

Cardiovascular disease to become leading cause of death worldwide by 2020

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Cardiovascular disease is projected to surpass infectious diseases to become the leading cause of death worldwide by 2020. The key to arresting this trend lies in preventing the development of thrombosis-prone plaques and identifying those patients who are at the highest risk of a second cardiovascular event, such as a heart attack or stroke.

While the mortality from atherosclerotic conditions, such as coronary heart disease (CHD) and stroke has declined by approximately 60% over the past 30 years, CHD remains the single largest killer of Americans. When considered separate from other cardiovascular diseases, heart disease and stroke still represent the first and third leading causes of death, respectively, among men and women in the US. In developed countries, cardiovascular disease (CVD) accounts for nearly 50% of all deaths, and within the next 15-20 years, cardiovascular disease is projected to surpass infectious diseases to become the leading cause of death worldwide.

Reducing an individual's risk is possible if they take aspirin (under a physician's guidance), eat a healthy diet, quit smoking and begin an exercise program. As obesity and diabetes also increase an individual's risk of atherosclerosis, these conditions need to be avoided or treated as well as reducing raised blood pressure and keeping cholesterol in check.

Coronary artery atherosclerosis is a progressive disease process that generally begins in childhood and manifests clinically in mid-to-late adulthood. It is a chronic, immunoinflammatory (stimulation of the immune system), fibroproliferative disease of large and medium-sized arteries fuelled by lipids, or fatty acids.

Atherosclerosis alone is rarely fatal; it is thrombosis (blood clotting), superimposed on a ruptured or eroded atherosclerotic plaque that precipitates the life-threatening clinical events such as acute coronary syndromes and stroke. Therefore, the vital question is not why atherosclerosis develops, but rather why none or only a few among plaques within a given person apparently pass through a thrombosis-prone and dangerous phase during a lifetime. The key is either preventing the development of thrombosis-prone plaques, or if they already exist, finding and treating those who harbor them and are consequently at high risk of a heart attack or stroke.

The importance of diagnosis

Biomarkers such as Lp-PLA2 and imaging technologies such as magnetic resonance imaging (MRI) are likely to be important for identifying vulnerable plaques (at risk of rupture), and thus identifying those patients who are at the highest risk of a second cardiovascular event.

Advances in diagnostic capabilities to identify vulnerable plaques have occurred in the areas of blood sampling, non-invasive imaging, and intracoronary diagnostic devices.

Rapid increases in accuracy of multislice computed tomography (MSCT) permit the detection of coronary calcification and imaging of coronary artery wall after only a peripheral injection of a contrast agent. In the future, both MRI and MSCT have the potential to provide a non-invasive method to detect vulnerable patients and vulnerable plaque.

For patients already undergoing cardiac catheterization for the treatment of coronary artery stenosis (narrowing or blocking of arteries), numerous intracoronary devices are being developed to provide improved plaque characterization from the excellent vantage point provided by the coronary artery lumen. The methods include intravascular MRI, modifications of intravascular ultrasound, near-infrared spectroscopy, nuclear methods, optical coherence tomography, palpography, and thermography.

Novel approaches are also possible for regional and local treatment of vulnerable plaques and vulnerable segments of arteries. Promising studies in animals indicate that inflammation can be safely eradicated in a region of an artery. New drug-eluting (drug-releasing) stents have reduced the rate of re-narrowing of the artery to such a level that stents may be considered for treatment of plaques that are not vulnerable to

abnormal narrowing, although the propensity of stents to cause potentially fatal thrombosis must be included in the risk-benefit analysis.

Advances in the understanding of vulnerable plaque, plus new methods that might enhance its diagnosis and treatment could make it possible to establish the goal of the complete eradication of subsequent coronary events in patients undergoing insertion of stents. The catheterization laboratory could become the place where stenoses are repaired, vulnerable plaques, vulnerable arteries and vulnerable patients are identified, and intensive preventive measures are initiated.

Unmet needs in the prevention of atherosclerosis

Plasma biomarkers are considered a ♦gadget♦ by some physicians who prefer to focus on atherosclerotic risk factors. Until they have been proven to improve therapeutic strategy over and above classical risk factors, they are likely to be too expensive and non-specific to be used routinely. The general consensus is that many plasma markers may find a niche use but not a general use. The difficulty is in identifying the niche.

All the emerging imaging techniques are believed to hold promise, but they also all have drawbacks at present, meaning further development is needed. In addition, access and expense are the main barriers to their wider use, particularly in Europe. Fortunately, advances are available that can provide improved risk stratification of patients. It is possible to envision a screening system based on three components; the standard risk factors, novel blood biomarkers, MSCT and nuclear methods.

Such a screening system is likely to identify some asymptomatic individuals at very high risk - a greater than 10% chance of a cardiac event in the ensuing year. But ultimately, raising public awareness of the risk factors for atherosclerosis is a key unmet need in primary prevention as well as improvements in medical therapies. In addition there is a need for studies showing that early identification of early atherosclerosis leads to improved outcomes. Only then is current treatment practice likely to change. 'End Intellixt