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[General]

Most coronary artery disease (CAD) is due to subintimal deposition of atheromas in the large and medium-sized arteries serving the heart. Risk factors and the pathogenesis of atherosclerotic lesions and CAD are discussed in Chs. 15 and 201. Less often, CAD is due to coronary spasm, which is usually idiopathic (with or without associated atheroma) or may be due to drugs such as cocaine. Rare causes include an embolus to the coronary artery, Kawasaki syndrome (see Ch. 265), and vasculitis (eg, in SLE).

The Merck Manual of Diagnosis and Therapy

Section 16. Cardiovascular Disorders

Chapter 202. Coronary Artery Disease

Topics

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Coronary atherosclerosis is characteristically insidious in onset, is often irregularly distributed in different vessels, and can abruptly interfere with blood flow to segments of the myocardium, most often due to rupture of an eccentric atheromatous plaque with consequent intraluminal thrombosis.

The major complications of CAD are angina pectoris, unstable angina, MI, and sudden cardiac death due to arrhythmias. In the USA, CAD is the leading cause of death in both sexes, accounting for about one third of deaths each year.

Although the precise pathogenesis of CAD is unclear, the risk factors are well known: high blood levels of low density lipoprotein cholesterol (LDL-C) and lipoprotein a, low blood levels of high density lipoprotein cholesterol (HDL-C) and serum vitamin E, and poor physical fitness. High blood levels of triglycerides and insulin reflecting insulin resistance may be risk factors, but the data are less clear. CAD risk is increased by tobacco use; diets high in fat and calories and low in phytochemicals (found in fruits and vegetables), fiber, and vitamin E and C or, at least in some persons, diets with relatively low levels of omega-3 polyunsaturated fatty acids (PUFAs); poor stress management; and inactivity. Several systemic diseases (eg, hypertension, diabetes, hypothyroidism) are also associated with increased CAD risk.

Recent studies have shown an association between CAD and a common variant of the platelet fibrinogen receptor (PIA²), found in 20% of Americans. The presence of this variant may be as strong a predictor of CAD as cigarette smoking and hypertension. Whether giving antiplatelet therapy to persons with this variant can prevent CAD remains to be established.

Homocysteine has recently been identified as a risk factor for coronary, peripheral, and

cerebral vascular disease. Patients with homocystinuria, a rare recessive disease, have plasma homocysteine levels 10 to 20 times above normal (hyperhomocysteinemia) and accelerated, premature vascular disease. Homocysteine has a direct toxic effect on endothelium and promotes thrombosis and oxidation of LDL. Normal values range from about 4 to 17 $\mu\text{mol/L}$. Modest elevations of total plasma homocysteine have multiple causes, including low levels of folic acid, vitamins B₆ and B₁₂, renal insufficiency, certain drugs, and genetically controlled variations in homocysteine metabolic enzymes. Patients with homocysteine values in the top 5% have a 3.4 greater risk of MI or cardiac death than those in the lower 90% after adjustment for other risk factors. Increased homocysteine levels are associated with increased risk regardless of etiology. Recent studies suggest a graded risk even in normal-range homocysteine; thus, reduction of normal plasma levels may be advantageous. The most simple and effective way to reduce plasma homocysteine is administration of folic acid 1 to 2 mg/day, which has essentially no side effects except in untreated vitamin B₁₂ deficiency. Many authorities recommend that patients with CAD be screened for plasma homocysteine levels and, unless the values are in the lower normal range, treatment be initiated with folic acid. (See also Hyperhomocysteinemia in Ch. 132.)

Patients with CAD undergoing atherectomy have biologic markers suggesting coronary artery localization of *Chlamydia* infection. The role of this and other putative infectious agents in the genesis of CAD is being investigated.