, but not all, cases, the IOP is elevated above the normal range (more than 2 standard deviations from the mean, or 21mm Hg), reflecting a reduced aqueous out-flow facility. [3]

Although elevated IOP is a clear risk factor, glaucomatous optic neuropathy can develop at virtually any level of IOP. [4] The existence of normal tension glaucoma (NTG) and the weak correlation between progression of the disease and the level of the IOP indicate that other factors might also be involved in the pathogenesis of glaucomatous damage. [5] Such factors may include ischemia of the ONH or nerve fiber layer, direct mechanical compression of the axons at the level of the lamina cribrosa, local toxicity, or some combination of these[3].

All glaucomatous optic neuropathies share the following features : [6]

1) Progressive death of the retinal ganglion cells manifested as,

2) A characteristic histo-pathologic alteration of the optic nerve, known as cupping which is functionally apparent as,

3) Sequential visual field deterioration in characteristic patterns.

Pathogenesis:

The most prevalent theories attempting to explain glaucomatous optic neuropathy (GON) are the mechanical theory and the vascular theory. The relative merits of either have been debated for more than 100 years.[2]

The mechanical theory states that elevated IOP causes direct compression on the axons of the retinal ganglion cells at the level of the lamina cribrosa. Alternately the raised IOP may attenuate the microcirculation to the ONH.[2]

On the other hand, the vascular theory suggests that eyes with inherently poor vascular supply to the ONH are more predisposed to damage by elevated or normal IOP.[2] It emphasizes the presence of a primary problem in the blood flow of the ONH as a result of a localized organic change[7] with or without a low perfusion pressure or vasospasm.[8] This vascular insult results in tissue death at the ONH and characteristic visual field loss. However, the cause-and-effect relationship between nerve damage and vascularity has not been established. As

neither theory could explain all cases of glaucoma, they are usually combined together.[2]

Another theory was proposed in the late 1960s and early 1970s that suggested that GON is due to interruption of the axoplasmic flow at the level of the lamina cribrosa with the resultant interruption of trophic factors to the ganglion cell bodies.[9,10] This causes the cells to initiate a suicidal response resulting in programmed cell death or apoptosis.[11]

The vascular theory (a historical review):

In 1892, Wagemann and Selzmann suggested the potential role of vascular factors in glaucoma. Convinced that ocular hypertension was not the sole contributing factor in glaucoma, Elschnig expanded on the vascular theory. After numerous examinations he observed that glaucomatous discs showed reduced capillary beds. He concluded that reduced nerve head perfusion resulted from the high IOP.[12]

The belief that vascular defects resulted from IOP insult remained for many years. Through the 1930s and 1940s various authors demonstrated that increased IOP could reduce retinal vascular pressure and visual fields.[13,14]

In 1940, Duke and Elder found evidence of sclerosis within the retro-bulbar vessels of glaucomatous eyes. This vascular defect had produced ischemia resulting in softening of the optic nerve tissue. This finding demonstrated the potential role of blood flow defects preceding and contributing to the type of neural damage observed in glaucoma.[8]