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PATHOGENETIC AND CLINICAL FEATURES OF ACUTE MYOCARDIAL INFARCTION IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Abstract
This article examines the pathogenetic and clinical features of acute myocardial infarction (AMI) in patients with type 2 diabetes mellitus (T2DM), a condition associated with a higher incidence of complications, a more severe clinical course, and a poorer prognosis compared with patients without diabetes. AMI remains one of the leading causes of mortality among individuals with T2DM, with its incidence being 2–3 times higher than in the general population. Disturbances in carbohydrate metabolism, insulin resistance, systemic inflammation, hyperglycemia, and accelerated atherosclerosis contribute to atherosclerotic plaque instability and an increased risk of thrombosis. In patients with T2DM, AMI often presents with an atypical clinical course, characterized by reduced pain perception, a higher risk of recurrent myocardial infarction, cardiac arrhythmias, and the development of heart failure.

Keywords: Acute myocardial infarction; type 2 diabetes mellitus; pathogenesis; insulin resistance; acute heart failure; atherosclerosis; thrombosis.

Introduction

Myocardial infarction (MI) is one of the most dangerous coronary events, frequently accompanied by sudden cardiac death (SCD) [1], and represents the

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most severe clinical manifestation of coronary artery disease (CAD) [2]. Clinically, MI is classified into two main categories: ST-segment elevation myocardial infarction (STEMI) and non–ST-segment elevation myocardial infarction (NSTEMI). In addition, unstable angina, which often precedes the development of MI, is also considered a component of acute coronary syndrome (ACS) [3].

According to data from the European Society of Cardiology, approximately 1 million cases of acute myocardial infarction are registered annually in European countries. Among these patients, 60–70% are diagnosed with STEMI, whereas 30–40% present with NSTEMI. In the United States, more than 800,000 cases of MI are diagnosed each year, with nearly 27% resulting in death within the first year [4]. Although MI is more frequently diagnosed in developed countries, its prevalence remains high in developing regions as well [5–8]. A large multicenter study involving 19,781 patients with CAD reported an MI prevalence of 23.3% [9].

The highest incidence of MI is observed in low- and middle-income countries, where access to advanced diagnostic and therapeutic technologies is limited. In contrast, developed countries have experienced a decline in the incidence of acute coronary events, including MI, owing to effective preventive strategies and the implementation of modern treatment approaches; nevertheless, the overall burden of MI remains substantial [10,11]. MI continues to be one of the leading causes of mortality and disability worldwide [12]. Despite a global reduction in MI-related mortality, the incidence of post-infarction heart failure (HF) remains high [13]. Post-infarction HF is associated with significant mortality and a high rate of complications [14,15], placing considerable pressure on healthcare systems. For example, in the United States, approximately 6 million individuals suffer from HF, which accounts for more than 300,000 deaths annually and healthcare expenditures of nearly USD 40 billion [16]. The economic burden of MI itself is also substantial: in 2010, more than 1.1 million hospitalizations for MI were recorded in the United States, with direct costs estimated at USD 450 billion [17].

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Pathogenesis and Clinical Course of Acute Myocardial Infarction in Patients with Type 2 Diabetes Mellitus

Obesity, a sedentary lifestyle, hypertriglyceridemia, and inflammatory markers such as high-sensitivity C-reactive protein (hs-CRP) are independent cardiovascular risk factors that are closely associated with insulin resistance [18]. Numerous studies have demonstrated a continued increase in the prevalence of major cardiovascular risk factors, including diabetes mellitus, hypercholesterolemia, obesity, and smoking [19–22].

Diabetes mellitus (DM), particularly type 2 diabetes mellitus (T2DM), is one of the most important risk factors for the development of cardiovascular diseases, including myocardial infarction (MI) [23]. T2DM represents one of the leading chronic non-communicable diseases, and its global prevalence has increased markedly over recent decades. In 2017, the worldwide prevalence of T2DM among adults was estimated at 8.8%, with projections suggesting an increase to 9.9% by 2045 [24,25]. According to the International Diabetes Federation (IDF), the number of individuals living with diabetes worldwide reached 537 million in 2021 and is expected to rise to 643 million by 2030 [26].

As the prevalence of T2DM continues to increase, its complications have had a profound impact on patients' quality of life and have become a major global public health concern. A pooled analysis of 22 prospective cohort studies involving more than one million participants in Asia demonstrated that patients with T2DM in this region have a significantly higher risk of mortality than those in Western countries, with an 89% increase in mortality compared with individuals without T2DM [27]. China currently has the highest prevalence of T2DM worldwide, with a prevalence of 10.9% among the adult population and a prediabetes prevalence of 35.7% [28]. By comparison, the prevalence of T2DM in the Russian Federation is estimated at approximately 6%; however, this figure may be underestimated due to incomplete population screening. Although T2DM predominantly affects individuals older than 40 years, recent years have seen a marked increase in its incidence among younger populations, largely attributable to lifestyle changes, including low levels of physical activity and unhealthy dietary habits [29].

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The prevalence of concomitant myocardial infarction (MI) and type 2 diabetes mellitus (T2DM) remains high. According to data from multicenter studies, diabetes mellitus is identified in 20–30% of patients hospitalized with acute myocardial infarction. In a considerable proportion of cases, diabetes is diagnosed for the first time during hospitalization for MI, which is largely attributable to the absence of prior screening and timely diagnosis [30].

Over recent decades, a consistent decline in mortality from myocardial infarction has been observed owing to the introduction of modern treatment strategies, including thrombolytic therapy, primary percutaneous coronary intervention (PCI), and the widespread use of antiplatelet agents, renin–angiotensin–aldosterone system (RAAS) inhibitors, and statins [31,32]. For example, in Denmark, the risk of cardiovascular mortality within one year after MI decreased from 18.4% to 7.6% between 2005 and 2021 [33]. Nevertheless, the coexistence of MI and T2DM markedly worsens prognosis, significantly increasing the risk of adverse outcomes such as heart failure, recurrent myocardial infarction, cardiac arrhythmias, and death [34].

Despite the overall trend toward reduced long-term mortality and complication rates following acute coronary events, patients with T2DM continue to exhibit substantially higher rates of mortality and complications during the acute phase of MI compared with the general population [35,36].

T2DM is characterized by a complex pathophysiological background that includes the development of insulin resistance, leading to disturbances in carbohydrate metabolism and persistent hyperglycemia. Chronic hyperglycemia promotes non-enzymatic glycation of proteins and lipids, resulting in vascular wall damage and accelerated atherosclerosis. In addition, systemic inflammation and oxidative stress associated with T2DM activate mechanisms that contribute to thrombosis and atherosclerotic plaque instability [37,38].

These pathophysiological mechanisms provide the basis for the development of coronary artery disease (CAD) and its complications, including myocardial infarction (MI). In patients with type 2 diabetes mellitus (T2DM), the risk of MI is approximately 2–3 times higher than in the general population [39,40]. MI represents the leading cause of death among individuals with T2DM. Importantly,

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the risk of major coronary events in patients with T2DM but without a prior history of CAD is comparable to that observed in patients with established CAD. The risk of a first MI within 10 years after the onset of T2DM exceeds 20%, which is similar to the risk of a second MI within 10 years in patients without T2DM but with a previous MI. Furthermore, the risk of recurrent MI in patients with T2DM and a history of MI exceeds 40% [41].

According to data from the PURE study (2018), one-year mortality after acute myocardial infarction (AMI) in patients with T2DM reaches 30%, which is significantly higher than in patients without diabetes [42].

Acute myocardial infarction in patients with T2DM is associated with a more severe clinical course, increased short- and long-term mortality, and a high incidence of complications. In addition to T2DM, several factors substantially contribute to an adverse prognosis following AMI, including chronic kidney disease (CKD), atrial fibrillation, reduced left ventricular ejection fraction at presentation, and advanced age [15,43–45]. Patients at high risk of complicated AMI have been enrolled in large-scale clinical trials evaluating the effectiveness of various treatment strategies, including acute surgical reperfusion, early invasive interventions, antiplatelet and antithrombotic therapy, as well as early initiation of renin–angiotensin–aldosterone system (RAAS) inhibitors, beta-blockers, and statins. The results of these studies have demonstrated that the incidence of adverse outcomes can be reduced in patients presenting with multiple risk factors at admission [46].

In light of these findings, a scientific statement from the American Heart Association (AHA), published in Circulation on April 13, 2020 [47], emphasized the need for more aggressive management of coronary artery disease (CAD) in patients with type 2 diabetes mellitus (T2DM) compared with those without diabetes in order to reduce the risk of myocardial infarction (MI). All contemporary clinical guidelines recommend glycemic control as an integral component of comprehensive cardiovascular risk management in patients with CAD. Moreover, accumulating evidence increasingly supports the concept that adequate glycemic control has a substantial impact on cardiovascular outcomes [48].

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The Guidelines on Diabetes, Prediabetes, and Cardiovascular Diseases issued by the European Society of Cardiology (ESC) in collaboration with the European Association for the Study of Diabetes (EASD) include a specific recommendation for mandatory screening for T2DM in all patients with cardiovascular disease. In addition, patients with cardiovascular disease complicated by T2DM should receive comprehensive management of cardiovascular risk factors, including control of blood pressure, serum glucose and lipid levels, optimization of antiplatelet therapy, and lifestyle modification [49].

To date, there are no studies providing a systematic evaluation of the prevalence of unrecognized abnormal glucose tolerance (AGT) and its impact on recurrent cardiovascular events in patients with myocardial infarction. However, a meta-analysis assessing the prevalence of AGT and the risk of recurrent major adverse cardiovascular events (MACEs) and mortality in patients with MI without previously diagnosed diabetes has been published. This analysis included 19 clinical studies (n = 541,509) with a mean follow-up duration of 3.1 years. The prevalence of newly detected AGT among patients with MI was 48.4%. Patients with prediabetes had a higher risk of mortality and MACEs than those with normal glucose tolerance (NGT). Similarly, patients with a short duration of T2DM exhibited a higher risk of death and MACEs compared with individuals with NGT [50].

Several clinical studies have examined the prevalence and prognosis of unrecognized myocardial infarction in patients with asymptomatic type 2 diabetes mellitus (T2DM) using delayed-enhancement magnetic resonance imaging (DE-MRI). Five-year follow-up of a cohort of 460 patients with T2DM demonstrated that the incidence of death or myocardial infarction was significantly higher among patients with unrecognized disease. These findings indicate that silent myocardial infarction is relatively common in patients with asymptomatic T2DM and no prior history of cardiovascular disease [51].

An increasing body of evidence suggests a strong association between prediabetes and an elevated risk of cardiovascular disease (CVD) and mortality [52–54]. In particular, a comprehensive meta-analysis evaluating the relationship between prediabetes and cardiovascular risk and mortality, which included 129

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studies with a total of 10,069,955 participants, demonstrated that prediabetes is associated with an increased risk of all-cause mortality and CVD-related death. These findings underscore the importance of prediabetes prevention as a key component of cardiovascular risk reduction in patients with or at risk for cardiovascular disease [55].

The results of a large-scale systematic review and meta-analysis assessing the association between T2DM and long-term (≥1 year) mortality after myocardial infarction have also been reported. This analysis included 10 randomized controlled trials and 56 cohort studies encompassing 714,780 patients, with a total of 202,411 deaths recorded over a median follow-up period of 2.0 years (range, 1–20 years). Long-term mortality among patients with T2DM was approximately 50% higher than among patients without diabetes, regardless of myocardial infarction phenotype and the use of contemporary treatment strategies [56]. Similar findings have been reported in other studies, which demonstrated that patients with T2DM experience less favorable short- and long-term outcomes compared with individuals without diabetes. Moreover, previously undiagnosed T2DM was significantly associated with increased mortality, particularly in patients in whom diabetes had not been identified at the time of hospitalization [41].

At present, percutaneous coronary intervention (PCI) with the use of drug-eluting stents (DES) is one of the standard treatment strategies for acute coronary syndrome (ACS), including myocardial infarction (MI) [57]. However, type 2 diabetes mellitus (T2DM) adversely affects both procedural outcomes and long-term prognosis after PCI. Available evidence indicates that 3–5 months after DES implantation, the incidence of stent occlusion is higher in patients with diabetes than in those without diabetes [58].

Increasing attention has been directed toward the long-term outcomes of PCI with DES in patients with new-onset diabetes mellitus (NODM). In a retrospective cohort study involving 6,048 patients who underwent PCI and were stratified according to the presence or absence of T2DM before the intervention, NODM developed during a mean follow-up period of 3.4±1.9 years in 436 (11.8%) of 3,683 patients with ACS who had no prior diagnosis of T2DM [59]. Independent

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predictors of NODM included high-intensity statin therapy, elevated body mass index (BMI), and increased levels of fasting plasma glucose (FPG) and triglycerides. The cumulative incidence of major adverse cardiovascular events (MACE) over 8 years of follow-up was significantly lower in the post-PCI NODM group (19.5%) than in patients with previously diagnosed T2DM (25%, p=0.003) and was comparable to that observed in patients without T2DM (20.5%, p=0.467).

Findings from another retrospective cohort study conducted in Taiwan, which included 30,665 patients with ACS who underwent PCI, demonstrated a 27% increase in the risk of developing NODM among patients receiving statin therapy compared with those who did not receive statins [60]. Nevertheless, the cardiovascular benefits of statins in reducing morbidity and mortality in patients with ACS have been consistently confirmed in multiple clinical studies. Therefore, decisions regarding statin therapy in patients with established cardiovascular disease should not be altered on the basis of the potential risk of NODM.

Knowledge of the mechanisms underlying the early onset and aggravated course of myocardial infarction (MI) in the context of type 2 diabetes mellitus (T2DM) has expanded considerably in recent years. Increasing evidence has clarified the pathways through which hyperglycemia and insulin resistance contribute to increased post-MI mortality [61]. Most patients with diabetes exhibit concomitant insulin resistance, hyperinsulinemia, and vascular calcification, which not only promote atherosclerosis but also accelerate the transition of stable atherosclerotic plaques into unstable lesions or plaque rupture, ultimately leading to thrombosis and adverse coronary events [62]. Key pathogenetic mechanisms, including diabetes-induced overproduction of reactive oxygen species, hypersecretion of pro-inflammatory cytokines, enhanced flux through the aldose reductase (AKR1B1) pathway, and activation of protein kinase C isoforms (β, δ, and θ), further contribute to the early development of MI in patients with T2DM [61].

Early and severe forms of MI are associated not only with T2DM itself but also with its chronic complications. A systematic review and meta-analysis of cohort studies, including 13 studies with a total of 17,611 participants, demonstrated that

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the presence of diabetic retinopathy (DR) significantly increases the risk of cardiovascular disease (CVD) and mortality among patients with diabetes [63]. A hospital-based cross-sectional study conducted in China involving 949 patients with T2DM (700 men and 249 women) showed that both non-proliferative DR (NPDR) and proliferative DR (PDR) were independently associated with increased cardio-ankle vascular index (CAVI) values, reflecting increased arterial stiffness [64]. In addition, other studies have reported that central atherosclerosis is closely associated with both the presence and severity of DR in patients with T2DM [65].

Type 2 diabetes mellitus is also a well-established independent risk factor for acute kidney injury (AKI), which in turn is associated with an unfavorable prognosis after MI. Data from a multicenter randomized controlled trial including 10,251 patients demonstrated a progressively increasing risk of adverse cardiovascular outcomes and all-cause mortality with the development of chronic kidney disease (CKD) and/or cardiovascular disease in patients with T2DM [66]. Moreover, experimental studies suggest that treatment with sodium–glucose cotransporter 2 inhibitors (SGLT2i) may protect the kidneys of patients with diabetes from MI-induced injury and reduce the risk of AKI [67].

Comprehensive and structured follow-up of patients with type 2 diabetes mellitus (T2DM) plays a pivotal role in the early identification of cardiovascular risk. A risk prediction model developed by Chinese investigators demonstrated that reductions in body weight, arterial blood pressure, and serum uric acid levels, as well as adequate control of diastolic blood pressure, can substantially reduce the risk of new-onset acute coronary syndrome (ACS) in patients with T2DM [68]. Data derived from large-scale electrocardiographic and cardiac magnetic resonance imaging screenings indicate a high prevalence of coronary artery disease (CAD) among patients with T2DM. However, standardized approaches to CAD screening in this population are currently lacking.

Recent studies suggest that routine screening for CAD using coronary computed tomography angiography (CCTA) may be reasonable for the early detection of subclinical CAD in asymptomatic patients with T2DM, particularly in those with a disease duration exceeding 10.5 years and systolic blood pressure greater than

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140 mmHg [69]. Such an approach may facilitate earlier identification of high-risk individuals and improve risk stratification.

In addition to imaging-based strategies, novel metabolic markers have been proposed for early cardiovascular risk assessment. One such marker is the triglyceride–glucose index (TGI), calculated as fasting triglycerides (mg/dL) × fasting plasma glucose (mg/dL) / 2. It has been shown that in patients with ACS undergoing percutaneous coronary intervention (PCI), the TGI is a more informative predictor of subsequent cardiovascular events than fasting plasma glucose or glycated hemoglobin (HbA1c) [70]. These findings highlight the potential utility of integrated metabolic indices in refining cardiovascular risk stratification beyond traditional glycemic parameters.

Statins remain a cornerstone of lipid-lowering therapy in patients with coronary artery disease. Nevertheless, accumulating clinical evidence suggests a possible association between statin therapy and the development of new-onset type 2 diabetes mellitus (NODM). In particular, studies have demonstrated that epicardial adipose tissue thickness is an independent predictor of NODM in patients with CAD receiving high-intensity statin therapy [71]. These observations may assist clinicians in developing targeted monitoring strategies and early interventions aimed at the timely identification of NODM, without compromising the well-established cardiovascular benefits of statin therapy.

In recent years, an increasing number of studies have focused on identifying genetic factors that predispose patients with type 2 diabetes mellitus (T2DM) to an elevated risk of myocardial infarction (MI). Among the genetic variants identified, the single-nucleotide polymorphism rs10830963 in the MTNR1B gene is one of the most extensively studied and has been shown to be closely associated with susceptibility to T2DM. A study based on data from the UK Biobank evaluated the association between the rs10830963 polymorphism and the risk of fatal and non-fatal MI in 13,655 participants with presumed T2DM over a mean follow-up period of 6.8 years. Using an additive genetic model, the investigators demonstrated that variation in the MTNR1B gene at rs10830963 was positively correlated with an increased risk of MI during follow-up, suggesting that this

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polymorphism may serve as a useful genetic marker for MI risk stratification in patients with T2DM [72].

The growing body of evidence regarding the genetic interplay between T2DM and cardiovascular disease is beginning to elucidate the shared biological pathways underlying both conditions [73]. In the future, more comprehensive genetic and multi-omics studies may provide deeper insights into the complex relationship between T2DM and MI, potentially enabling more precise risk prediction and the development of personalized preventive and therapeutic strategies.

Conclusion

Acute myocardial infarction (AMI) in patients with type 2 diabetes mellitus (T2DM) is characterized by a higher rate of complications, a more severe clinical course, and a poorer prognosis compared with patients without diabetes. AMI remains one of the leading causes of mortality among individuals with T2DM, with the incidence of myocardial infarction being 2–3 times higher than in the general population. Disturbances in carbohydrate metabolism, insulin resistance, systemic inflammation, hyperglycemia, and accelerated atherosclerosis contribute to atherosclerotic plaque instability and an increased risk of thrombosis. In patients with T2DM, AMI often presents with an atypical clinical course, including reduced pain perception, a higher risk of recurrent myocardial infarction, cardiac arrhythmias, and the development of heart failure.

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