Obesity: A Risk Factor for Acute Myocardial Infarction with Angiographically Patent Epicardial Coronary Vessels in an Adolescent

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Introduction

Obesity among youthful populations is a common problem in the Gulf area [1–4]. Patients with acute myocardial infarction (AMI) and normal epicardial coronary arteries tend to be young and have relatively few coronary risk factors; however, they often have a history of cigarette smoking and a positive family history for coronary artery disease greater than older patients. The clinical, laboratory and electrocardiographic features of AMI are otherwise indistinguishable from those present in the overwhelming majority of patients with AMI who have atherosclerotic coronary heart disease [5]. Recent data from surveillance studies of myocardial infarction (MI) suggest that excess weight is the most common cardiovascular risk factor in patients with MI and that its prevalence has increased over time [6]. The underlying mechanism of MI in obese patients in the absence of other risk factors is not clear.

Case Report

A 17-year-old Syrian male patient living in Qatar since birth, who is a nonsmoker, nondiabetic and normotensive with no family history of coronary artery disease and no acknowledged drug abuse, presented to the emergency department with central chest pain during the previous night. The pain was crushing in nature,
Radiating to the left shoulder and arm, increasing in severity the night prior to admission. It was not associated with sweating, shortness of breath or palpitation. Physical examination revealed: morbidly obese; weight 139.5 kg, height 170 cm (BMI 48.3), afebrile and no lower limb edema. The abdomen was soft and lax, and the chest was clear. Cardiovascular examination showed: blood pressure 100/62 mm Hg, pulse 82 beats/min, jugular venous pressure was not elevated, normal S1S2 with no murmur. ECG showed low voltage, normal sinus rhythm, ST segment elevation in the inferior leads (fig. 1). Cardiac enzymes were elevated: CPK (normal 5–232 U/l), 575, 212; CPK-MB (normal <5 ng/l), 29.9, 17.3, 8.5; troponin (normal <0.1 ng/l), 2.6, 0.78, respectively; C-reactive protein (normal <6 mg/l), 12.5; ESR (normal <15 mm/h), 16. Cholesterol: 4.95 (normal <5.1 mmol/l); triglycerides: 2.1 (borderline: 1.7–2.1 mmol/l); HDL: 0.77 mmol/l, and LDL: 3.22 mmol/l. Serum levels of ethanol and cocaine were undetectable. An echocardiogram showed mildly impaired left ventricular systolic function (ejection fraction: 45%), trivial mitral regurgitation, hypokinesia of the inferior wall and normal right ventricular systolic pressure. There was no evidence of intracardiac shunt, mass or thrombi. He was admitted to the coronary-care unit as a case of inferior MI with late presentation; he received aspirin and heparin, tirofiban and isosorbide dinitrate infusion. A coronary angiogram was performed on the second day of admission and revealed patent epicardial coronary arteries (fig. 2). The patient was followed up by a dietitian and endocrinologist, who started short- and long-term plans for weight reduction and family counseling. The hospital stay was uneventful, and he returned home on the fourth day on a regimen of aspirin 150 mg once daily, isosorbide dinitrate retard 20 mg once daily, diltaizem 60 mg 3 times per day and atorvastatin 20 mg once daily.

**Discussion**

Obesity is a global problem, affecting an estimated 300 million people worldwide. Its prevalence is increasing in both developing and developed countries throughout the

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**Fig. 1.** ECG on arrival. **a** Low-voltage complexes and ST segment elevation in limb leads 2, 3 and aVF. **b** ECG on the fourth day showing resolution of ST segment elevation.

**Fig. 2.** The coronary angiogram reveals patent left (a) and right (b, c) coronary arterial systems.
world, pertaining to people of all ages and all socio-economic levels [7]. It has been estimated, using age- and sex-specific BMI criteria, that 10.4% of children 2–5 years of age, 15.3% of children 6–11 years of age and 15.5% of adolescents 12–19 years of age are overweight [8]. Compared to the international standard [1], 10-year-old male children from the United Arab Emirates had 1.7 times the rate of obesity and 1.9 times at 18 years, while female children from the United Arab Emirates have 1.8 times the rate of obesity at 10 and 18 years of age. The overall level of overweight and obesity among adolescent Kuwaitis aged 10–14 years is 30.9% [2] and 26% among Saudi adolescents aged 10–14 years [3]. In a sample of 800 boys in the age group of 14–19 years in Qatar, 33.1% were overweight, while the cardiovascular disease risk factor of obesity was more prominent among women (528, 78.3%) than among men (334, 68.9%) in older patients [4].

Excess weight is associated with higher mortality and cardiovascular events [9]. About 10% of all patients with AMI and nearly 20% of those under 35 years of age do not have coronary atherosclerosis demonstrated by coronary angiography, necropsy or both. Around half of nonatherosclerotic AMI patients have angiographically normal coronary arteries [10]. The possible reasons include poor angiographic technique so that a significant coronary artery stenosis is missed, small-vessel disease, hypercoagulable states, coronary embolism, coronary trauma and coronary vasospasm. Intravascular ultrasound studies documented atherosclerotic plaques that failed to encroach on the lumen of the involved coronary artery. In these patients, it was hypothesized that one of these small, intramural atherosclerotic lesions had ruptured, thereby leading to coronary arterial thrombosis that subsequently lysed, leaving the normal lumen intact [5, 10, 11]. Nevertheless, evidence has not been produced to show that obesity may be a risk for AMI in adolescents in the absence of the other risk factors, i.e. smoking, diabetes, hypertension, hyperlipidemia or drug abuse. Our case demonstrates an adolescent with angiographically normal epicardial coronary arteries and inferior AMI. This patient was not given thrombolytic therapy because of late presentation. His electrocardiographic changes normalized, and the chest pain subsided after starting antianginal medications. The patient does not have a significant risk factor apart from morbid obesity. The mechanisms whereby excess body fat affects the cardiovascular system include not only an indirect effect on the vascular system through risk factors like dyslipidemia, hypertension, obstructive sleep apnea or insulin resistance, but also an enhanced inflammatory state, a high turnover of free fatty acids with a lipotoxic effect on myocardial cells and the potential effects of high levels of leptin [10, 12]. Obesity has been shown to be independently associated with coronary endothelial dysfunction in patients with normal or mildly diseased coronary arteries [11]. There was no evidence that obesity might initiate coronary artery spasm. The provocation of coronary artery spasm during the acute stage of MI carries a high risk so that in most cases the diagnosis was based on the patency of the coronaries during elective coronary angiography in addition to the clinical and electrocardiographic evidence of acute coronary events. Lack of an intravascular ultrasound study in our case is one of the limitations. In this case MI may be due to coronary spasm, invisible ruptured plaque or re-canalized thrombus, and all are related to the obesity effect. However, it is an interesting case for further assessment that may re-address the role of obesity in AMI in such an age group.

Conclusion

In the absence of well-known risk factors for coronary artery disease, obesity might be the sole risk for MI at a young age. This hypothesis warrants further supportive studies that in turn will have major implications on therapy and risk stratification in obese teens. Public education and awareness of this complication at a young age are warranted.
References


