

LIVER CIRRHOSIS – ULTRASOUND ASPECTS OF PORTAL CIRCULATION

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Abstract

Doppler ultrasound is a non-invasive method for assessing vascular port system. The aim is to study the changes in structure of the liver with the portal flow in patients with cirrhosis. Evolution of liver cirrhosis with portal hypertension makes the essential changes of portal vein. One of the objectives of the study was the investigation of the Doppler axis spleno-portal hemodynamics, evaluated and analyzed in relation to liver functional reserve, which we estimated classified as Child - Pugh. Our study followed 187 patients with cirrhosis and portal hypertension. The study is retrospective, based on analysis of inpatient observation sheets in County Emergency Hospital, Timisoara, during the last five years. Sectional area of the hepatic portal vein is another criterion for evaluation of patients with cirrhosis area we reported a study in functional classes: Child class "A" → VP sectional area = 175 mm² class Child "B" → sectional area VP = 236 mm²; Child class "C" → VP sectional area = 183 mm².

Key words: vascular port system, Doppler ultrasound, portal hypertension.

Introduction

Liver cirrhosis represents the tenth death cause worldwide, according to the latest statistical data. The frequent complications which may appear in this disease are: ascites (50% of the patients develop ascites in a period of 10 years since the diagnosis), hepatic encephalopathy and variceal bleeding (25 % of the patients), while portal hypertension is the result of the increased intrahepatic resistance and portal blood flow. (1)

The incidence of the liver cirrhosis is not well known in Romania. The majority of the patients who come to the doctor due to the ascitic syndrome, have liver cirrhosis (75%), the other etiologies being rarely come across: malign

tumors (10 % of the cases), heart failure (3%), peritoneal tuberculosis (2%), chronic pancreatitis (1%) etc.

Doppler Ultrasound represents an invasive method of evaluation for the vascular port system. The purpose of the study is that of changes in liver's structure determination along with those of portal blood flow, in the case of patients with liver cirrhosis. Several diagnostic elements are considered in favor of liver cirrhosis:

- Hepatic structure: it is modified at 1/ 2 of the cases; it is heterogeneous and scratchy.
- Liver surface: it is wavy, micro or macro wane (nodules bigger than 5 mm);
- Caudate lobe hypertrophy: in about 70-80 % of the cirrhosis, the anterior-posterior diameter is > 35-40 mm;
- The presence of the portal hypertension signs: hepatic portal vein dilation over 14 mm (the normal value is up to 13 mm), lack of variability in hepatic portal vein in forced inhale or exhale , enlargement of the splenic vein > 10 mm (preaortic), umbilical vein , re-permeability of the umbilical vein ,the presence of ascites (2).
- Ascites and splenomegaly are not always specific to liver cirrhosis;

The latest data invoke a relatively high frequency of the portal thrombosis to cirrhotic patient. The role of this phenomenon in the development of portal hypertension's complications is not well defined nowadays, fact which requires further research in this domain (3, 4).

Material and method

Ultrasonography is an invasive, anatomic and functional examination method, which allows the simultaneous viewing of both the parenchyma and the hepatic vessels.

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Ultrasound examination of the vascular system port has the following indications: the diagnosis of portal hypertension, etiologic diagnosis, and evaluation of portal and systematic shunt surgery, evaluation of hepatic transplant even in pharmacological and dynamic studies of portal hypertension (5).

The evolution of hepatic cirrhosis with portal hypertension leads to essential changes of hemocirculation in the hepatic portal vein basin. One of the study's objectives is that of hemodynamics' Doppler research on the spleno-portal axis evaluated and analyzed in relation to

functional liver reserves, which we estimated according to Child- Pugh classification, with the severity score assessment of liver function disorders. (CHART 1).

Our group had 187 patients with liver cirrhosis and portal hypertension; they were endoscopically treated for hemorrhages of esophageal varices and for ascitic syndrome associated with splenomegaly and hypersplenism. The study is a retrospective one, being based on the analysis of the observation sheet of the patients which have been hospitalized in County Emergency Clinical Hospital of Timisoara during the last 5 years.

CHART 1: Child - Pugh criteria of liver functional reserve assessment.

INDEX	VALUE		
	1 point	2 points	3 points
Albumin (g/l)	>35	30 - 35	<30
Bilirubin (mmol/l)	<35	35 - 50	>50
Prothrombin (%)	>70	40 - 70	<40
Ascites	absent	insignificant	moderate/severe
Encephalopathy	absent	insignificant	moderate/severe

According to this classification, the patients of Child "A" category gather 5 or 6 points, Child "B" → 7 - 9 points, Child "C" → 10-15 points; this classification was obtained depending on the score of the estimated factors, which are to be seen in the above chart.

Results and discussions

We have studied 187 patients with liver cirrhosis (111 males and 76 females) (Diagram 1).

With the age between 18 and 82 years old; 115 patients were from urban and 72 were from rural; the diagnosis to these patients was established on the basis of clinical, biochemical, endoscopic, imaging and morphological criteria.

The etiology of liver cirrhosis was made by the identification of serological markers of viral hepatitis to all patients. Cirrhosis of viral etiology was come across in 102 cases-representing 54,5% of the total.

The etiological specification certified the essential role of chronic viral infection which certainly leads to chronic, persistent and often worsening clinically asymptomatic hepatitis in its future development. Serological investigations did not notice a statistically significant difference regarding the etiology of liver cirrhosis and patients' distribution by gender. Thus, in our group, the viral factor was identified to 64 (57.7%) out of 111 investigated males and respectively, to 45 (59,2%) of 76 females.

There were studied the particularities of portal blood circulation depending on the hepatic functional reserve.

The appreciation of hepatic functional reserves according to Child- Pugh classification of the patients with acute hemorrhages (due to esophageal varices) found compensated hepatic functions → Child "A" in 29 cases

(15,5%), sub compensated → Child „B" – 102 cases (54,4%) and decompensated → Child „C" in 56 cases (30,1%) (Diagram 2). Thus, in the group of the patients with hepatic cirrhosis, complicated with active hemorrhages, due to esophageal varices, remarked a certain prevalence of patients with sub compensated and decompensated functional hepatic status (84,5%).

We found out that to Child "A" and "B" patients, it is established a spleno-portal flow with large hyperkinetic volume and while the cirrhosis progresses to the decompensation stage, these indexes essentially decrease.

The asymmetry phenomenon of intrahepatic portal blood circulation was demonstrated and manifested by right liver lobe hemodynamic deprivation and prevalence of left portal branch circulation at the same time with the increase of splenic venous inflow (6).

The area of hepatic portal vein constituted another criterion of the patients' evaluation, area which we reported to the studied functional classes: Child „A" class → sectional area VP = 175 mm² Child „B" class → sectional area VP = 236 mm²; Child „C" class → sectional area VP = 183 mm² (Diagram 3).

Once with the decrease of hepatic functional reserves, one could notice a tendency to hypertrophy of the left liver lobe. Thus, to the patients in Child A, B, C categories, the ultrasound dimensions of LHS were the following: 74 mm / 81 mm / 87 mm (Diagram 4).

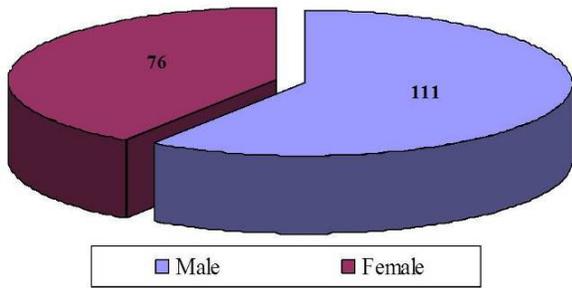


Diagram 1: Distribution by gender.

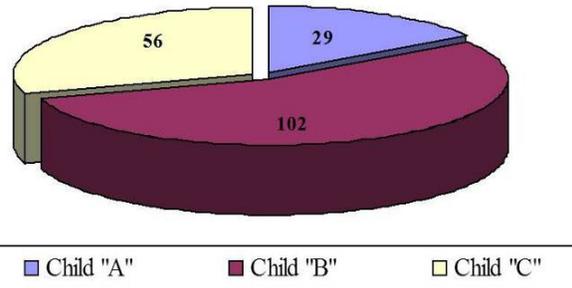


Diagram 2: Distribution of our group according to Child- Pugh classification.

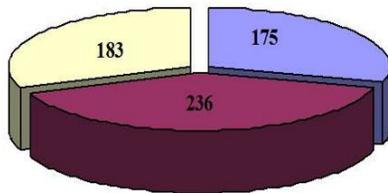


Diagram 3: Sectional area of the hepatic portal vein to the studied functional classes.

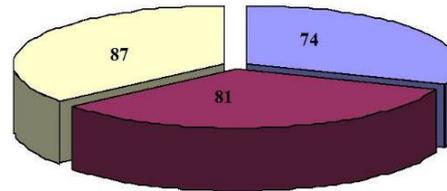


Diagram 4: Ultrasound dimensions of the liver left lobe, in Child-Pugh categories.

The ultrasound should always be interpreted in the clinical context of the patients. The information anterior to examination is extremely important in performing the ultrasound and drawing the attention of the doctor who performs the ultrasound to a certain organ or region.

The liver can be sonographically normal or increased with the hepatic portal vein and main branches thickening which become highly echogenic, especially around the

hepatic portal vein. Splenic veins can be dilated and, if there is portal hypertension, there is also splenomegaly. An increase in collateral circulation can occur around the splenic hilum and along the medial edge of the liver (Figure 1, Figure 2). This can be seen under the form of rigid vascular structures, without echo, which must be distinguished from fluid-filled intestines.



Figure 1: The ultrasound aspect in hepatic cirrhosis (I.S., 47 years old, diagnosed with alcoholic liver cirrhosis).

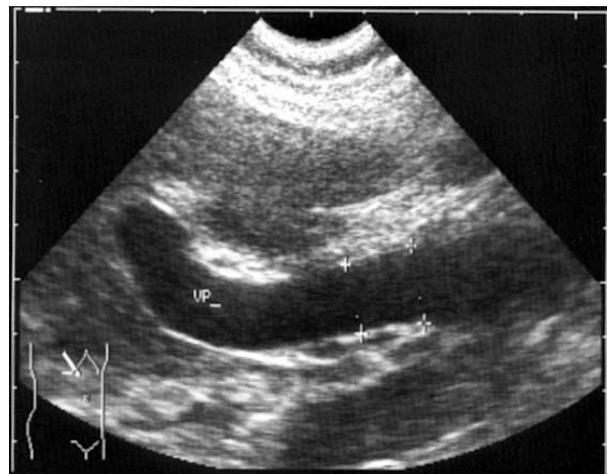


Figure 2: Signs of portal hypertension: VP dilation (21 mm) (C. A., 32 years old, diagnosed with virus liver cirrhosis).

All the patients were endoscopically evaluated for the staging of esophageal varices: 12 patients did not have esophageal varices (6,4 %), 31 patients had esophageal

varices of first rank (16,6 %), 101 patients had esophageal varices of second rank (54%) and 43 patients had esophageal varices of third rank (23%) (Diagram 5).

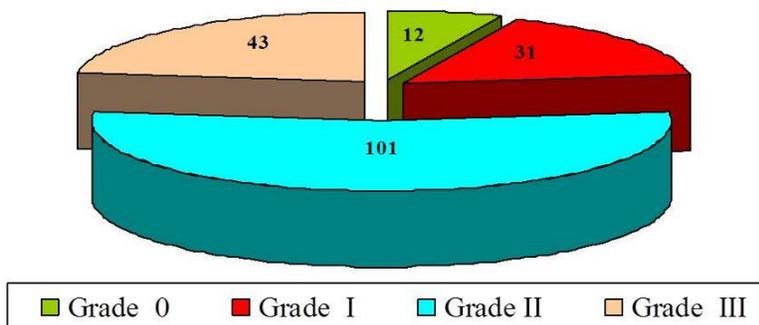


Diagram 5: Distribution of the group according to the rank of esophageal varices.

Conclusions

1. Extremely useful, noninvasive, without the risk of nephrotoxicity given by the contrast substance (as it happens in the case of computer tomography), cost-good efficiency, unlaborious and available- it is the abdominal ultrasound, the first investigation to be performed in the case of cirrhosis suspicion.
2. The portal hypertension is determined by multiple and diverse etiopathogenic factors, primarily based on angioarchitectonic and morphological denaturation of liver.(7)
3. The gradual changes of liver parenchyma lead to an increased vascular resistance to the portal flow, at the same time with a splanchnic hyperemia with excessive blood inflow. These mechanisms, initially adaptive and compensatory are then reflected by expressive disturbance of portal hepatic hemo -circulation and adversely affect the liver status.
4. In portal hypertension a part of the portal blood volume is derived through portal-systemic shunts, intra and extra hepatic, thus, the functional hepatic flow stream being reduced; it is a hemodynamic phenomenon which introduces the decompensation of liver functions.
5. Once with the diminution of functional liver reserves and increase of intrahepatic vascular resistance, there occurs a decreased portal blood flow and liver functional current. (8). That finds its explanation through the emergence, development and functioning of multiple intrahepatic portal –systemic anastomosis with a substantial deviation and redirection of the portal blood flow to the systemic one within portal hypertension.
6. According to Gorka and Plestina (9, 10), the splenic flow venous report in relation to the portal one

represents a predictive factor in the development of esophageal and gastric varices and in digestive haemorrhages bleeding by triggering their eruption and intensifying portal gastropathy, altogether with worsening of secondary portal hypertension splenopathy; this fact was also found in our studied group.

7. By estimating the role of splenic venous hemocirculation in portal hypertension's progressing, Kutlu and his co-workers (11) record a right liver lobe hemodynamic deprivation altogether with a decrease in velocity and in portal blood flow on this intrahepatic branch , while mentioning a marked venous congestion. Even in the case of our group, I noticed a tendency of hypertrophy of the left liver lobe.
8. The hemorrhages in the case of patients with liver cirrhosis and portal hypertension have certain specific features to this pathology, because they trigger in extreme conditions of portal- hepatic circulation, distorted by congestion and regional vein stasis, trophic disorders of the gastro-esophageal mucosa, associated with dysfunctions of the coagulation system.
9. Esophageal varices occur in the case of an excessive portal pressure and result in shunting the venous flow in submucosal esophageal veins, while gastric varices are caused by portal blood flow derivation throughout the short gastric veins (12, 13).
10. Digestive bleeding by esophageal and gastric varices rupture is one of the worst complications of liver portal hypertension.
11. The analysis of laboratory indexes in our study group found significant changes in the values of prothrombin, bilirubin and liver enzymes.

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