

# HOSPITAL PHYSICIAN®

## INTERNAL MEDICINE BOARD REVIEW MANUAL

### STATEMENT OF EDITORIAL PURPOSE

The *Hospital Physician Internal Medicine Board Review Manual* is a peer-reviewed study guide for residents and practicing physicians preparing for board examinations in internal medicine. Each manual reviews a topic essential to the current practice of internal medicine.

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## Primary Prevention of Coronary Disease; Management of UA/STEMI; Management of STEMI

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# Section 1—Primary Prevention of Coronary Artery Disease

Nehal N. Mehta, MD

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## CASE: INITIAL PRESENTATION

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A 42-year-old man presents to the emergency department with substernal chest pressure and nausea that awoke him from sleep at approximately 5 AM. He had returned home late from work the previous evening and had eaten a meatball sandwich before retiring to bed. The patient has no significant past medical history but has a 15 pack-year smoking history. His electrocardiogram and measurements of cardiac biomarkers are normal, and a stress test the following morning reveals good exercise tolerance with no evidence of ischemia. The patient expresses his concern about his risk of having a myocardial infarction (MI), noting that his brother had his first MI in his late 40s. He is eager to do whatever he can to reduce his personal risk for cardiovascular disease.

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## INTRODUCTION

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Coronary artery disease (CAD) and its associated complications of acute myocardial infarction (AMI) and congestive heart failure continue to be the leading cause of death in the developed world. In less than 20 years, it is projected that CAD will replace infectious diseases as the worldwide leading cause of death. Given the tremendous morbidity and mortality resulting from CAD, major focus has shifted from treatment of CAD to its prevention.

Primary prevention is defined as both the prevention of disease before it occurs and the reduction of its incidence. One of the most important advances in medicine over the past quarter century has been identification of major risk factors for development of CAD. These include elevated low-density lipoprotein cholesterol (LDL), elevated total cholesterol, reduced high-density lipoprotein cholesterol (HDL), hypertension, diabetes, cigarette smoking, family history, and age (Table 1).<sup>1</sup> In 2001, the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) published a report summarizing these “traditional” risk factors and treatment guidelines for them.

Traditional risk factors, such as elevated LDL, high total cholesterol, and reduced HDL, help to identify high-risk groups for CAD. They do not account for all cases of atherosclerosis. Emerging evidence indicates that in addition to traditional risk factors, metabolic abnormalities, defined as insulin resistance/metabolic syndrome, and an inflammatory immune response play pivotal roles<sup>2,3</sup> in the pathogenesis of atherosclerotic cardiovascular disease (ASCVD). Since ASCVD has been considered a disease of inflammation, new markers such as C-reactive protein (CRP) and fibrinogen have been proposed as predictors of atherosclerosis and its complications among the general population.<sup>4,5</sup> In addition, hyperhomocysteinemia has emerged as a risk factor for both CAD and peripheral vascular disease. With better understanding of the link between inflammation and CAD, high-risk groups for development of premature CAD are emerging, including HIV-positive patients treated with highly active antiretroviral therapy and patients with insulin resistance with or without the metabolic syndrome.

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## ESTABLISHED RISK FACTORS FOR CAD

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The NCEP has identified lipid factors that contribute to overall CAD risk and have shown that, if untreated, these significantly contribute to morbidity and mortality associated with CAD. These lipid factors include an elevated LDL and a reduced HDL. Because the majority of circulating cholesterol is LDL, high total cholesterol has also been linked to increasing CAD. LDL elevations alone, as demonstrated by genetic disorders involving defective LDL receptor in familial hypercholesterolemia, accelerate atherosclerosis and increase risk for AMI in the absence of other CAD risk factors.

Abundant data show that lowering of LDL reduces CAD; therefore, LDL is used as a primary marker for those at increased risk of CAD-related events.<sup>6</sup> The ATP III guidelines identify LDL as the primary target of cholesterol-lowering therapy, with LDL goal based on category of risk (Table 2). Of note, diabetes mellitus and