CONCLUSION

Atherothrombosis is defined as atherosclerotic plaque disruption with superimposed thrombosis, is the leading cause of mortality in the Western World. It is a diffuse disease affecting different arterial beds but with a similar aetiopathogenesis .Atherosclerosis, a chronic inflammatory disease, involves both innate and adaptive arms of immunity which modulate lesion initiation, progression, and potentially devastating thrombotic complications.

Coronary atherosclerosis and its complications are eminently preventable. The assessment of CHD risk factors and screening for asymptomatic coronary or carotid atherosclerosis are helpful in identifying individuals at risk of adverse CV events. Aggressive normalization of CV risk factors such as dyslipidemia, hypertension, sedentary lifestyle, and tobacco smoking is the most effective means of improving prognosis in the setting of chronic coronary artery disease.

In the field of clinical imaging, ongoing technologic advances will involve high-field MRI, improved coil technology, or dual X-ray source MDCT. These modalities will improve our ability to evaluate the arterial system in one examination, to quantify plaque burden, and to identify areas where targeted, high-resolution MRI, in combination with molecular contrast agents, can provide complete characterization of specific lesions. Molecular imaging will be increasingly employed to study in vivo atherogenesis, detect subclinical disease, or even target delivery of therapeutic agents. Developments in MRI contrast molecules, some of which have already been tested in vivo will widen the possibilities for this technique.So The use of noninvasive imaging technology makes it possible to detect atherothrombotic disease in a preclinical stage and, therefore, to implement adequate preventive interventions instead of use of invasive technique.

Atherothrombosis is a major global health problem and represents a growing burden on society. Therefore, adequate prevention and treatment strategies are essential. Antiplatelet therapy with agents such as aspirin and clopidogrel has been shown to greatly improve outcomes in patients with acute coronary syndromes, stroke, and peripheral arterial disease, especially when their synergistic effects are exploited. The GP IIb/IIIa antagonists are also beneficial when given early (intravenously), especially in patients undergoing PCI, but have proved unsuitable for long-term (oral) maintenance therapy. There is still much to learn, but as the elucidation of the coagulation and inflammatory processes mediated by platelets continues, it is hoped that the fine-tuning of our use of vital antiplatelet therapies to prevent and treat atherothrombotic disease will also progress with respect to the potency of platelet inhibition has also brought increased bleeding tendency. To shift the balance from causing excess bleeding toward preventing vascular occlusion, dose adjustments are necessary in low-weight patients for drugs such as prasugrel and ticagrelor. As age is another factor found to increase bleeding, elderly patients still await a registered drug to address their needs. In contrast to the studies on clopidogrel and prasugrel, ticagrelor, besides its stronger platelet inhibition, did not increase the risk of major bleeding; thus, it may become an antiplatelet option for older patients, although it may not be possible for those with respiratory problems or symptomatic bradicardia. Also, with regard to bleeding, the group of thrombin receptor inhibitors is particularly promising, as it is hypothesized that the pathway they block may play a role only in pathologic thrombosis, leaving physiologic hemostasis intact.

Statin(3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors) are currently regarded as the cornerstone of dyslipidaemia management in secondary prevention, based on their established efficacy in reducing LDL cholesterol in a series of major prospective clinical trials and meta-analyses. Raising HDL

concentrations is likely to become the next major therapeutic target with the PPAR (peroxisome proliferative activated receptor) (fibrates, thiazolidinediones and nicotin receptor agonists). cholesteryl ester transfer protein (CETP) (Torcetrapib ,dalcetrapib and anacetrapib) another potential strategy towards raising HDL-C.