

Chapter 8

Acute Coronary Syndrome

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ABSTRACT

Acute coronary syndrome (ACS) is a common and sometimes lethal event, usually precipitated by sudden rupture and thrombosis of an atherosclerotic plaque. Patients presenting with ACS can be rapidly risk stratified based on signs, symptoms, electrocardiogram, and biomarkers. There is a new generation of potent and reliable antiplatelet drugs, which in concert with anticoagulation and rapid revascularization, can preserve myocardium and save lives. When choosing how to revascularize, hemodynamically stable patients with diabetes mellitus and complex coronary disease benefit more from coronary artery bypass grafting rather than percutaneous coronary intervention. Despite optimal treatment, ACS can result in deadly complications such as cardiogenic shock. Supportive care is paramount, but despite its widespread use, the utility of intraaortic balloon counterpulsation is uncertain. In the future, advanced coronary imaging may enhance preventative care, novel molecular targets will help expand treatment options, and cell-based regenerative therapies may aid myocardial recovery after acute coronary syndrome.

INTRODUCTION

This chapter aims to provide the reader with an in-depth, evidence-based understanding of the principles behind the current understanding and management of acute coronary syndrome. The mechanisms of plaque rupture and thrombosis are explored in some depth, as these are key to understanding principles of pharmacologic therapy. A discussion on risk stratification follows, as does the basis for choosing between treatment modalities. The most important decision is whether to pursue a conservative or early-invasive treatment strategy, and this directly follows from individualized risk stratification. An evidence-based review of pharmacologic agents follows, with a focus on several of the newer antiplatelet agents, which are beginning to replace clopidogrel as mainstays of therapy. This is followed by a discussion of revascularization strategies and the factors which would make a patient a better candidate for coronary artery bypass grafting (CABG) versus percutaneous coronary intervention (PCI). Post-ACS care is es-

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sential, as there are a number of potentially lethal complications which can be anticipated and managed to varying degrees. Finally, the discussion looks to the horizon of acute coronary syndrome care and potential future therapies.

Chapter Objectives

By the end of the chapter, the reader will be able to:

- Understand basic mechanisms of ACS at the molecular and cellular level
- Risk stratify patients with ACS and use this assessment to appropriately tailor therapy
- Understand the mechanisms and clinical evidence underlying the use of current antiplatelet, anti-coagulant, and adjunctive medications in treatment of ACS
- Individualize the optimal choice and timing of coronary revascularization
- Anticipate and treat complications of acute coronary syndrome
- Appreciate the frontiers and limitations of advanced coronary imaging, new therapeutic targets, and regenerative myocardial stem cell treatments

BACKGROUND

Epidemiology

Despite recent advances in prevention and therapy, acute coronary syndrome (ACS) – a broad term which includes unstable angina (UA), non-ST segment elevation myocardial infarction (NSTEMI), ST segment elevation myocardial infarction (STEMI) and sudden cardiac death due to myocardial infarction - remains a tremendous source of morbidity and mortality. Current estimates suggest that there will be 515,000 new acute myocardial infarctions (AMI) and 205,000 recurrent AMIs in 2014 in the United States, with significant associated mortality (American Heart Association, 2014). Coronary disease accounted for roughly 15% of all deaths in the United States in 2010, a third of which were caused by acute MI. This equates to one death every 83 seconds due to acute MI (Alexander, et al., 2011)

Overall death rates due to coronary disease declined 59% in the period from 1950 to 1999. This decline in mortality correlated with modest improvements in modifiable risk factors as well as advances in medical therapy. Development and use of evidence-based medical therapies such as aspirin, novel anti-platelet agents, beta blockers, and revascularization have helped decrease in-hospital mortality from AMI (McGovern, et al., 2001). Despite this progress, acute coronary syndrome represents a common and dangerous phenomenon, requiring rapid diagnosis and treatment.

Pathogenesis

With a few notable exceptions (see Table 3), acute coronary syndromes develop in the setting of existing atherosclerotic plaques which are typically not flow-limiting prior to rupture. In contrast to larger, more fibrotic and occlusive lesions, which tend to cause stable angina, these “softer,” non-occlusive plaques are more prone to sudden rupture and resultant thrombosis, leading to ACS (Libby, 2013).

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