Myocardial Infarction

Etiology

Most heart attacks are caused by a clot that blocks one of the coronary arteries, which are the blood vessels that bring blood and oxygen to the heart muscle. The clot usually forms in a coronary artery that has been previously narrowed from changes related to atherosclerosis. (Atherosclerosis is a disease of the arteries caused by fatty materials being deposited in the vessel walls, which leads to narrowing of the vessel walls and blood flow impairment.) The arterial wall sometimes cracks, and this triggers the formation of a clot, also called a thrombus. A clot in the coronary artery interrupts the flow of oxygen and blood to the heart muscle, which leads to death of heart cells in the area where the clot exists. This damaged heart muscle will then lose its ability to contract, and the remaining heart muscle has to compensate for the weakened area. Sometimes, sudden overwhelming stress can also trigger a myocardial infarction.

Epidemiology

Heart attacks account for one out of every five deaths. It is a major cause of sudden death in adults. It is estimated that approximately one million patients visit the hospital each year with a heart attack. Risk factors for coronary artery disease and heart attack include: smoking, high blood pressure, consuming too much fat in the diet, poor blood cholesterol levels (high LDL and low HDL), and having diabetes. In addition, many of these factors are related to being overweight. Males are also more at risk; especially men aged 40-70 years. Heredity plays an important role in who is at risk for myocardial infarction. Women are harder to diagnose as having myocardial infarction because their symptoms are different from men. Women also are at higher risk for myocardial infarction after they go through menopause. Blacks have a higher incidence of high blood pressure, which can lead to heart attacks. American Indians are also at a higher risk.

Treatments

Anticoagulation Therapies: Anticoagulants decrease the clotting ability of the blood and therefore help to prevent harmful clots from forming in the blood vessels. These medicines are sometimes called blood thinners, although they do not actually thin the blood. They also will not dissolve clots that have already formed, but they may prevent the clots from becoming larger and causing more serious problems. They are often used as treatment for certain blood vessel, heart, and lung conditions.

Aspirin: If there is plaque buildup in a vessel, it can rupture. When this happens, substances within the blood vessel lining can form a blood clot, blocking blood flow through the artery to the heart muscle, and causing a heart attack. Aspirin improves blood flow by reducing the stickiness of the platelets—the cells that cause the blood to clot. Regular aspirin use helps prevent clots from forming as readily and helps to keep arteries open. Aspirin will not stop the progression of coronary artery disease, but I can help stoop blood clots from forming, lessening the risk of an additional heart attack.

Dicumarol: Dicumarol prevents the liver from producing the factors that thicken or clot the blood. It acts by inhibiting the hepatic synthesis of vitamin k-dependent coagulation factors (prothrombin and factors VII, IX, and X). The depression of three of the four vitamin K-dependent coagulation factors (factors II, VII, and X) results in decreased prothrombin levels and a decrease in the amount of thrombin generated and bound to fibrin. This reduces the thrombogenicity of clots.

Thrombolytic Therapy: Urokinase is used for the treatment of pulmonary embolisms. Urokinase is an enzyme (protein) produced by the kidney, and found in the urine. Urokinase acts on the fibrinolytic system. It converts plasminogen to the enzyme by cleaving the Arg/Val bond. Plasmin decreases fibrin clots as well as fibrinogen and some other plasma proteins. This helps eliminate blood clots or arterial blockages that cause myocardial infarction. The mode of action is Streptokinase is very similar. It also cleaves the Arg/Val bond in plasminogen to form the enzyme plasmin. Plasmin in turn decreases the fibrin matrix of the thrombus, thereby exerting its thrombolytic action.