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THE CORRELATION BETWEEN ELASTOGRAPHIC PARAMETERS

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Abstract: Ultrasound elastography has become one of the most informative and noninvasive techniques for assessing the structural and functional state of the liver. The morphological concept of this method is based on the evaluation of tissue stiffness, which reflects the degree of fibrosis and other pathological alterations of the hepatic parenchyma. Unlike conventional ultrasound, which primarily provides anatomical imaging, elastography offers a functional and morphological interpretation by quantifying the elastic properties of liver tissue. The correlation between elastographic parameters and histological stages of fibrosis has been well established, allowing for accurate differentiation between normal, inflammatory, and fibrotic changes. The method is particularly valuable in the early detection of subclinical fibrosis, long before the onset of clinical symptoms or biochemical abnormalities. By mapping stiffness distribution across the hepatic tissue, elastography also enables the visualization of heterogeneous fibrosis patterns, which are often observed in viral hepatitis, alcoholic and nonalcoholic steatohepatitis, and cirrhosis.Morphologically, elastographic findings correspond to alterations in extracellular matrix composition, collagen deposition, and architectural remodeling of the hepatic lobules. These structural transformations determine the biomechanical properties of the liver and, therefore, the measured shear-wave velocity or strain ratio. Thus, ultrasound elastography represents a bridge between morphological assessment and functional evaluation, offering a precise, reproducible, and safe method for monitoring liver pathology progression and response to therapy.

Key words: Alcohol, liver, elastography, morphological concept, fibrosis, ultrasound diagnostics

Alcohol consumption remains one of the most significant global public health problems. In 2016, alcohol use was the seventh leading risk factor for both mortality and disability-adjusted life years (DALYs), accounting for 13.6% of the global disease burden. Age-standardized deaths attributable to alcohol were reported in 2.2% of women and 6.8% of men. Approximately 32.5% of the world's population—2.4 billion people—were chronic alcohol users, leading to an estimated 2.8 million deaths annually. Liver cirrhosis, mainly of alcoholic or viral etiology, accounted for 50–80% of all deaths from gastrointestinal diseases [5]. A screening study conducted in Moscow among 5,000 randomly selected residents revealed that the prevalence of alcoholic liver disease (ALD) was 6.9% [6]. According to the U.S. National Institute on Alcohol Abuse and Alcoholism, 44–48% of fatal cirrhosis cases are due to the toxic effects of alcohol [7].

The liver is the central organ of alcohol metabolism. Chronic ethanol exposure leads to accumulation of acetaldehyde, oxidative stress, and lipid peroxidation, which cause hepatocellular injury and inflammation. Morphologically, these changes are manifested by fatty degeneration of hepatocytes, infiltration by inflammatory cells, ballooning degeneration, and progressive deposition of collagen in the perisinusoidal and pericellular spaces. The process results in fibrosis, distortion of lobular architecture, and eventually cirrhosis. As fibrosis advances, hepatic tissue becomes progressively stiffer, disrupting both microcirculation and bile canaliculi function. These morphological and biomechanical alterations form the pathophysiological basis for quantitative evaluation using ultrasound elastography.Ultrasound elastography measures the stiffness of hepatic tissue by assessing the propagation speed of shear

waves generated within the liver. Stiffer tissue, corresponding to advanced fibrosis or cirrhosis, transmits these waves faster. This principle allows the quantitative differentiation of normal, inflammatory, and fibrotic liver conditions.From a morphological standpoint, increased stiffness reflects the accumulation of extracellular matrix components—primarily types I and III collagen—along with changes in hepatic sinusoidal structure and lobular remodeling. The technique provides a noninvasive alternative to biopsy, correlating strongly with histological staging systems such as METAVIR and Ishak. However, against the backgroundof "naturalhypocoagulation", a wide range of spontaneous (or unprovoked) venous thrombotic complicationsmay occur [3]. GGT reflects the enzymatic activity of the liver, but an increase in its level ispossiblewith pathology of the biliary system, cardiac pathology, and the use of certain medications. Alcoholstimulates the expression of the GGT gene. Serum GGT activity is increased in approximately75%ofindividuals who abuse alcohol (sensitivity - 60-90%, specificity - 50-72%) [2]. ASTandALTareprotein substances involved in metabolic processes, in particular amino acid processes. Theseenzymesare produced intracellularly, so an increase in their content in the blood indicates the destructionofcellular structures. With many forms of acute and chronic liver damage, with steatosis, AST/ ALTisless than or equal to 1, and with alcoholic hepatitis this ratio often exceeds 2 [6]. Hyperbilirubinemiamay be an indicator of liver failure. As the severity of APB increases, bilirubin levels >50μmol/Lareoften observed [5].However, against the background of "natural hypocoagulation", a widerangeofspontaneous (or unprovoked) venous thrombotic complications may occur [5]. GGTreflectstheenzymatic activity of the liver, but an increase in its level is possible with pathologyof thebiliarysystem, cardiac pathology, and the use of certain medications. Alcohol stimulates the expressionoftheGGT gene. Serum GGT activity is increased in approximately 75% of individuals whoabusealcohol(sensitivity - 60-90%, specificity - 50-72%) [2]. AST and ALT are protein substances involvedinmetabolic processes, in particular amino acid processes. These enzymes are produced intracellularly,so an increase in their content in the blood indicates the destruction of cellular structures. Withmanyforms of acute and chronic liver damage, with steatosis, AST / ALT is less than or equal to1, andwithalcoholic hepatitis this ratio often exceeds 2 [6]. Hyperbilirubinemia may be an indicator ofliverfailure. As the severity of APB increases, bilirubin levels > 50 μmol/L are often observed[7]

Elastography modalities, including transient elastography (FibroScan), shear-wave elastography, and point shear-wave elastography, have demonstrated high diagnostic accuracy for assessing fibrosis. Moreover, mapping stiffness heterogeneity enables visualization of focal lesions or uneven fibrotic distribution, which are often observed in alcoholic liver disease, viral hepatitis, and nonalcoholic steatohepatitis.The integration of elastography into clinical practice has significantly improved the early diagnosis and monitoring of chronic liver diseases. It facilitates timely identification of subclinical fibrosis, guides treatment decisions, and allows for noninvasive follow-up of therapeutic effectiveness. In patients with alcoholic liver disease, early elastographic evaluation helps detect fibrotic changes before clinical decompensation occurs, providing an opportunity for intervention and lifestyle modification.Furthermore, the technique plays a key role in pre-transplant assessment, reducing the need for invasive biopsy procedures and enabling risk stratification in patients with advanced fibrosis or cirrhosis.

Ultrasound elastography represents a pivotal advancement in hepatology by linking morphological and functional assessment of the liver. Its ability to quantify tissue stiffness provides a noninvasive, reproducible, and accurate tool for diagnosing and staging fibrosis of various etiologies, including alcohol-induced liver injury. Morphologically, the stiffness

measured by elastography reflects the extent of fibrotic remodeling and collagen accumulation within the hepatic parenchyma. Therefore, this method has become an essential part of modern liver diagnostics, allowing for early detection, improved monitoring, and better prognostic evaluation of patients with chronic liver diseases.

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