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قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها

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لم ترد بالأصل

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Effect of Atherosclerotic risk factors and its modification on endothelial function in patients with coronary artery disease. A study using Brachial artery Duplex scanning.

Thesis

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بسم الله الرحمن الرحيم

قالوا سبحانك لا علم لنا إلا ما علمتنا
إنك أنت العليم الحكيم

صدق الله العظيم

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NOS	:	Nitric oxide synthase.
PAI-I	:	Plasminogen activator inhibitor I.
RF	:	Resting flow.
RHD	:	Reactive hyperemia diameter.
SD	:	Standard deviation.
SE	:	Standard error.
TC	:	Total cholesterol
TG	:	Triglycerides.
VCAM	:	Vascular cell adhesion molecule.
VPF	:	Vascular permeability. Factor.
VSMC	:	Vascular smooth Muscle cell.
NS	:	Not significant.
PDGF	:	Platelet derived growth factor.
RH	:	Reactive hyperemia.
SBP	:	Systolic blood pressure.
SOD	:	Super oxide Dismutase.
TGFβ	:	Transforming growth factor β.
VLDL	:	Very low density lipoprotein
YR	:	Year.

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INTRODUCTION

INTRODUCTION

The disease process of atherosclerosis begins in childhood. The first histological signs are lipid deposition in the intema of systemic arteries (1).

Many of risk factors operate from early life and may cause vascular damage long before disease is clinically evident. However, the presence of risk factors is correlated only imperfectly with development of vascular disease (2).

If the effect of risk factors on the arterial wall could be studied in the long preclinical phase of atherosclerosis, there would be an opportunity to assess interventions in high risk patients in the hope of altering the natural history (3).

Endothelial dysfunction is an early event in atherogenesis (4). Studies in vivo have shown that the endothelium is abnormal in the earliest stages, before clinical detection of the disease, and that endothelial injury predisposes to thrombosis, leucocyte adhesion and proliferation of smooth muscle cells in the arterial wall (5). An important functional consequence of endothelial dysfunction is the inability to release endothelium derived relaxing factor (EDRF) (6). Cigarette smoking, hypertension, diabetes mellitus, and hypercholesterolemia are major risk factors in coronary artery disease (7). They are involved in the development of endothelial dysfunction (due to impaired endothelial release of EDRF), atherosclerosis, thrombogenesis, vascular occlusion, and coronary constriction due to catecholamine release(8).

Celemajer et al (3) developed a non-invasive means of studying early changes in vascular physiology in systemic arteries. High resolution ultrasound is used to follow up changes in vessel diameter in response to increased flow and to nitroglycerine (NTG). In arteries lined by healthy endothelium, increased flow causes dilatation of the vessel (8), via release of EDRF. This mechanism fails in endothelial dysfunction (9,10). By contrast, NTG causes vasodilatation by direct action on the smooth muscle, its effect is therefore independent of the endothelium. This endothelium dependent dilatation has been shown to be abnormal in young, asymptomatic patients, such as children with familial hypercholesterolemia, or young smokers(11-13).

REVIEW

PHYSIOLOGY OF ENDOTHELIUM

Structure of the vessel wall:

Arterial wall is made of three layers: the innermost intema, the media and the outermost adventitia. The intema is composed of a single layer of endothelial cells embedded in an extracellular matrix. The media is separated from the intema by the internal elastic lamina and consists of smooth muscle cells, elastic lamina, bundles of collagen fibers and elastic fibrils, all embedded in an extracellular matrix.

The adventitia contains dense fibroelastic tissue, nutrient vessels and nerves.

The actual composition of each of these layers varies with the type of blood vessel. Large, conduit arteries are typically referred to as elastic arteries because of their high ratio of elastic laminae to smooth muscle cells.

Muscular arteries are generally smaller and have a prevalence of smooth muscle cells, while arterioles consist of only one or two layers of smooth muscle cells. The smallest vessels are the capillaries made up of a single layer of endothelial cells that are occasionally opposed to pericytes-smooth muscle like cells- that serve a contractile function. The venous system has a similar architecture to that of that arterial system, the main difference being the orientation of the smooth muscle cells within the wall.