



COMMENTARY

Sudden Cardiac Death in Adult Patients with Stable Ischemic Heart Disease

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Do Stable Ischemic Heart Disease Patients Die Suddenly, yet Remain Stable?

There are close to one half million patients with stable ischemic heart disease (SIHD) in the United States, so this is not a trivial issue. What is written below is my own opinion about why sudden cardiac death occurs in patients with stable ischemic heart disease. In my view, it seems highly likely that patients with stable ischemic heart disease who die suddenly do not remain stable, and this may be due to rupture of plaques resulting in an acute coronary syndrome, i.e., unstable angina or myocardial infarction. Thus, stable ischemic heart disease itself probably is not a cause for sudden cardiac death, but it may be that the patients who develop an acute coronary syndrome (myocardial infarction or unstable angina) are the ones who have severe ischemia or arrhythmia as the cause of their sudden cardiac death. Sudden death in patients with stable ischemic heart disease is not a common occurrence and is sparsely reported.

Reported SCD in SIHD Patients

A study by Benchimol et al. reported 319 consecutive stable angina patients without clinical heart failure or a recent myocardial infarction [1]. There were 25 sudden deaths during a follow-up period of 97 ± 29 months (7.8%). Risk factors for these

patients include peripheral vascular disease, high LDL, left ventricular hypertrophy and low left ventricular ejection fraction. Most of the risk factors for sudden cardiac death were time-dependent with the exception of low ejection fraction. All of these patients had proven coronary artery disease, but the coronary arteries were not described in detail using the Syntax Scoring System. I was not able to determine the angiographic characteristics of those patients who died suddenly, nor was I able to find autopsy data on these patients.

Hjemdahl et al., in a registry-based follow-up, (Angina prognosis study in Stockholm-APISIS) reported 809 patients with stable angina pectoris [2]. Patients who were excluded from this registry were those who had a myocardial infarction within the past three years, an anticipated need for revascularization within one month, significant valve disease, or severe congestive heart failure, and/or other severe diseases, or poor compliance. In the APISIS study, signs of ischemia or previous manifestations of coronary artery disease, i.e., myocardial infarction or revascularization, were found in 69% of both male and female patients at baseline. Angiography was not done in all patients, and I could not detect any evidence that autopsies were performed on patients who died.

A total of 123 (15.2%) patients died and 72 did not die but had a non-fatal myocardial infarction. The mortality was higher than of the reference population.

Why the patients who had a myocardial infarction died is not explained, i.e., the occurrence of arrhythmias, heart failure, recurrent myocardial infarction, etc. were not noted. The authors of this article concluded that patients with stable ischemic heart disease had a favorable prognosis, but they identified patients with multi-vessel disease, signs of heart

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failure, and age of more than 60 years as patients exhibiting increased risk of mortality.

Based on what I can gather from these two studies and personal experience, I must conclude that stable angina patients are, by definition, stable and do not fall into the category of high risk unless they evolve an acute coronary syndrome or develop a serious arrhythmia.

Arrhythmias and SCD in SIHD patients

The arrhythmias that occur in patients with stable ischemic heart disease may be bradycardia or tachycardia. Tachycardia can be either ventricular tachycardia, ventricular fibrillation, or other tachyarrhythmias such as atrial fibrillation with rapid ventricular response resulting in myocardial ischemia. Stable ischemic patients at highest risk are those who have had a previous myocardial infarction and those with LV dysfunction generally related to myocardial infarction.

Patients who have increased heart rate (without the development of an ACS), from any cause, e.g., atrial fibrillation may trigger myocardial ischemia which may result in ventricular arrhythmias and sudden cardiac death. Thus, cardioversion or slowing of the heart rate with beta blockers may prevent SCD by decreasing the amount of myocardial ischemia.

Approach to Management

My approach to management of the patients with stable ischemic heart disease who continue to have ischemic symptoms is to vigorously use sublingual glycerol trinitrate at the onset of ischemia. If myocardial ischemia is anticipated, e.g., prior to exertion, I also recommend the prophylactic use of glycerol trinitrate prior to the exertion.

It seems to me that patients with stable ischemic heart disease and multiple risk factors can make them more prone to acute myocardial infarction or

unstable angina, mainly because they enhance the progression of coronary artery pathology, i.e., even minor plaques may disrupt and result in unstable angina or occlusive coronary disease which then may result in acute myocardial infarction [3]. Once this occurs, the patient may move on to possible sudden cardiac death.

Coronary Pathology during SIHD and Acute Coronary Syndrome

It would be interesting to retrospectively review all patients admitted to hospital with a myocardial infarction and/or sudden cardiac death who had previous stable angina to investigate the pathologic changes in the coronary circulation during stable angina vs. during an episode of acute myocardial ischemia. Little and colleagues published a retrospective study in order to determine if coronary angiography can predict the site of a future coronary occlusion resulting in a myocardial infarction [3]. The answer to the question posed is no, since 66% (not 100%) of patients had an acute myocardial infarction because of an occlusion of a coronary artery that previously had less than a 50% angiographic coronary stenosis. Infarction due to occlusion also occurred at the sites with high grade stenoses and not only at the “minor” lesion sites. Their observations support the concept that “minor” coronary stenoses can be disrupted and eventually occlude the artery. Sudden cardiac death was not evaluated in this report.

Angioplasty of Minor Lesions in SIHD Patients

I wonder if performing angioplasty at these “minor” sites might have prevented the subsequent myocardial infarction. This is a large step, but I know that a few agree with performing angioplasty (not stent) in patients with minor stenoses. The rationale is that restenosis may occur but that then can be dealt with by repeat angioplasty and stenting.

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