The Pharma Innovation Journal 2020; 9(6): 311-313

www.ThePharmaJournal.com

The Pharma Innovation

ISSN (E): 2277- 7695
ISSN (P): 2349-8242
NAAS Rating: 5.03
TPI 2020; 9(6): 311-313

www.thepharmajournal.com
Received: 07-04-2020
Accepted: 09-05-2020

Juraeva Khafiza Iskandarovna
Phd, the Assistant to Department of Internal Diseases and Endocrinology of The Bukhara State Medical Institute, Bukhara, Uzbekistan

Badridinova Barno Kamalidinovna
Assistant to Department of Internal Diseases and Endocrinology of the Bukhara State Medical Institute, Bukhara, Uzbekistan

Corresponding Author:
Juraeva Khafiza Iskandarovna
Phd, the Assistant to Department of Internal Diseases and Endocrinology of The Bukhara State Medical Institute, Bukhara, Uzbekistan

Relationship of myocardial infarction with metabolic syndrome

Juraeva khafiza iskandarovna and Badridinova barno kamalidinovna

Abstract
Annotation: In patients with metabolic syndrome, the synergism of the pathogenetic mechanisms of the metabolic syndrome, coronary heart disease and arterial hypertension determines the high prevalence and severity of acute coronary pathology. For patients with myocardial infarction on the background of the metabolic syndrome, severe coronary lesion and poor prognosis are characteristic. The article discusses the factors influencing the course and outcome of myocardial infarction in patients with metabolic syndrome.

Keywords: myocardial infarction, diabetic mellitus, arterial hypertension, dyslipidemia obesity

Introduction

In a number of developed countries, diseases of the cardiovascular system are leading among the causes of morbidity, disability and mortality, although their occurrence in different regions is subject to variability. According to the WHO, approximately 3.8 million men and 3.4 million women die from cardiovascular causes every year, with 1/4 of the deaths being people under the age of 65 [2, 7]. To date, several clinical studies have been carried out demonstrating the widespread prevalence of metabolic syndrome (MS) among patients with myocardial infarction. Metabolic syndrome is a complex of metabolic, hormonal and clinical disorders, which are based on insulin resistance (IR) and compensatory hyperinsulinemia (GI), abdominal obesity, impaired lipid, purine metabolism and arterial hypertension (AH). Clinicians of various specialties pay close attention to MS due to the high prevalence, multicomponent nature of this syndrome, and a high risk of developing cardiovascular complications.

The prevalence of MS among the population in various regions is quite high (10-24%), and in economically developed countries the frequency of MS among the population reaches 35-40% - [1]. To date, the number of patients with MS is 2 times greater than the number of patients with diabetes, and in the next 20 years it is expected to increase by 50%. The presence of MS increases the risk of developing cardiovascular events by 2-4 times, even with the exception of diabetes patients from the analysis [7, 8]. MS is detected in approximately 20% of adults and 45% of individuals older than 50 years. The prevalence of MS in myocardial infarction (MI) varies from 37% (Japan) to 50% (USA, France) [7, 10, 12, 13]. It should be noted that the incidence of MS in patients with myocardial infarction younger than 45 years increases and is about 60% [3, 4]. When analyzing the individual effect of each of the components of MS on the risk of developing complications of myocardial infarction, it was found that hyperglycemia is an independent predictor of the development of cardiogenic shock, and hyperglycemia and low values of high density lipoprotein cholesterol (HDL cholesterol) - acute heart failure [19]. When studying the effect of MS on the prognosis of myocardial infarction, it was found that the presence of MS significantly increases the risk of death in the next 3 years by 29%, and cardiovascular events, including fatal arrhythmic complications, by 23% [11].

The mortality rate in the acute period of myocardial infarction (MI) in the group of patients with MS is 2 times higher than in the group of patients who do not have a combination of risk factors for the diagnosis of MS [10].

Regarding the influence of MS on the risk of developing recurrent MI, there are somewhat conflicting data. According to some authors, MS does not cause an increase in the incidence of recurrent MI and fatal ventricular arrhythmias [10].

On the other hand, the presence of MS in patients with acute coronary syndrome (ACS) leads to an increase in the relative and absolute risk of developing sudden cardiac death,

Recurrent MI, recurrent myocardial ischemia by 34 and 5%, respectively [13].

Improving diagnostic capabilities at the present stage has led to the introduction in clinical practice of new methods that allow not only to diagnose myocardial infarction, but also to judge the degree of cardiac muscle necrosis - the size of myocardial infarction. In order to determine the magnitude of myocardial necrosis, a quantitative determination of cardioselective enzymes (CPK-MV, troponin), myocardial scintigraphy with Tc99, computed tomography are used. Using the above methods, it was established that the presence of MS in patients with MI is associated with an increase in the size of MI and is accompanied by a regular decrease in the ejection fraction of the left ventricle [16, 17]. The causes and mechanisms underlying the predisposition of MI patients with MS to a larger myocardial damage have not been sufficiently studied, but it is obvious that the basis of these processes is a complex of metabolic, hormonal and clinical disorders linked to the pathobiochemical and pathophysiological level that are characteristic of MS.

At present, IR is considered as a key etiological category in the pathogenesis of MS. [4, 5, 6]. According to the article "Analysis of the condition and treatment of the metabolic syndrome at the primary health care level", excess insulin production increases the activity of the SNA, causes vasoconstriction and increases the minute volume of blood circulation, increases the synthesis of VLDL, forms atherogenic dyslipidemia and obesity [1]. And also, plays a fundamental role in the formation of arterial hypertension (AH). In addition, it is known that insulin resistance is interrelated with the nature of the distribution of adipose tissue in the body and contributes to the accumulation of fat deposits mainly in the abdomen: in the omentum and around the internal organs, i.e., leads to the formation of visceral obesity [6, 8, 9, 14]. It has been established that adipose tissue of the visceral region is an active endocrine organ and secretes more than 90 biologically active substances, including adipokines, anti-inflammatory cytokines, angiotensinogen, plasminogen activator inhibitor 1 (PAI-1), etc. [7,16,19]. The pathogenetic and prognostic role in MS of some of these substances has been established, but their additional significance is possible under conditions of MI development. Adiponectin regulates food intake and body weight, glucose and lipid metabolism, has anti-inflammatory and anti-atherosclerotic properties, positively affects lipid metabolism and blood coagulation properties [27, 28]. It realizes its effects by binding to specific adiponectin receptors. Adiponectin levels are lower in women than in men, with obesity and diabetes. A strong negative correlation between the level of adiponectin and the amount of visceral fatty tissue, body mass index (BMI) was revealed. Low values of adiponectin in blood serum are associated with an increased risk of IR, diabetes mellitus, and cardiovascular diseases [31, 32]. There is evidence that in patients with MI, the level of adiponectin is associated with the degree of necrosis of the heart muscle; it is assumed that adiponectin has a positive effect on the ischemic myocardium during revascularization and subsequent remodeling processes [33, 34]. Adiponectin has protective properties in relation to the development of systolic dysfunction of the left ventricle, and inhibits the processes of appoptosa cardiomyocytes in MI [33].

In 1994, a new adipocyte hormone, leptin, was discovered, which is secreted by white adipose tissue in amounts proportional to body weight. Leptin is an adipocytokine that is involved in the regulation of saturation and energy consumption [28.]. Plasma leptin levels increase during the development of obesity and decrease with weight loss. The leading function of leptin is to protect peripheral tissues from the accumulation of fat - the regulation of adipose tissue homeostasis.

The level of leptin directly correlates with BMI, blood pressure and heart rate (HR), affects the aggregation properties of platelets. The level of leptin is associated with the severity of outcomes of cardiovascular diseases, its high values are associated with the development of MI and stroke, regardless of other cardiovascular risk factors and the degree of obesity [29]. In the case of developing MI, high serum leptin values during the first 6 hours from the onset of pain in the chest are predictive in terms of the inefficiency of subsequent thrombolytic therapy [20].

An important place in the modern neuroendocrine theory of the development of MS and diseases of the cardiovascular system is given to the tumor necrosis factor (TNF-α), which normally plays a fundamental physiological role in immunoregulation, but in some cases it can have a pathological effect, taking part in the development and progression of inflammation, microvascular hypercoagulation, hemodynamic disturbances and metabolic depletion in various human diseases of both infectious and non-infectious nature. Values of TNF-α positively correlate with IR, which determines the ability of this cytokine to be an early marker of the development of diabetes mellitus. It was shown that TNF-α disrupts insulin signals in muscle and adipose tissue and thereby contributes to the development and progression of IR [21, 22, 23, 24]. It is suggested that in patients with MI, TNF-α triggers a cascade of pathological biochemical reactions, leading ultimately to the induction of cellular apoptosa cardiomyocytes [21]. An increase in serum levels of TNF-α in MI has been established, and its potential role in the development of restenoses after coronary stenting is considered [22]. Studies have shown a relationship between the level of TNF-α in the blood and the incidence of cardiogenic shock in MI [25]. Of undoubted interest are the data demonstrating the effect of TNF-α on the frequency of development of ventricular rhythm disturbances in the acute period of MI in laboratory animals. It is believed that TNF-α increases the level of intracellular calcium in cardiomyocytes and thereby provokes the development of arrhythmias [24]. In patients with MS, the synergism of the pathogenetic mechanisms of MS, IHD and AH determines the accelerated development and severity of acute forms of IHD. For the majority of patients with myocardial infarction on the background of MS, severe coronary lesion and poor prognosis are characteristic. It seems necessary to timely detect MS, determine the degree of cardiovascular risk and indications for myocardial revascularization, as well as the development of algorithms for the diagnosis, treatment and prevention of ACS in patients with MS. Treatment of MI in patients with MS should be carried out in accordance with modern approaches to the treatment of MI: when determining a high risk, an early invasive strategy is justified, in other cases, conservative therapy, including thrombolytics, if indicated, anticoagulants, antiplatelet agents, β-blockers, statins, inhibitors ACE, if necessary - nitrates [10, 12, 14, 26,].

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