How much does prior myocardial infarction increase risk of sudden cardiac death in the young?

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Received 29 June 2012; accepted after revision 3 July 2012; online publish-ahead-of-print 23 July 2012

This editorial refers to ‘Prior myocardial infarction in the young: predisposes to a high relative risk but low absolute risk of a sudden cardiac death’ by B. Risgaard et al., on page 48

Sudden cardiac death (SCD) is a major public health problem in western countries and is particularly devastating in young subjects. Clearly, understanding the causes of SCD is paramount to design preventive strategies. Importantly, causes of SCD may vary between age groups. While most SCD cases in patients >35 years occur in the setting of coronary artery disease, the role of coronary artery disease is not so clear in younger SCD victims. For instance, SCD in persons aged 18–40 years is often caused by inherited heart disease which can be structural (e.g. hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy) or primary electrical (e.g. long QT syndrome, Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia). To establish the role of coronary artery disease in young (18–35 years) SCD victims, Risgaard et al. in this issue of the journal report data on SCD after myocardial infarction (MI) in 18–35-year-old individuals. By combining data from several Danish registries (National Cause of Death Registry and National Patient Registry), they found 1234 such patients with prior MI, 96 of whom died between 2000 and 2006; of these, 50 died of cardiac disease, including 10 from SCD (30.5 per 100 000 person-years). By comparing this number with the rates of SCD in non-MI patients of similar age (4.1 per 100 000 person-years), they calculated that prior MI was associated with a hazard ratio of 55.0 for SCD. This hazard ratio is much higher than published data for older post-MI patients, suggesting that prior MI is of even greater importance among the young. However, the absolute risk for SCD in post-MI patients remains clearly lower among young persons than among older persons. Moreover, one should note that the very high hazard ratio in young post-MI patients may be due to the fact that the risk of SCD among young non-MI patients to be very low (4.1 per 100 000 patient years vs. 60–89 per 100 000 patient years in the general population). Also, it may be of importance that it was not truly established in all patients that death was caused by SCD. For instance, autopsy was not performed in any of the 10 SCD cases post-MI, while 3 of them had asystole as electrocardiogram (ECG)-documented rhythm during cardiac arrest, and one had no ECG documentation.

Despite these limitations, it is clear that prior MI is also an important risk factor for SCD in patients 18–35 years of age. At present, one can only speculate about the causes of a significant increase in SCD risk conferred by prior MI in young persons. For instance, it may stem from concomitant SCD-associated conditions that are relatively prevalent in the young. While one of the 10 SCD victims reported by Risgaard et al. had epilepsy, sudden unexplained death in epilepsy typically affects young persons. Another patient had complex congenital heart disease which is associated with an increased risk for lethal arrhythmias and is clearly more prevalent in younger patients. Finally, concomitant inherited heart disease may have been present, but not been recognized. All these concomitant conditions are likely to play an important role because they may be associated with pathophysiological changes in the heart, which may play a permissive or even amplifying role in the occurrence of lethal cardiac arrhythmias.

How can we prevent SCD in young individuals? One may consider implantable cardioverter defibrillator (ICD) therapy as a possible therapeutic strategy for the prevention of SCD. Yet, ICD therapy can potentially be disadvantageous. Thus, risk stratification is crucial to optimize the benefit-to-harm ratio. Patients with prior MI have traditionally been regarded as patients at high risk of SCD. In the thrombolytic era, risk of arrhythmic death after MI has been as high as 5% per year. More recent data show that SCD rate after primary coronary intervention for MI has drastically decreased to 0.7–1% per year. The risk for SCD in the young post-MI patients reported by Risgaard et al. is even lower at only one-tenth of the presently accepted threshold for ICD therapy (3% yearly risk).
Clearly, at present, other preventive strategies must be sought. Strategies based on identification and modification of the causative factors for SCD may be more effective. Evidently, primary prevention of MI and atherosclerosis plays an important role. Premature atherosclerosis is often caused by a combination of traditional risk factors, but can also be caused by inherited conditions such as familial hypercholesterolaemia. In young patients with MI, screening for atherosclerosis in relatives is advised, but often forgotten. In addition, one should consider the role of concomitant inherited heart disease. Clearly, patients with prior MI are at increased risk for recurrent acute ischaemia and may suffer SCD as a result (this possibility must be strongly considered in this study population as 3 of 10 patients had angina in the year before SCD). Sudden cardiac death during acute ischaemia is a complex trait in which heritable factors play an important role as they may amplify the arrhythmia-provoking effects of the acute ischaemia insult. Thus, prevention of SCD must include screening of relatives of the SCD victim for heritable factors, e.g. inherited heart disease. Importantly, a recent study showed that diagnostic workup in relatives of sudden unexpected death victims <50 years may reveal inherited heart disease with increased SCD risk in as many as 33%.

Conflict of interest: none declared.

References