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RISK FACTORS FOR ATHEROSCLEROSIS

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ABSTRACT

A number of hypothetical risk factors for atherosclerosis and cardiovascular disease have emerged in recent years. Numerous research have been undertaken worldwide looking at the involvement of the risk factors such as infection, inflammation in coronary arteries due to the association of the factors associated with cardiovascular disease. It has been demonstrated that prior interventions can regulate the risk factors to lower the incidence and progression of coronary artery disease. The involvement of traditional risk factors such as smoking, hypertension, obesity in the development of cardiovascular diseases, has been thoroughly established. Recently, it is also discovered that a number of novel risk factors have an impact on the onset of CAD. Finding novel risk factors for atherosclerosis and CAD has kept a lot of attention in recent years. Homocysteine is one of the most recent risk factors whose importance has been proven, however there are still research being conducted to support the significance of other novel risk factors in patients suffering from coronary artery syndrome.

Keywords: Atherosclerosis, homocysteine, CAD, lipoprotein, ACE gene polymorphism

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INTRODUCTION

The last few years has seen the emergence of a number of notional risk factors for atherosclerosis with cardiovascular disease. A number of non-traditional risk factors like smoking, hypertension, diabetes mellitus has been reported to influence the development of CAD. timely interventions can control these risk factors to reduce the incidence and progression of artery disease (CAD).

HOMOCYSTEINE

McCully (1969) was first to describe the presence of atherosclerotic disease in arteries of people suffering from homocysteinemia an inborn error of metabolism due to deficiency of CBS (cystathionine-B synthetase). [1] Elevations in plasma homocysteina are typically caused by genetic defects in cystathionin β-synthase N5 NIO MTHFER. Nutritional deficiency in the vitamin cofactors required for protein metabolism may promote hyperhomocysteinemia.

HOMOCYSTEINE METABOLISM

Homocysteine is an amino acid formed during the metabolism of methionine. It may partially explain accelerated atherosclerosis in end stage renal disease, hypothyroidism and pernicious anaemia. The role of vitamins like folate, cobalmin, pyridoxal phosphate has been subject of many studies. [2]

HAEMOSTATIC FACTOR AND CORONARY ARTERY DISEASE

Fibrinogen may be a marker for cardiovascular disease as it reflects an inflamed condition of the vascular wall which is associated with increased levels of other inflammatory cytokines including 11-6 and TNF-alfa. Elevated levels may contribute to cardiovascular risk by increasing blood viscosity.[3]

INFECTION AND CORONARY ARTERY DISEASE

CRP (c-reactive protein) is a sensitive but non-specific acute phase reactant. It selectively binds to LDL and VLDL from plasma and could participate in their atherogenic accumulation. Low-grade inflammation participates in the pathogenesis of atherosclerosis.

LIPOPROTEIN(a) AND CORONARY ARTERY DISEASE

Elevated plasma levels of LP(a) have been linked to the development of premature CAD. LP-(a) is like LDL particle but is attached to apo(a). The association is due to accumulation of apo(a) in the vascular wall and atherosclerotic plaques, and the potential inhibition of plasminogen and fibrinogen.

SMALL DENSE LDL

Low-density lipoprotein (LDL) particles have been implicated as a risk factor for cardiovascular disease. LDL particles are heterogenous in size, composition and physico chemical properties. The Quebec cardiovascular study was the first to study the association between these particles and CAD.[5]

APO-LIPOPROTEIN PHENOTYPE

APO-E and APO-C III are thought to play a role in the clearance of lipoproteins including chylomicrons and VLDL-remnants, IDL, LDL and HDL by receptor pathways. The apo-E appears in 3 major iso-forms E2, E3, E4 which are coded by Corresponding alleles E2, E3 & E4.

TRIGLYCERIDE & CORONARY ARTERY DISEASE

Triglyceride has not traditionally been considered as a major lipid risk factor for CAD. Castelli analyzing the findings of the Framingham study, found that triglyceride was a powerful predictor of CAD in women over 50.[6] Another potent evidence for the association of triglyceride and CAD was seen from Stockholm Ischemic Heart Disease Secondary Prevention Study.[6]

INSULIN LEVELS AND MARKERS OF INSULIN RESISTANCE

Insulin resistance and hyperinsulinemia are commonly observed in patients with hypertension and related cardiovascular diseases. Hypertriglyceridemia and low HDL levels frequently coexist and are referred as the insulin resistance syndrome. The study on the Quebec Cardiovascular study population clearly showed that increased intake of Plasma insulin significantly increased the risk of CAD. [7]

ACE GENE POLYMORPHISM

The ACE gene is characterized by a polymorphism based on the presence [Insertion (i)] or absence [Deletion (D)] within intron 16 of a 287 - base pair IU repeat sequence. It results in 3 genotypes [DD or II homozygote and ID heterozygote]. The DD genotype has been linked to an increased risk of MI

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and a study on the Caerphilly Heart study population showed that this confers a risk. [8]

LOW BIRTH WEIGHT: THE BARKER'S HYPOTHESIS

In 1989, Barker hypothesized that environmental influences that impair growth & development in early life may be risk factors for CAD [9]. People who are thin at birth with low placental weight have high death rates from cardiovascular disease (CAD) in later life, according to research published in the British Journal of Clinical Epidemiology and Biomarkers. [10]

CONCLUSION

Coronary artery disease is the number one killer disease in the modern world. A great deal of interest has been focused in discovering new risk factors for atherosclerosis & CAD. The role of some of the newer factors like homocysteine has been firmly established. It is important that therapeutic interventions against these risk factors may further cause a decline is coronary artery disease.

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