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Chronic Stable Angina I: Risk Factors and Evaluation

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I. INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death in the United States and worldwide. In 1997, CVD was the cause of 41% of all deaths in the United States. Almost 60 million North Americans have CVD, and an estimated 6.3 million have angina pectoris. From 1987 to 1997, CVD death rates have declined, resulting in increased prevalence rates of heart disease as the population lives longer. Among ethnic minorities in the United States, the effect of CVD is similar to the general population trends: among African-Americans, CVD was the leading cause of death, accounting for 34% of male deaths and 41% of female deaths; among Hispanics, CVD accounted for 28% of male deaths and 34% of female deaths; among American Indian/Alaskan Natives, CVD accounted for 26% of male deaths and 29% of female deaths.¹

Approximately 350,000 new cases of angina occur each year. Prevalence rates are greater for women than men in each ethnic group. Although CVD mortality rates have been declining for both men and women, the rate of decline for women has been slower. Since 1984, more women than men have died yearly from CVD. This statistic is partly accounted for by the slower rate of decline in mortality, the longer life expectancy, and 10 year age-lag in onset of coronary artery disease (CAD) for women when compared with men.

This is the first of a 2-part review on angina. The second part will be published as “Chronic Stable Angina II:

Treatment and Case Studies” (Volume 7, Part 2, the *Hospital Physician Cardiology Board Review Manual*).

II. PATHOPHYSIOLOGY

GENERAL PRINCIPLES

Stable angina is most frequently caused by atherosclerotic obstruction of the epicardial coronary arteries, resulting in inadequate supply of oxygen and blood to an area of heart muscle at a time of increased demand. Less common causes of angina, not covered extensively in the scope of this review, include coronary vasospasm, aortic stenosis, idiopathic hypertrophic subaortic stenosis, hypertensive heart disease, coronary anomalies, and cardiomyopathy. Although atherosclerotic obstruction is the fundamental mechanism underlying exertional angina, great advances in vascular biology in recent years have enabled us to develop a more detailed understanding of the process of atherosclerosis and endothelial function. The focus of this review is stable angina; however, a grasp of the mechanisms resulting in plaque formation and plaque instability are essential to understanding treatment rationales that often focus on plaque stabilization and improved endothelial function.

Although a plaque causing a tight stenosis is more prone to cause complete obstruction and to result in unstable angina or myocardial infarction (MI), most MIs are caused by rupture of a nonobstructive plaque with less than 50% stenosis.² This is true because nonobstructive plaques are so abundant. Less obstructive