Ocular Manifestations Of Systemic Hypertension

An essay submitted
For partial fulfillment of
Master Degree of Ophthalmology
Ain Shams University

By

Mariam Talaat Ayad
M.B.B.CH
Ain Shams University

6/681
617.7
M.T.

Supervisors

Prof.Dr/ Ahmed Abou El Naga
Professor of Ophthalmology
Faculty of Medicine
Ain Shams University

Dr./Ahmed Darwish
Assistant Professor of Ophthalmology
Faculty of Medicine
Ain Shams University

A. Darwish

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أ.د. صباح عبد العظيم سعود
أ.م.د. عبد الستار سالم
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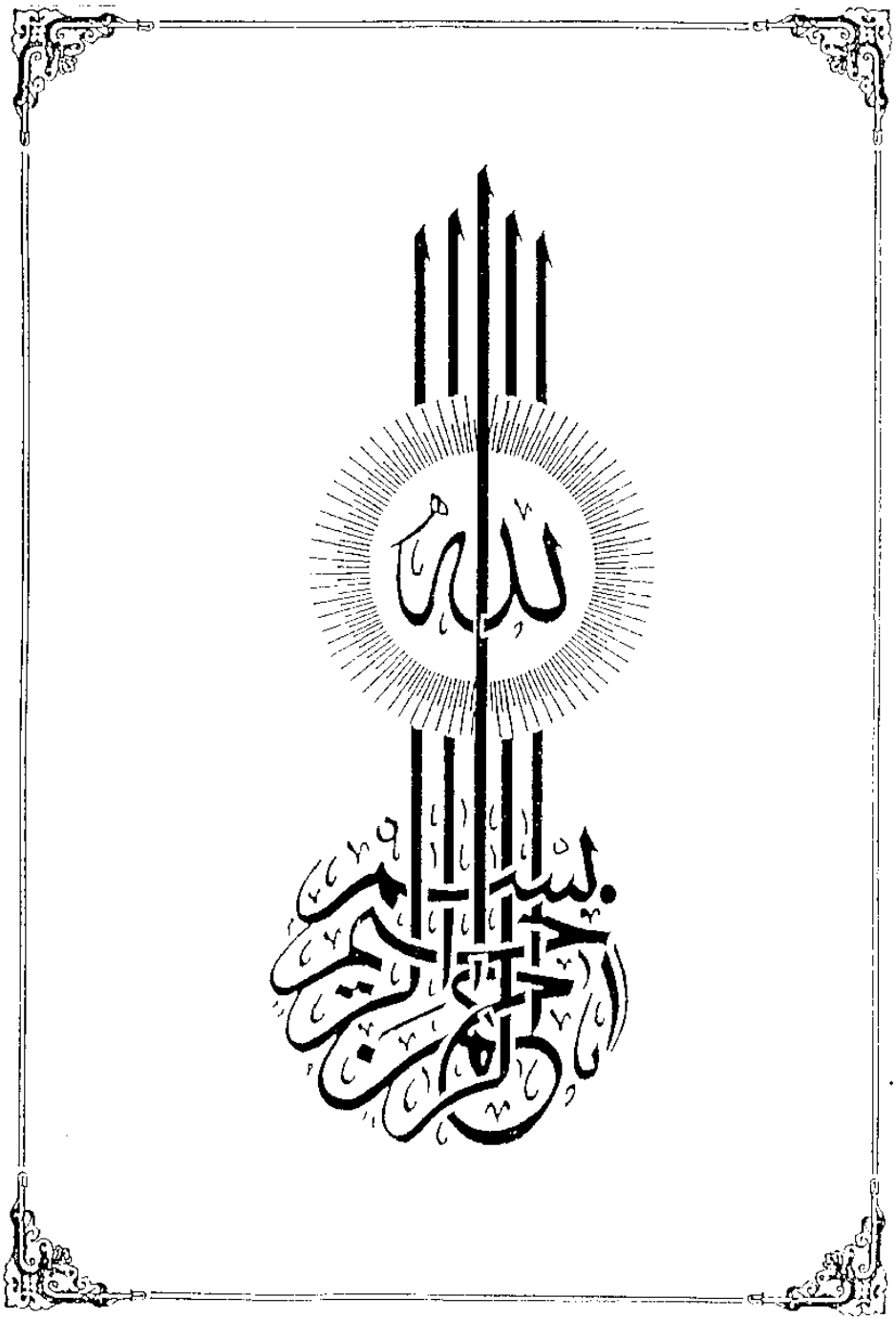

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Acknowledgment

I am extremely grateful to prof. Dr. Ahmed Abou El Naga and Dr. Ahmed Darwish for their continuous help & support throughout this work & for taking the time & care to review it & give many helpful suggestions.

I would like to thank them since without their Knowledge and guidance this work could not have been written..

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List of abreviations

ACTH	:	Adrenocorticotrophin hormone
AION	:	Anterior ischemic optic neuropathy
ARMD	:	Age-related macular degeneration.
A - V	:	Arterio - Venous
BP	:	Blood pressure
BRB	:	Blood retinal barrier
BRVO	:	Branch retinal vein occlusion
CF	:	Counting fingers
CNS	:	Central nervous system
CRAO	:	Central retinal artery occlusion
CRF	:	Corticotropin- Releasing factor.
CRVO	:	Central retinal vein occlusion
CSF	:	Cerebro spinal fluid.
DIC	:	Dessiminated intravascular coagulopathy
DM	:	Diabetes mellitus
ECG	:	Electro cardiogram
Echo	:	Echocardiography
ERG	:	Electro retinogram
FIPT'S	:	Focal intra retinal periarteriolar transudates
GCA	:	Giant cell arteritis
HDL	:	High density lipoproteins
HELLP	:	Hemolysis, elevated liver enzymes, low platelet count
ICGV	:	Indocyanine green videoangiographic
IDL	:	Intermediate density lipoproteins
IOP	:	Intra ocular pressure
IPCV	:	Idiopathic polypoidal choroidal vasculopathy
IRISs	:	Inner retinal ischemic spots
IRMA	:	Intraretinal microvascular abnot malities
LDL	:	Low density lipoproteins
LVH	:	Left ventricle hypertrophy
NVD	:	Neovessels at the disc
NVE	:	Neovessels elsewhere

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Malignant hypertension usually refers to a blood pressure level greater than 200/140 mmHg. Accelerated hypertension refers to a significant elevated blood pressure rise over previous levels. (Beilin and Puddey, 1992)

Primary hypertension (essential hypertension)

From 90-95 % of systemic hypertension is classified as essential hypertension, the cause of which remains unknown. The condition occurs in 10-15 % of white adults and 20-30 % of black adults in the USA.

The onset of essential hypertension is usually between ages 25 and 55. Hypertension is uncommon before age 20. In young people it is commonly caused by renal insufficiency, renal artery stenosis, or coarctation of the aorta. (Cutler, et al., 1991)

The pathogenesis of essential hypertension is multi-factorial. Genetic factors play an important role. Children with one-and even more so with two- hypertensive parents tend to have higher blood pressure. Abnormal cation exchange in RBCs has been suggested to be a potential marker of a genetic defect.Environmental factors also appear to play an important role; a combination of too much salt plus a genetic predisposition is required. Chloride may be as important as sodium in the pathogenesis of hypertension. Other factors that may be involved in the pathogenesis of essential hypertension are the following (Beilin and Puddey, 1992):

1- Sympathetic Nervous system hyperactivity:

It is most apparent in younger hypertensives, who may exhibit tachycardia and an elevated cardiac output. Insensitivity of the baroreflexes may play a role in the genesis of adrenergic hyperactivity. Sympathetic activity

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may also play a role in "labile" hypertension, characterized by marked blood pressure fluctuations under differing, or even similar conditions.

2- Renin-Angiotensin system:

Renin is secreted by the Juxta glomerular cells surrounding afferent arterioles in response to a number of stimuli, including decreased renal perfusion pressure, decreased intravascular volume, circulating catecholamines, increased sympathetic nervous system activity, increased arteriolar stretch, and hypokalemia.

$$\text{Angiotensinogen} \xrightarrow{\text{renin}} \text{angiotensin I}$$

$$\text{Angiotensin I} \xrightarrow[\text{enzyme}]{\text{angiotensin-converting}} \text{angiotensin II}$$

Angiotensin II is a potent vaso constrictor and major stimulant of aldosterone release from adrenal glands.

Black hypertensives tend to have lower plasma renin activity, hence higher intravascular volumes. Plasma renin activity levels can be classified in relation to dietary sodium intake or urinary sodium excretion. Approximately 10 % of essential hypertension patients have relatively high levels, 60 % have normal levels, and 30 % have relatively low levels.

3- Defect in natriuresis:

Normal individuals increase their normal sodium excretion in response to increased arterial pressure and to a sodium or volume load. Hypertensive patients, particularly when their blood pressure is normal, exhibit a decreased ability to excrete a sodium load. However, during chronic hypertension, a sodium load is usually handled normally.

4- Intracellular Na^+ and Ca^{2+}:

There is growing evidence that intracellular Na^+ is elevated in blood cells and other tissues in essential

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hypercholesterolemia, and a higher level of systolic pressure (Medical Research Council Trial of treatment of mild hypertension, 1985)

Pathophysiology:

Blood pressure depends on the degree of blood flow or cardiac output and the vascular peripheral resistance to the blood flow (Schwartz, 1992; and the 1988 report of the JNCDV).

An increase in either parameter can lead to the development of hypertension. When a defect to excrete excess sodium exists, and the intake of dietary sodium is high, the total sodium level in the body becomes elevated. Chronic sodium retention promotes increased intracellular sodium levels, which in turn leads to increased intracellular calcium. This causes vascular contraction and increased arteriolar tone, with the result of increased vascular resistance and cardiac output. Subsequently, the blood pressure becomes elevated (Williams and Braunwald, 1987; Lund- Johansen, 1989·
)

Vascular contraction, and the increase in peripheral resistance, may also be initiated by a pressor mechanism. The two pressor systems thought to primarily contribute are the renin- angiotensin – aldosterone system and the sympathetic nervous system (Schwartz, 1992; and Kaplan, 1992). Angiotensin II is a potent vasoconstrictor and stimulates the release of aldosterone, which causes impaired sodium excretion from the kidneys.

Stimulation of the sympathetic nervous system releases epinephrine and norepinephrine, causing vasoconstriction, increased cardiac output, release of renin, and impairment of renal sodium excretion. Epinephrine; norepinephrine, and angiotensin II also promote vascular wall hypertrophy.

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