FUNCTIONAL STATUS OF THE LIVER IN CARDIOVASCULAR DISEASES

Sharapova Nozima Erkinjonovna

Teacher of the Department Fundamental Medicine Disciplines Asia International University.

E-mail: *sharapovanozimaerkinjonovna@oxu.uz*

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Abstract. *Cardiovascular diseases and liver diseases are closely related. Non-alcoholic fatty liver disease has the same risk factors as those for atherosclerotic cardiovascular disease and may also be a risk factor for atherosclerotic cardiovascular disease on its own. Heart failure causes liver fibrosis, and liver fibrosis results in worsened cardiac preload and congestion. Although some previous reports regard the association between cardiovascular diseases and liver disease, the management strategy for liver disease in patients with cardiovascular diseases is not still established.*

Keywords: *liver disease; heart failure; atherosclerotic cardiovascular disease; non-alcoholic fatty liver disease.*

ФУНКЦИОНАЛЬНОЕ СОСТОЯНИЕ ПЕЧЕНИ ПРИ СЕРДЕЧНО-СОСУДИСТЫХ ЗАБОЛЕВАНИЯХ

Аннотация. *Сердечно-сосудистые заболевания и заболевания печени тесно связаны. Неалкогольная жировая болезнь печени имеет те же факторы риска, что и атеросклеротические сердечно-сосудистые заболевания, и может также быть фактором риска атеросклеротических сердечно-сосудистых заболеваний сама по себе. Сердечная недостаточность вызывает фиброз печени, а фиброз печени приводит к ухудшению сердечной преднагрузки и застоя. Хотя некоторые предыдущие отчеты рассматривают связь между сердечно-сосудистыми заболеваниями и заболеваниями печени, стратегия лечения заболеваний печени у пациентов с сердечно-сосудистыми заболеваниями все еще не установлена.*

Ключевые слова: *заболевание печени; сердечная недостаточность; атеросклеротическое сердечно-сосудистое заболевание; неалкогольная жировая болезнь печени.*

Despite initiation of new therapies, both the short- and long-term mortality rate of patients with heart failure is still high. This may be at least partly attributable to frequently present comorbidities.

Heart failure itself is characterized by impaired organ perfusion resulting from both forward failure and increased central venous pressure (backward failure). We recently showed that both forward and backward failure are the most important determinants of renal dysfunction

in heart failure. Liver function abnormalities are frequently found in patients with heart failure and are related to a poor outcome. Individual small reports have highlighted the importance of either high central venous pressure or reduced hepatic perfusion. However, the relative contribution of reduced perfusion (forward failure) or venous congestion (backward failure) in causing alterations in specific markers of liver function has not been established.

In patients with non-alcoholic fatty liver disease (NAFLD), (cardiovascular diseases) CVD is the most important comorbidity as well as liverassociated complications. CVD is the most common cause of death in these patients. ASCVD and NAFLD share the same risk factors, such as dyslipidemia, diabetes mellitus, and obesity; however, NAFLD itself is considered a promoting factor for ASCVD. NAFLD is associated with dyslipidemia, oxidative stress, microbiome disturbances, chronic inflammation, insulin resistance, coagulation disorders, and endothelial dysfunction, and these factors might also be associated with CVD. Owing to these factors, NAFLD has a strong association with prognosis in CVD.

NAFLD is associated with abnormal lipid metabolism and altered glucose metabolism.

Patients with NAFLD tend to have high triglyceride concentrations, high low-density lipoprotein concentrations, oxidated low-density lipoprotein and remnant cholesterol, and low high-density lipoprotein concentrations, which are established risk factors for ASCVD.

Dyslipidemia can become one of the causes of NAFLD and also be worsened by NAFLD. Elevated plasma insulin and glucose levels, which are caused by diabetes mellitus and insulin resistance, and excess plasma lipid lead to increased de novo lipogenesis and decreased intracellular triglyceride hydrolysis. This increases hepatic triglyceride content, which causes increased plasm triglyceride levels. In addition, increased intracellular cholesterol in the liver reduced membrane-bound low-density lipoprotein receptor and low-density lipoprotein uptake to the liver. These changes contribute to increased large very-low-density lipoprotein 1 and small dense low-density lipoprotein, which finally accelerate atherosclerosis. Notably, the Mediterranean diet and antioxidant formulation efficiently improve anthropometric parameters, lipid profile, and insulin sensitivity and reduce hepatic fat accumulation and liver stiffness in patients with NAFLD as preventive management.

Because the therapeutic strategy for NAFLD has not been established yet, diet therapy may be one of the few efficient therapies for patients with NAFLD.

Liver Function Testing Laboratory measurements were extracted from samples drawn within 3 days before catheterization. Liver function tests that were extracted included aspartate aminotransferase (AST, upper limit of normal [ULN] 40 U/L), alanine aminotransferase (ALT, ULN 30 U/L), alkaline phosphatase (ALP, ULN 120 U/L), g-glutamyl transpeptidase (GGT, ULN 65 U/L), lactate dehydrogenase (LDH, ULN 235 U/L), direct bilirubin (Bili dir, ULN 5

mmol/L), and total bilirubin (Bili tot, ULN 26 mmol/L). Abnormal liver function tests were defined as values above the upper limit of normal. To account for confounding by drug-induced liver injury, we investigated medication use of all patients that showed either a hepatocellular profile (ALT O3 ULN), a cholestatic profile (ALP O2 ULN, ALT/ALP !2) or a mixed profile (ALP and ALT OULN) of liver injury, according to Chang et al.19 In addition, we also screened patients with liver function tests values higher than 5 times ULN. After exclusion of subjects on possible hepatotoxic medication, a history of hepatitis or substance abuse, and missing laboratory samples within 3 days before right heart catheterization, 323 heart failure patients were available for the present analysis.

Medical Therapy for CVD and Liver Damage Several drugs which are widely used for the treatment of CVD are known to be able to cause liver damage [8]. Amiodarone, which is classified as a class III antiarrhythmic drug, has multiple electrophysiologic properties.

Amiodarone has been used for the treatment of life-threatening ventricular arrhythmias and atrial fibrillation in patients with HF. Amiodarone is extensively concentrated in tissues including the liver, which explains its organ specific adverse effect. Amiodarone has been used for the treatment of life-threatening ventricular arrhythmias and atrial fibrillation in patients with HF. Amiodarone is extensively concentrated in tissues including the liver, which explains its organ-specific adverse effects.

Regarding adverse effects on the liver, abnormalities in liver function tests, hepatitis, and cirrhosis may occur. Amiodarone induces liver steatosis histologically resembling alcohol-induced liver injury. Amiodarone interferes with oxidative phosphorylation, which causes adenosine triphosphate depletion. Adenosine triphosphate depletion leads to the reduced activity of the smooth endoplasmic reticulum Ca2+ pump, which produces endoplasmic reticulum stress and lipid accumulation. Because amiodarone is lipid-soluble and has a prolonged half-life, the adverse effects can continue for a long duration even after its withdrawal. Thus, its usage needs cautious monitoring including liver function tests. Enalapril, one of the angiotensin-converting enzyme inhibitors that are widely used as a treatment of HF or antihypertension, has been reported to possibly cause liver damage. Whereas several drugs cause liver damage, there are drug treatments for HF that are reported to improve liver conditions. Sodium-glucose co-transporter-2 inhibitors are oral antidiabetic drugs that inhibit renal proximal tubules from reabsorbing glucose and increase urinary glucose excretion. Sodium-glucose co-transporter-2 inhibitors have some benefits beyond their glucose-lowering effects, such as promoting natriuresis and osmotic diuresis based on glycosuria. These effects advantageously affect patients with HF as they decrease cardiac preload and improve prognosis.

Thus, current guidelines for the management of HF recommend the use of sodium-glucose co-transporter-2 inhibitors for patients with both HF with reduced ejection fraction and HF with preserved ejection fraction regardless of the presence of diabetes mellitus. Because sodium-glucose co-transporter2 inhibitors have noninsulin-dependent glucose-lowering effects, their advantages as a treatment for diabetes mellitus are also expected for patients with impaired insulin resistance such as obesity. For these patients, sodium-glucose co-transporter-2 inhibitors have also been reported to improve liver conditions. Verapamil, which is a calcium channel blocker with negative inotropic, chronotropic, and dromotropic effects, is reported to have a protective effect against liver damage. Previous studies showed verapamil reduced inflammation, insulin resistance, and liver steatosis in mice models of high-fat diet-induced obesity. Nifedipine, which is one of the representative calcium channel blockers as antihypertensive drugs, is also reported to improve liver damage. In a previous report, nifedipine decreased fibrosis and the serum level of aspartate aminotransferase by upregulating the peroxisome proliferator-activated receptor-γ receptor in rats with NAFLD induced by an L-methionine and choline-deficient diet. Although these medication therapies have not been established in patients with comorbidity of both CVD and liver disease, it may be expected that they can be optional treatment strategies to prevent the development of CVD and improve their prognosis.

In conclusion, CVD and liver disease interact with each other, and the evaluation of both diseases is important. It may be sometimes difficult to distinguish their interaction and confounding effects between them in a clinical setting because these diseases share similar risk factors and basic pathologies. Although this limitation remains, the importance of both CVD and liver disease has been increasing in the current era of ageing populations. Especially, early detection and prevention of CAD are thought to be important, considering its high impact on health prognosis. Non-invasive and convenient approaches for the assessment of liver conditions such as the FIB-4 index may be efficient for daily practice to help the management of CVD. Thus, cardiologists must have a knowledge of liver diseases to understand the pathologies and risks associated with CVD. However, whether management and interventions for liver disease improve the prognosis of CVD has not been fully understood. As above mentioned, several treatments, such as diet therapy and medication therapy, including sodium-glucose co-transporter-2 inhibitors, are reported to possibly improve the liver condition. In addition, several anti-fibrotic drugs are currently under investigation.

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