Journal of Advanced Medical and Dental Sciences Research

@Society of Scientific Research and Studies ***NLM ID:*** *101716117*

Journal home page: www.jamdsr.com doi: 10.21276/jamdsr Index Copernicus value = 85.10

(e) ISSN Online: 2321-9599; (p) ISSN Print: 2348-6805

Review Article

Ultrasound Diagnostics of Portal Hypertension

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***ABSTRACT*:**
Portal hypertension is one of the main causes of severe gastroesophageal bleeding in children, often leading to death. In addition, hepatic or renal failure may be the outcome of portal hypertension in children with liver disease. In this regard, it is extremely important to timely recognize the syndrome itself and establish the causes of its development.
Keywords: portal hypertension, echo examination, ultrasound diagnostics, diagnostics.

Received: October 24, 2020 Accepted: November 28, 2020

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This article may be cited as: Yangieva S. Ultrasound Diagnostics of Portal Hypertension. J Adv Med Dent Scie Res 2020;8(12):118-120.

INTRODUCTION

Portal hypertension in patients with chronic liver disease leads to serious complications such as bleeding from gastroesophageal varices, ascites, and portosystemic encephalopathy. The gold standard methods for assessing portal hypertension and its complications include hepatic venous pressure gradient measurement and endoscopy, but they are invasive and not available at all centers. Therefore, non-invasive alternative diagnostic methods have become the subject of extensive research over the past 20 years. Hemodynamically portal hypertension is determined by an increase in the gradient of venous pressure in the liver, calculated from its inflow through the portal vein compared to its outflow through the hepatic veins [1].

Like any other vascular system, portal pressure is the result of two independent factors: resistance to blood flow and volume of flow, as defined by Ohm's law:

Pressure = Resistance × Blood flow.

An increase in resistance to portal blood flow is the initial factor that leads to an increase in portal pressure. This resistance can be located at any point in the hepatic blood flow, that is, at the prehepatic, intrahepatic, or posthepatic levels. About 90% of all cases of portal hypertension are caused by progressive chronic liver disease or cirrhosis. Portal hypertension may remain asymptomatic for many years, but imaging and laboratory testing may indicate its presence. Splenomegaly is a very common consequence of portal hypertension, usually resulting in thrombocytopenia due to hypersplenism [2], and is often the first manifestation of portal hypertension.

Clinically, portal hypertension is important because of its severe complications, which include gastroesophageal bleeding, ascites, spontaneous bacterial peritonitis, hepatorenal syndrome, and hepatic encephalopathy [3]. Early identification of patients with an increased risk of developing clinical complications is extremely important, since it can be effectively used to correct portal pressure in these patients by medical, surgical or other alternative methods [4, 5].

Currently, the main methods for diagnosing extrahepatic portal hypertension in pathology of the pancreatoduodenal zone are non-invasive, with high sensitivity and specificity. Ultrasound examination using color duplex scanning makes it possible to assess the state of the pancreas and anatomically interconnected organs, to reveal compression or thrombosis of the portal veins, and to visualize dilated venous collaterals.

PATHOGENESIS OF PORTAL HYPERTENSION

The pathogenesis of portal hypertension in different types of blockade of the portal system is different. The most common cause of the hepatic form of hypertension is cirrhosis of the liver, when, against the background of inflammation, sclerosis occurs, followed by fibrosis of the portal tracts, the

development of regeneration nodes, disruption of the structure of the hepatic lobules, compression of vascular structures, including intrahepatic branching of the portal vein [6, 7].

Violation of the organization of the lobules leads to a change in the intralobular circulation and is the cause of the development of necrosis in the center of the lobule, fibrosis and pronounced cellular infiltration with the proliferation of Kupffer cells. The latter can go into the lumen of the sinusoids, narrow them, leading to an increase in pressure in the portal system. Obstruction of blood flow through the portal vein system and an increase in portal pressure lead to an increase and hyperplasia of the spleen.

Further, portal hypertension causes the development of collaterals of both natural and new portocaval connections: through the coronary vein of the stomach to the venous plexus of the esophagus and then through the azygos and semi-unpaired veins with the superior vena cava system. The veins of the esophagus expand. Vv.epigastricae participate in the development of portocaval anastomoses, forming the "jellyfish head", which is more often observed in adult patients. Cruveillier-Baumgarten syndrome is most common in childhood with congenital liver fibrosis [8].

With the development of portocaval collaterals through v. Mesentericae inferior, hemorrhoidal veins increase. Stasis in the portal and lymphatic systems of the abdominal organs and impaired inactivation of corticosteroids, in particular, aldosterone, play a role in the occurrence of ascites developing in portal hypertension. In contrast to the mechanism of intrahepatic portal hypertension, extrahepatic, at first glance, seems to be simpler.

The virtual absence of the trunk and branching of the portal vein in congenital genesis of development, obliterated or narrowed its lumen as a result of thrombosis or extravasal compression leads to an increase in blood flow resistance, leading to a stable increase in portal pressure up to 300-500 mm of water column) and the development of a hyperdynamic type of hemocirculation in the vessels of the portal vein basin, with pronounced arteriovenous shunting in the organs, increasing the load on the vessels, with their subsequent morphological changes. Their severity depends on the duration and severity of the course of the disease [9, 10].

The development of portal hypertension with adrenal blockade of blood circulation is most often caused by restrictive pericarditis, narrowing of the hepatic veins or inferior vena cava above its confluence with the hepatic veins (Chiari disease), narrowing of the inferior vena cava and / or hepatic veins as a result of a congenital septum, thrombus, tumor, hypertrophied caudate lobes of the liver, which ultimately leads to a disorder of blood and lymph circulation in the organs and tissues of the underlying sections [11, 12, 13].

ULTRASONIC DIAGNOSTICS OF PORTAL HYPERTENSION

The main methods of studying patients with suspected portal hypertension, in addition to the generally accepted physical ones, are angiographic, endoscopic and ultrasound in combination with Doppler sonography (in color and normal modes). All of these methods complement each other. Relative competition can be discussed between angiography and Doppler imaging [14].

However, given the harmlessness of the ultrasound method and the relative ease of its use, it must be recognized that it should be used first in every child with an enlarged spleen and other signs that suggest portal hypertension. And then, focusing on the results obtained, decide on the conduct of X-ray contrast studies. The most common form of portal hypertension in children compared to others is extrahepatic blockade of the portal circulation, the frequency of which in the differential diagnosis of portal hypertension reaches 50-88%. Moreover, in adults, this figure is within 10% [1, 2]. This difference is explained by the fact that the main reason for the development of hypertension is the primary lesion of the veins of the portal system, which in some cases is accompanied by fibrosis of the liver parenchyma.

Echographic examination of the abdominal organs in children with suspected extrahepatic form of portal hypertension in almost 100% of cases allows correct diagnosis. The liver is usually of normal or reduced size. The latter is due to a significant decrease in portal blood flow with the development of parenchymal fibrosis, observed in 60-70% of children. In congenital fibrosis, multiple small strong echo signals are recorded from the parenchyma, scattered over the entire area of the organ cut. The portal vein is changed, while there may be a lack of lumen of its trunk or main branches, the presence of multiple thin convoluted vessels with hyperechoic walls, collected together - angiomatous transformation, the presence of a thrombus that narrows or closes the lumen of the vessel, or local narrowing of the lumen, which leads to the development of collaterals visible as anechoic narrow, convoluted tubular structures adjacent to the main trunk. Such changes in the portal vein lead to a significant slowdown in blood flow. Color Doppler imaging facilitates a qualitative assessment of the state of the portal vein, allowing to determine the direction of blood flow (hepatopetal - to the liver or hepatofugal - from the liver) [15].

Portal hypertension in its extrahepatic form gradually leads to a number of secondary changes. The spleen enlarges, there is a small-focal or diffuse small-focal compaction of the parenchyma, hardening of the walls of small vessels, some may be thrombosed. The splenic vein is dilated, often twisted in the area of the gate. The blood flow velocity through it, as a rule, remains, but the volumetric blood flow rate increases significantly. The splenic artery is slightly dilated, which leads to an increase in the resistance index and

indicates an increase in peripheral resistance, indirectly confirming the echography data on the state of the spleen parenchyma [16, 17, 18].

In severe portal hypertension, venous collaterals develop, some of which can be seen: these are coronary, gastroesophageal, defined between the left lobe of the liver and the head, the body of the pancreas as anechoic round or tubular structures. Esophageal varices can be identified by longitudinal scanning in the aortic region. Due to the different direction of the blood flow due to the tortuosity of the vessels, color duplex examination demonstrates its mixed nature. When examining the liver with fibrosis, a passable umbilical vein is noted, which sometimes has uneven walls and is directed from the region of the portal vein bifurcation to the liver surface in the cephalic-lateral direction [19, 20].

CONCLUSION

Thus, with a complicated course of liver cirrhosis, violations of the rheology of blood, bile, caused by structural and morphological changes in blood cells, hepatocytes, are determined. In the diagnosis of portal hypertension, the most optimal and informative are non-invasive, invasive and angiographic research methods.

ACKNOWLEDGEMENTS

We are grateful to the staff members of Tashkent Pediatric Medical Institute for the cooperation and support in our research.

CONSENT

Written informed consent was obtained from all participants of the research for publication of this paper and any accompanying information related to this study.

CONFLICT OF INTEREST

The authors declare that they have no competing interests.

FUNDING

No funding sources to declare.

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