HEART

Introduction

Advances in unstable angina: the role of low molecular weight heparins

The clinical syndromes of unstable angina and non-Q wave myocardial infarction (MI) have been the focus of much interest in recent years and, as a result, our understanding of the pathology and management of these conditions has increased dramatically. It is now clear that these conditions are more complicated than originally considered.

The sudden rupture of an atheromatous plaque, leading rapidly to thrombosis and subtotal arterial occlusion, has long been established as a cause of unstable angina. This explained how unstable angina might easily progress to full MI but was incompatible with subsequent findings that plaque rupture is not a necessary prerequisite for the development of instability. Furthermore, it failed to explain why the disease progresses to acute MI in some patients while settling spontaneously in others. It is now apparent that other mechanisms, particularly inflammation, have critical roles in the aetiology of these conditions. The wide variations that exist between patients with the same clinical syndrome are now recognised. The balance of the different aetiological factors in any individual may be critical in determining both the optimal clinical management and the eventual outcome.

Despite the recognition of other pathological mechanisms, thrombosis remains central to the understanding of acute coronary syndromes. The Veterans Administration study of 1983 was the first to show that treatment with oral aspirin reduces the subsequent rate of ischaemic events. Newer antiplatelet agents such as the glycoprotein IIb/IIIa receptor inhibitors have also demonstrated efficacy in a number of trials, both as an adjunct to revascularisation and as part of a conservative regimen.

A number of studies have also been performed to evaluate direct thrombin inhibitors such as hirudin. To date, no conclusive evidence has been presented showing an acceptable combination of safety and efficacy. However, results from the OASIS-2 (organization to assess strategies for ischaemic syndromes) trial are expected shortly and may provide more conclusive findings. (The results of the OASIS-2 trial have now been published and are discussed elsewhere in this supplement.)

Unfractionated heparin (UFH) is a well established part of the standard management of unstable angina and non-Q wave MI, even though the evidence supporting treatment with intravenous UFH is less compelling than that for aspirin. Recently, the results of trials of low molecular weight heparins (LMWHs) have indicated that these agents are not only easier to use than UFH, but that they may also be more effective. This improved efficacy can be shown to be cost effective despite the higher price of LMWHs. LMWHs may therefore provide a good alternative to UFH in the management of unstable angina and non-Q wave MI.

This supplement represents the proceedings of a symposium on unstable angina and its aetiology and management, presented at the XIIIth World Congress of Cardiology, Rio de Janeiro, Brazil on 27 April 1998.

E BRAUNWALD

Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts, USA

E GURFINKEL

Fundación Favaloro, Av Belgrano 1746, Buenos Aires, Argentina

Publication of this supplement is made possible by an unrestricted educational grant from Rhône-Poulenc Rorer