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Case Report

Acute Myocardial Infarction in a 25-Years-Old Male: Understanding the Risk and Comprehensive Management

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ABSTRACT

Background: Acute coronary syndrome (ACS) in young adults is a rare entity, yet it occurs. Acute Coronary Syndrome (ACS) mainly observed in the older population (>40 years old). Recently, there is an increase of ACS reported in the younger population even without abundant risk factor
Objective: This case report was an attempt to look for the risk factors most prevalent in young patients and its management prior to and during the hospital stay.
Case Presentation: We present a case of a man 25 years old with no history of certain diseases, who suddenly come up with ST elevation myocardial infarct.
Discussion: Young patients have different risk factors, clinical features, and prognoses as compared to elderly patients. The diagnosis of ACS is also often overlooked in this subset of the population
Conclusion: Acute coronary syndrome (ACS) in young adult it constitutes an important problem because of the devastating effect of this disease on the more active lifestyle of young adults.

1. Introduction

Atherosclerotic lesions are now beginning to appear at an early age.[1-3] In a study of 760 young adults, ages 30-34 years who underwent autopsy, atherosclerotic lesions are found in 20% of men and 8% of women.[3] Along with these findings, also revealed that 50% of people under the age of 34 have evidence of atherosclerotic intima.[4] Furthermore, Acute Coronary Syndrome (ACS) mainly observed in the older population (>40 years old). Recently, there is an increase of ACS reported in the younger population even without abundant risk factor. However, the cause of the development of atherosclerosis at a such young age is still under research. It has been suggested that the mechanism involved in the early coronary disease at a young age, is not explained exclusively by the presence of a large number of risk factors.

2. Case Illustration

A 25-year-old man who has no past medical history came with complaints of heavy sensation chest pain while playing football. Chest pain was radiating to the back with a duration of more than 20 minutes, it did not improve with rest. Chest pain was also accompanied by sweating and nausea and vomiting. The patient was then brought by his family to the emergency room of a private hospital. The patient was then diagnosed as acute ST-elevation Myocardial Infarction (STEMI). He was given a standard protocols medication such as a loading dose of asetylsalisilatacid (ASA) 320 mg, a loading dose of clopidogrel 300 mg, and sublingual nitrate. Then, he was referred to our hospital for further management.

Further anamnesis revealed that he had no family history of cardiovascular disease. He was an active smoker but denied the use of drugs or alcohol. Physical examination was within normal limit with no sign of acute heart failure. He was overweight with body mass index 29.8 (Weight: 86 kg, height: 1.7 m. The chest X-Ray showed normal cor and pulmo. Electrocardiogram (ECG) showed sinus rhythm 97bpm, Q wave in lead V1-V6 with ST elevation lead V2-V5 (Figure 1). Cardiac enzymes were measured and provide exceptional presentation for creatine kinase-MB: 377 ng / ml and troponin I: 47.6 ng / ml. Reproducible measurement of cardiac enzymes at 3 hours after the initial presentation and revealed the level of creatine kinase-MB is 261 ng / ml and troponin I of 74.5 mg / ml.

The chest pain was still persisted, and we immediately performed primary percutaneous coronary intervention (PPCI). The result showed total occlusion in osteal left anterior descending (LAD), TIMI flow was 0, other coronary arteries showed non-significant 40% stenosis in the proximal Right Coronary Artery (RCA) and 30% stenosis at distal Left Circumflex (LCx) artery. A Drug eluting stent (DES) was implanted at osteal-distal LAD. The final angiographic evaluation showed TIMI III flow and no residual stenosis (Figure 2).

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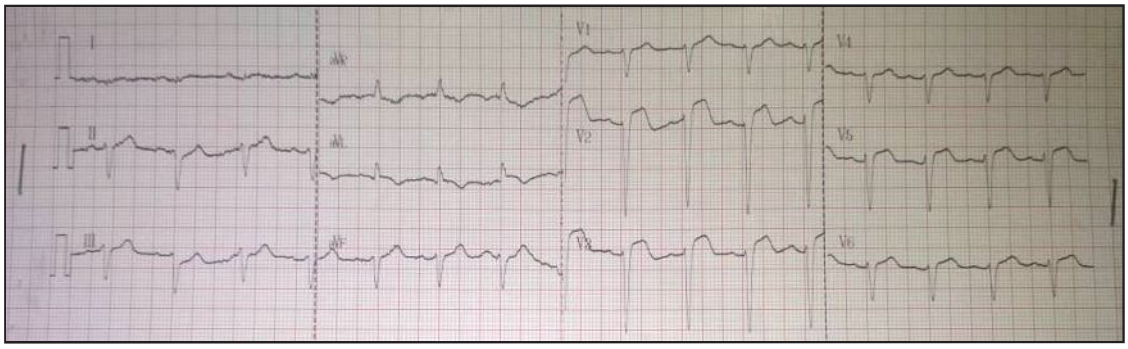
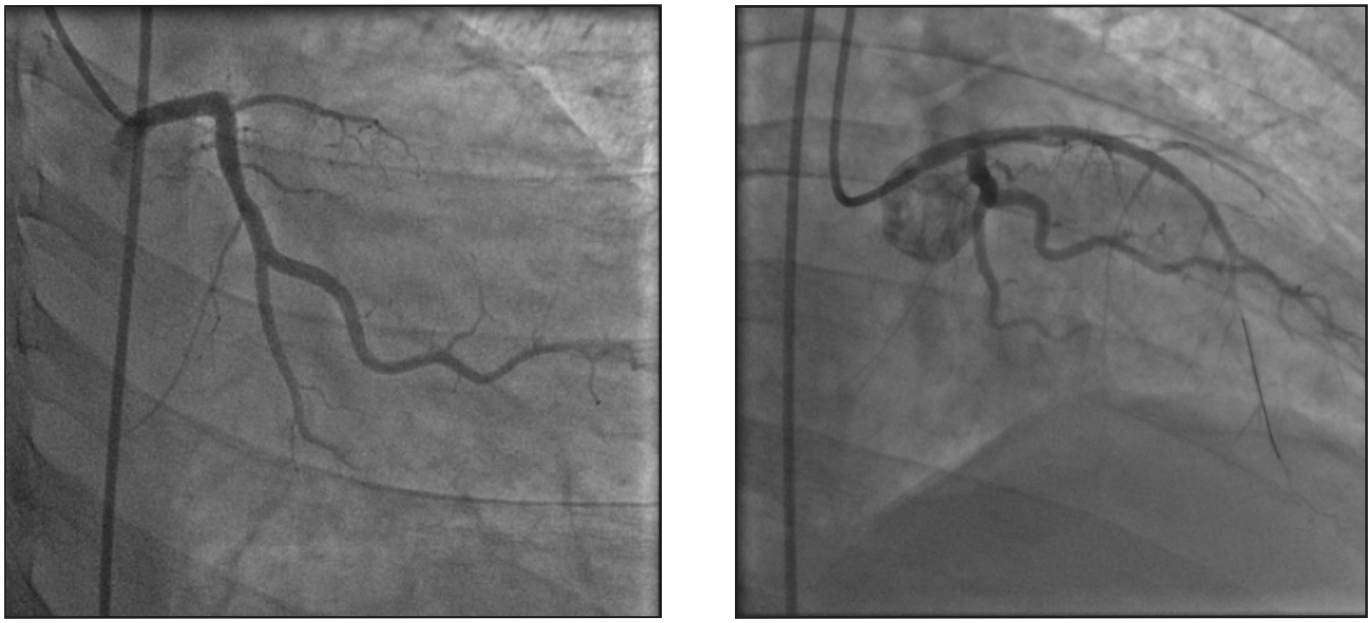


Figure 1. Initial electrocardiogram, showing evidence of acute inferior myocardial infarction

Then, patient was clinically stable, he had no chest pain nor dyspnea. Afterward, he was treated with Ticagelor 90 mg/12 hours, ASA 80 mg/24 hours, Atorvastatin 40 mg/24 hours, Laxative 10 ml/24 hours as daily medicine. ACE inhibitor and beta blocker was also initiated. ECG showed no evolution. Laboratory result showed normal lipid profile (Triglyceride 97mg/dl, Total Cholesterol 137 mg/dl, high-density lipoprotein 37 mg/dl and low-density lipoprotein 107 mg/dl), and normal fasting glucose result (Glucose 89mg/dl)

Echocardiography showed decreased contractility left ventricle (LV) function with ejection fraction (EF) 43% (Biplane), with akinetic in anteroseptal and LV apex, hypokinetic at mid anterior and anteroseptal, and normal valves. He remained in the hospital for three days after stent placement and experienced no further chest pain. He was discharged with prescriptions for double antiplatelet agent, an ACE inhibitor, a beta blocker, and a high intensity statin, and he agreed to stop smoking.



(A) (B)

Figure 1. A) Diagnostic Coro angiography revealed acute total occlusion at ostial LAD (arrow). B) Angiographic evaluation after DES implantation at ostial-distal LAD show TIMI Flow 3.

4. Discussion

According to recent data, between 2 and 10% of all acute myocardial infarction patients hospitalized were under the age of 455. An autopsy found that 50% of people under the age of 34 had atherosclerotic intima. Young age patients have different risk factor profile, clinical presentation, pattern of coronary artery involvement and clinical outcome than older patients. We will discuss in following sections.

4.1 Risk Factor of ACS in Young Population

Several studies show that young age patients have different risk factor profile compared to older population. A prospective case-control study of 100 patients with ACS younger than 40 years old declare that the most prevalent risk factor was obesity; as 86 patients were either overweight or obese. The second most prevalent risk factor was smoking (62%)6. Hypertension and DM were found in nearly one-quarter of patients each while family history of ACS was positive in 24% of patients. This data shows different from older population, as in this population, the most prevalent risk factor was hypertension and diabetes mellitus. Another study from India concluded that Acute Coronary Syndrome in people younger than 40 years showed a significant male predominance with a sedentary lifestyle being a major modifiable risk factor, with tobacco smoking was the second highest.[7]

Some data suggest smoking may be the most important modifiable risk factor among young Myocardial Infarction (MI) patients.[8] Yusuf et al. identified it as one of the most important risk

factors associated with young MI9. They suggested the association of smoking and MI in the young has an odds ratio (OR) of 3.33 (99% confidence interval (CI), 2.86-3.87) compared to controls9. This was significantly higher than older individuals (OR 2.44: 99% CI, 2.86-3.87). In comparison to older patients, young MI patients smoked a greater number of cigarettes per day but had a lower pack year history as expected due to their younger age.[8] Of young MI patients presenting with ST-elevation myocardial infarction (STEMI), the rate of smoking was found to be highest among the youngest.

The results of these studies are in accordance with our patient. Our patient was overweight, and he was an active smoker. There was no other risk factor we found in our patient. Obesity is itself an independent risk factor for ACS in both men and women. Obese individuals have a higher incidence of cardiovascular risk factors, such as hypertension, dyslipidemia and DM. Therefore, this group of patients has higher morbidity and mortality associated with diseases of the cardiovascular system. In regard to obesity, the guidelines recommend an ideal BMI of 25 kg/m2 and suggest a reduction in body weight if BMI > 30 kg/m2 or when waist circumference is >102 cm for men and >88 cm for women.[6]

Dyslipidemia is one of the major modifiable ischemic heart disease risk factors. Our patient had high level of LDL (107 mg/dl). Akhtar et al reported dyslipidemia in their 63.2% of patients. Sedentary lifestyle seems to be contributing to the increased prevalence of dyslipidemia the young population. A meta-analysis of 16 prospective epidemiologic studies suggests that an elevated triglyceride concentration is an independent risk factor for coronary artery disease. So, we may infer that high triglyceride level, which may be the primary lipid abnormality with or without low HDL levels and normal/low LDL levels is a matter of concern in young adults.[10]

Our patient also an active cigarette smoker. Study by Mirza et al found that among Acute myocardial infarction patients 90%6. Smoking was the leading risk factor (65%) as seen in the study by Yusuf et al.[9] All this evidence points out smoking as an important modifiable risk factor and preventable cause of CAD in young adults.

4.2 Clinical presentation of ACS in Young Population

The clinical presentation of acute myocardial infarction in younger populations may differ from that in older patients. Although chest pain was present in our patient, a staggering high proportion of young patients do not experience angina pectoris, and frequently the first manifestation of coronary artery disease is acute myocardial infarction. Klein et al[11] found that young patients seldom experience angina pectoris before myocardial infarction, but that angina pectoris quickly progresses to acute myocardial infarction. In recent studies, only 12% of young elevation myocardial infarction patients experienced angina pectoris before ST elevation myocardial infarction, a significantly smaller percentage than in old ST elevation myocardial infarction patients. ST elevation myocardial infarction in young patients generally has no ischaemic pre-conditioning and occurs and progresses faster than it does in older patients. Fournier et al[12] demonstrated that acute myocardial infarction and angina pectoris were the first ischaemic manifestations in 48 and 26% of all patients in this subset of the population. This emphasizes the increased importance for prescreening, given the high risk of developing unpredictable and morbid events.

Up to two-thirds of young MI patients will present with non-ST elevation myocardial infarction (NSTEMI) with approximately a third presenting with STEMI. It appears that, overall, the incidence of STEMI is reducing among the young but the proportion of young patients diagnosed with STEMI is increasing.[13] Most young MI patients do not report a history of previous angina, MI or congestive heart failure and they report this less frequently in their histories than their older counterparts. Egiziano et al. reported only about 25% of young MI patients complained of chest pain in the month prior to their acute presentation for MI. The rate was even lower among young women.[14] By way of comparison, in a study of all-comers with MI, chest pain was reported among two thirds of patients and those presenting with chest pain had a median age of 67 years.[15]

Coronary angiography usually reveals less extensive disease in young MI patients than older patients. Zimmerman et al. reported normal coronary arteries in 16% of men and 21% of women. By comparison only 2% of older men and 11% of older women had normal coronary arteries.[16] Three vessel disease is infrequent with Fournier et al. reporting it in less than 10% of young MI patients.[12] In that study there was no report of left main coronary artery stenosis in young MI patients which is supported by other authors. Single vessel disease is more frequent among young MI patients compared to their older counterparts and the left anterior descending artery is most commonly affected.[16] Spontaneous coronary artery dissection is not an infrequent finding at angiography in young MI patients. Tweet et al. described the occurrence of spontaneous coronary artery dissection in a group of Myocardial Infarction in the "young" patients with a mean age of 43 years, who were mostly female.[17] Approximately 50% of these patients presented with STEMI and conservative management was associated with an uncomplicated in-hospital course.[17]

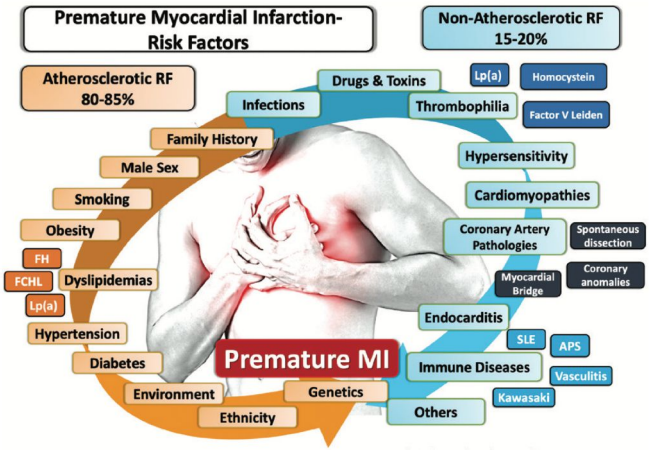


Figure 3. Risk factors of Premature CAD in Young Patients.

4.3 Precipitating Factor

Our patient felt sudden chest pain while he was playing football. Vigorous physical activity could lead to acute Myocardial Infarction (MI) and Sudden Cardiac Death (SCD) in susceptible populations. In general, physical activity can be categorized according to metabolic equivalent (METs). METs is a useful, convenient and standardized way to describe the absolute intensity of a variety of physical activities. Intensity of physical activity could be divided according to the METs category into light intensity (2.0-2.9), moderate (3.0 - 5.9), and vigorous (≥ 6.0)18. Depending on the various previous experience and the fitness level of each individual, the intensity of different forms of physical activity might also vary.

Vigorous exercise is defined as an absolute exercise work rate for at least 6 metabolic equivalent (METs).[19] Vigorous exercise could increase the risk of cardiovascular events during or immediately after exercise and it happens frequently in the afternoon or in early evening. Vigorous-intensity-exercise or high-intensity- exercise is a physical activity which requires a large amount of effort resulting in a higher heart rate, and rapid breathing, such as jogging or running.[19]

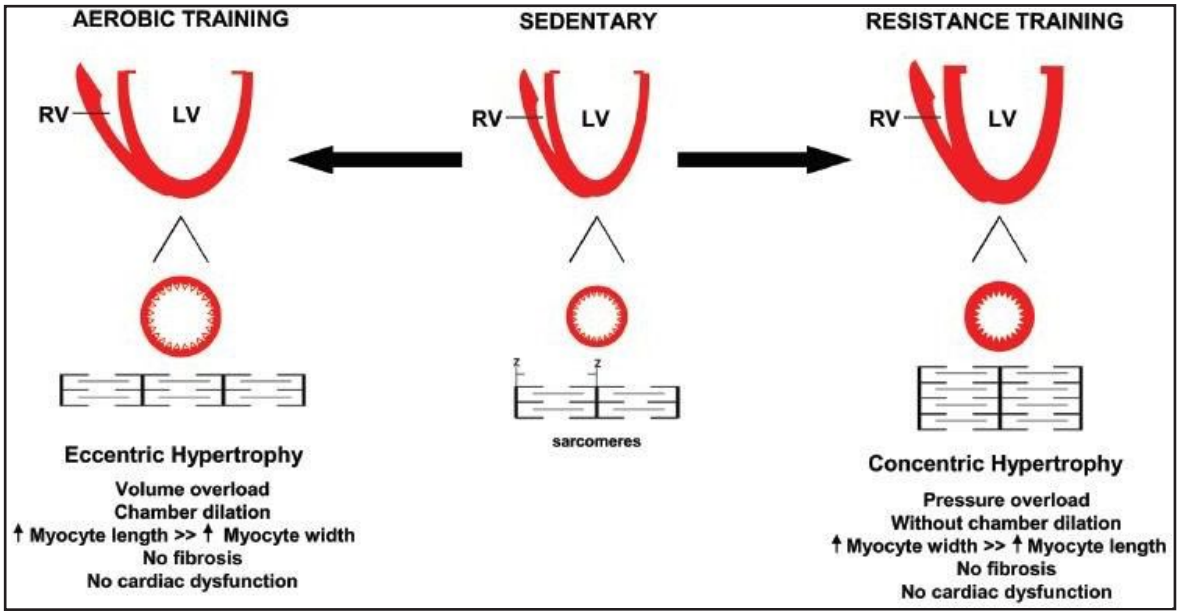


Figure 4. Cardiac Remodelling due to particular training.[26]

It is well known that physical activity is important to reduce the risk of cardiovascular disease. The American Collage of Sport Medicine – American Heart Association (ACSM-AHA) Primary Physical Activity Recommend that all healthy adult age 18 to 65 years old should participate in moderate to high intensity aerobic activity for a minimum of 20 minutes on 3 days a week or moderate intensity aerobic Physical Activity for a minimum of 30 min on 5 day a week.[20] Nevertheless, vigorous physical activity also have a potential decremental effect. Some studies estimated that an individual's risk of AMI onset was transiently increased 5-fold within 2 hours of an episode of vigorous exercise (METs ≥ 6) and 1.6-fold within 2 hours of moderate exercise compared lower levels of exercise. Interestingly, compare with indoors activity, strenuous outdoor activity was more associated with an increase of AMI symptom onset. Vigorous intensity exercise has a small but measurable acute risk of cardiovascular complication. Therefore, it is important to mitigate this risk in susceptible individuals.[21]

The exact mechanism in which vigorous activity led to AMI was poorly understood. But it is thought to be a triggering mechanism including increased wall stress, coronary spasm, the thrombotic occlusion. The increase of wall stress is causing an increase of the heart rate and blood pressure. Vigorous exercise could also trigger the coronary spasm of the diseased artery segment and increase the flexing of atherosclerotic epicardial coronary arteries which lead disruption of plaque and thrombotic occlusion.[22] The other mechanism is thrombosis by deepening existing coronary fissure, augmenting catecholamine-induced platelet aggregation. Mildly fissure coronary plaque requires some exacerbating event to induce coronary thrombosis not despite an increases of myocardial oxygen demand.[23] Vigorous exercise can also induce simultaneously shortening diastole and coronary perfusion time resulting in myocardial ischemia.[24]

La Gerce et al. found that youngster who had a greater prior exposure to vigorous exercise will develop a fibrosis and scarring in the interventricular septum. Cardiac-MRI can show the patchy scanning and myocardial fibrosis. Myocardial fibrosis is a result of the extreme training with more than five-years of continuous intense training.[25]

In some studies, vigorous training could also lead to cardiac hypertrophy. The physical training defines into dynamic and static. Both of them lead into two different kinds of chronic cardiac pressure that can induces morphological changes of the heart, such as eccentric and concentric physiological cardiac hypertrophy.[26]

A dynamic or endurance training such as swimming, running, cycling (or any other aerobic exercise) changes the hemodynamic by increasing heart rate and stroke volume. Furthermore, endurance exercises increase the skeletal muscle pump and venous return to the heart resulting eccentric left ventricular hypertrophy due to overload of volume. It is characterized by an increase of cardiac cell length and mass with increased chamber volume.[27] Based on Mc-Ardle, et al., aerobic exercise leads an increase of the average cardiac output from 5 litres per minute to a maximum of 35 litres per minute. This change put a strain on the heart causing the dilatation of right ventricle (RV) and right atrium (RA).[27]

On the other hand, the static training (e.g., Bodybuilding, wrestling, hammer throwing, and weight lifting) with no or less movement can develop the strength. This type of activities is also called resistance training which is purposed to strengthen power and muscle. Both cardiac and skeletal muscle has a response to this type of training. This type of training causes an elevation of blood pressure leading to pressure overload in the heart. This training also increase the thickness of left ventricular without reducing the internal cavity's diameter in diastole (concentric hypertrophy).[26]

Interestingly, not all people who exercise vigorously lead to myocardial infarction. It is presumed that cardiovascular risk factors play a part in this matter.[28] The most important risk factor observed and reported in the young adult with MI was cigarette smoking.[29] It is proven that smokers are more susceptible to myocardial infarction compared to non-smokers.[30] It was concluded based on the fact that cigarette smoking has a role in the development of atherosclerosis and vascular spasm by increasing the number of mLDL (which is the key to atherosclerosis formation), platelet aggregation, endothelial dysfunction, inflammation and decreasing number of Nitric Oxide (NO).[31]

4.4 Management

The management of MI generally is not dependent on age and guideline-suggested therapies are just as applicable to younger patients as they are to their older counterparts.[32-33] With respect to

STEMI management, the benefits of primary angioplasty over thrombolysis are as applicable in young patients as they are in older individuals and no particular age cohort has a greater relative benefit.[34] Young age is an independent predictor for favourable prognosis following thrombolysis.[35] and hence thrombolysis should still be utilised where timely primary angioplasty cannot be offered. Given the longer expected survival of younger patients, the rate of repeat revascularisation would be expected to be high. One study suggests a rate of about 50% at a median of 4.7 years.[36]

In addition to medical treatment of acute events, risk factor modification is of utmost importance in any patient post-MI. As highlighted above, smoking is one of the most important modifiable risk factors among young MI patients; addressing this may yield the highest reward. Critchley et al. studied the benefit of smoking cessation in patients with CHD in a systematic review. They report a 36% reduction in crude relative risk of mortality for patients with CHD who quit smoking compared to those who continued to smoke (relative risk 0.64: 95% CI, 0.58-0.71).[37] This benefit did not appear to be affected by age37. Recurrent coronary events also appear to be reduced by smoking cessation. Rea et al. demonstrated a relative risk of 1.51 (95% CI, 1.10-2.07) for recurrent coronary events among continued smokers compared to non-smokers.[38] Smoking cessation is a difficult task for patients and healthcare professionals. It often requires multiple strategies including counseling, personalized prescription and management of co-occurring mental health conditions.[39]

4.5 Prognosis

In-hospital and short-term outcomes are generally favourable in young MI patients. In-hospital and six-month mortality has been shown to be 0.7% and 3.1%, respectively.[40] This compares favourably to their older counterparts who's in-hospital and six-month mortality were 8.3% and 12%, respectively.[40] There is significant reduction of health-related quality of life post MI in young MI patients. Depression is common after MI41 with Denollet et al. reporting post-MI depressive symptoms in approximately 47% of patients with a mean age of 54 years.[42] Hence, identifying and managing depression following MI in young patients is important. Angina is also a significant contributor to lower health-related quality of life post MI but it appears improving control of angina leads to greater improvement in health-related quality of life in older patients only.[43] Heart failure is a potentially debilitating complication of MI in young patients. They are often at the peak of their productive lives and may have multiple dependents. Heart failure is also an important predictor for long-term prognosis.

5. Conclusion

This case demonstrates the significance of including acute myocardial infarction in the differential diagnosis for young population with acute chest pain. Smoking was a risk factor for this young man's sudden myocardial infarction. However, risk factor screening recommendations and knowledge of the illness mechanisms and progression in this demographic together move us closer to the objective of making a diagnosis on the young population. Regardless of age, people experiencing chest pain should be evaluated for acute myocardial infarction. Exercise is one of the recommended activities to prevent coronary heart disease. But, exercise vigorously or too much could also lead to myocardial infarction which could happen even in healthy young adults. While coronary revascularization is still the mainstay for definitive therapy, it is also important to address cardiovascular risk factors such as overweight, dyslipidemia and cigarette smoking that have a role as catalyst in the development of acute myocardial infarction.

6. Declarations

6.1. Ethics Approval and Consent to participate
Not applicable

6.2. Consent for publication
Not applicable.

6.3. Availability of data and materials
Data used in our study were presented in the main text.

6.4. Competing interests
Not applicable.

6.5. Funding source
Not applicable.

6.6. Authors contributions
Idea/concept: AG, CT. Design: AG, CT. Control/supervision: CT, SW, ER. Data collection/processing: AG, CT. Analysis/interpretation: AG, CT. Literature review: CT, SW, ER. Writing the article: AG, CT. Critical review: CT, SW, ER. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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