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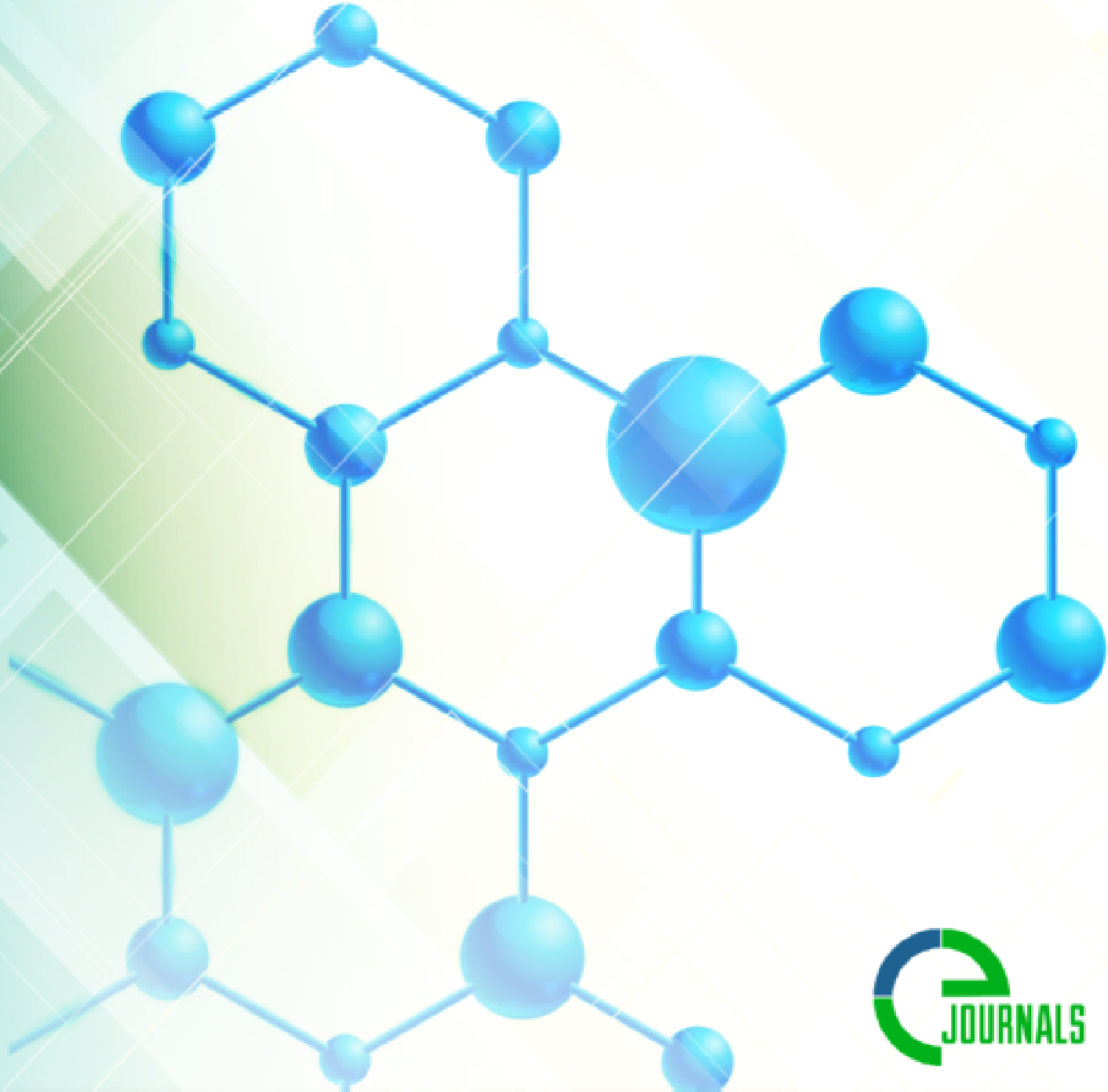

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ANGIOGRAPHIC INDICATORS IN PATIENTS WITH CORONARY ARTERY DISEASE AFTER REVASCULARIZATION AGAINST THE BACKGROUND OF LIVER DYSFUNCTION

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Abstract. Patients with coronary artery disease (CAD), particularly those with metabolic disorders, including liver dysfunction, belong to a group at very high risk of developing cardiovascular events. This literature review provides a detailed analysis of studies examining the clinical and angiographic indicators in CAD patients with impaired liver function.

Worldwide, over the past decades, cardiovascular events have been the primary etiological factor in morbidity and mortality among both men and women. According to statistical data, in the Republic of Uzbekistan in 2016, mortality from circulatory system diseases amounted to 179.8 per 100,000 population. The European Society of Cardiology has provided substantial evidence demonstrating that myocardial revascularization reduces the risk of mortality and CAD progression.

However, in practice, managing patients with comorbid pathologies presents significant challenges. Liver dysfunction is particularly notable among associated diseases and is considered a risk factor for the progression of cardiometabolic pathology. In all forms of liver failure, there is a disruption of metabolic, detoxification, and other hepatic functions, which can lead to central nervous system impairment, up to the development of hepatic coma. Chronic liver failure progresses over months or years, characterized by a gradual and slow onset of clinical manifestations.

The etiological factors contributing to liver dysfunction include chronic progressive liver diseases such as alcoholic hepatitis, viral hepatitis, autoimmune hepatitis, liver cirrhosis, tumors, fatty hepatosis, helminthiasis, and even tuberculosis. Liver dysfunction may also develop as a result of gallstone disease. In some cases, the condition may be caused by chronic heart failure or portosystemic shunting surgeries. Regardless of the underlying cause, chronic liver failure always involves prolonged and slowly developing hepatonecrosis, which is initially compensated by physiological mechanisms. However, acute exacerbation can be triggered by infections, alcohol consumption, physical overexertion, intoxications, or high doses of certain medications (e.g., diuretics).

Clinically, liver dysfunction may present as hepatodepression, which occurs with impaired liver function in the absence of encephalopathy, or hepatic failure, where hepatic encephalopathy develops. Chronic liver failure progresses from a compensated stage to a decompensated and terminal stage, ultimately leading to hepatic coma.

Early clinical signs of chronic liver failure include dyspeptic symptoms such as nausea, vomiting, diarrhea, and anorexia. Other manifestations may include intermittent fever, jaundice, and skin lesions (hemorrhages, weeping and dry eczema, palmar erythema).Peripheral edemaand ascites may develop early in the course of the disease. A distinctive feature of chronic liver failure is the presence of endocrine disorders, such as infertility, decreased libido, testicular atrophy, gynecomastia, alopecia, and atrophy of the mammary glands and uterus. Neurological and psychiatric disturbances manifest as depression, memory impairment, alternating drowsiness and insomnia, anxiety, episodes of confusion, disorientation, inappropriate behavior, aggressiveness, and irritability.

Due to the prolonged course and nonspecific symptoms of chronic liver dysfunction, especially in its early stages, diagnosis is often delayed. Early laboratory indicators of chronic liver failure include a gradual increase in bilirubin and transaminase levels, hypoglycemia, and reduced cholesterol levels, as well as signs of hypocoagulation in coagulation tests. Urine tests may show elevated bilirubin and urobilin, giving it a yellowish-brown color.

To assess liver dysfunction, hepatoscintigraphy is used. Comprehensive diagnostic evaluations also involve MRI of the liver and multislice computed tomography (MSCT) of the abdominal cavity.

In chronic liver failure, a strict dietary restriction of protein and salt intake is recommended. Treatment aims to eliminate ammonia intoxication, including the use of cleansing enemas and saline laxatives, antibiotic therapy (neomycin), lactulose (which inhibits ammonia formation in the intestines), glutamic acid, and ornithine-containing medications to bind absorbed toxic substances. Electrolyte imbalances are corrected with intravenous infusions of saline solutions and glucose, as well as transfusions of fresh frozen plasma. Hepatoprotectors, B vitamins, cocarboxylase, vitamin K, and folic acid are used to support liver function. Detoxification procedures such as hemoperfusion, plasmapheresis, and hemodialysis are also indicated. In cases of progressive chronic liver failure, liver transplantation is considered.

Early diagnosis and treatment of chronic liver failure improve quality of life and prolong survival. The presence of cirrhosis, ascites, and a history of alcohol consumption worsens the prognosis. In cases of hepatic encephalopathy, mortality reaches 80-90%. Preventing the progression of chronic liver failure requires avoiding triggering factors, such as excessive protein intake, uncontrolled medication use, and alcohol abuse. Patients with chronic liver failure should be under the supervision of a gastroenterologist or hepatologist.

Thus, accurately assessing the risk of cardiac complications in patients with liver dysfunction is crucial for the efficient allocation of healthcare resources and for improving both short-term and long-term clinical outcomes. Current screening and diagnostic tests remain limited in their ability to accurately detect coronary artery disease and myocardial dysfunction in individuals with liver dysfunction.

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