

Laboratory and Instrumental Characteristics of Chronic Hepatitis C

Shamsiyeva Muattar Axmatovna

Bukhara State Medical Institute Named After Abu Ali Ibn

Sina Uzbekistan Bukhara, Gijduvon Street 23,

<https://orcid.org/0009-0003-1895-3352>

shamsiyeva.muattar@bsmi.uz

e-mail: info@bsmi.uz



Abstract

In terms of fibrosis and disease progression, the relationship between HCV and steatosis is unclear. HCV genotype 3 is clearly associated with steatosis. In some patients, steatosis may be associated with the development of fibrosis. The mechanisms underlying this association are unknown. This article describes and discusses the main and clinical-laboratory and instrumental aspects of the relationship between the development of steatosis and fibrosis and the response to treatment in patients with chronic hepatitis C.

Keywords: Chronic hepatitis C, hepatotropic virus, hepatocytes, fibrosis tissue.

Introduction

Hepatitis C (HCV) is a hepatotropic virus that disrupts liver metabolism and leads to progressive liver damage. [1-4]. The mechanism of origin of steatosis occurs when the accumulation of fat in the liver exceeds the amount of fatty acid that can be released as a lipoprotein of very low density or oxidized as an energy source. The main causes of fatty acid accumulation in the liver are listed below. Increase the absorption of fatty acids by the liver. [5-8]. Fatty acids are mobilized from peripheral adipocytes under the influence of the hormone-sensitive lipoprotein lipase. Insulin resistance leads to increased lipolysis with the accumulation of circulating fatty acids; this is followed by increased passive intake in hepatocytes, and as a result, increased glycolysis, insulin resistance hyperinsulinemia stimulates glycolysis with fatty acid synthesis, which is caused by a decrease in fatty acid removal due to a violation of mitochondrial β -oxidation density as low lipoproteins, a decrease in the export of fatty acids excreted from apolipoprotein B-linked hepatocyte causes steatosis. [9-12]. Currently, chronic hepatitis C is among the leading causes of liver transplantation, and it is estimated that 242,000 people worldwide die each year from hepatitis C-related complications. HCV itself can create hepatic steatosis through viral genotype-dependent mechanisms. [13-16]. Both mild and chronic hepatitis C can independently cause liver-related complications, and their coexistence can have additional effects on liver health. The chronic form of hepatitis C with steatosis is a chronic inflammatory process of the liver in which fat accumulation (steatosis) may occur in liver cells. [17-20]. This condition is one of the common

schemes of liver disease and necessitates a detailed study of its clinical, laboratory and instrumental appearance. Hepatitis C is mainly caused by a viral infection as well as other factors (such as obesity, high alcohol consumption, Type II diabetes and hyperlipidemia). [21-28]. In humans, hepatitis C virus (HCV) genotype 3 is more associated with steatosis. In vitro studies suggest that HCV core protein (genotype 1) can induce lipid accumulation within hepatocytes. Chronic hepatitis C with steatosis may experience the following clinical symptoms: patients often experience fatigue, undescribed pain, and abdominal discomfort. In some cases, yellowing may appear on their skin. [29-35].

The purpose of the scientific work:

To investigate whether steatosis can be associated with the development of fibrosis and whether it can be genotype-specific through clinical-laboratory and instrumental investigations.

Methods:

Palpation, blood tests, laboratory indicators, ultrasound, computed tomography, magnetic resonance imaging are planned.

Results:

The results that we recorded in 40 people with the disease through the following examinations: - Blood tests: even in 40 of our patients, the level of infection is determined by the detection of an antithela-antigen complex (e.g. anti-HCV, HBsAg) on hepatitis viruses.

- Laboratory Indicators

Transaminases (ALT and AST) are enzymes that show damage to liver cells, and we can see that steatosis — induced hepatitis C has changed in 20 of our patients. Gammaglutamyltransferase (GGT) is an enzyme that has been observed to increase in high levels in steatosis as well [36-43]. Triglycerides—an increase in liver fat can lead to an increase in triglyceride levels. Also liver enlargement (hepatomegaly), sometimes splenomegaly was found in 22 (55 %) of our patients. In all 40 of our patients, an Uzi test revealed an accumulation of fat.

Computed tomography (CT) was carried out in 15 of our patients, and the results once again proved the conclusion of the Ultrasound examination. In this case, information was obtained about the amount and distribution of fat by X-rays of the liver.

Conclusion:

There are many unanswered questions and patients with hepatitis C need large prospective studies using multidimensional analysis, taking into account steatosis, fibrosis and confusing factors to respond to treatment. A meta-analysis of individual patient data is ongoing to study the relationship between steatosis, inflammation, and fibrosis in chronic hepatitis C. Chronic hepatitis C often occurs in conjunction with hepatic steatosis. This aggravates the course of the disease and can reduce the effectiveness of antiviral treatment. Hepatic steatosis is characterized by the

accumulation of fat in hepatocytes in the hepatitis C virus. It has been proven through investigations that this can lead to the development of inflammation and fibrosis

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