

DO YOU KNOW HOW YOUNG YOUR HEART IS?

Tase Cristina Ramona^{1,2}, Cojocaru Lucia^{1,3}, Rusali Andrei^{1,3}, Suta Cristina^{1,4}

¹ Faculty of Medicine, University "Ovidius" of Constanta

² Emergency Clinical County Hospital of Constanta, Emergency Department

³ Emergency Clinical County Hospital of Constanta, Cardiology Clinic

⁴ Emergency Clinical County Hospital of Constanta, Internal Medicine Clinic

Lucia Cojocaru

Emergency Clinical Hospital of Constanta, Cardiology Clinic,
Tomis Boulevard No 145, Constanta, Romania
email: bostanlucia@yahoo.com
phone: +40 722370635

ABSTRACT

We present the case of a 25 years old patient who was submitted to our unit with a first time acute coronary syndrome. Despite his young age he had multiple cardiovascular risk factors. Although the chest pain was atypical and the electrocardiogram on presentation had unspecific changes, repeated investigations established the diagnosis of anterolateral myocardial infarction. Per primam angioplasty with stent implantation in the proximal segment of left anterior descending artery was performed, with good clinical outcome. Awareness is the key in establishing the diagnosis of myocardial infarction in young patients.

Keywords: myocardial infarction, young adults, low HDL-cholesterol, family history

Introduction

Coronary heart disease (CHD) is the leading cause of death worldwide, being responsible for 20% of all deaths in Europe alone (1-3). Although CHD primarily occurs in patients over 40, younger people can be affected (4,5).

In the past two decades, due to the primary and secondary prevention strategies, there has been a reduction in the incidence of cardiovascular (CV) events and mortality in the general population (6,7). However, in the young adults, the incidence of acute myocardial infarction (MI) remained stable and CV disease is still a major cause of death (6-8).

As a clinician, there are real challenges in the proper approach and management skills in prevention and treatment of CHD among young individuals, the majority of them being unaware of their risk factors, presenting atypical symptoms that may delay presentation in the emergency department and have higher rates of medication non-adherence (9-12). The different risk factor profiles, clinical presentations and prognoses should be taken into consideration when treating young patients with CHD (4,5).

Clinical Case

We present the case of a 25-years old male patient, smoker (3.5 pack-year), without past medical history, but with a positive family history for cardiovascular disease (father with sudden death after MI at 32 years old), presented to the emergency department for severe precordial stabbing chest pain. The pain occurred at rest, lasted for approximately 3 hours and was relieved after one tablet of nitroglycerin given by the ambulance crew. He described having had, in the previous 3 months, repetitive episodes of stabbing left sided chest pain during effort with spontaneous remission at rest (in than 5 minutes) for which he had an medical exam that established the diagnosis of atypical chest pain and recommended an stress test, which he didn't performed.

On the admission, the patient was free of pain, in good clinical condition. The clinical exam revealed a normoponderal patient with a blood pressure of 120/60 mmHg in both arms, a heart rate of 56 bpm, without heart murmurs, gallop or pericardial friction rub and without signs of pulmonary or systemic congestion.

Electrocardiogram (ECG) on admission revealed sinus bradycardia, 52 bpm, QRS axis = + 30°, slight ST elevation in V2 (0.5mm), biphasic T waves in V1-V2 and negative T wave in aVL (Figure 1).

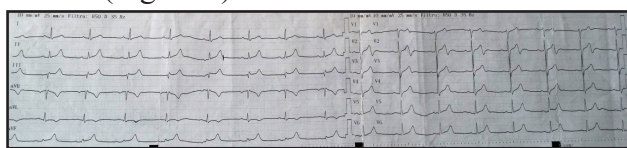


Figure 1: Electrocardiogram on admission - sinus bradycardia, 52 bpm, QRS axis = + 30°, slight ST elevation in V2 (0.5mm), biphasic T waves in V1-V2 and negative T wave in aVL.

Emergency echocardiography revealed normal cardiac chambers size, normal left ventricular (LV) systolic and diastolic function (left ventricle ejection fraction - LVEF of 65%), no significant valvulopathy, and no signs of pulmonary hypertension, no pericardial fluid and no signs of aortic dissection. During the echocardiographic exam the patient had another episode of precordial stabbing pain accompanied by akinesis of LV apex and apical

half of interventricular septum. The repeated ECG during the pain showed Q waves in V1-V3, 1mm ST elevation in V3 and 0.5mm elevation in DI, aVL, V2, with ST depression in DII, DIII, aVL and negative T wave in aVL (Figure 2); this rapidly evolved to significant elevated ST segment in DI, aVL, V2-V5 (Figure 3).

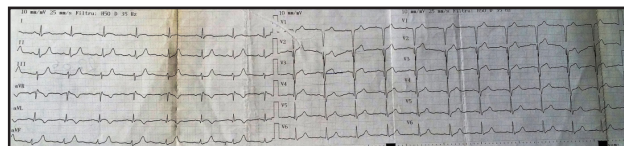


Figure 2. Electrocardiogram during chest pain – sinus rhythm, 75bpm, Q waves in V1-V3, 1mm ST elevation in V3 and 0.5mm elevation in DI, aVL, V2, with ST depression in DII, DIII, aVL and negative T wave in aVL

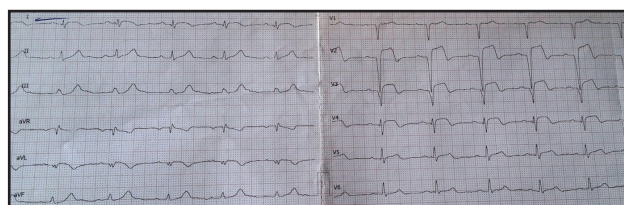


Figure 3. Electrocardiogram during chest pain (prior to angiography) – sinus rhythm, 62bpm, QS in V1-V3 and poor R in V4, Q in aVL, elevated ST segment in DI, aVL, V2-V5 that encompasses the T wave in V2-V4, negative T wave in DI, aVL

Laboratory tests revealed positive myocardial necrosis markers (with typical myocardial necrosis-associated rise during hospital stay, peak CK-MB=209U/L and peak TnT 2488ng/mL), mixed dyslipidemia with normal LDL-cholesterol (82mg/dl), low HDL-cholesterol (27mg/dl) and high triglycerides levels (196 mg/dl), and mild leukocytosis with neutrophilia. The liver function tests, renal function tests and ionogram were in normal limits.

The diagnosis of acute anterolateral MI was established and the patient received a loading dose of unfractionated heparin and double antiplatelet and was sent to the cath lab. The coronarography revealed a 50-70% stenosis in the proximal left anterior descending artery (LAD) with the aspect of a ruptured plaque (Figure 4) and less than 50% stenosis in the medial segment of LAD and ostium of first diagonal artery. Emergency angioplasty with stenting (drug-eluting stent) of the culprit lesion was performed and post-interventional

TIMI 3 flow was established (Figure 5).



Figure 4. Angiography - 50-70% stenosis in the proximal left anterior descending artery with the aspect of a ruptured plaque (arrow)

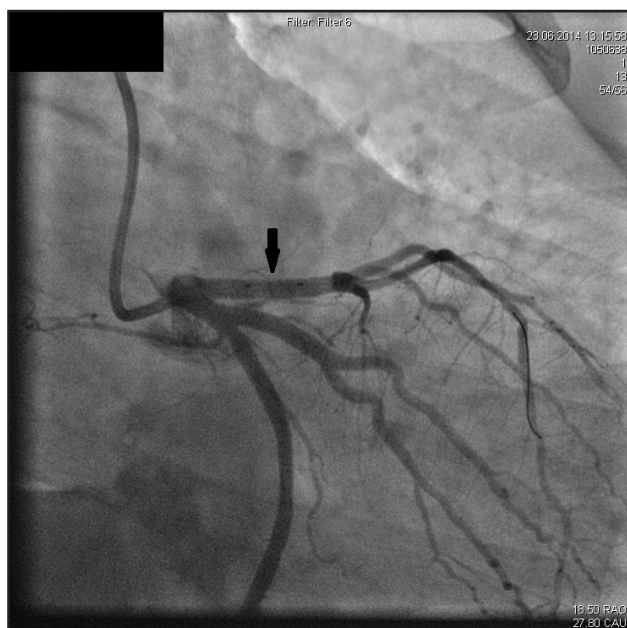


Figure 5. Angioplasty with stenting (arrow) of the culprit lesion in the proximal left anterior descending artery with distal TIMI 3 flow

During hospitalization in the cardiology unit, under pharmacological treatment with double antiplatelet therapy, beta-blocker, angiotensin-converting-enzyme inhibitor and statin, the clinical evolution was good and the patient was discharged free of pain, without signs of heart failure. Three years after the acute MI the patient

is in good clinical condition, asymptomatic, with a proper adherence to treatment but he resumed smoking and has a sedentary lifestyle. The laboratory tests reveal optimal LDL-cholesterol (68mg/dL) and triglyceride (90mg/dL) levels, but the HDL-cholesterol remains low (30mg/dL). The ECG shows no R waves in V1-V3 and at echocardiography there is a mild LV apical hypokinesia (LVEF of 55%) as his only evidence of the previous MI; the ECG stress test is negative for ischemia. Given his family history, smoking habit, sedentary lifestyle and low HDL-cholesterol the patient remains at high-risk of subsequent ischaemic events.

Discussion

The prevalence of CHD in younger subjects is difficult to establish because is frequently a silent process (4,5). There are limited data on the frequency of MI in younger subjects, studies showing that 4 to 10% of patients with MI were under 40 or 45 years of age (4,5,13-15).

Although CHD is an uncommon entity in young patients, it is an important public-health issue given the negative impact on physical, mental, social, and financial health, and greater healthcare utilization among affected individuals (4-6).

Young patients with MI usually have multiple risk factors for CHD (5). Studies found that 90 to 97% of them had one or more traditional risk factors for atherosclerosis (4,5,16-18). Cigarette smoking is the most common and most modifiable risk factor in young patients, being present in 65 to 92 percent of young patients with MI, compared to 24 to 56 percent of patients older than 45 years of age (4,5,17). Younger patients with CHD more often have a family history of premature CHD (41 to 64 percent) and the offspring of patients with premature CHD are more likely to have coronary risk factors than those without such family history (4,5,17,19,20). This association between family history and premature CHD can be due to both genetic and environmental factors (4,5). Lipid abnormalities are another risk factor for CHD in young patients. When compared to older patients, young patients have lower HDL-cholesterol concentrations, higher triglycerides

and the same prevalence of hypercholesterolemia (4,5,21). Arterial hypertension and diabetes mellitus appear to be less common in young patients with MI (4,5,19,22). However, young patients frequently have subtle problems with glucose metabolism, more than half of them having decreased oral glucose tolerance and hyperinsulinemic response to oral glucose challenge that is also a risk factor for CHD (4,5). Obesity is another important coronary risk factor and it appears to be an independent risk factor for coronary atherosclerosis, at least in young men (4,5). Other risk factors that have been identified in young patients with MI are: oral contraceptive use, factor V Leiden, frequent cocaine use, smoking marijuana and psychosocial factors, such as anger (4,5). In our patient, risk factors for CHD have been smoking, the positive family history for premature CHD and low HDL-cholesterol levels.

Current CV risk calculators are less applicable to younger patients where they underestimate the risk (6,23). The YOUNG-MI study gave criteria for new risk factor calculators and awareness of CHD events in patients < 50 years, knowing that most patients do not cross the threshold for primary prevention defined by current guidelines and consequently they are not considered candidates for preventive therapies (6,23).

When considering the clinical presentation, young adults are more likely to have atypical symptoms, as was the case of our patient (4,5). When compared to older patients with CHD, a higher proportion of young patients do not experience stable angina, and, in the majority of cases, an acute coronary syndrome is the first manifestation of CHD (4,5,13,21). Among those who have preceding chest pain, the first episodes often occur only in the week prior to MI (4,13). The most important differential diagnosis of MI in young patients is acute myocarditis. This disorder can mimic the MI and must be considered in young patients with a clinical presentation of acute coronary syndrome but with normal coronary angiogram (4,5,24,25).

The management of young patients with acute MI varies with the type: ST-elevation or non-ST elevation. The overall approach is generally the same regardless the age of

the patient, in accordance to current clinical practice guidelines. Young patients with acute ST-elevation MI should be treated with primary angioplasty or, if not available, thrombolytic therapy (4,26). When compared to older patients, young patients do better regardless of the type of reperfusion they received (4). The angiographic findings in young patients also differ from those of older ones. Younger patients have a higher incidence of normal coronary arteries, mild luminal irregularities, and single vessel coronary artery disease than do older patients (4,5,18,21,22,27). Spontaneous coronary artery dissection and Kawasaki disease are two rare causes of MI that occur more commonly in the young and must be taken into consideration when assessing these patients (4).

When compared to older patients, younger patients have lower in-hospital mortality, better long-term outcome and same rate of reinfarction (4). In the treatment of MI survivors, risk factor reduction plays a central role (4).

Conclusions

Although symptomatic CHD is uncommon in young people, with increasing rates of traditional CV risk factors we can expect CHD to become even more prevalent in this age group (6). We must keep in mind that current available risk calculators underestimate the CV risk in young population and most of them will not benefit from primary prevention, increasing the incidence of CV events. No matter the patient's age, the age of his heart will be given by the cluster of his CV risk factors.

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