REVIEW

Present-day Investigations Cannot Adequately Determine the Risk of Acute Coronary Syndromes

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SUMMARY

It is an important irony that present-day clinical stress testing methods including exercise electrocardiogram, stress echocardiography and even coronary angiography are not able to demonstrate vulnerable coronary plaques at risk of rupture. A vulnerable plaque may in fact be invisible on clinical stress test and perhaps only visualized directly through less available techniques such as coronary angiography. Landmark pathological studies have deepened our understanding of the mechanisms behind acute coronary syndromes over the last decade. Thrombosis plays a key role and is a unifying feature in the pathogenesis. Platelet-rich thrombus superimposed over the disrupted atherosclerotic plaque or eroded plaque endothelium, with or without fibrin-thrombus extension, is evident in post-mortem necropsy and angiographic studies. However features which contribute to the risk of acute events lie in the atherosclerotic plaque itself. Plaque content and not plaque size is the important factor. Clinical stress testing demonstrates plaque size but not plaque content. A plaque will be prone to rupture if it has only a thin cap and a proportionally larger lipid core. In such a plaque there is preponderance of activated macrophages and T-lymphocytes, and high activity of matrix metalloproteinases. Smooth muscle cell proliferation and collagen synthesis are downregulated. These features may serve as possible targets for devising clinical methods to detect plaques at risk or for reversing the risk in vulnerable plaques. (Clin. Lab. 2001;47:257-263)