Coronary angioplasty for acute coronary syndromes

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Introduction

Acute coronary syndrome (ACS) is a term of relatively recent origin, encompassing all clinical conditions that are manifestations of acute myocardial ischaemia. The label of “unstable angina” was first applied in 1971, when it was recognized that patients with new or worsening angina were at increased risk for cardiovascular morbidity and mortality compared to those with more chronic symptoms. Further, it was obvious that there was a need to define diagnostic criteria for this group of patients in order to develop risk stratification and treatment strategies, particularly to identify those who would benefit from urgent surgical revascularization. A variety of terms have been associated with this clinical presentation, including preinfarction angina, crescendo angina, acute coronary insufficiency, and intermediate coronary syndrome. Among the large number of patients who receive these labels is a subgroup of patients at increased risk for acute myocardial infarction and cardiovascular death; careful evaluation is necessary to separate those who will benefit most from aggressive treatment, particularly coronary revascularization.

The strategy of early revascularization in patients with unstable angina developed from the larger experience with surgical coronary revascularization for stable angina in the 1970s, and from the relative limitations of the available medical therapy. By the mid-1980s, coronary artery bypass surgery was a primary therapy for patients with unstable angina, with low operative mortality, and excellent short- and long-term results. Shortly after Greuntzig’s report of “non-operative” revascularization for stable angina in 1979, angioplasty emerged as an alternative to bypass surgery, particularly in patients with refractory symptoms, proximal lesions, and single vessel disease.

Patients with ACS are quite diverse with regard to demographics, signs and symptoms, and the results of diagnostic testing at the time of their presentation. Once identified, however, the approach to treatment has been distilled to a fairly consistent set of principles. These principles have developed through careful observation, examination of the pathophysiology of this clinical entity and by large, prospective randomized trials. This chapter focuses specifically on the use of percutaneous revascularization in this patient group, a therapy which has now surpassed surgical coronary bypass as the mainstay of treatment.

Angioplasty in patients with ACS is generally considered “high risk”, as it is associated with more complex lesion morphology, higher rates of acute closure, periprocedural creatinine kinase (CK) release, and restenosis. In spite of these complicating factors, the widespread acceptance of percutaneous coronary intervention (PCI) in patients with ACS has resulted from three important factors:

1. early evidence of the efficacy of angioplasty for symptom control;
2. the progressive improvement in patient selection, angioplasty techniques, device technology, and adjunctive pharmacotherapy that has improved the safety and durability of PCI in this patient subgroup;
3. the large body of evidence that early, aggressive revascularization in patients who present with signs and symptoms of acute ischaemia is associated with improved clinical outcomes.
This last factor has led to an emphasis on risk stratification and identification of those patients who will receive the most benefit from early invasive evaluation. In this chapter, I identify several papers which illustrate each of these developmental factors.

Current American Heart Association/American College of Cardiology (AHA/ACC) guidelines reflect the rapid advancement in our understanding of the identification, risk stratification, and treatment of this diverse group of patients. At present we are able to control ischaemic symptoms effectively in almost all patients who present with ACS using a combination of medical therapy and percutaneous revascularization, reserving surgery for those with significant left main coronary obstruction or particularly diffuse three vessel disease. Furthermore, the phenomenal advancements in risk factor modification, in particular treatment of lipid disorders, begin during the index hospitalization and substantially alter the natural history of these patients in the months and years following discharge.

In spite of the dramatic advances in the interventional treatment of unstable angina, our understanding of the pathophysiology remains incomplete. Evidence for the role of inflammation in acute manifestations of coronary atherosclerosis is relatively new. Immune competent cells reside within both healthy and diseased arteries, and respond to influences as diverse as hyperlipidemia, exercise, and emotion. Therapies which target these cells and the cytokines that regulate their behaviour are being tested in a variety of “immune-mediated” diseases, and undoubtedly these therapies will be translated to patients with unstable angina. The use of stents as platforms for local delivery of antiproliferative and immunosuppressive drugs is arguably the largest technological advance in interventional cardiology. The integration of data regarding atherosclerotic inflammation into risk stratification and interventional treatment of patients with acute coronary ischaemia will facilitate the next major set of advances in this exciting field.