Review Article

Distinctive characteristics, risk factors, and prevention of premature myocardial infarction: A narrative review

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Abstract

The proportion of young individuals that present with acute myocardial infarction is a major problem that keeps increasing. The specific characteristics of premature coronary artery disease and its differences between young and older individuals need to be elucidated. Although risk factors are similar in different age categories, there is a great difference in their prevalence. The vast majority of young patients are males and there is a higher prevalence of cigarette smoking, family history of premature coronary artery disease, lipid disorders, and illicit drug use, while the prevalence of hypertension and diabetes mellitus is lower. Young individuals with acute coronary syndrome usually present either with ST-segment elevation or non-ST-segment elevation myocardial infarction. It is not unusual for young patients to present with atypical symptoms to the office of primary care physicians, leading occasionally to incorrect or delayed diagnosis. Therefore, prompt and correct diagnosis is necessary to implement the specific management as quickly as possible. A literature research of studies was conducted for the last 10 years, regarding the risk factors and prevention of premature myocardial infarction. As databases, we used PubMed and peer reviewed journals. The aim of this review is to raise awareness among family medicine and primary care physicians, regarding the clinical presentation of young patients with acute myocardial infarction, to provide optimal medical attention.

Keywords: Coronary artery disease, ischemic heart disease, premature myocardial infarction, prevention, young

Introduction

Coronary artery disease (CAD) remains one of the most common causes of death in the world and its prevalence keeps increasing, particularly in developing countries. It may manifest as stable CAD, acute coronary syndrome (ACS), or sudden cardiac death. ACS is classified in the ST-segment elevation myocardial infarction (STEMI) which accounts for ~30% of ACS and the non-ST-segment elevation ACS, which accounts for ~70% of ACS.[1] The latter is further classified as non-ST-segment elevation myocardial infarction (NSTEMI) and unstable angina[1] [Figure 1]. Unstable angina is a disappearing entity nowadays, due to the widespread application of high-sensitivity cardiac troponin assays which can detect minimal myocardial necrosis.[2] In the following text, under the term acute myocardial infarction (AMI) both STEMI and NSTEMI are included.

Coronary atherosclerosis starts developing silently in early years and progresses over the course of one's lifespan. AMI is responsible for ~1.8 million deaths, around 20% of all deaths in Europe with some variations between countries,[3] and affects both women and men, although its development in women delays on average of 7–10 years compared to men. The mean age of the first AMI in the United States of America (USA) is roughly calculated at 65.6 years for men and 72 years for women.[4]

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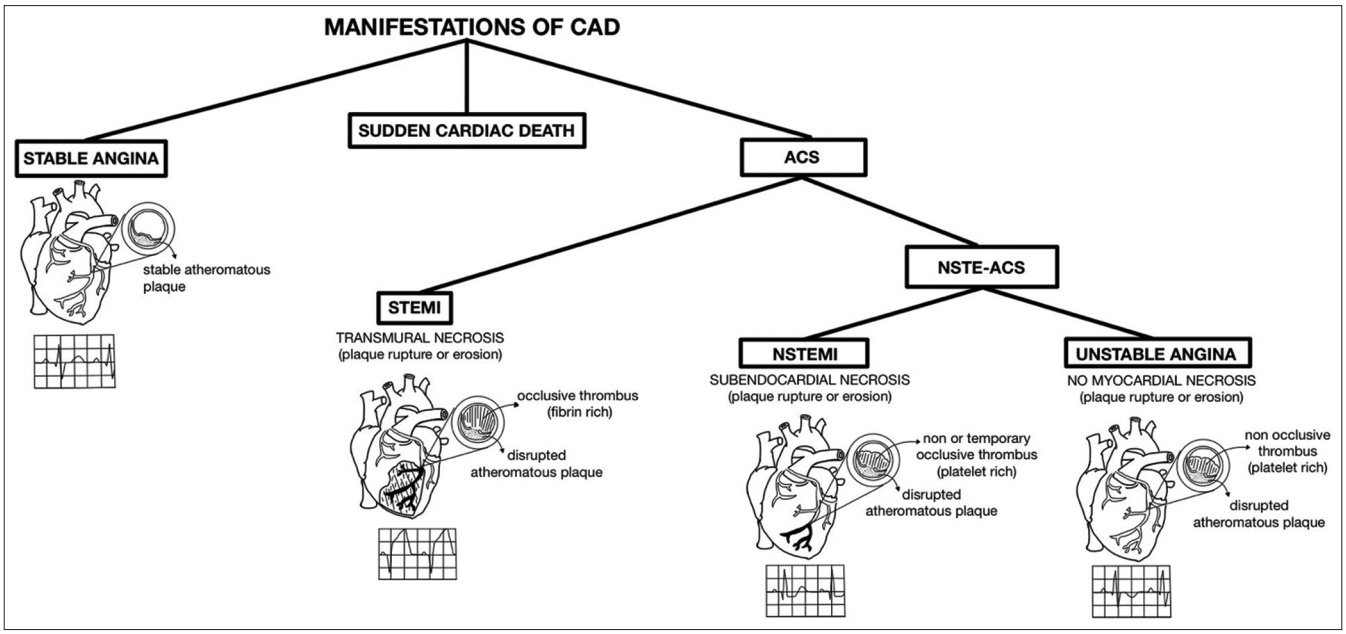

Figure 1: Manifestations of coronary artery disease. A summary diagram of the manifestations of coronary artery disease. CAD, coronary artery disease; ACS, acute coronary syndrome; STE, ST-segment elevation; STEMI, ST-segment elevation myocardial infarction; NSTEMI, non-STEMI

Premature AMI is relatively uncommon but its annual incidence is steadily rising over the past two decades in contrast to the reduction of all AMIs occurring in the same period.[5]

The consequences of premature AMI are detrimental in young patients due to the impact on their physical capability, psychology, and socioeconomic status.[6] This is because this population is supposed to be professionally active for more than 20 years after the coronary event and theoretically has a longer life expectancy compared to the patients who experience AMI at older ages. Therefore, the differentiation between younger and older AMI patients is important, because there are substantial differences in the risk factors profile, clinical presentation, and prognosis between these two subgroups.

The aim of this review is (a) to examine the specific characteristics of premature AMI regarding the clinical presentation along with the risk factors profile and to raise the awareness of primary care doctors on the possibility of AMI in a young individual with chest pain and (b) to highlight the role of primary care and family medicine doctors to convey the message of healthy lifestyle habits, such as healthy diet, abstain from smoking, avoid excess weight, regular physical activity in all young individuals.

Methods

A literature search was conducted through various studies of the last 10 years (2013–2023) about the risk factors of young patients experiencing an AMI, as well as the possible ways for prevention. For the purpose of this narrative review, we used PubMed, some peer-reviewed journals, such as the European Heart Journal, the Journal of the American College of Cardiology, and some important websites such as the American Heart Association. During the search in PubMed, we used Boolean operators, such as AND, to receive more accurate papers from this database. We used the English language for the specific retrieved papers and journals. We also scanned both titles and abstracts to find more relevant articles. For the research in PubMed, we applied a different set of keywords that were related to premature CAD ("premature," "MI," "premature coronary artery disease," "risk factors," and "prevention").

Epidemiology

CAD causes ~17.8 million deaths per year globally.[7] In more developed areas around the world, such as the USA, the mortality rate from CAD was ~75–103 deaths per 100,000 people in the year 2019. However, in developing countries of Central Asia, the mortality rate from CAD was ~132 deaths per 100,000 people in the same year.[7] This difference can be attributed to ethnological and lifestyle differences and also to limited access to healthcare professionals or appropriate treatment in developing countries.

Regarding the epidemiology of premature AMI, it is difficult to define it, due to the great variability in the age thresholds that have been applied. Although the rate of admissions for AMI is declining during the last decades, the incidence of premature AMIs is constantly growing and constitutes one of the leading causes of premature death worldwide.[8-10] It is estimated that approximately 4–10% of all AMIs occur under the age of 45 years.[5]

Most data on the prevalence of premature AMI have been provided mainly by USA studies. In the decade 2001–2010, more than 1 million hospital admissions in the USA were due to 30–50-year-old AMI patients.[10,11] Although short-term prognosis is more favorable in patients suffering an AMI at a young age

compared with older patients, long-term prognosis is not benign with young women having worse long-term survival compared to men.[12,13]

Risk factors

The risk factors of patients who experience AMI at a young age are almost the same as in older patients but with major differences in their prevalence. It has been shown that male gender, cigarette smoking, family history of premature CAD, lipid disorders, and drug abuse display a higher prevalence, while diabetes mellitus (DM) and arterial hypertension present with lower prevalence in patients with premature AMI compared to older patients.[14] Obesity is also associated with the risk of premature AMI.[8]

Among all risk factors, cigarette smoking is the most important modifiable risk factor among young patients with AMI, with the prevalence ranging from 70 to 95%.[15] Smoking is followed by lipid disorders. Approximately, 10–20% of young patients have heterozygous familial hypercholesterolemia.[16] This is an autosomal dominant-inherited genetic lipid disorder characterized by high cholesterol levels from birth which if remains untreated is associated with a very high risk of developing premature CAD. Another inherited lipid disorder with a similar natural history is familial combined hyperlipidemia, with a prevalence of ~12.5%.[17] Finally, lipoprotein(a) [Lp(a)], which is mainly determined genetically, is also a risk factor for premature CAD and it has been reported that 30% of patients with AMI ≤50 years have elevated Lp(a) levels, i.e. Lp(a) >50 mg/dL.[18,19]

Another important risk factor in young patients, which is almost negligible in the older ones, is substance abuse. Substances, such as cocaine and marijuana that can trigger AMI, have a higher prevalence among young patients compared to their older counterparts. It has been reported that marijuana and cocaine use was present in almost 11% of patients who suffered from AMI ≤ 50 years of age.[20] In particular, cocaine apart from its acute vasoconstrictive and thrombogenic effect, its chronic use exerts an atherogenic effect contributing to the acceleration of coronary atherosclerosis.[21,22]

Finally, the impact of thrombophilias, particularly in the setting of low atheromatous coronary burden and the absence of traditional risk factors, should be considered.[15,23] The summary of the most important studies, along with their findings, is shown in Table 1.

Pathophysiology

In this section, we discuss type 1 AMI which is caused by thrombus formation on a disrupted atherosclerotic coronary plaque.[24,25] It is described as a sudden ischemic necrosis of the myocardial tissue caused by inadequate blood supply due to thrombotic total occlusion of the coronary artery in STEMI or to transient or subtotal intraluminal occlusion and/or distal embolization in NSTEMI. Thrombus formation is caused by the disruption (rupture or erosion) of an unstable/vulnerable atherosclerotic coronary plaque.

A distinct anatomic characteristic of premature AMI is the lower coronary atheromatous burden compared to older patients. It is estimated that the prevalence of myocardial infarction with nonobstructed coronary arteries (MINOCA) plaques, i.e. lesions in coronary arteries causing <50% reduction in lumen diameter, ranges from 10 to 20% among young patients while in older patients is 5–6%. The prevalence of MINOCA is even higher among young women and NSTEMIs.[26]

Disease presentation

The typical presentation of AMI is characterized by a severe, crushing, retrosternal pain usually described by the patient as a compressing, squeezing, or even burning sensation. The pain lasts >30 minutes and is not relieved by nitroglycerin or rest. It is also usually accompanied by nausea, vomiting, and sweating and may radiate to the upper extremities, neck, lower jaw, shoulders, interscapular area, or epigastrium.[1]

The clinical signs of AMI are usually poor and not specific, i.e. perspiration, sinus tachycardia, or 4th heart sound.[26] However, in the setting of complicated AMI, i.e. acute left heart failure due to extensive AMI or mechanical complications, there may be hypotension, signs of pulmonary congestion, systolic cardiac murmur, or signs of inadequate perfusion.

The clinical presentation of AMI in young individuals is quite similar to that of older patients. However, there are some differences which pose difficulties for the early diagnosis.[27] In young patients, a history of angina symptoms, before the occurrence of an AMI, is less common and only appears in one-fourth of patients.[27] There is an increased likelihood (~20%) for young patients, particularly women, to present with shortness of breath, palpitations, or fatigue without the presence of chest pain.[14] The atypical presentation and their young age may lead the examining physician to underestimate their cardiovascular risk and it is not unusual for the young patients to suffer a delay before the correct diagnosis is reached.

Diagnosis

The diagnostic criteria for AMI do not differ between young and older patients. These criteria are based on the detection of a rise and/or fall of a myocardial necrosis marker (preferably cardiac troponin) that should also be accompanied by at least one of the following features: symptoms of acute myocardial ischemia, new ischemic changes on the electrocardiogram (ECG), imaging demonstration of new loss of viable myocardium or new regional wall motion abnormalities in a pattern suggestive of ischemia and visualization of an intracoronary thrombus by angiography or autopsy.[25]

As for the type of AMI in young individuals, most studies report a predominance of STEMI. In the Mass General Brigham

Table 1: Literature summary table with studies regarding risk factors in young myocardial infarction patients

Authors/date	Aim of the study	Type of the study	Sample size	Study population	Main findings/ conclusion	Limitations
Rallidis LS, Triantafyllis AS, Tsirebolos G, *et al.* (2016)	Explored the prevalence of HeFH in patients with early STEMI, the use of statins, and their impact on long-term prognosis.	Prospective study	320 (279 males, 41 females)	Patients who had their first STEMI at ≤35 years of age.	20% of patients with STEMI <35 years have definite or probable HeFH and are characterized by a high risk of recurrence of coronary events.	There was no genetic molecular analysis for the correct identification of HeFH patients.
Vikulova DN, Trinder M, Mancini GBJ, *et al.* (2021)	Examined the prevalence of FCHL, FH, elevated Lp(a), and their impact regarding the management of patients presented with premature CAD.	Prospective cohort study	263 (All males)	Males ≤50 years and females ≤55 years with obstructive CAD.	Among 263 participants, 9.1% met the criteria for FH, 12.5% for FCHL, and 19.4% had elevated Lp(a).	Small population size and lack of established diagnostic criteria for FCHL.
Berman AN, Biery DW, Singh A, *et al.* (2021)	Assessed the prevalence of elevated Lp(a) levels among patients who suffered MI ≤50 years.	Retrospective cohort study	2,097 (1699 males, 398 females)	Patients with a first MI at age ≤50 years Median age: 45±3	Lp(a) was measured in 21% of 2,097 patients with MI ≤50 years and Lp(a) >50 mg/dL was found in ~30% of them.	There were two different assays for Lp(a) measurements and this could decrease the internal and external validity of the work.
DeFilippis EM, SinghA,DivakaranS *et al.* (2018)	Assessed the prevalence of marijuana and/or cocaine use in patients with first MI ≤50 years and determined its contribution to long-term outcomes.	Retrospective cohort study	2,097 (1692 males, 405 females)	Patients with type 1 MI at≤50 years Mean age: 44.0±5.1 years	Marijuana and/or cocaine use was found in 10.7% of young MI patients and was associated with worse cardiovascular and all-cause mortality during a median follow-up of 11.2 years.	The association between substance abuse and cardiovascular outcomes could be driven by confounders such as lifestyle and behavioral factors.
Chen DH, Kolossváry M, Chen S, *et al.* (2020)	Investigated the effects of cocaine use on coronary artery plaque volume by using coronary computed tomography angiography.	Pilot cross-sectional study	48 (39 males, 9 female)	Individuals ≥21 years 15—noncocaine users 33—chronic cocaine users	Prolonged use of cocaine was associated with higher total LAD plaque volume.	Small study size and unbalanced data (69% were chronic cocaine users, 81% were men, and 81% were HIV positive).
Mahtta D, Ramsey D, Krittanawong C, *et al.* (2021)	Assessed the association of recreational substances in regard to premature and extremely premature atherosclerotic disease.	Cross-sectional study	1,248,158 (1,221,057 males, 27,101 females) with premature and nonpremature ASCVD	Veterans (males, females) that had: Premature ASCVD Mean age: 49.63±5.79 years nonpremature ASCVD Mean age: 69.58±8.92 years	Recreational substances were independently associated with an increased probability of premature and extremely premature atherosclerotic disease.	No adjustment for additional confounders such as dose and duration of recreational substance use.
Rallidis LS, Gialeraki A, Tsirebolos G, *et al.* (2017)	Examined the prevalence of prothrombotic polymorphisms (FV Leiden and G20210A prothrombin gene), deficiencies of protein C, protein S, and antithrombin III and APS in patients with early STEMI.	Case–control prospective study	655 Patients: 255 (224 males, 31 females) Control: 400 (149 males, 251 females)	Patients who survived their first STEMI, occurring at <36 years. A control group of healthy individuals. Patients mean age: 32.4±3.5 years Controls group mean age: 32.7±3.1 years	G20210A polymorphism of prothrombin gene was linked with a higher risk of STEMI in young people, particularly in smokers. All other thrombotic disorders were only minimally associated with premature STEMI.	Measurements regarding the diagnosis of APS were not made in accordance with the proposed Sapporo criteria and the prevalence of APS could have been underestimated.

Legend: MI: myocardial infarction, HeFH: heterozygous familial hypercholesterolemia, CAD: coronary artery disease, FCHL: familial combined hyperlipidemia, FH: familial hypercholesterolemia, Lp(a): lipoprotein(a), STEMI: ST-segment elevation myocardial infarction, LAD: left anterior descending artery, APS: antiphospholipid syndrome, ASCVD: atherosclerotic cardiovascular disease, HIV: human immunodeficiency virus

YOUNG-MI registry, 53% of individuals who presented with AMI ≤50 years of age had STEMI, while studies from Asia reported an even higher incidence of STEMI.[11,14] On the contrary, other studies reported that two-thirds of all AMIs in young patients were NSTEMI.[14] These discrepancies may be due to ethnological differences between the populations.

In general, the diagnosis of STEMI in the Emergency Department is usually straightforward. The presence of a pathological ST-segment elevation on the ECG in association with the clinical presentation sets the diagnosis of STEMI in the vast majority of cases **[Illustrative Case Story 1].** However, this is not the case for NSTEMI, where the diagnosis apart from the

clinical symptoms and the ECG changes (usually nonspecific) always requires the measurement of the cardiac troponin, preferably the high-sensitivity cardiac troponin.[27]

It should be mentioned that not infrequently there is a delay for young individuals in seeking a medical advice for chest pain because they underestimate the severity of their symptoms due to their young age.

There are a few cardiac diseases that should be in the mind of all family medicine and primary care practitioners because they can mimic the presentation of AMI and need further tests to make the correct diagnosis. This is the case with acute myocarditis and rarely with acute pericarditis.

Acute myocarditis is relatively uncommon, but most commonly affects young individuals. The patient may present with intense chest pain, elevated cardiac troponin, a history of virous infection, and in some cases with ST-segment elevation requiring differentiation from STEMI. The definite diagnosis of myocarditis is based on the exclusion of obstructive CAD by performing a coronary angiography and the demonstration of subepicardial necrosis by cardiac magnetic resonance imaging.[28]

Acute pericarditis may rarely mimic the clinical presentation and the ECG pattern of STEMI. The diagnosis is based on clinical presentation (chest pain that deteriorates with inspiration), auscultation of the pericardial friction rub, presence of pericardial fluid (detected by echocardiogram), elevated C-reactive protein, and characteristic ECG findings. The ST-segment elevation is widespread, typically concave with a characteristic evolution, since T waves become reversed after ST-segment returns to the isoelectric line while in STEMI the reversion occurs before the return of the ST-segment.[29] **[Illustrative Case Story 2].**

Management

The treatment of AMI depends on the type of ACS and is the same, both for the older and younger ones with the exception of some additional measures for the latter group.

In STEMI, the mainstay of treatment is the restoration of the patency of the occluded artery as soon as possible, either by primary percutaneous coronary intervention (PCI), i.e. balloon inflation to open the occluded artery followed by implantation of a drug-eluting stent or by fibrinolytic treatment. Primary PCI is the reperfusion therapy of choice, provided that it can be performed within 120 min of the patient's presentation at the initial hospital. If primary PCI is not feasible, fibrinolytic treatment with alteplase, tenecteplase or reteplase should be administered. After fibrinolysis, the patient should be transferred within 6–24 h to a PCI-capable hospital to have coronary angiography and reperfusion therapy according to the coronary anatomy.[1] Prior to the institution of reperfusion therapy, the patient should receive a dual antiplatelet treatment to decrease platelet aggregation. Antiplatelet treatment involves the administration of 150–300 mg of the nonenteric coated form of aspirin, that is chewed by the patient and remains in the oral cavity to achieve quicker effect. The second antiplatelet is a $P2Y_{12}$ receptor inhibitor given at loading dose, i.e., ticagrelor 180 mg, prasugrel 60 mg, or clopidogrel 300–600 mg. The latter is preferable in cases of fibrinolysis. Along with antiplatelets, heparin is given (usually low molecular weight heparin, such as enoxaparin) to inhibit the coagulation cascade on the ruptured plaque. The patient should also receive oxygen if dyspneic and morphine intravenously to relieve the pain. After reperfusion therapy, the patient will receive beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, and high-intensity statins and continue dual antiplatelet treatment.[30] Furthermore, a subset of them will receive a mineralocorticoid receptor antagonist [Figure 2].

In NSTEMI, the treatment approach is mostly the same. The patient will receive dual antiplatelet therapy (aspirin immediately while a $P2Y_{12}$ receptor inhibitor is better given during catheterization), heparin, nitrates, beta-blockers, morphine, and in most cases an invasive approach will be offered within 24 h with coronary angiography followed by revascularization according to the coronary anatomy. Note that in NSTEMI, fibrinolytic treatment is contraindicated [Figure 3].

There is a lot of speculation regarding the use of β-blockers in AMI patients after cocaine exposure. It has been hypothesized

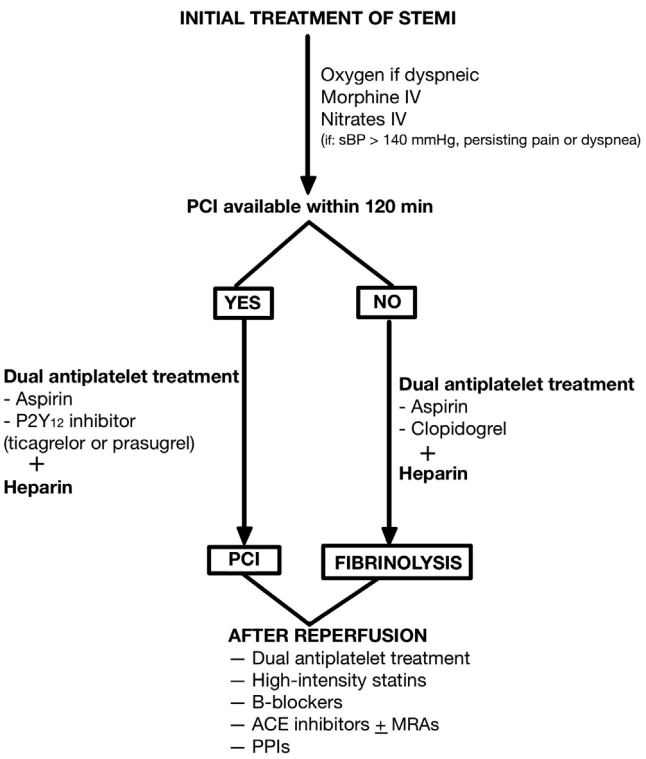

Figure 2: Initial treatment for ST-segment elevation myocardial infarction. A summary diagram that shows the actions need to be taken for the treatment of ST-segment elevation myocardial infarction. ACE, angiotensin-converting enzyme; STEMI, ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; IV, intravenous; sBP, systolic blood pressure; PPIs, proton pump inhibitors; MRAs, mineralocorticoid receptor antagonists

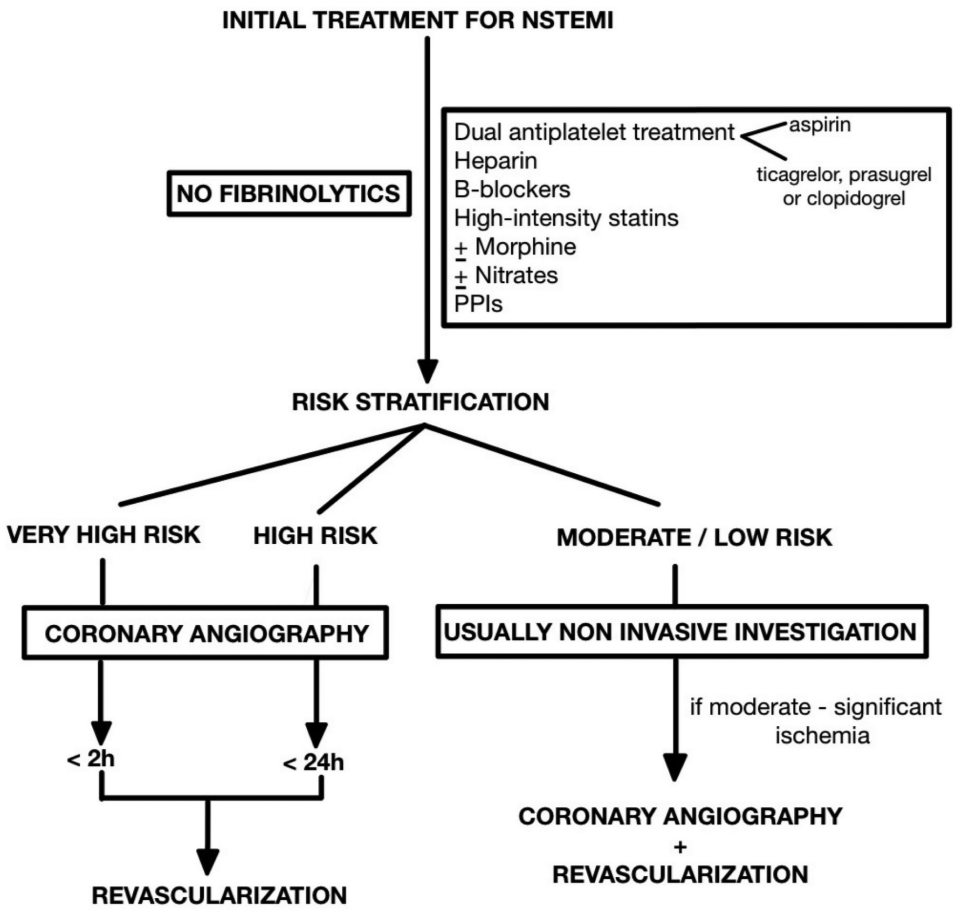

Figure 3: Initial treatment for non-ST-segment elevation myocardial infarction. A summary diagram that shows the actions need to be taken for the treatment of non-ST-segment elevation myocardial infarction. PPIs, proton pump inhibitors; NSTEMI, non-ST-segment elevation myocardial infarction

that β-blockers might cause hypertensive response and coronary artery vasoconstriction via unopposed α-receptor stimulation.[31] However, this has not been confirmed by human studies[32] and several experts recommend the consideration of nonselective β-blockers use in AMI patients after cocaine exposure.[33]

Prevention

The measures for prevention of AMI and in general, of cardiovascular disease (CVD) are almost the same for the general population as for the younger, with only minor differences.

For primary prevention, the latest European Guidelines on CVD prevention encourage individuals to have a healthier lifestyle.[34] This has to include a healthy diet, such as the Mediterranean, that is considered the cornerstone of CVD prevention. It is also advised to reduce the salt intake as it is highly associated with the elevation of blood pressure (BP) and also to increase potassium intake through fruits and vegetables as it has a protective effect over BP. Apart from dietary modifications, individuals are advised to perform at least 150–300 min a week of moderate intensity exercise, i.e. walking at moderate pace, or even better 75–150 min a week of vigorous intensity aerobic activity, i.e. swimming or jogging.

Regarding smoking, individuals are recommended to stop any form of tobacco. It should be emphasized that smoking is more detrimental in young individuals compared to the elderly and it has been shown that it increases by five-fold the risk of developing AMI among young individuals.[35] For this reason, family medicine and primary care physicians should highlight the importance of smoking cessation in their young patients.

For secondary prevention, patients should be educated on the importance of lifestyle interventions for long-term compliance. They should be reminded about it through their family medicine and primary care professionals and access to reliable resources, such as medical brochures and sites with simple information about CVD. It is "obligatory" to abstain from smoking since continuation of smoking is one of the strongest predictors for the recurrence of CAD events. It has been shown that smoking cessation in young AMI patients is the single most effective intervention to improve their long-term prognosis.[36] They should also show adherence to their checkups to monitor the progress of CVD and stay on medications, such as high-intensity statins, aspirin, $P2Y_{12}$ receptor inhibitor (the first year after the event), and, most of them on β-blockers and ACE inhibitors lifelong. These interventions in addition to the adherence to

lifestyle changes are even more mandatory in young patients due to their longer life expectancy compared to elderly patients. Also, cardiac rehabilitation programs are an integral component of secondary prevention. However, patients from low-income countries do not have the same access to healthcare professionals for checkups, reliable information, rehabilitation programs, and drugs compared to high-income countries.[37] It has also been shown that patients with low education level, especially in low- and middle-income countries, have a higher risk of cardiovascular events. This highlights the importance of the communication skills that family medicine and primary care professionals should utilize during their consultations. This will lead to improved patient education and understanding of their current condition [Figure 4].

Conclusions

The incidence of premature AMI is rising over the last decades despite the reduction of AMI observed in older patients. Family medicine and primary care physicians should be able to identify without delay these patients, by being aware of the specific characteristics of the presenting symptoms, to initiate quickly the appropriate treatment. Distinctive risk factors that differentiate young individuals from the general population, such as illicit drug use, and familial lipid disorders should be on the mind of the physicians for a more comprehensive approach. The general population, especially the younger ones, should be educated by their family medicine and primary care practitioners, about the ways of preventing CAD events, through adopting healthy lifestyle habits, i.e. healthy diet, abstain from smoking, avoid excess weight, and regular physical activity.

Highlights

- AMI is a rising threat among young individuals and is characterized by an unfavorable long-term prognosis
- Smoking is the most important modifiable risk factor for premature AMI
- >30% of patients with premature AMI have inherited lipid disorders, such as high cholesterol or high Lp(a) levels
- Consider cocaine or marijuana use among young individuals with AMI and request urine analysis to detect their metabolites
- The most powerful secondary preventive measure of premature AMI is smoking cessation
- Young individuals should be educated to adopt healthy lifestyle habits, abstain from smoking, avoid excess weight, and exercise regularly.

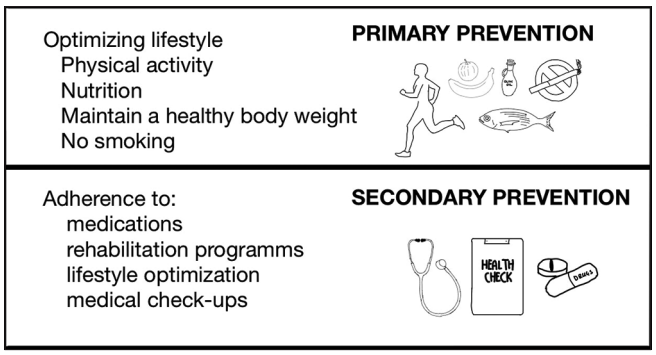

Figure 4: Preventive measures at the individual level. A summary of the ways an individual can intervene in each type of prevention for coronary artery disease

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Conflicts of interest

There are no conflicts of interest.

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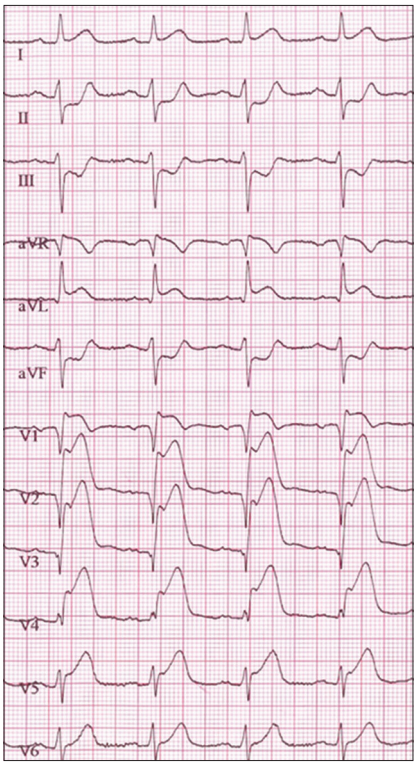
Illustrative cases stories

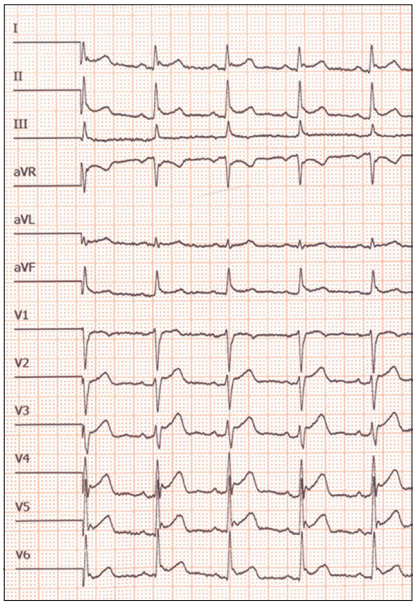
Case 1: A 37-year-old male presented to the medical office complaining of a retrosternal burning sensation. He is a smoker (two packs/day), reported cocaine use 2 h before, and has a family history of premature CAD (lost his father from a heart attack at the age of 48 years). ECG showed ST-segment elevation in V_1-V_5 and aVL leads with mirror ST-segment depression in inferior leads suggestive of an anterolateral STEMI



Key messages

1) Cigarette smoking, cocaine use and a family history of premature CAD expose young individuals to high risk for AMI and when they complain of chest pain, an ECG should always be performed immediately. In young patients with AMI>70% are smokers, ~10% are cocaine and/or marihuana users and ~40% have a family history or premature CAD. In particular, the risk of MI is high in the first hour of cocaine consumption. Note that metabolites of cocaine can be detected in urine for 24–48 h after ingestion.
2) A patient with STEMI carries a significant risk for ventricular fibrillation within the first hours of pain onset. Therefore, before the institution of any therapy, he should be connected to a defibrillator monitor.
3) The diagnosis of STEMI dictates the administration at the medical office of 250 mg nonenteric aspirin (to be chewed and swallowed—exclude prior allergy to aspirin or recent gastrointestinal bleeding) and the transportation of the patient with an ambulance (not with a private car) as soon as possible to a PCI-capable hospital to have a PCI. A prerequisite for this is to arrive to the hospital<120 min.
4) If a PCI-capable hospital cannot be reached<120 min, the patient should receive fibrinolysis. Prehospital fibrinolysis should be considered if paramedics or prehospital providers are appropriately trained. Note that the main absolute contraindications for fibrinolysis are: history of intracranial hemorrhage, ischemic stroke or significant closed-head/facial trauma within the previous 3 months, active bleeding (excluding menses), and suspicion of aortic dissection.

Case 2: A 22-year-old male presented to the medical office with a retrosternal sharp pain that deteriorates with inspiration. He does not smoke but had symptoms of viral infection for the last 2 days. Heart auscultation was normal. ECG showed concave ST-segment elevation in I, II, aVF, V_2–V_6 suggestive of acute pericarditis. There was also PR-segment depression more evident in I, II, V_2–V_4



Key messages

1) In most cases, acute pericarditis complicates a viral infection, particularly in young individuals.
2) Heart auscultation reveals the pericardial friction rub in ~30% of cases. Therefore, the absence of pericardial rub does not exclude the diagnosis.
3) ST-segment elevation in acute pericarditis is typically concave upward (saddle-shaped) and widespread (almost in all leads except for aVR and V_1). PR-segment depression is a less frequent but more specific finding.
4) If acute pericarditis is suspected based on the clinical presentation and ECG, as in the presented case, the patient should be advised to visit a medical center to have

—basic biochemical profile, i.e. full blood count, C-reactive protein (elevation supports the diagnosis), and high-sensitivity cardiac troponin (to exclude myocardial involvement, i.e. myopericarditis) measurements. Note that if cardiac troponin is elevated the patient should be admitted for hospitalization

—echocardiography to assess the presence and the amount of pericardial fluid. Note that in ~50% of cases, there is no fluid (dry pericarditis).

5) Reassure the patient that acute pericarditis is a benign disease in most cases.