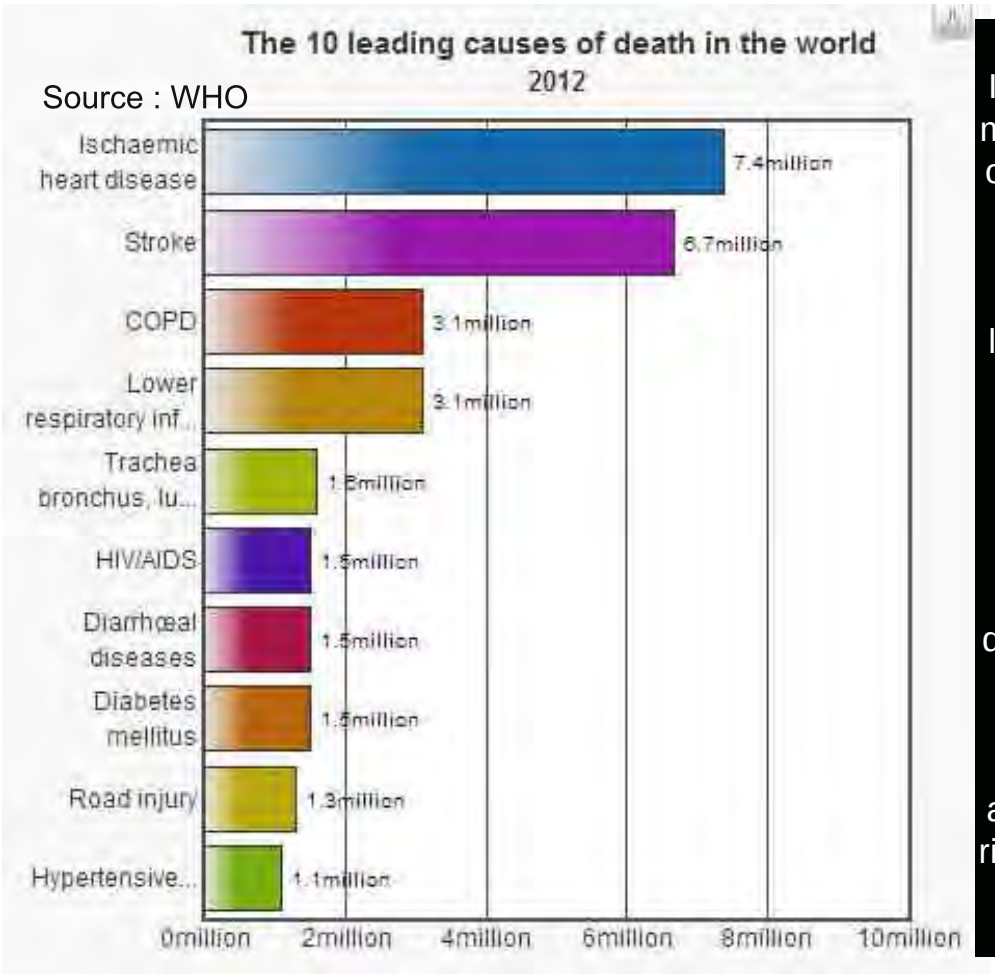
COVER ARTICLE

Deadliest Diseases of the world

ISCHAEMIC HEART DISEASE

Coronary artery disease (CAD) also known as atherosclerotic heart disease, coronary heart disease, or ischemic heart disease (IHD), is the most common type of heart disease and cause of heart attacks.



It was as of 2012 the most common cause of death in the world and a major cause of hospital admissions. There is limited evidence for population screening, but prevention (with a healthy diet and sometimes medication for diabetes, cholesterol and high blood pressure) is used both to prevent IHD and to decrease the risk of complications.

Ischemic heart disease (IHD) is the generic designation for a group of closely related syndromes resulting from myocardial ischemia—an imbalance between the supply (perfusion) and demand of the heart for oxygenated blood. Ischemia comprises not only insufficiency of oxygen, but also reduced availability of nutrient substrates and inadequate removal of metabolites

In more than 90% of cases, the cause of myocardial ischemia is reduction in coronary blood flow due to atherosclerotic coronary arterial obstruction. Thus, IHD is often termed coronary artery disease (CAD) or coronary heart disease. In most cases, there is a long period (decades) of silent, slowly progressive, coronary atherosclerosis before these disorders become manifest. Thus, the syndromes of IHD are only the late manifestations of coronary atherosclerosis.

History

The cardiologist tends to envisage the history of coronary heart disease in terms of anatomy, physiology, pathology and clinical features, whereas the historian approaches it chronologically as Dr. Leibowitz has done, but by passing backwards and forwards in time in the text, he has succeeded in presenting a pleasantly readable account of the growth of our knowledge of arterial disease, angina pectoris and cardiac infarction.

Until Harvey described the third and separate coronary circulation (1649), and recognized the nutritive function of the arteries as opposed to veins, no conception of coronary disease was possible, and until Heberden identified angina pectoris over a century later.

Dr. Leibowitz's painstaking search of the primary sources of medical knowledge, however speculative some of his conclusions must be, makes a valuable contribution to cardiological history. Dr. Leibowitz's review (1970) covers most of the familiar landmarks and adds some which are less familiar such as Vulpian's case of cardiac infarction and Nicholls's account of the ruptured heart of King George II whose role in the history of cardiac infarction is comparable with that of King Edward VII in the history of appendicitis.

Epidemiology

IHD in its various forms is the leading cause of death for both males and females in the world.

CVDs are the number one cause of death globally: more people die annually from CVDs than from any other cause .

An estimated 17.3 million people died from CVDs in 2008, representing 30% of all global deaths. Of these deaths, an estimated 7.3 million were due to coronary heart disease and 6.2 million were due to stroke. Low- and middle-income countries are disproportionally affected: over 80% of CVD deaths take place in low- and middle-income countries and occur almost equally in men and women.

The number of people who die from CVDs, mainly from heart disease and stroke, will increase to reach 23.3. million by 2030. CVDs are projected to remain the single leading cause of death.

Clinical manifestations

The clinical manifestations of IHD can be divided into four syndromes:

1.Myocardial infarction (MI), the most important form of IHD, in which the duration and severity of ischemia is sufficient to cause death of heart muscle.

2. Angina pectoris, in which the ischemia is less severe and does not cause death of cardiac muscle. Of the three variants—stable angina, Prinzmetal angina, and unstable angina—the latter is the most threatening as a frequent harbinger of MI.

3. Chronic IHD with heart failure.

4. Sudden cardiac death.

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acute myocardial infarction, unstable angina, and sudden cardiac death are sometimes referred to as acute coronary syndromes.

Risk factors

Coronary artery disease has a number of well determined risk factors. The most common risk factors include smoking, family history, hypertension, obesity, diabetes, high alcohol consumption, lack of exercise, stress, and hyperlipidemia. Smoking is associated with about 54% of cases and obesity 20%. Lack of exercise has been linked to 7–12% of cases.

Pathogenesis

The dominant influence in the causation of the IHD syndromes is diminished coronary perfusion relative to myocardial demand, owing largely to a complex and dynamic interaction among fixed atherosclerotic narrowing of the epicardial coronary arteries, intraluminal thrombosis overlying a disrupted atherosclerotic plaque, platelet aggregation, and vasospasm.

More than 90% of patients with IHD have atherosclerosis of one or more of the coronary arteries. The clinical manifestations of coronary atherosclerosis are generally due to progressive encroachment of the lumen leading to stenosis (chronic, "fixed" obstructions) or to acute plaque disruption with thrombosis (generally both sudden and dynamic), which compromises blood flow. A fixed obstructive lesion of 75% or greater (i.e., only 25% or less lumen remaining) generally causes symptomatic ischemia induced by exercise; with this degree of obstruction, the augmented coronary flow provided by compensatory vasodilation is no longer sufficient to meet even moderate increases in myocardial demand. A 90% stenosis can lead to inadequate coronary blood flow even at rest. Slowly developing occlusions may stimulate collateral vessels over time, which protect against distal myocardial ischemia and infarction even with an eventual high-grade stenosis.

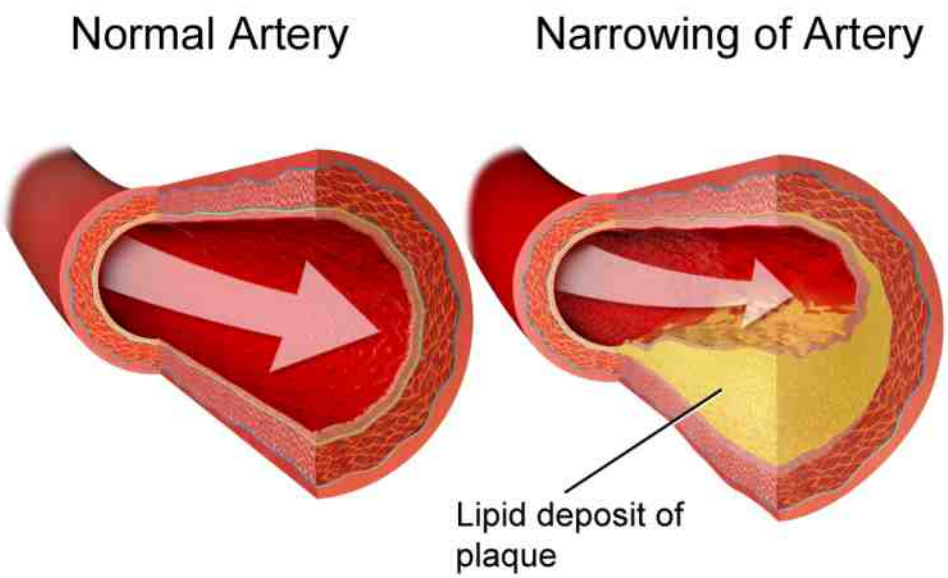
Although only a single major coronary epicardial trunk may be affected, two or all three—lateral anterior descending (LAD), left circumflex (LCX), and right coronary artery (RCA)—are often involved. Clinically significant stenosing plaques may be located anywhere within these vessels but tend to predominate within the first several centimeters of the LAD and LCX and along the entire length of the RCA. Sometimes the major secondary epicardial branches are also involved (i.e., diagonal branches of the LAD, obtuse marginal branches of the LCX, or posterior descending branch of the RCA), but atherosclerosis of the intramural branches is rare. However, as mentioned above, the onset of symptoms and prognosis of IHD depend not only on the extent and severity of fixed, chronic anatomic disease, but also critically on dynamic changes in coronary plaque morphology.

Diagnosis

MI is diagnosed classically by typical symptoms, biochemical evidence, and by the ECG pattern. In about 10% to 15% of MI patients, the onset is entirely asymptomatic and the disease is discovered only later by ECG changes, usually consisting of new Q waves. Such "silent" MIs are particularly common in patients with diabetes mellitus and in elderly patients.

Laboratory evaluation is based on measuring the blood levels of intracellular macromolecules that leak out of fatally injured myocardial cells through damaged cell membranes; these molecules include myoglobin, cardiac troponins, creatine kinase (CK), lactate dehydrogenase, and many others. Although these markers have become increasingly sensitive indicators of myocardial damage, they do not reflect its mechanism.[65] From a biochemical perspective, the diagnosis of myocardial injury is established when blood levels of sensitive and specific biomarkers, such as cardiac troponin and the MB fraction of creatine kinase (CK-MB), are increased in the clinical setting of acute ischemia. The preferred biomarkers for myocardial damage are cardiac-specific proteins, particularly Troponin-I (TnI) and Troponin-T. Troponins are proteins that regulate calcium-mediated contraction of cardiac and skeletal muscle. These markers have nearly complete tissue specificity and high sensitivity. TnI and TnT are not normally detectable in the circulation, but after acute MI, levels of both cardiac troponins rise at 2 to 4 hours and peak at 48 hours. Troponin levels remain elevated for 7 to 10 days after the acute event. Formerly the "gold standard," cardiac creatine kinase (CK-MB) remains the best alternative to troponin measurement. Creatine kinase is an enzyme that is highly concentrated in brain, myocardium, and skeletal muscle and is composed of two dimers, designated "M" and "B.

source: Times of India



Coronary Artery Disease

" CK-MB activity begins to rise within 2 to 4 hours of onset of MI, peaks at about 24 hours, and returns to normal within approximately 72 hours. Although the diagnostic sensitivities of cardiac troponin and CK-MB measurements are similar in the early stages of MI, persistence of elevated troponin levels for approximately 10 days allows the diagnosis of acute MI long after CK-MB levels have returned to normal.

C-reactive protein (CRP) may serve as a marker to predict the risk of myocardial infarct in patients with angina, and the risk of new infarcts in patients who recover from infarcts.[49][50] Using highly sensitive methods, serum CRP, levels of more than 3 mg/L are associated with the highest risk of cardiovascular disease, while levels of 1 to 3 mg/L are associated with moderate risk.

Other diagnostic modalities such as echocardiography (for visualization of abnormalities of regional wall motion), radioisotope studies such as radionuclide angiography (for chamber configuration), perfusion scintigraphy (for regional perfusion), and magnetic resonance imaging (for structural characterization) sometimes provide additional anatomic, biochemical, and functional data.

Treatment

Although coronary heart disease (CHD) cannot be cured, treatment can help manage the symptoms and reduce the risk of further problems.

There are three main treatment options for coronary artery disease:

Medical treatment

Drugs (e.g. cholesterol lowering medications, beta-blockers, Nitrates, ACE (angiotensin-converting enzyme) inhibitors, Statins, Antiplatelets, nitroglycerin, calcium antagonists, etc.);

Coronary interventions as angioplasty and coronary stent;

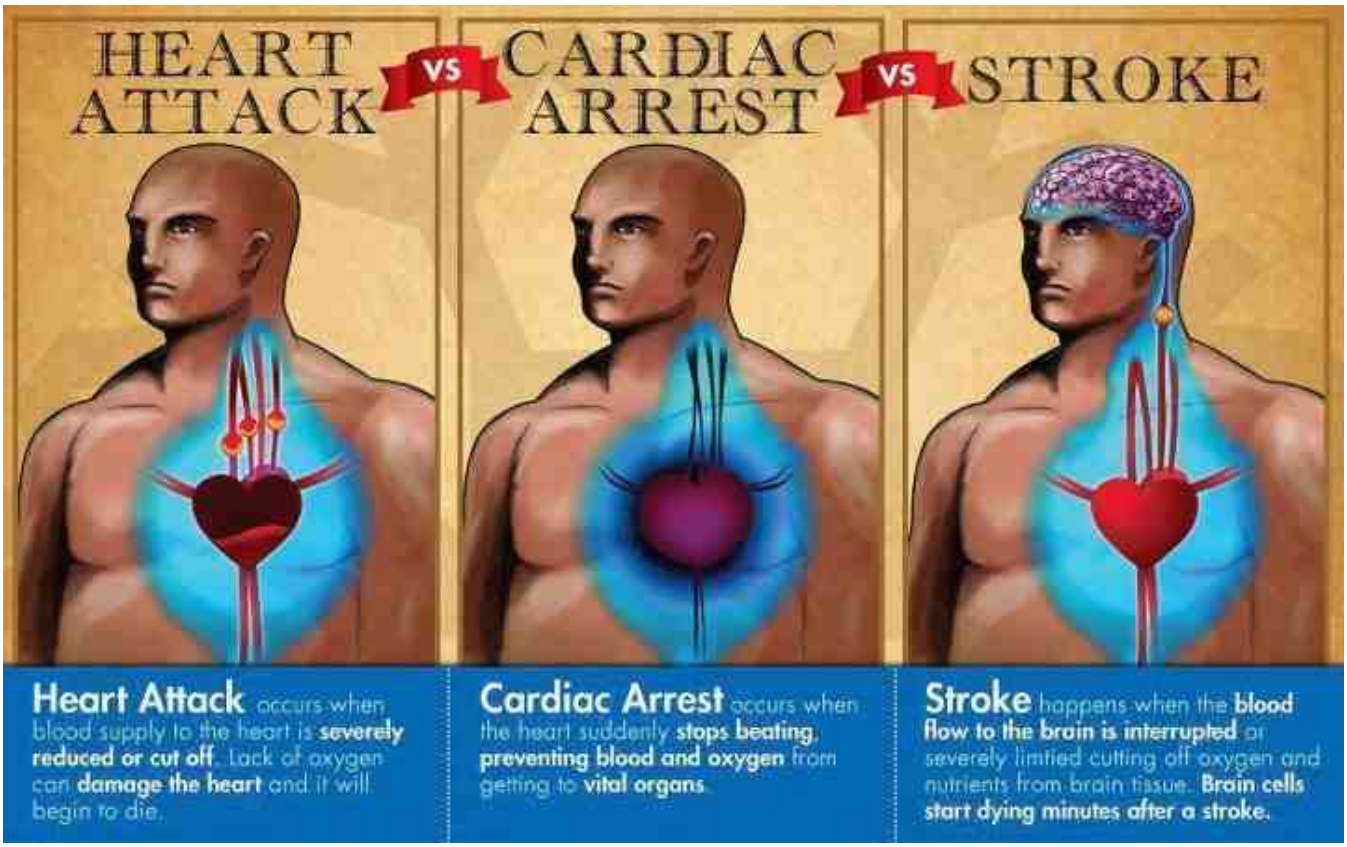
Coronary artery bypass grafting(CABG)

Angioplasty

Angioplasty is a nonsurgical procedure that opens blocked or narrowed coronary arteries. This procedure also is called percutaneous (per-ku-TA-ne-us) coronary intervention, or PCI. A thin, flexible tube with a balloon or other device on the end is threaded through a blood vessel to the narrowed or blocked coronary artery. Once in place, the balloon is inflated to compress the plaque against the wall of the artery. This restores blood flow through the artery. During the procedure, the doctor may put a small mesh tube called a stent in the artery. The stent helps prevent blockages in the artery in the months or years after angioplasty.

Coronary Artery Bypass Grafting

CABG is a type of surgery. In CABG, arteries or veins from other areas in your body are used to bypass (that is, go around) your narrowed coronary arteries. CABG can improve blood flow to your heart, relieve chest pain, and possibly prevent a heart attack.



Heart transplant

In a small number of cases, when the heart is severely damaged and medicine is not effective, or when the heart becomes unable to adequately pump blood around the body (heart failure), a heart transplant may be needed. A heart transplant involves replacing a heart that is damaged or is not working properly with a healthy donor heart.

Alternative therapies for the treatment of heart disease and heart failure, such as stem cell transplantation, are being investigated to complement current pharmacological therapies, primary angioplasty, and cardiac surgery. The procedure involves collecting stem cells from a patient's own blood or bone marrow and using them to repair damaged tissues in the patient's heart and arteries.

A team of Cochrane researchers, based in the UK and working with the Cochrane Heart Group, set out to assess the effectiveness and safety of stem cell therapies as a complement to standard treatments for heart disease and heart failure. The research team examined data involving 1,255 people from 23 randomised controlled trials, where all participants received standard treatments. Compared to standard treatment alone or with placebo, stem cell therapy using bone marrow cells resulted in fewer deaths due to heart disease and heart failure, reduced the likelihood of patients being readmitted to hospital, and improved heart function.

Dr Enca Martin-Rendon, lead author of the Cochrane Review and based at NHS Blood and Transplant and the University of Oxford, UK, said: “It is encouraging that stem cell therapy may have some benefits for heart disease patients. However, the evidence is of low quality at present because the studies are too small, with very low numbers of events overall, and the estimate of the effect is likely to change as further research becomes available. We also need to find out why the treatment seems to work for some patients but not for others.” ■

Dr. Seema P. Upadhye