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ORIGINAL ARTICLE

The Association between Cardiovascular Risk Factors and Angiographic Patterns in Young Patients with Myocardial Infarction

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ABSTRACT

Acute myocardial infarction (AMI) is a leading cause of mortality. Although AMI is less common in persons under 45 years old than in older adults, young individuals have been become clinically interesting due to the risk of long term damage and death. Young individuals had an incidence of AMI as low as 2-6%, but it has recently started to grow. The mechanism and disease course of AMI in young people are probably different from that in old population. The protection offered by young age is countered by increased prevalence of risk factors for coronary artery disease (CAD), such as impaired glucose tolerance and obesity, in adolescence. Identifying and controlling cardiovascular risk factors at an early age may prevent cases of young myocardial infarction (MI). This study aims to evaluate the Risk factors during acute Myocardial infarction in young individuals. Conclusion: Smoking, obesity, dyslipidemia, and hypertension were the most common risk factors for acute MI. Smoking was the most common risk factor in males **Keywords:** AMI, risk factors; Young patients

INTRODUCTION

The primary cause of death globally is cardiovascular illnesses (CVD), which mostly comprise rheumatic heart disease (RHD), cardiomyopathy, and coronary heart disease (CHD). When the 20th century began, less than 10% of deaths globally were attributable to cardiovascular disease (CVD); by 2001, that number had risen to 30%. 80% of deaths in nations with low and intermediate incomes are attributable to CVD. By 2020, CVD became the leading cause of death and disability in low and middle-income countries. In a year, mortality of CVD accounts ~ 9% **[1].**

Cardiovascular disease (CVD) is a broad category of disorders that includes diseases of the vascular and cardiac muscles. Diabetes, high low-density lipoprotein cholesterol, tobacco use, physical inactivity, hypertension, and a number of linked metabolic risk factors are potential risk factors for CVD. The idea of risk factors, which connects the existence of high cholesterol, tobacco use, hypertension, and diabetes mellitus to potential CVD, was initially presented by the Framingham Heart Study in 1961. **[2].**

The role of traditional risk factors for MI in young patients:

Tobacco:

Compared to older MI patients, younger patients have a higher likelihood of currently smoking (80% vs. 57%). In young individuals with ST-segment elevation MI, smoking is very common. There is a dose-effect relationship between smoking and MI; patients who smoked more than 25 cigarettes per day had eight times the risk of MI compared to non-smokers. Compared to older first-time MI patients, younger patients smoked cigarettes and used water pipes more frequently. Since it can amplify coronary vasoconstriction, smokeless tobacco has been linked to decreased levels of high-density lipoprotein (HDL) and greater levels of total cholesterol, making it potentially atherogenic and thrombogenic **[3].**

There is still much to learn about the precise mechanism linking cigarette smoking to cardiovascular disease. It is highly recommended that smoking has two effects on platelets: (i) a major acute potentiation of platelet activation that happens soon after a cigarette is smoked, and (ii) a chronic cell desensitization to activating agents that happens in between cigarettes and causes Type 1 MI **[4].**

Dyslipidemia:

Dyslipidemia is an established MI risk factor among all age groups MI risk factor for people of all ages is dyslipidemia. In survivors of premature MI, Hovingh et al. found a high frequency of familial-combined hyperlipidemia (FCHL) at 10%, whereas levels of low-density lipoprotein (LDL-C) remained >70 mg/dL regardless of statin usage. To be more precise, FCHL was linked to an adjusted risk of MI that was 24 times higher, with non-HDL and very low-density lipoproteins acting as aggravating factors for MI incidence**[5].**

Patients with high levels of LDL-C (60% prevalence in "young" MIs) and/or a family history of premature CAD (20% prevalence in young MIs) are more likely to have FCHL. The incidence of Type 1 MI is more strongly correlated with dyslipidemia in older adults (43% vs. 36%) as compared to younger adults **[6].**

However, cross-sectional investigations discovered that while HDL-C levels were lower, triglyceride, LDL-C, and apolipoprotein B levels were considerably greater in younger MI patients compared to older MI patients. Teenagers whose parents experienced early MI have higher levels of lipoprotein-a [Lp(a)]. Similarly, it has been reported that a high level of Lp(a) is an independent risk factor for MI across all age groups **[7] Figure 1.**

For people under 45 Years old, high levels of Lp(a) triple the risk of MI; the relationship is less pronounced for those between 45 and 60 years of age. A rise in results of 10 mg/dL was linked to a 4% increased relative risk of Type 1 MI at a younger age (less than 45 years old) and a 2% increased risk in middle age (45–60 years old) **[9] Figure 2**.

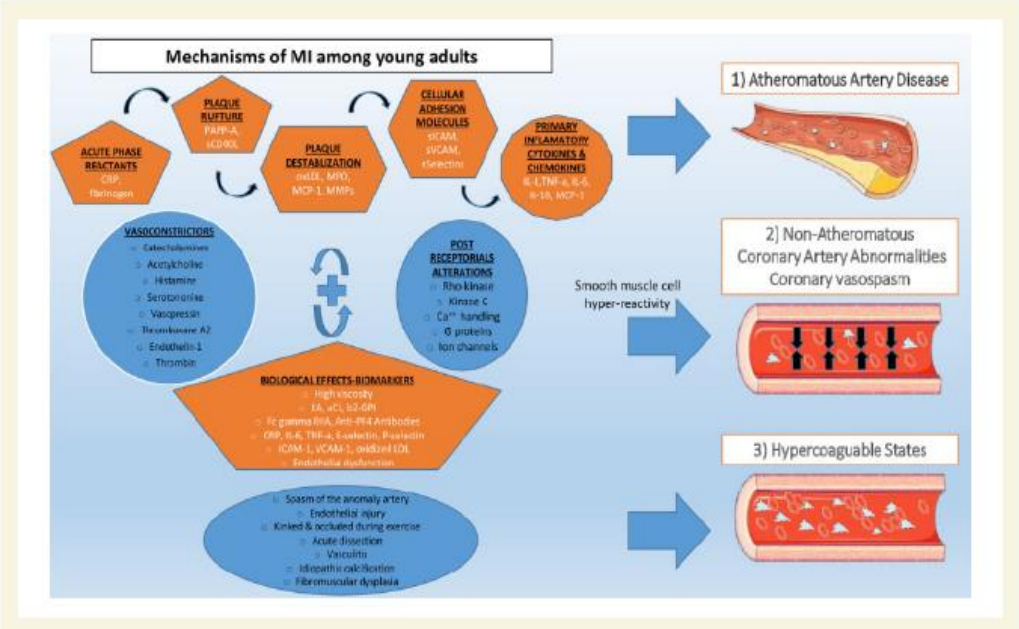

Figure 1: Pathophysiology of MI among young adults. MI, myocardial infarction; C-reactive protein (CRP), PAPP-A, pregnancy-associated plasma pro-tein-A; sCD40L, soluble CD40 ligand; oxLDL, oxidized low-density lipoprotein; MPO, myeloperoxidase; MCP-1, monocyte chemoattractant protein-1;MMPs, matrix metalloproteinases; sICAM, circulating soluble intercellular adhesion molecule; sVCAM, circulating soluble vascular cell adhesionmolecule;IL-1/IL-6/IL-18, interleukin-1/-6/-18; TNF-a, tumor necrosis factor-a; LA, lupus anti-coagulant; aCL, anticardiolipin antibodies; Anti-b2–GPI, anti-beta2-glycoprotein I; Fc gamma RIIA, Fc gamma RIIA/CD32a recombinant protein antigen; Anti-PF4, anti-platelet factor 4 **[8].**

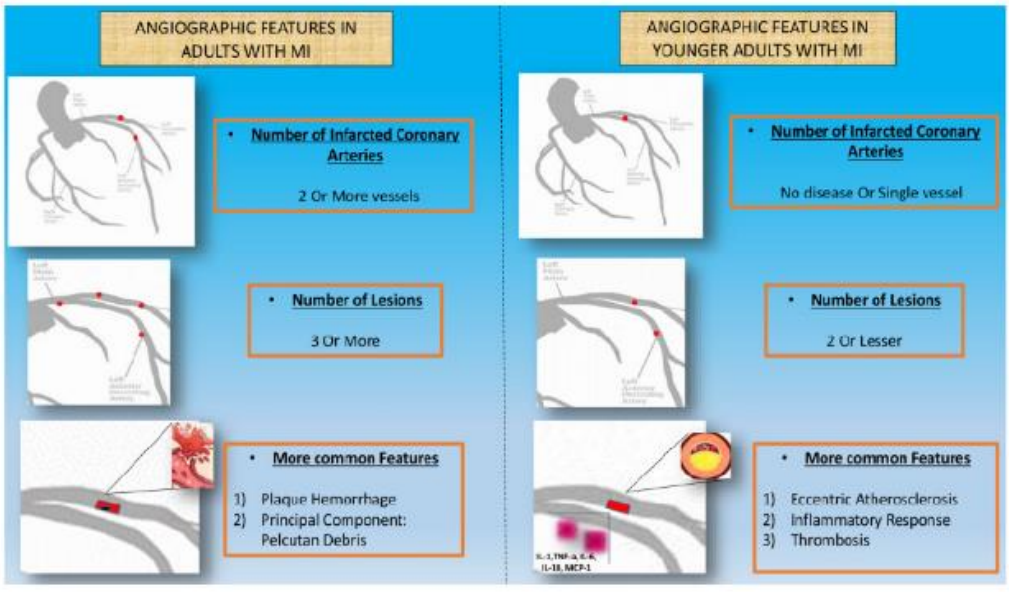

Figure 2: Comparison of angiographic features between young and elderly patients. IL-1/IL-6/IL-18, interleukin-1/-6/-18; TNF-a, tumour necrosis factor-a; MCP-1, monocyte chemoattractant protein-1 **[8].**

DISCUSSION

Obesity:

The relationship between healthy lifestyle factors (HLFs) and MI in younger patients was examined by Liu et al. These HLFs included (i) an average body mass index of less than 25 kg/m^2, (ii) no or moderate alcohol consumption, (iii) a higher score for a healthy diet, (iv) a higher score for physical activity, and (v) never smoking. For those with 0–1, 2, 3, 4, and 5 HLFs, the prevalence of CAD in combination with age, sex, and race was 3.0%, 14.6%, 29.5%, 39.2%, and 60.7%, respectively, with comparable graded associations being seen for each sex-race group. Younger MI patients had a higher body mass index (31 kg/m2 vs. 29 kg/m2) and were more likely to be male than older (>45) patients **[9].**

Adipocyte enlargement, reduced blood supply to adipose tissue, changed oxygen levels in the tissue, a persistent low-grade inflammatory state, and impaired lipid metabolism are the components of adipopathia. The incidence of early Type 1 MI is expected to rise in tandem with rising obesity rates, necessitating more study on the impact of adiposopathy in juvenile populations**[10].**

Sex:

Young male MI patients predominate. Young women tend to come with more unusual symptoms and fewer incidences of STEMI, while men tend to have higher levels of cardiac biomarkers and more classic ECG findings. In particular, female patients reported experiencing palpitations, dyspnea, and epigastric discomfort more frequently than male patients did when they presented with chest pain among MI patients. Furthermore, female patients with acute MI may have a poorer outcome if they present to the hospital later than expected because female soften mistake their anginal symptoms for elevated anxiety **[11].**

According to recent data, the prevalence of MI in young women appears to be on the rise. A considerable proportion of STEMI cases are caused by spontaneous coronary artery dissection**[12].**In addition to having greater rates of anxiety and changed mental and physical condition, young women with MI are also more likely to have congestive heart failure, chronic obstructive pulmonary disease, morbid obesity, diabetes, hypertension, and renal failure **[13].**

When it comes to shifts in patterns, smoking among young women has gradually decreased while the prevalence of hypertension has increased **[11].** It has been proposed that women's natural estrogen status prior to menopause has a protective effect, as they encounter their first MI 6–10 years later than men. A less atherogenic lipid profile and a better distribution of fat have been linked to female sex hormones **[14].**

There is a need for more study since some studies attempt to determine the protective function of estrogen on the cardiovascular system **[15].**There are significant differences between the sexes when it comes to management. In particular, regardless of the type of MI, young women are less likely than their male counterparts to undergo a nearly invasive strategy, primary percutaneous coronary intervention or CABG, even though improvements are seen with time **[16].**

When compared to male patients, this results in a worse outcome in terms of significant bleeding, vascular complications, and in-hospital death. It's interesting to note that, compared to older men, females who grew older had better results.

Diabetes mellitus:

DM is linked to an increased risk of MI in both sexes, despite being uncommon in younger MI patients. Among older patients, the prevalence of diabetes, hypertension, dyslipidemia, and prior MI was higher (37%,

60%, 43%, and 42%, respectively), compared to 10%, 24%, 36%, and 25% in the younger population **[17].**

Age of onset and gender are significant predictors of survival and MI outcomes in young participants with Type 1 DM. In particular, the hazard ratio for women who got Type 1DM before the age of ten was 91.07, while the comparable hazard ratio for men was 15.11. Though the lowest hazard ratio for MI was found in women with Type 1 diabetes whose condition began between the ages of 26 and 30, these differences were less pronounced as the age of onset increased **[18, 19].**

Tight glycemic control may be crucial for the prevention of cardiovascular disease, as shown by the activity of glycosylated hemoglobin A1c (HbA1c), which is defined as an independent factor for microvascular perfusion and may account for the aforementioned finding**[20, 21].**

Risk factors for MI more specific at a younger age:

Anabolics and stimulants:

One of the less frequent risk factors for MI is substance misuse, including abuse of drugs like cocaine and cannabis **[22].** Particularly for cannabis, the most often consumed drug, it has been found to be associated with MI incidents without regard to conventional cardiovascular risk factors; this association is stronger in younger patients **[23].**

Similarly, cocaine use has been linked to a higher risk of cardiovascular death and is documented in 10% of MIs in their early years. The Type 2 MI criteria are met by the underlying mechanisms, which include elevated myocardial oxygen demand, decreased peripheral vascular reflex response, and coronary artery vasospasm. Unlike cannabis, cocaine does not have a dose-dependent risk of MI. Furthermore, both professional and recreational sportsmen frequently use stimulants, primarily androgenic anabolic steroids (AASs), with excessive dosages having a variety of negative effects. Due to a substantial increase in LDL-C and apolipoprotein B (up to 20%) and a decrease in HDL-C and apolipoprotein A1 (by 20–70%), AASs raise the risk of MI in young patients. In addition, prolonged usage of AASs may result in the development of hypertension and elevated C-reactive protein levels **[24].**

Erythrocytosis (a 9.6% rise in hematocrit during 26 weeks of usage), thrombocytosis, and platelet hyperactivity are the primary causes of thrombosisAdditionally, AAS raise homocysteine levels, pro-coagulant factors (particularly fibrinogen, factor VIII, and X), and endothelium release of proteins C and S. Their pro-thrombotic action is further enhanced by prostacyclin synthesis and decreased fibrinolytic activity, which includes decreased levels of a-2-macroglobulin and plasminogen activator inhibitor 1 as well as increased levels of tPA and plasminogen **[25].**

Derivatives of erythropoietin, known as erythropoiesis-stimulating agents (ESAs), have been extensively employed as performance-enhancing medications. Blood transfusion in the form of homologous or autologous injection, erythropoietin and analogues, and synthetic biomaterial particles that imitate red blood cells can all be used to induce erythrocytosis **[26].**Although there is little information available about ESAs and CAD and MI, it is hypothesized that using them in conjunction with physical activity-induced dehydration may increase the risk of adverse cardiovascular events.As per the Type 1 MI criteria **[27],** the use of AASs and ESAs can cause acute coronary atherothrombosis through numerous mechanisms.

Thrombotic/fibrinolytic factors:

The interactions between fibrinolytic and thrombolytic pathways are specific and intricate. High concentrations of factor V or

VII in serum plasma have been linked to an increased risk of MI, as demonstrated by Maor et al. the risk increased by 50 -fold when smoking or arterial hypertension were present. Premature MI Type 1 has been linked to factor V Leiden, the most prevalent inherited hypercoagulability factor; young MI survivors have also been linked to a greater activity of factor XIII **[28].**

Furthermore, factor XI is acknowledged as a separate risk factor for MI since it has the ability to inhibit the anti-coagulant tissue factor pathway inhibitor and activate coagulation factors X, V, and VIII **[29].**An unfavorable lipidaemic profile may be linked to the increased risk associated with these hypercoagulable conditions, as evidenced by aberrant Lp(a) levels in a group of patients with antiphospholipid syndrome **[30].**

Additional risk factors for MI were also shown to include the Factor II 20210A allele, the Factor XIII-ALeu34 variant, and their combined effect. In the context of stable CAD, a recent meta-analysis revealed the detrimental influence of hypercoagulable states with prior MI without such a connection **[31].**

Increased oxidative damage results from hyperhomocysteinemia's production of pro-inflammatory cytokines, such as intracellular adhesion molecule-1, tumor necrosis factor-a, interleukin-1band -6, and monocyte chemoattractant protein 1 **[32].**The relationship between homocysteine levels and the occurrence of CAD is not entirely clear, as reports on the subject vary. Several investigations have demonstrated the link between hyperhomocysteinaemia and an elevated risk of MI, designating it as a separate risk factor and potential indicator of a preclinical disease state. Younger infarcted patients had greater homocysteine levels than elderly patients, according to certain investigations **[25].**

Genetic factors:

There is compelling evidence linking a patient's genetic history and early-life CAD. The problem is complicated by the non-Mendelian inheritance of CAD and MI **[33].**

A 2017 study that looked at prothrombotic risk variables found that polymorphisms in the prothrombin [FII] gene, namely G20210A, are linked to a higher risk of premature ST-segment elevation MI. Prothrombin (FII), the precursor of thrombin, is a glycoprotein that is dependent on vitamin K. Its main job is to activate factor XIII and change fibrinogen into fibrin, which helps clots become more resistant to fibrinolysis. When the mutation G20210A is expressed, prothrombin levels are slightly elevated. This is because prothrombin is easily converted to thrombin when needed, which makes it more likely that hypercoagulable states would develop**[34].**

A combination of the aforementioned polymorphism with casual smoking increases the risk by 22 times (95% CI: 9.192–66.517). Prothrombin [FII] gene polymorphism G20210A is strongly linked to premature MI, regardless of whether the allele is present in one or two copies **[35].** The polymorphism increases the risk of MI in a manner proportional to age, with younger people incurring the highest risk (OR = 1.76, 95% CI: 1.32–2.35), according to a recent meta-analysis **[36].**

A meta-analysis revealed that, in individuals with a mild degree of coronary atherosclerosis on angiography, the G20210A prothrombin gene polymorphism can be a moderate but significant risk factor for MI at an early age (<45 years) (OR= 2.3, 95% CI: 1.27–4.59), favoring the manifestation of ischemic heart disease. Additional polymorphisms that appear to be important include those related to glycoprotein VI (GP6, 13254 TC, Ser219Pro), plasminogen activator inhibitor1 polymorphism 4G/5G, and factor V Leiden—particularly the homozygote

phenotype. In most cases, factor X is activated by FV during the prothrombin conversion to thrombin. Thrombin activates FV, and the active risk factors profile of myocardial infarction patients, both older and younger, has been shown to do so**[10].**

Research indicates that the etiology of disease is influenced by factors such as urbanization, dietary westernization, and rising rates of obesity, diabetes, and smoking. The past several decades have seen a decrease in CVD-related mortality as a result of efforts made to control the disease. Hypertension, tobacco use, physical inactivity, high low density lipoprotein cholesterol, diabetes, and several connected metabolic risk variables are potential risk factors for cardiovascular disease (CVD) **[37].**

Even after these risk factors have been identified, many patients never gained sufficient control over them. In addition, the increasing rates of obesity and type 2 diabetes mellitus (Type 2 DM) pose a danger to the advancements made in CVD. Significant increases in the prevalence of other critical CVD risk factors, such as insulin resistance, dyslipidemia, hypertension, and type 2 diabetes, have been attributed to the rising incidence of obesity. Numerous studies have established that blood cholesterol is the main factor contributing to CVD and the mortality that is associated with it in younger participants. Low HDL cholesterol levels raise the risk of heart disease, while high HDL cholesterol levels appear to protect against CVD **[38].**

Homocysteine, coagulation markers like fibrinogen, D-dimer, plasminogen activator inhibitor-1 (PAI-1), and thrombin/anti-thrombin III complex; and different inflammatory markers such CRP, interleukin (IL), serum amyloid A (SAA), MMP, and adhesion molecule are examples of nontraditional risk markers. Pharmacologic treatments, such as endo-cannabinoid receptor antagonists and peroxisome proliferator inhibitors that control glucagon-like peptide-1 activity, are already accessible to address specific CVD risk factors and are undergoing evaluation **[38].**

CONCLUSIONS

Smoking, obesity, dyslipidaemia, and hypertension were the most common risk factors for acute MI. Smoking was the most common risk factor in males.

Declaration of interest

The authors report no conflicts of interest. The authors along are responsible for the content and writing of the paper**.**

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