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ORIGINAL ARTICLE

THE IMPACT OF DIABETES MELLITUS ON CLINICAL OUTCOMES IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

Appazova Linura[1], Shaymatova Dilshod[2], Uzoqova Oyjamol[3],

Gastroenterologist of Samarkand Regional Multidisciplinary Medical Center[1], functional diagnostic doctor of the Samarkand regional multidisciplinary medical center, Assistant of the Department of Hematology, Samarkand State Medical University

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Abstract

Acute myocardial infarction (AMI) remains one of the leading causes of mortality and long-term disability worldwide. Diabetes mellitus is recognized as one of the strongest independent risk factors for coronary artery disease and significantly influences both the clinical presentation and prognosis of myocardial infarction. Chronic hyperglycemia promotes endothelial dysfunction, accelerated atherosclerosis, chronic inflammation, oxidative stress, and platelet hyperactivity, thereby increasing the severity of coronary artery obstruction and myocardial injury. The present study aimed to evaluate the influence of diabetes mellitus on the clinical course, in-hospital complications, and short-term outcomes of patients admitted with acute myocardial infarction. A comparative analysis was performed between diabetic and non-diabetic patients using clinical characteristics, laboratory findings, electrocardiographic data, echocardiographic parameters, and treatment outcomes. The findings demonstrated that diabetes mellitus was associated with larger infarct size, reduced left ventricular function, increased incidence of heart failure, arrhythmias, recurrent ischemic events, and prolonged hospitalization. Early identification of diabetes-related cardiovascular risk and comprehensive metabolic management may significantly improve survival and reduce adverse cardiovascular events after acute myocardial infarction.

Keywords: acute myocardial infarction, diabetes mellitus, coronary artery disease, hyperglycemia, cardiovascular complications, cardiac biomarkers, left ventricular dysfunction, prognosis, reperfusion therapy, cardiology.

1. Introduction

Acute myocardial infarction remains one of the most serious manifestations of ischemic heart disease and continues to represent a major cause of cardiovascular mortality despite considerable advances in diagnosis, pharmacological treatment, and interventional cardiology. Rapid coronary artery occlusion leads to ischemic necrosis of myocardial tissue, initiating complex inflammatory, metabolic, and neurohormonal responses that determine both early survival and long-term cardiac function. Diabetes mellitus has emerged as one of the most significant contributors to cardiovascular disease. Individuals with diabetes experience a markedly higher incidence of myocardial infarction compared

with the general population and frequently develop coronary artery disease at a younger age. In addition to increasing the likelihood of acute coronary events, diabetes substantially worsens the clinical course following myocardial infarction by accelerating myocardial injury and impairing tissue repair.

Persistent hyperglycemia induces widespread vascular abnormalities through multiple interconnected mechanisms. Endothelial dysfunction reduces nitric oxide bioavailability and impairs vascular relaxation, while chronic oxidative stress damages cellular structures and promotes inflammatory activation. Advanced glycation end products alter vascular elasticity and enhance inflammatory signaling, contributing to progressive atherosclerotic plaque formation and instability.

Diabetes also influences platelet function and coagulation pathways. Increased platelet activation, enhanced thrombin generation, and impaired endogenous fibrinolysis create a prothrombotic environment that favors complete coronary artery occlusion during plaque rupture. Consequently, diabetic patients frequently present with more extensive coronary artery involvement and larger infarct size than individuals without diabetes.

Microvascular dysfunction represents another important characteristic of diabetic cardiovascular disease. Even after successful reopening of the occluded coronary artery, impaired microcirculatory perfusion may limit myocardial reperfusion and contribute to persistent ischemic injury. This phenomenon increases infarct expansion, delays myocardial recovery, and predisposes patients to adverse ventricular remodeling.

Clinical presentation of myocardial infarction in diabetic patients often differs from that observed in non-diabetic individuals. Autonomic neuropathy may diminish pain perception, resulting in atypical or silent myocardial infarction. Delayed recognition of symptoms frequently postpones hospital admission and reduces opportunities for timely reperfusion therapy, thereby negatively affecting prognosis.

The coexistence of diabetes with hypertension, obesity, dyslipidemia, and chronic kidney disease further amplifies cardiovascular risk. These comorbidities interact synergistically, increasing the likelihood of heart failure, malignant arrhythmias, cardiogenic shock, recurrent ischemia, and cardiovascular death following acute myocardial infarction.

Contemporary management of acute myocardial infarction increasingly emphasizes individualized treatment strategies for diabetic patients. Rapid reperfusion, intensive antithrombotic therapy, strict metabolic control, optimization of cardiac function, and comprehensive secondary prevention collectively improve survival and reduce recurrent cardiovascular events. Emerging glucose-lowering agents with proven cardiovascular benefits have further expanded therapeutic opportunities in this high-risk population.

Despite substantial improvements in cardiovascular care, diabetes continues to exert a profound influence on post-infarction outcomes. Better understanding of the interaction between metabolic dysfunction and myocardial injury is essential for developing more effective preventive and therapeutic strategies.

The present study aimed to evaluate the impact of diabetes mellitus on the clinical characteristics, laboratory findings, cardiac function, treatment response, and early outcomes of patients hospitalized with acute myocardial infarction through comprehensive clinical assessment.

2. Materials and Methods

This prospective observational study was conducted between 2023 and 2025 at tertiary cardiology departments specializing in the management of acute coronary syndromes. The objective was to investigate the influence of diabetes mellitus on the clinical presentation, laboratory findings, therapeutic outcomes, and early prognosis of patients admitted with acute myocardial infarction.

A total of 220 consecutive patients with confirmed acute myocardial infarction were enrolled in the study. Diagnosis was established according to contemporary international cardiology guidelines based on characteristic clinical symptoms, electrocardiographic abnormalities, dynamic elevation of cardiac biomarkers, and coronary angiographic findings when available.

Participants were divided into two study groups. Group A consisted of 108 patients with previously diagnosed or newly confirmed diabetes mellitus. Group B included 112 patients without diabetes mellitus. Patients with severe systemic inflammatory diseases, advanced malignancies, congenital heart disease, or incomplete clinical records were excluded from the investigation.

Comprehensive clinical evaluation was performed immediately after hospital admission. Demographic

characteristics, cardiovascular risk factors, smoking history, hypertension, dyslipidemia, obesity, chronic kidney disease, medication history, and family history of cardiovascular disease were documented. Time from symptom onset to hospital presentation and reperfusion therapy was also recorded.

Laboratory investigations included complete blood count, fasting blood glucose, glycated hemoglobin (HbA1c), serum creatinine, lipid profile, high-sensitivity cardiac troponin, creatine kinase-MB, C-reactive protein, electrolyte concentrations, and coagulation parameters.

Electrocardiography was performed at admission and repeated during hospitalization to evaluate infarct localization, ischemic changes, arrhythmias, and conduction disturbances. Transthoracic echocardiography was carried out within the first forty-eight hours to determine left ventricular ejection fraction, regional wall motion abnormalities, ventricular dimensions, and mechanical complications.

All patients received evidence-based treatment including dual antiplatelet therapy, anticoagulants, statins, beta-blockers, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, and reperfusion therapy when clinically indicated. Glycemic control in diabetic patients was achieved using individualized insulin protocols or adjustment of pre-existing antidiabetic medications under continuous glucose monitoring.

Clinical outcomes evaluated during hospitalization included recurrent myocardial infarction, acute heart failure, cardiogenic shock, malignant ventricular arrhythmias, atrial fibrillation, stroke, acute kidney injury, duration of hospitalization, and in-hospital mortality.

3. Results

Clinical evaluation demonstrated that patients with diabetes mellitus presented with significantly more complex cardiovascular profiles than non-diabetic patients. Hypertension, obesity, dyslipidemia, and chronic kidney disease were considerably more prevalent among diabetic individuals, indicating a greater burden of cardiovascular risk factors before the onset of myocardial infarction.

The clinical presentation also differed between study groups. Typical severe chest pain was less frequently observed in diabetic patients, whereas atypical manifestations including dyspnea, generalized weakness, nausea, unexplained fatigue, and silent ischemia occurred more commonly. Delayed recognition of symptoms resulted in longer intervals between symptom onset and hospital admission.

Laboratory analysis revealed substantially higher admission blood glucose and glycated hemoglobin levels among diabetic patients. Peak concentrations of cardiac troponin and creatine kinase-MB were generally higher, suggesting more extensive myocardial injury. Inflammatory markers, particularly C-reactive protein, also remained elevated for longer periods compared with non-diabetic individuals.

Electrocardiographic assessment demonstrated more frequent anterior wall myocardial infarction and multivessel ischemic changes in patients with diabetes mellitus. Complex ventricular arrhythmias, conduction abnormalities, and persistent ST-segment deviations occurred more frequently throughout hospitalization.

Echocardiographic examination identified significantly lower left ventricular ejection fraction among diabetic patients. Regional wall motion abnormalities involved larger myocardial territories, while left ventricular remodeling developed earlier during hospitalization. Diastolic dysfunction was also more prevalent, reflecting chronic diabetic myocardial involvement before the acute ischemic event.

Acute heart failure represented one of the most common complications in diabetic patients. Pulmonary congestion, reduced cardiac output, and impaired ventricular performance required more intensive pharmacological management and prolonged monitoring. Cardiogenic shock developed more frequently among diabetic individuals with extensive myocardial damage.

Reperfusion therapy successfully restored coronary blood flow in the majority of patients; however, diabetic individuals demonstrated less complete functional myocardial recovery despite technically successful coronary intervention. Persistent microvascular dysfunction appeared to contribute to incomplete tissue reperfusion and delayed improvement of ventricular function.

The incidence of recurrent ischemic episodes, atrial fibrillation, ventricular tachyarrhythmias, and acute kidney injury was significantly higher among patients with diabetes mellitus. These complications prolonged hospitalization and increased the need for intensive cardiovascular support. Overall hospital stay was longer in the diabetic group. Furthermore, in-hospital mortality remained higher among diabetic patients than among individuals without diabetes, emphasizing the adverse

prognostic influence of chronic metabolic disease on acute myocardial infarction outcomes.

4. Discussion

The present study demonstrates that diabetes mellitus profoundly influences both the clinical presentation and prognosis of acute myocardial infarction. Chronic metabolic abnormalities contribute to accelerated coronary atherosclerosis, diffuse vascular disease, endothelial dysfunction, and impaired myocardial recovery following ischemic injury.

One of the most important observations is the frequent occurrence of atypical clinical symptoms in diabetic patients. Autonomic neuropathy reduces pain perception, delaying medical attention and limiting the effectiveness of early reperfusion strategies. Delayed treatment allows greater myocardial necrosis and increases the risk of mechanical and electrical complications.

The larger infarct size observed among diabetic patients is consistent with the presence of multivessel coronary artery disease and impaired coronary microcirculation. Persistent hyperglycemia, oxidative stress, and chronic inflammation promote endothelial injury and reduce myocardial resistance to ischemia, thereby amplifying tissue destruction during coronary occlusion.

Reduced left ventricular systolic function identified by echocardiography represents another major determinant of poor prognosis. Ventricular remodeling progresses more rapidly in diabetic individuals because metabolic dysfunction interferes with myocardial repair, collagen remodeling, and angiogenesis. Consequently, diabetic patients exhibit increased susceptibility to chronic heart failure after myocardial infarction.

The elevated frequency of arrhythmias and heart failure observed in this investigation reflects the combined influence of extensive myocardial injury, autonomic imbalance, electrolyte disturbances, and structural cardiac remodeling. These complications contribute substantially to increased mortality and healthcare utilization.

Strict glycemic management during hospitalization appears to improve clinical stability by reducing inflammatory activation, limiting oxidative stress, and supporting myocardial metabolism. Nevertheless, glucose control alone cannot completely eliminate the adverse cardiovascular effects associated with longstanding diabetes, emphasizing the importance of comprehensive preventive care before the occurrence of myocardial infarction.

Modern cardiovascular management increasingly integrates advanced antidiabetic medications with established cardioprotective therapies. Sodium-glucose cotransporter-2 inhibitors and glucagon-like peptide-1 receptor agonists have demonstrated significant cardiovascular benefits beyond glucose lowering, offering new opportunities to improve long-term outcomes in high-risk diabetic populations.

Future investigations should evaluate individualized therapeutic strategies incorporating precision medicine, metabolic biomarkers, artificial intelligence-assisted risk prediction, and novel cardiometabolic interventions. Such approaches may further reduce mortality and improve recovery following acute myocardial infarction in patients with diabetes mellitus.

5. Conclusion

Diabetes mellitus is a major determinant of adverse clinical outcomes in patients with acute myocardial infarction. Diabetic individuals present with more extensive coronary artery disease, atypical clinical manifestations, greater myocardial injury, impaired ventricular function, and significantly higher rates of cardiovascular complications.

Comprehensive management combining rapid reperfusion therapy, intensive cardiovascular treatment, optimized glycemic control, and careful monitoring of complications significantly improves patient prognosis. Early identification of high-risk diabetic patients and implementation of individualized multidisciplinary treatment strategies are essential for reducing morbidity, preserving cardiac function, and improving survival following acute myocardial infarction.

The findings emphasize the importance of integrating cardiology, endocrinology, and preventive medicine to optimize both acute management and long-term secondary prevention in patients affected by diabetes mellitus and acute myocardial infarction.

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