CAROTID ARTERY DISEASE IN PATIENTS WITH PERIPHERAL ARTERIAL DISEASE

Thesis

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بسم الله الرحمن الرحيم
وَقُــل رَّبِّ زِدْنِــي عِلْمـًا
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LIST OF ABBREVIATIONS

Abbrev.	Meaning
ABI	: Ankle/brachial pressure index
ACA	: Anterior cerebral artery
ACAS	: Asymptomatic carotid atherosclerotic study
AKA	: Above knee amputation
BKA	: Below knee amputation
CABG	: Coronary artery bypass graft
CAD	: Coronary artery disease
CAS	: Carotid artery stenting
CCA	: Common carotid artery
CCU	: Coronary care unit
CEA	: Carotid endarterectomy
CT	: Computed tomography
CVD	: Cerebrovascular disease
ECST	: European carotid surgery trial
EDV	: End diastolic velocity
GSM	: Grey scale median
HDL	: High density lipoprotein
IC	: Intermittent claudication
ICA	: Internal carotid arterty
ICAS	: Internal carotid artery stenosis
IDL	: Intermediate density lipoprotein
LDL	: Low density lipoprotein
MCA	: Middle cerebral artery
MRI	: Magnetic resonance imaging
NASCET	: North American symptomatic carotid endarterectomy trial
NO	: Nitric oxide
PAD	: Peripheral arterial disease
PDGF	: Platelet derived growth factor
PSV	: Peak systolic velocity
PTCA	: Percutaneous coronary angiography
SMC	: Smooth muscle cell
SWMA	: Segmental wall motion abnormality
TCD	: Transcranial Doppler
TIA	: Transient ischemic attack
VLDL	: Very low density lipoprotein

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INTRODUCTION

Atherosclerosis is a systemic and generalized disorder of the arterial tree, which results in localized plaque deposition at selected sites within the vascular bed **(Libby, ٢٠٠٢).**

Epidemiological studies have revealed a number of risk factors that predispose to pathogenesis of atherosclerosis, although the final expression of the disease appears to be governed by the interaction of a number of genetic and environmental factors which may exert a different effect on the arterial tree in question. The pathophysiological basis of atherosclerosis is now well accepted to involve a close interplay between endothelial dysfunction and inflammation, which in turn modify the vascular response to oxidative stress, and platelet-endothelial interaction. The evolution of atherosclerotic plaques is associated with important anatomic and hemodynamic adaptive responses, and it is when these compensatory mechanisms fail that complications of atherosclerosis such as stenosis, plaque ulceration, embolization and thrombosis appear **(Libby et al., ٢٠٠٢).**

Stroke is the third leading cause of death in the United States each year. It is the second leading cause of cardiovascular death and the most common cause of death as a result of neurologic disorders. The incidence of new stroke is approximately ١٦٠ per ١٠٠,٠٠٠ population per

year. In addition to death, the disability following cerebral infarction must be considered from the standpoint of the crippling effect on the patient as well as the socioeconomic burden on the patient, his or her family, and society. Reviews of the financial impact of stroke for calendar year ١٩٩٩ were estimated to be $٤٥.٣ billion of direct and indirect cost (**Wesley, ٢٠٠٥).**

The carotid bifurcation plaque, responsible for approximately ٧٠% of all ischemic strokes, is found in various stages of evolution in both symptomatic and asymptomatic patients. In its least complex form it is composed almost entirely of fibromuscular intimal thickening, grossly pearly white with a smooth lumenal surface made up of flattened cells, probably transformed smooth muscle cells, forming a nonthrombogenic surface. Its characteristic location, best observed in early and intermediate stages of development, is at the lateral surface of the carotid bulb and origin of the internal carotid artery, where it may produce degrees of stenosis varying from barely perceptible to almost total occlusion. At this fibrous stage of development, symptoms occur probably entirely as the result of flow restriction, which, by causing marked stasis of blood, may finally end in thrombosis of the vessel. Referred to as "hard" plaque, it is sonaropaque and appears to pose lesser risks for symptoms to occur than do "soft," sonar-lucent plaques, until occlusion occurs **(Fisher and Ojemann, ١٩٨٦).**

The correlation between peripheral arterial disease (PAD) and cerebrovascular disease (CVD) is not as strong, as compared to coronary artery disease (CAD). Amongst patients with intermittent claudication, ٢٦% have carotid artery disease, by duplex Doppler. Aronow and Ahn found that ٣٣% of patients with CVD also had PAD **(Aronow and Ahn, ١٩٩٤).**