Premature Myocardial Infarction: An updated Overview

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Abstract

Although myocardial infarction (AMI) is usually the disease of older people over 45 years of age, an increasing number of younger patients is being recorded. Additionally, AMI in very young patients aged 35 years and younger has been poorly described. Besides atherosclerotic coronary artery disease, non-atherosclerotic coronary artery diseases or hypercoagulability should be considered for young cases of myocardial infarction. The predominance of angiographic single-vessel disease and myocardial infarction with normal coronary arteries in these patients primarily suggest that premature myocardial infarction probably result from a rapid progressive event, such as thrombosis or plaque rupture, rather than a gradually evolving process, such as atherosclerosis. In the future, atherosclerotic burden, hemostatic function, characterization of stressors and inflammation will be important targets for research in this group of patients.

An Overview

Advancing age is a well-acknowledged risk factor for AMI in both men and women. Although myocardial infarction (MI) is usually the disease of older people over 45 years of age, an increasing number of younger patients is being recorded presenting with premature myocardial infarction (1). It is estimated that about less than 10% of individuals presenting with documented CAD are Although CHD is an uncommon entity in young patients, it constitutes an important problem for both the patient and the physician because of the devastating effect of this disease. More importantly, these patients have different risk factor profiles, clinical presentations, and prognoses than older patients. Myocardial infarction presenting at a young age shows a different clinical, angiographic findings and pathophysiological profile compared to older (2, 6-11). Additionally, MI in very young patients (aged 35 years and younger) has been poorly described (12).

Traditional risk factors of CAD seen in the elderly age group do not apply to younger age group (13-15). Young patients tend to have less extensive atheromatous lesions and the contribution of prothrombotic and inflammatory indices may be particularly implicated in the initiation and development of such atherosclerotic lesions (8,9). It is also known that the long-term prognosis and functional status of young patients who have acute myocardial infarction (MI) is not benign (2,6,16).

Myocardial Infarction with Normal Coronary Arteries (MINCA) is an important subgroup of myocardial infarction in young patients with a frequency of at least 3-4% of all myocardial infarctions. The proposed mechanisms for MINCA include coronary vasospasm, coronary thrombosis in situ or embolization from a distal source with spontaneous lysis, cocaine abuse, viral myocarditis, aortic dissection, hypercoagulable states, autoimmune vasculitis and carbon monoxide poisoning (17).

Moreover, the predominance of angiographic single-vessel disease in these young patients primarily suggest that premature myocardial infarction probably result from a rapid progressive event, such as thrombosis or plaque rupture, rather than a gradually evolving process, such as atherosclerosis, and thus substantiate the need for an intense and aggressive approach directed towards primary and secondary prevention of premature cardiovascular disease (11). It is unknown whether the incidence of MI at an early age incorporates a grave prognosis or warrants an approach that is different from that used in older patients (12). Nevertheless, it is unclear whether AMI at the younger age can be considered an autonomous representation of the infarct process or a more premature and accelerated expression of the same atherosclerotic process witnessed in older population. Attempts to study this aspect have been stalled by the relative infrequency of AMI in the young, as well as the inconsistencies regarding the definition of youth (10).

Intravascular thrombogenesis is influenced by a complex interplay of procoagulant, anticoagulant, fibrinolytic, endothelial damage/dysfunction and inflammatory factors. Abnormalities of these biological systems would contribute to coronary artery disease presenting at a young age (9). Clinical features suggesting underlying arterial thrombophilia in Premature CAD include: 1) Previous arterial thrombosis and age ≤ 50 (male) or ≤ 55 (female); 2) Age ≤ 55(male) or ≤ 60 (female) with no other traditional risk factors present; 3) No significant
coronary stenosis on angiography; and 4) Age ≤ 55 (male) or ≤ 60 (female) with strong family history (defined as at least one 1st-degree relative affected at age 50 years if male or 55 years if female) (4,18). In the future, atherosclerotic burden, hemostatic function, characterization of stressors and inflammation will be important targets for research in this group of patients (17).

References

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