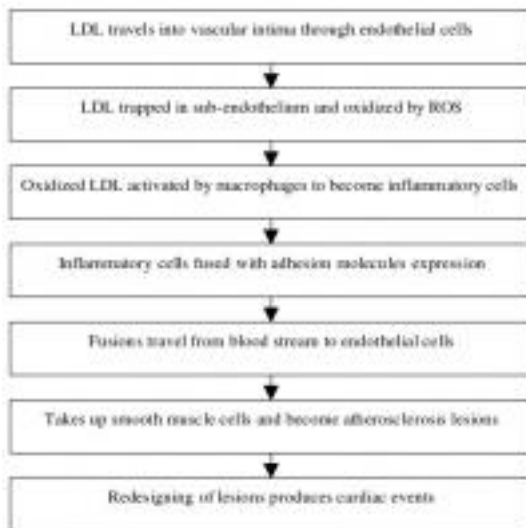




Atherosclerosis in Myocardial Infarction

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Fig. 1. Flowchart of the progression of atherosclerosis



MYOCARDIAL INFARCTION- DETECTION THROUGH CARDIAC ENZYMES Myocardial infarction (MI) is commonly known as heart attack. It is classified as ischemic heart disease, together with 3 other conditions. These clinical syndromes are also known as atherosclerosis stenosis or occlusion of coronary arteries because of the association with narrowing of coronary arteries. The first and the least severe of these conditions is angina pectoris which includes stable and unstable angina. Myocardial infarction ranks second while more extensive myocardial scarring and heart failure is the third condition better known as chronic ischemic heart disease. The last and most severe is sudden cardiac death. Atherosclerosis is the main cause of most of these ischemic heart diseases (98% of the time). Nevertheless, when a young person is just diagnosed with myocardial infarction, the causes are more likely among others to be infected cardiac valve or secondary effect from cocaine.

Atherosclerosis develops through a number of

mechanisms, some of them inter-related, and others though causes that are not yet clearly defined. One of the main mechanisms involves dietary low density lipoprotein (LDL) and the effects generated along its passageway.

LDL together with other macromolecules travels into the vascular intima through the endothelial cells. LDL gets trapped in the sub-endothelium, where it is vulnerable to be attacked by reactive oxygen species (ROS) (Wentworth et al 2003). These oxidized LDL is then taken up by macrophages which give it an active form ultimately leading to chronic inflammation (Alavi et al 2003). Inflammatory cells are attracted to adhesion molecules expression (VCAM-1, P- and E-selectin). These fusions enable them to travel from the blood stream and settle at the endothelial cells. Smooth muscle cells (SMC) are then taken up and stimulated to release collagen. Finally atherosclerosis lesion develops which does not continue to grow but undergoes rigorous redesigning. During these processes, intervals of activities that might produce cardiac events occur (Scott 2004).

MI happens when the demand of the heart muscle exceeds the supply. This process happens when there is blockage in the coronary arteries. These blockages might be caused by clots that are formed. Previously, it is thought that only complete blockage causes MI but recently it was found even minor blockages causes MI. The passage for blood flow becomes narrow and the blood flow becomes slow. This closure of the artery causes lack of oxygen and nutrients that is transported to the involved heart muscle. This ultimately leads to irreversible death or necrosis of some heart muscle.