**"OVIDIUS" UNIVERSITY OF CONSTANȚA**

**DOCTORAL SCHOOL OF MEDICINE**

# INVASIVE AND NONINVASIVE EVALUATION OF THE VENOUS PORTAL SYSTEM IN NORMAL AND PATHOLOGICAL CONDITIONS

**PHD THESIS SUMMARY**

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## CONTENTS

## INTRODUCTION - The importance of the chosen topic and the aim of the study

The portal venous system is a vast, complex subject that has shown a real interest over time, and research in this area is still vigorous, dynamic.

The evaluation of the venous port system is a necessity for any physician who is treating hepatic pathology (gastroenterologist, surgeon or medical imaging specialist). Classical methods are either invasive (measuring the pressure gradient in the hepatic veins, superior digestive endoscopy) or without high accuracy (2D standard abdominal ultrasound, color Doppler and pulsed). In recent years, however, non-invasive methods of assessing the venous system of the port are used in clinical practice, both morphologically (computed tomography or nuclear magnetic resonance), and functionally, hemodynamic (biological indexes and hepatic elastography - methods that have shown a certain correlation with the level of portal hypertension, the presence and severity of esophageal varices).

In this study, I aimed to evaluate the role of noninvasive methods (biological scores, hepatic elastography, abdominal ultrasound, computer tomography) in the descriptive and functional evaluation of the venous port system in normal and pathological conditions (the latter referring to CHRONIC liver disease: chronic hepatitis and compensated and decompensated hepatic cirrhosis) and to compare the noninvasive methods with invasive methods (upper digestive endoscopy, endoscopic ultrasound) in order to evaluate the possibility of replacing invasive classical invasive screening with non-invasive methods.

The secondary objectives were: to identify certain features of the portal vein formation, caliber, portal vein tributaries, to characterize the portal circulation hemodynamics in normal and pathological conditions, to study the porto-systemic shunts in patients with portal hypertension, to compare the diagnostic performance between modern endoscopic examination techniques examination such as virtual chromosomescopy with i-scan and standard endoscopy in the diagnosis of portal hypertension complications (esophageal varices and hypertensive portal gastropathy), to assess the role of endoscopic ultrasound in the evaluation of esophageal, parasophageal, gastric and eso-gastric varicose veins.

The study of portal hypertension has begun since the Gastroenterology Residency with a variety of cases. This allowed me to explore the invasive and non-invasive methods of investigating the portal venous system.

The general part begins with anatomy, which I consider particularly important, that in correlation with the physiological mechanisms helps to understand this complex system. As early as 1543, Vesalius created an anatomical image of the venous portal system, to which there was not much added. In the 1650s, 25 years after Harvey discovered blood circulation, Glisson determined that the portal vein represented the vessel through which blood was collected from the gastrointestinal tract and brought into the systemic circulation. In the anatomy section, I approached and described the portosystemic anastomoses, whose investigation, description is still occurring, being a topic widely debated by researchers since the 1930s.

In the personal part, I described the group of patients and the methods I used, describing the morphological and functional parameters I followed. In the next chapter, at discussions I compared the personal results with those described in the literature.

The last chapter refers to the findings of this research, focusing on the new, current non-invasive methods of investigating the portal system.

Finally, I would like to thank my mentor, Professor Bordei Petru and my resident coordinator, Professor Dumitru Eugen for their support and permanent help in carrying out this work.

## MATERIAL AND METHOD

The subjects analyzed in this study were patients admitted to the Gastroenterology Department of the Clinical Medical I, Constanta County Hospital between 01.09.2013 and 31.08.2016 (3 years) with the diagnosis of liver cirrhosis. The study was prospective.

All patients signed an informed consent when performing any invasive procedures such as upper gastrointestinal endoscopy or endoscopic ultrasound. For comparative analysis of results, a control group consisting of patients admitted to other gastroenterological diagnoses not related to chronic liver disease (e.g., patients with dyspepsia, gastritis, irritable bowel syndrome, hemorrhoidal disease, etc.) was used. Patients with active upper gastrointestinal bleeding, hepatic encephalopathy, spontaneous bacterial peritonitis, hepatorenal syndrome, primitive or secondary malignant liver tumors were excluded. Demographics, clinical elements and results of biological and imaging investigations were collected in Microsoft Excel files and were statistically analyzed.

*Diagnosis of liver cirrhosis.*

Diagnosis of liver cirrhosis was established based on anamnestic data (alcohol consumption, hepatitis B virus infection, C history), clinical (inferior margin of the liver, splenomegaly, ascites, edema, jaundice, spider naevi, abdominal collateral circulation ) and paraclinic (thrombocytopenia, hepatic insufficiency syndrome, hepatocytolitic syndrome, cholestasis syndrome, mesenchymal inflammation syndrome, viral markers, specific echographic or tomographic aspect). Patients were classified according to the CHILD class.

*Characteristics of the batch of patients studied.*

The patient population comprised 315 patients (178 male, representing 56.5% and 137 female, representing 43.5%), aged 37 to 75 years, diagnosed with cirrhosis of the liver.

*Biological investigations (haematological, biochemical, immunological, serological).*

Laboratory tests were performed at the Synevo Laboratory in the Clinical County Hospital of Constanta.

*Abdominal ultrasound.*

All patients enrolled in the study as well as patients in the control group were examined in the morning, or after 6 hours without eating. A Hitachi Aloka ProSound Alpha 7 ultrasound

with a UST-9147 convex multifrequency transducer and a Siemens Antares Premium ultrasound with a convex CH 6-2 transducer were used. Devices were provided with appropriate software for calculating hemodynamic parameters based on the Doppler spectral waveform.

All scans were performed with patients in dorsal decubitus for 15 minutes. Patients were analyzed by standard 2D ultrasound and Doppler color Doppler and pulsed Doppler. The examination began with the examination of gray-scale parenchyma. Subsequently, the examination followed with spectral Doppler. The problem of intestinal gas was overcome by prolonging scanning time or resuming the next day's examination. The haemodynamic parameters provided by the Doppler ultrasound can be classified as qualitative, quantitative and semi-quantitative. Qualitative data include assessing the presence, direction and characteristics of blood flow. Quantitative data include calculating velocity and flow in large vessels, such as the portal vein. To estimate changes to the Doppler path, I used semicantitative parameters, namely the pulsatility index (PI) and the resistivity index (RI) that provide hemodynamic information downstream from the site where they were measured.

*Hepatic elastography.*

Elastography is a non-invasive method that measures rigidity in a volume of the liver, which is approximately 100 times higher than that of a liver biopsy, and can be less prone to sampling error than biopsy. The hepatic resistance was measured with the Fibroscan in the dorsal decubitus position, in a jeun patients. Only results with a success rate over 60% and an IQR (interquartile range) of up to 30% of the mean hepatic elasticity were considered. The result is expressed in kiloPascals (kPa).

*Computed tomography.*

Computed abdominal tomography with contrast substance was performed in all cases where alpha-fetoprotein (AFP) was greater than 50 ng / mL for liver tumor screening. Of the total of 315 patients in the study, a contrast-enhanced CT was performed in only 72 patients (22.8%); these patients constituted the sublot of patients who entered the final analysis of the morphological imaging results of the venous port system.

Computed tomographies (CT) were performed at the Medimar Medical Center at the Emergency Clinical Hospital in Constanta. They were performed on a GE BrightSpeed Select 16 slice CT scanner. The abdominal stage examination technique included native acquisition and postcontrast intravenous iodine substance. Multidetector computed tomography (MDCT)

has allowed images to be acquired in arterial, portal, parenchymal phase imaging. All patients had a cubital peripheral venous approach and received a 1.5 mL / kg contrast substance (100-150 mL nonionic iodine contrast) by single shot injection on the injection site at a flow rate of 4 ml / sec. Acquisitions were initiated at 25-30 seconds for the arterial phase, 50-70 seconds for the portal phase, and 180-200 seconds for the parenchymal phase. CT images were purchased with a 5 mm section and then transferred to the workstation to achieve 2.5 mm thick rectangular and coronal reconstructions.

The results from CT were correlated with the presence of documented esophageal varices in endoscopy and the hepatic stiffness measured by FibroScan.

*Superior digestive endoscopy.*

Superior digestive endoscopy is a useful tool for screening because it is widely available. In addition, endoscopy identifies the risk of bleeding related to varicose veins and the presence of red spots, and in addition to esophageal varices, endoscopy identifies other lesions with bleeding potential related to PHT, such as gastric varices, portal hypertension gastropathy and gastric antral vascular ectasia. In the present study, superior digestive endoscopy was the reference standard.

Endoscopy was performed with a Pentax EPK-1000 digital videoendoscope, EG-290Kp videogastroscope, sometimes under mild sedation (midazolam or propofol IV), and included a complete examination of the esophagus, stomach and proximal duodenum as portal hypertension can be found in all three regions. All patients were endoscopically examined after they agreed and signed the informed consent.

To assess the endoscopic aspects of portal hypertensive gastropathy, a double endoscopic examination, initially in white light and then with i-scan technology, was performed in some patients. The existing endoscopic signs described by the Baveno criteria were noted and for each sign the diagnostic sensitivity, specificity and accuracy were calculated. It was followed by signs of portal hypertension (esophageal varices, gastric varices, hypertensive portal gastropathy).

*Endoscopic ultrasound (EUS).*

The examination was performed in some of the patients in the study group. Inclusion criteria: PHT patients due to cirrhosis (diagnosis based on clinical, biological and ultrasound

criteria). Exclusion criteria: Serious complications of liver cirrhosis (active upper gastrointestinal bleeding, hepatic coma), patients with esophageal varices ligation.

While the patient was under light sedation, a linear OLYMPUS GF UCT 140 side view with 13mm top view with an ultrasonic probe of 7.5-12 MHz was inserted into the esophagus and then into the stomach and an ultrasound ALOKA Prosound Alpha 5. Axial images were performed that revealed all 5 layers of the esophagus and stomach in hyperecogenic and hypoecogenic alternating layers, beginning with the hyperecogenic mucosa.

**Personal findings on the morphological aspects of the venous port system resulting from computed tomography under normal and pathological conditions.**

The study was conducted in a group of 72 patients (22.8% of the 315 patients in the 3-year study) only those patients who had alpha-fetoprotein ( AFP) greater than 50 ng / mL, where computed tomography was performed for liver tumor screening.

I studied the origin of the portal vein in 15 CT angiography cases. The formation of the portal vein by joining the superior mesenteric vein and splenic vein, classified as type I, occurred in 14 patients (8 women, 6 males) (Table 6).Type I formation was subdivided into two subgroups depending on the completion of the inferior mesenteric vein. Cases where the inferior mesenteric vein drained into the splenic vein were noted as type Ia. Cases where the inferior mesenteric vein drained directly into the superior mesenteric vein were classified as type Ib.

**Table 6 Types of portal vein formation.**

| Type | Female cases | Male cases | Total cases |
|---|---|---|---|
| I | 8 (53.33%) | 6 (40%) | 14 (93.33 %) |
| II | 1 (6.66%) | 0 | 1 (6.66 %) |

The formation of the portal vein by joining the splenic vein, the upper mesenteric vein and the inferior mesenteric vein, classified type II, was found in 1 cases (6.66 % of cases).

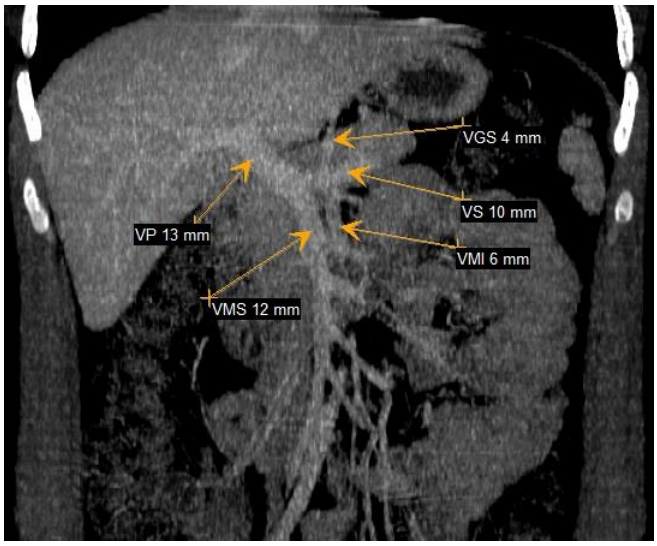


**Fig. 9 Forming the portal vein by joining the splenic vein with the upper mesenteric vein. IMV drains into SV (type IA)**

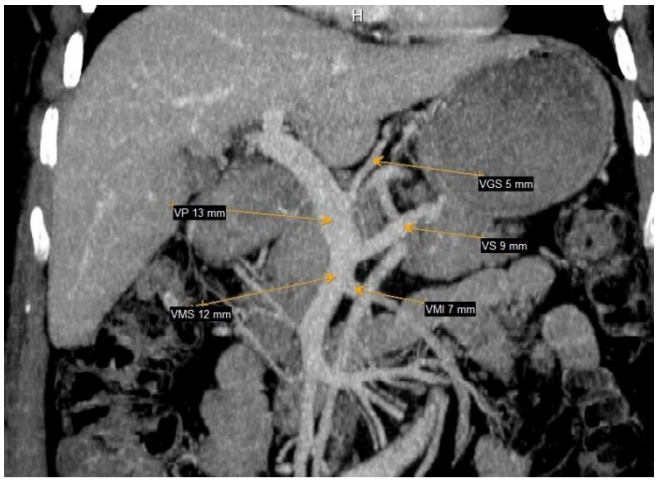


**Fig. 11 Forming the portal vein by joining the splenic vein with the upper mesenteric vein. IMV flows into the SMV (type IB)**

The mean value of the portal vein caliber measured in the hepatic hilum was 16.9 ± 2.3 mm, splenic vein was 10.3 ± 3.7 mm, superior mesenteric vein 12.1 ± 3.1 mm, inferior mesenteric vein 9.7 ± 3.8 mm, and left gastric vein of 8.2 ± 2.4 mm. The other numerical values are shown in Table 8.

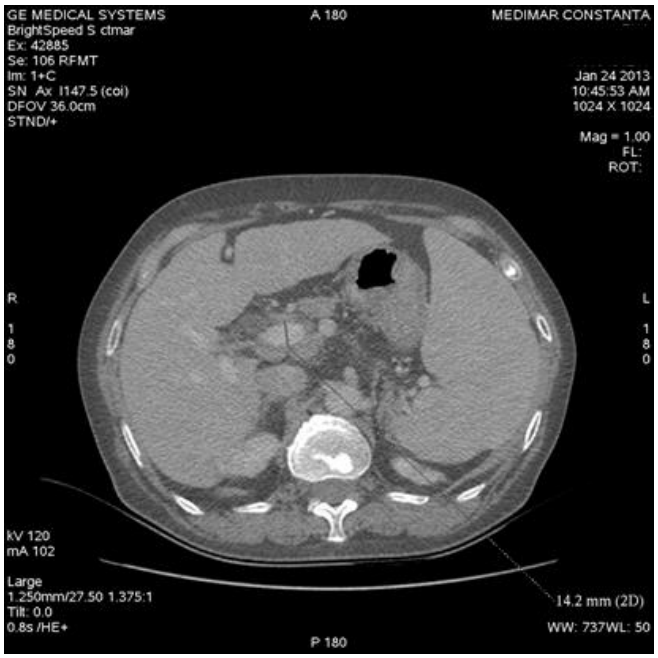


**Fig. 1. Measurement of portal vein caliber in the hepatic pedicle**

Three-dimensional CT scan is useful to analyze the origin of the portal vein and the types of venous collateral in portal hypertension.

Computed tomography is useful to visualize venous collateral circulation beyond the reach of endoscope. In this respect, the tomographic examination of the patients in the studied group demonstrated the presence of esophageal varices in 58 (80.5%) patients (Table 9) and the venous collateral circulation of the venous system, especially in the perigastric, perihepatic, paracolic, but also in the smaller percentages in the perisplenic, intramezenteric and spleno-renal regions (Table 9). The recanalized umbilical vein was described in 19% of cases examined by CT.

**Table 1 Oesophageal varices and collateral vein circulation of the venous portal system (described in abdominal CT).**

| **Anatomical territory with collateral vein circulation:** | **No. pt. (%)** |
|---|---|
| Intramural esophageal (esophageal varices) | 58 (80.5%) |
| Perigastric | 62 (86%) |
| Perihepatic (pericapsular) | 60 (83%) |
| Paracolic | 37 (51%) |

| | |
|---|---|
| Perisplenic (pericapsular) | 31 (43%) |
| Spleno-renal shunt | 14 (19%) |
| Intramesenteric | 10 (14%) |
| Umbilical vein recanalization | 14 (19%) |

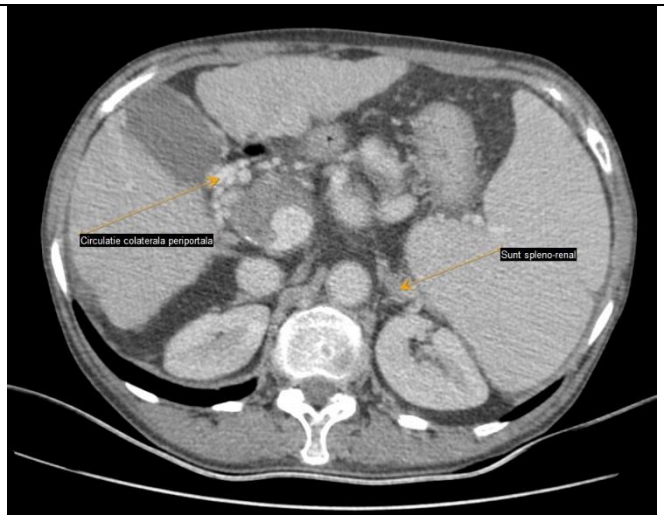


**Fig. 2 Spleno-renal shunt**

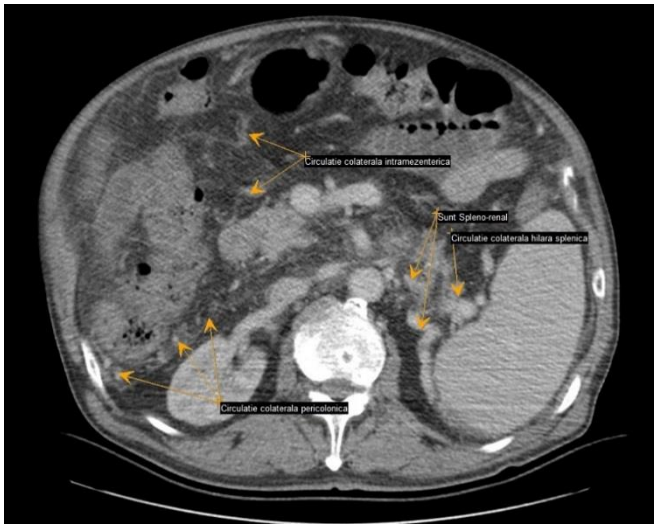


**Fig. 3 Spleno-renal shunt, hilar splenic collateral circulation, intramesenteric, pericolonic**

According to CT results, cases were classified into:

- Group I: 14 patients without esophageal varices (19.5%) including 8 males and 6 females

- Group II: 33 patients with small varices (57%), including 18 men and 15 women

- Group III: 25 patients with large varices (43%) including 14 men and 11 women

Upper digestive endoscopy detected 9 cases without esophageal varicose veins (EV), 40 cases with small EV and 23 large EV cases (Table 10).

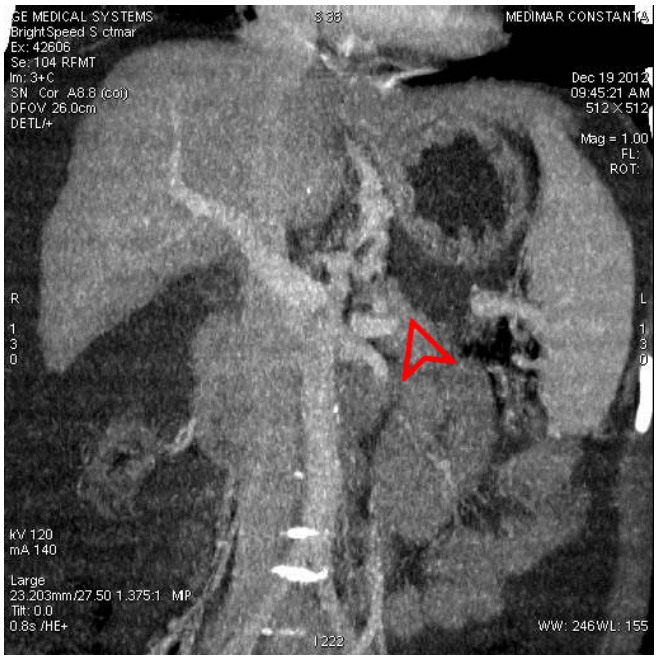


**Fig. 4 Large esophageal varices**

CT sensitivity for EV detection was 92.06%, 100% specificity, 100% positive predictive value and 64.29% negative predictive value (Table 11) compared to upper digestive endoscopy as the reference standard.

CT sensitivity for large EV cases (100%) was higher than for cases with small EV (80%) (Table 13).

There was no significant difference in the presence of splenomegaly and splenic varicose veins between small EV cases and large VE cases ($p > 0.05$) (Table 15, graph 4).

There was no significant difference in ascites (Chart 6) between cases with small and large VE ($p > 0.05$).

There was no significant difference in the presence of gastric varicose veins (Table 18, Graph 7) and paraoesophageal varicose veins (Table 19, Figure 8) between small EV cases and large EV cases ($p > 0.05$).

**Personal findings on portal haemodynamics resulting from abdominal ultrasound, in normal and pathological conditions.**

In order to study the importance of hemodynamic parameters in the characterization of patients with portal hypertension (54 patients) compared to a control group (55 patients) and their possible correlation with the severity of oesophageal varices and the degree of hepatic impairment classified as Child, I evaluated a group of patients with liver cirrhosis and compared the results with a control group.

**The portal vein diameter** is higher in patients with cirrhosis compared to the control group (14.2 ± 2.1 mm vs 11.2 ± 1.8, p <0.05), but this does not correlate with the severity of cirrhosis assessed by the CHILD class.

**The mean blood velocity through the portal vein** was significantly lower in patients with cirrhosis compared to the control group (11.5 ± 2.7 cm / sec vs. 19.1 ± 3.2 cm / sec, p <0.05), and there was a decreasing tendency of the velocity as the severity of liver cirrhosis increases, the smallest velocities occurring in CHILD B and C classes (12.1 ± 1.9 cm / sec, 10.2 ± 2.8 cm / sec).

Regarding **the blood flow through the portal vein**, it was significantly low (857.2 ± 86.8 mL / sec) only in advanced liver cirrhosis (CHILD C) without significant differences between the control group and patients with compensated cirrhosis CHILD A) (1253.7 ± 65.2 mL / sec vs. 1279.3 ± 53.7 mL / sec, p> 0.05). Only patients with CHILD B cirrhosis had a downward trend in portal blood flow, but this was not significant (1126.1 ± 98.5 mL / sec, p> 0.05).

The **congestion index of the portal vein** was increased in patients with cirrhosis versus the control group, patients with decompensated cirrhosis (CHILD C) with the highest congestion index (0.175 ± 0.028 cm x sec vs 0.086 ± 0.011 cm x sec, p < 0.05).

**Hepatic pulsatility and resistivity indices** were significantly higher in patients with CHILD B and C cirrhosis compared to controls and CHILD A patients.

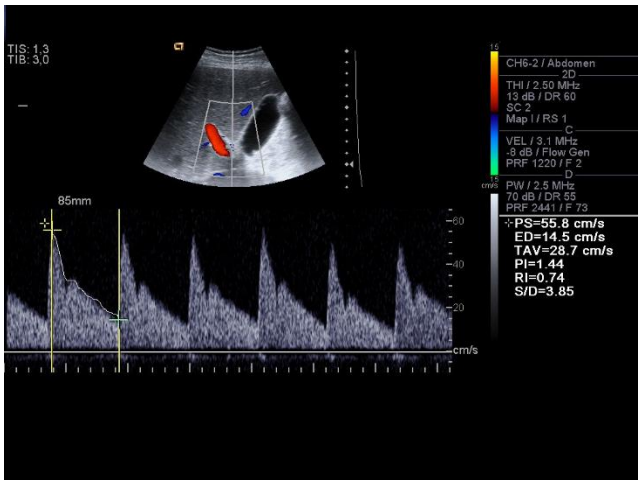


**Fig. 30 Measurement of pulsatilitz and resistivity indices in the hepatic artery in a healthy patient (PI = 1.44, RI = 0.74)**

Regarding the **splenic artery** Doppler analysis, there were no statistically significant differences between the pulsatility index (PI) or the splenic arterial resistivity index (RI) in patients with cirrhosis versus the control group nor between these indices and the CHILD scores of liver cirrhosis (Table 20).

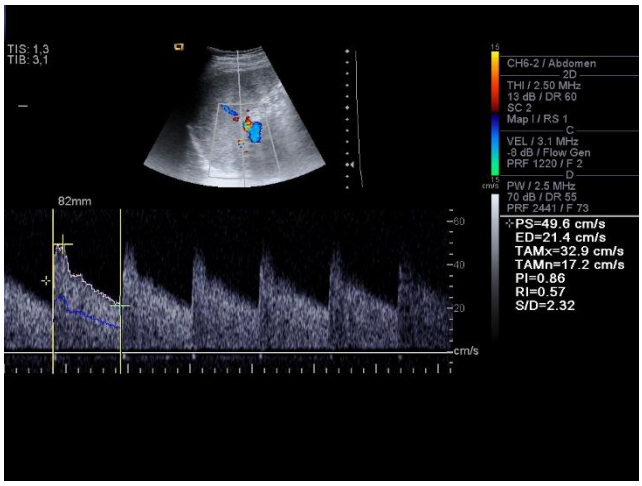


**Fig. 5 Measurement of splenic impedance indices in a healthy patient (PI = 0.86, RI = 0.57)**

**The liver vascular index (LVI)** was significantly lower in patients with severe CHILD C cirrhosis (8.36 ± 3.13) compared to patients with CHILD compensated cirrhosis A (15.08 ± 2.63) and control group (17.52 ± 2.76) ($p < 0.05$). Although LVI was lower in CHILD B class (10.14 ± 3.98), the value did not reach statistical significance ($p > 0.05$).

The results of pulsed Doppler haemodynamic parameters analysis based on the size of the esophageal varices can be found in Table 21. There is no correlation of these parameters with the esophageal varices, except PV flow which is significantly lower in patients with third grade EV (843.4 ± 91.3 mL / sec), and liver vascular index (LVI), also significantly lower in these patients (8.21 ± 3.20) ($p < 0.05$).

**Correlations of hepatic stiffness and other clinical-biological parameters with the presence or size of esophageal varices.**

Of the 315 patients in the data base over the 3 years of the study, only a group of 128 patients with liver cirrhosis (%) was able to perform an accurate measurement of hepatic and splenic rigidity. These patients were also examined by upper gastrointestinal endoscopy for the presence of esophageal varices, and the results of these correlations are summarized below.

The mean hepatic stiffness measured by FibroScan was significantly higher in the group of patients with large EV compared with the group of patients without varicose veins or with small EV (35.3 kPa vs 16.8 kPa vs 13.7 kPa, respectively). The cut-off of hepatic stiffness for predicting esophageal varices was 18.6 kPa (92% sensitivity, 61% specificity). The cut-off of hepatic stiffness for prediction of large esophageal varices was 27.4 kPa (100% sensitivity, 72% specificity).

Similar to the previously presented results that refer to hepatic stiffness, we performed an analysis of the results of splenic rigidity and the presence or size of esophageal varices, and the results are presented below. The mean splenic stiffness measured by FibroScan was significantly higher in the group of patients with large EV, compared to the group of patients without varicose veins or small EVs (31.6 kPa vs 18.2 kPa vs 15.1 kPa, respectively). The cut-off value of splenic rigidity for predicting esophageal varices was 21.6 kPa (94% sensitivity, 69% specificity). The cut-off value of splenic rigidity for prediction of large esophageal varices was 31.2 kPa (100% sensitivity, 75% specificity).

In this study, we also performed an analysis of other prediction parameters for esophageal varices. Thus, we found that platelet count, longitudinal spleen diameter (measured at ultrasound) and the ratio between them are significantly correlated with the presence of esophageal varices ($p < 0.05$ for each). The cut-off for platelet counts, splenic diameter, and their ratio for VE prediction is 92,000 / mmc, 13.8 cm and 651 respectively, while the large varicella cut-off was 64,000 and 529, respectively.

**The role of high-definition digestive endoscopy and virtual chromoendoscopy in the diagnosis of portal hypertension complications, with particular reference to portal hypertensive gastropathy.**

This was a prospective, observational, analytic, transversal and cohort study. The study group included 50 consecutive patients diagnosed with PHT of any etiology, admitted to the Gastroenterology Department, Internal Medicine Clinic I, Constanţa between November 2014 – July 2015. The diagnosis was based on clinical, laboratory, and imaging criteria (history of liver disease; altered liver tests; ultrasound changes in the liver, spleen, and portal venous system), according to the current guidelines. We did not include patients with active upper gastrointestinal bleeding because the blood in the stomach would have prevented a better visualization of the gastric mucosa. We searched for signs of PHT (esophageal varices, gastric varices, PHG).

We diagnosed 39 patients with PHG using endoscopic examination in white light, in accordance with the Baveno criteria. After applying i-scan endoscopy, we found that another 6 patients presented signs of PHG, that amounted to a total of 45 patients with PHG. Six (12%) patients were considered normal by classic endoscopy, but were diagnosed with PHG by i-scan endoscopy (table II). The strength of agreement between the two methods was moderate (k=0.565; 95%CI 0.271 - 0.859; p<0.001).

The mosaic pattern was misclassified by endoscopy in white light in 8 (16%) patients (table III). In 3 (6%) cases the mosaic pattern was absent at the classic endoscopy, but the i-scan 1 endoscopy revealed the existence of a mild mosaic pattern. In 5 (10%) patients the mosaic pattern was described as mild by the classic endoscopy, but the i-scan 1 endoscopy classified it as severe. The strength of agreement between the two methods was good (k=0.800; 95%CI 0.669 – 0.932; p<0.001).

The i-scan 2 endoscopy had a lower specificity for the mucosal surface even when compared to the endoscopy (table IV). In 5 (10%) patients i-scan 2 endoscopy failed to recognize the presence of a mild mosaic pattern, and in 4 (8%) patients it misclassified the severe mosaic pattern as mild. But the strength of agreement between the two methods was still good (k=0.785; 95%CI 0.652 – 0.917; p<0.001).

Red spots were discovered at the same patients by both classic and i-scan 1 endoscopy (table V). The strength of agreement between the two methods was perfect (k=1; p<0.001). The red spots were missed by endoscopy in white light in 3 (6%) patients (table VI). In 6 (12%) cases the red spots was considered as isolated at the classic endoscopy, but the i-scan 2

endoscopy described them as confluent. The strength of agreement between the two methods was almost perfect (k=0.863; 95CI 0.776 – 0.950; p<0.001).

The presence of GAVE was described in 4 (8%) patients by all the endoscopic methods. The strength of agreement between the any i-scan endoscopy and endoscopy with white light methods was perfect (k=1; p<0.001).

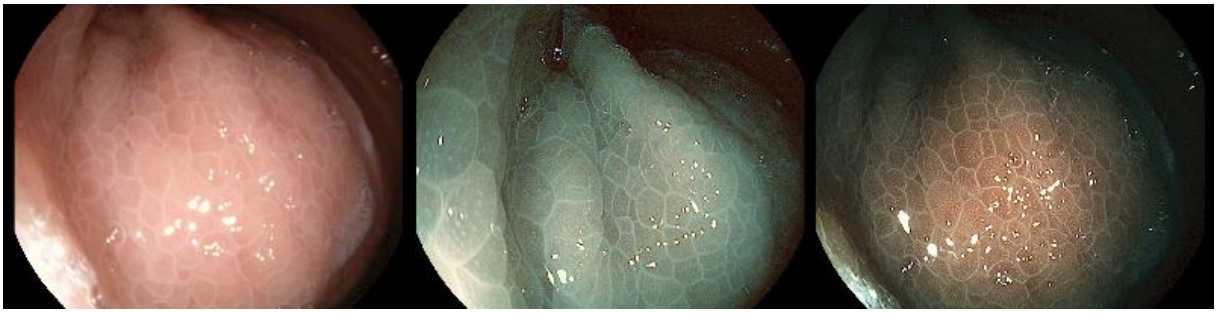

**Fig. 6 Mild mosaic pattern of portal hypertensive gastropathy (A. white light endoscopy; B. i-scan 1; C. i-scan 2). The image accuracy increases with i-scan in comparison with white light examination.**

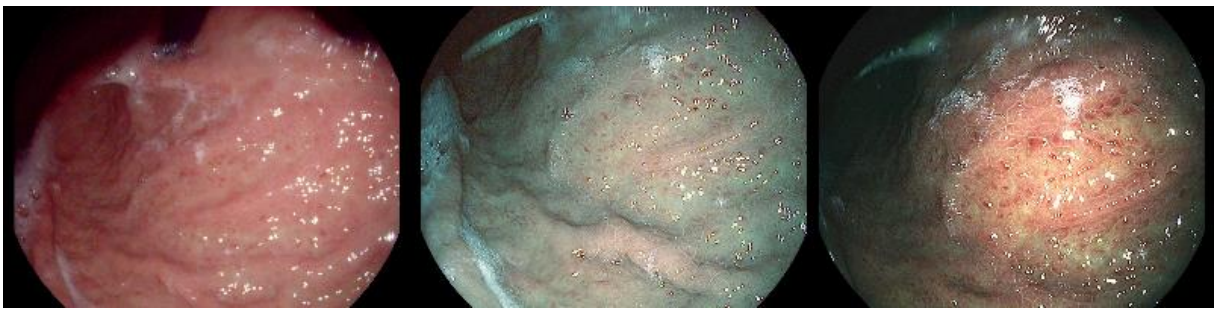

**Fig. 7 Confluent red spots in portal hypertensive gastropathy (A. white light endoscopy; B. i-scan 1; C. i-scan 2). The visibility of red spots is increased with i-scan 2.**

**Comparative study between non-invasive methods (CT angiography) and invasive (endoscopy / ecoendoscopy) in the assessment of porto-systemic anastomoses and other manifestations of portal hypertension.**

Another subchapter of the current study was comparing the results obtained by a non-invasive method such as abdominal CT with those obtained by the standard method (EDS) or by potentially more efficient methods (endoscopic ultrasound, EUS), but still invasive in the study of portal circulation in pathological conditions. The study focused on the evaluation of the three methods in detecting vascular changes associated with portal hypertension as well as their correlation with the occurrence of PHT complications (upper gastrointestinal bleeding,

death). Inclusion criteria were: patients with cirrhosis (diagnosis based on clinical, biological and ultrasound criteria). Exclusion criteria were: the presence of serious complications of liver cirrhosis (active upper gastrointestinal bleeding, hepatic coma), patients with EV ligation, patients with other general contraindications to upper endoscopy or EUS. The study consisted of 15 patients with a mean age of 54.8 ± 12.5 years (age range 45-63 years) of various etiologies, especially from the CHILD A and B classes.

At endoscopy, we looked for the presence of esophageal and gastric varices, classified following the most recent recommendation of the American Association for the Study of the Liver (AASLD) in absent, small (<5 mm) or large (> 5 mm). EUS monitored intramucosal and deep varicose veins (peri / para esophageal varices, peri / para-gastric varicose veins), classified according to the same criteria as well as hemodynamic elements (blood velocity). The same goals (venous collaterals of the venous port system) were followed at CT, from which their presence and size were noted.

Endoscopy identified esophageal varices in 11/15 patients and gastric varices in 4/15 patients (Table 36). Regarding EV identification, computed tomography had a good diagnostic sensitivity, comparable to endoscopy, only for large EVs (> 5 mm). In the case of small EV (<5 mm), CT showed a lower sensitivity to endoscopy (Table 37).

In terms of gastric varices, EUS has a higher sensitivity than endoscopy in fornix varicose veins (4 endoscopy patients, 7 patients in the EUS), with the same accuracy as CT (Table 38). Para and peri-esophageal varices were identified in CT in 6 cases (40%). Their mean diameter was 7.4 ± 3.6 mm. No correlation was found between the EV grade at endoscopy and the diameter of the para- and peri-esophageal veins.

Endoscopic ultrasound can detect perforated veins and can detect the direction of blood flow through varicose veins as well as its velocity. EUS and CT have a similar accuracy in the detection of peri-gastric and peri esophageal varicose veins and their classification according to the diameter of the vessels. There was a direct correlation between the diameter of the esophageal varices and the blood velocity through them ($r = 0.758$). Five patients developed upper gastrointestinal bleeding (UGIB). Blood velocity in esophageal varices correlated positively with the risk of UGIB (mean velocity of 12.4 cm / sec in UGIB versus 7.2 cm / sec in without UGIB $p < 0.01$).

**Personal findings on the role of CT in predicting the occurrence of higher digestive haemorrhage (UGIB) and hepatic encephalopathy (EH) in patients with portal hypertension (HTP).**

The 72 patients (22.8% out of a total of 315 patients in the 3-year database) who underwent computed tomography for liver tumor screening (the characteristics of this subgroup of patients are shown in Table 5) were monitored prospectively for the detection of complications, for upper digestive haemorrhage (UGIB) and hepatic encephalopathy (EH).

Patients were evaluated at 6 months and 12 months after inclusion in the database.

Oesophageal varices (VE) were graded at CT as: small (<5 mm), large (> 5 mm):

- Group I: 14 patients without esophageal varices (19.5%) including 8 males and 6 females

- Group II: 33 patients with small varices (57%), including 18 men and 15 women

- Group III: 25 patients with large varices (43%) including 14 men and 11 women

At the 6-month evaluation, it was found that upper gastrointestinal haemorrhage through esophageal varices rupture occurred in 5 patients (6.9%), all of them were registered with large esophageal varices. No patient in the small esophageal varices or esophageal varices group experienced digestive haemorrhage at this time (Table 43, Figure 15). At the 12-month evaluation, it was found that upper gastrointestinal haemorrhage by esophageal varicose rupture occurred in 8 patients (11.1%), all of them were registered with large esophageal varices. No patient in the small esophageal varices or esophageal varices group experienced digestive haemorrhage at this time (Table 44, graph 15).

If the presence of esophageal varices and gastric varices is directly related to the occurrence of upper gastrointestinal haemorrhage, the presence of collateral venous circulation may influence the appearance of hepatic encephalopathy by making porto-systemic shifts. Therefore, I analyzed the role of portal venous collateral circulation as a risk factor in the development of hepatic encephalopathy. Patients were divided into two categories, depending on the presence or not of collateral circulation in CT in at least 4 of the following 7 vascular areas analyzed (collateral circulation in more than 50% of the territories). The first group of patients (with venous collateral circulation in more than 50% of the territories) consisted of 41 patients (56.9%) and the second group of patients (with venous collateral circulation in less than 50% of the territories) of was made up of 31 patients (43.1%). At 6 months of follow-up,

hepatic encephalopathy was recorded in 4 patients (5.5%), all of whom were in the large vein collateral circulation group (Table 45, Figure 17). At 12 months of follow-up, encephalopathy occurred in 9 patients (12.5%), 7 in the large collateral circulation group (group 1), and 2 in the group with collateral circulation in less than 50% of the territories ( group 2) (Chart 47, Table 46).

## DISCUSSIONS

### Discussions on personal findings about morphological aspects of the venous portal system resulting from computed tomography in normal and pathological conditions.

Regarding the **origin of the portal vein**, I have encountered the most frequent type I, in which type I A in 66,66 % of cases of the portal vein formation through the anastomosis of the splenic vein with the superior mesenteric vein, and the superior mesenteric vein draining into the splenic vein.

**Table 2 Comparative results on the origin of the portal vein**

| Study | Type I | | Type II |
|---|---|---|---|
| | I A | I B | |
| **Douglas** | 38% | 29.3% | 32.7% |
| **Purcell** | 44% | 53% | 3% |
| **Chaijarookhanarak** | 58,46% | 26,15% | 15,38% |
| **Rajashree** | 30% | 22.5% | 47.5% |
| **Iorga** | 81.58% | 7,89% | 10,53% |
| **Personal study** | 66,66 % | 26,66 % | 6,66 % |

By comparing the results in the literature, as it can be seen in the table, Douglas found a 38% frequency of type IA, Chaijarookhanarak of 58.4%. At Purcell dominated type IB, and Rajashree encountered type II more frequently in 47.5% of cases.

**Portal vein caliber** at angio CT was found to be 11.9 ± 0.87 mm. The results from the personal study are similar to those of Chaijarookhanarak. The Kamina obtained a smaller caliber, unlike Testut, Rouvière and Matusz that have identified a larger caliber.

Under normal circumstances, I identified a **left gastric vein** diameter similar to Li, Wachsberg. Dilation of the left stomach vein more than 5-6 mm in diameter at CT is a sign of portal hypertension. Under pathological conditions, I have identified a left gastric diameter greater than normal, being larger than that described by Wachsberg, Li, Zhou.

Bolognesi demonstrated that the portal vein diameter and splenic vein diameter are diagnostic criterion for portal hypertension. I identified a caliber greater than 14 mm in the portal vein, the results being similar to the Haag, Zhou (2012). Shateri and Zhou described a smaller diameter. With regard to the splenic vein, I obtained a caliber similar to the Haag, unlike Zhou who described a higher value.

The umbilical vein is obliterated after birth and becomes the round ligament. With this ligament there are additional smaller paraombilic vessels. These veins may hypertrophy in the presence of elevated portal venous pressure and become paraumbilical recanalized veins (varicose veins). The paraumbilical vein originates from the left port vein, flows along the falciform ligament and usually extends to the umbilicus. In the personal study, the paraumbilical venous collaterals were identified in 19% of the cases, less than Cho.

Among the numerous port-systemic shunts are left gastro-renal shunts. The path of this shunt is usually through the left renal vein, which becomes dilated and tortuous.

The splenornal shunt we identified in a smaller percentage than Cho, Wu.

Upper digestive endoscopy is the gold standard for assessing esophageal varices and the risk of upper digestive haemorrhage. It assesses the existence of esophageal varices, which can be graded for prognosis.

The use of single detector and multidetector CT was evaluated as a screening method for esophageal varices (Kim YJ, Kim SH, Perri RE). In all three studies, the performance of CT esophagography was compared with upper digestive endoscopy, which served as a gold standard. When examining varicose veins, the CT esophagography had a sensitivity of 64% to 93% and the specificity between 76% and 96.6%. For detection of varicose veins, sensitivity ranged between 56% and 92%, and the specificity was between 84% and 92%.

In my study, I obtained results similar to Moftah and collaborators who described a CT sensitivity for the diagnosis of 96% oesophageal varices, a 100% specificity, a positive predictive value of 100% and a negative predictive value of 66.6%.

In the current study, we performed an analysis of the results provided by abdominal CT in the detection of esophageal varices and their measurement. CT scan had a sensitivity of 92.06%, a 100% specificity, a 100% positive predictive value (PPV), and a negative predictive value (NPV) of 64.29%, upper digestive endoscopy being the reference standard. CT sensitivity to high VE cases (100%) was higher than that in patients with small EV (80%). Kim and colleagues in 2007 achieved the following results: 92% sensitivity, 84% specificity, 55% VPP and 98% VPN. During the endoscopy, the esophageal lumen was inflated with air to visualize varices. This technique could have a negative effect on the ability to accurately visualize and measure small varices. Studies conducted by Kim and collaborators in 2007 and by Ba-Ssalamah and collaborators in 2009 included the use of an oral contrast agent, so that the residual contrast material covering the luminal surface could also interfere with detection small varices.

CT also allows the evaluation of extra-luminal oesophageal pathology that influences the prognosis of patients with liver cirrhosis. Para-esophageal varices can be seen as venous dilations close to the outside of the esophagus. Para-esophageal and gastric varices were found in 21.3% and respectively, 86.0%, in my patients. There was no significant statistical difference in the distribution of age, gender, ascites, splenomegaly, splenic varicose veins and gastric varicose veins in patients with large EVs compared to patients with small VâEVs. In my study, I used a MDCT with a 2.5 mm slice thickness on a batch of 72 patients. We identified esophageal varices in 80.5% of cases, approximately equal to Takashi's study.

**Discussions on personal findings on portal haemodynamics resulting from abdominal ultrasound, in normal and pathological conditions.**

The results of this study demonstrate that the **portal vein diameter** was significantly higher in patients with cirrhosis than in the control group, similar results being described by Yan. The diameter of the portal vein has not been shown to be a useful parameter for differentiating between the severity classes of cirrhosis and there has been no correlation with the size of the esophageal varices. A 2012 study by Shateri also shows the weak and unnecessary correlation in practice between the diameter of the portal vein and the CHILD or MELD classes of liver cirrhosis, although there are studies that support the opposite, for example Yan’s. According to our study, this parameter cannot be used in clinical practice for assessing the risk of bleeding or prognosis of patients with cirrhosis, a possible explanation

being that the diameter of the portal vein depends on the number and size of open vein collateral in the port system, and this is a feature with great individual variability.

In contrast, the blood **velocity through the portal vein** was significantly decreased in patients with decompensated cirrhosis (CHILD B and C) compared to the control group and CHILD A patients, similar results being observed in other studies like Haktanir, Yan, Chawla. Gaiani and other authors reported similar results, namely: the portal vein blood velocity was significantly decreased in cirrhotic patients compared to the control group and was significantly lower in patients with Child B and C cirrhosis than in Child A. In my study I obtained lower values of portal vein velocity than those of Haag, Cioni. Under normal circumstances, we have identified higher vein velocity values than those reported by Moriyasu, Zoli, Ohnishi, and Gaiani-like. In cirrhotic patients we obtained higher values than those described by Moriyasu, Zoli.

However, the velocity of the portal vein is not a constantly correlated parameter with the severity of cirrhosis because there are studies in which the results were opposite, like Shateri, Schneider.

Piscaglia says that these inconsistent results are obtained due to the existence of some intra- and inter-conservative variability, as well as due to the existence of venous collaterals.

The **portal vein flow** depends on PV diameter (which increases with the severity of the cirrhosis) and the blood velocity through the PV (which decreases with the severity of the cirrhosis), therefore, the PV flow does not experience significant changes in the evolution of patients with cirrhosis. However, in advanced stages as well as in patients with grade III varices, PV blood flow was significantly low, indicating the value of this index in identifying patients with severe prognosis and increased risk of decompensation and digestive haemorrhage.

Similarly, there is a **portal vein congestion index**, which in my study and in other studies of Shateri, Moriyashu, was significantly elevated in patients with CHILD C and those with grade III varices.

A significant increase in **hepatic pulsatility and resistivity indices** was identified in patients with cirrhosis compared with the control group, as in the study of Haktanir . Moreover, these indices correlated with the severity of cirrhosis, being significantly elevated in the CHILD B and C classes, but they did not correlate with the size of the esophageal varices. Sacerdoti demonstrated that PI and RI indices of the hepatic artery measured at the intrahepatic level are significantly higher in cirrhosis than in the control group. Bolognese evaluated Doppler **splenic indices** in patients with portal hypertension and a control group. They found that the pulsatility

index was higher in cirrhotic than control in patients with cirrhosis and vein thrombosis than in those without thrombosis.

In my study, there was no good correlation between esophageal varices and any of the static or hemodynamic ultrasound parameters, with the exception of the group of patients with grade III varices that had a significantly reduced blood flow through the portal vein and the LVI, and the congestion index of the portal veins significantly increased, which corresponds to similar results in the literature, like Zardi. Taourel explained this by the existence of various porto-systemic anastomoses that drain portal blood circulation through other vascular territories, not only through esophageal varices.

**Discussions on the correlation of hepatic stiffness and other clinical-biological parameters with the presence or size of esophageal varices.**

In the group of patients studied by me, the Child score was much higher in patients with cirrhosis and esophageal varices than in patients with cirrhosis without esophageal varices, this finding being in agreement with Madhotra. He also claimed that there was a correlation between the presence of esophageal varices and an elevated Child Score. It can be said that more advanced the liver damage, the more likely it is to have esophageal varices.

In my study, the platelet count was significantly lower in patients with esophageal varices (mean value was 98,000 / mmc) than in patients without esophageal varices (on average 138,000 / mmc) ($p < 0.05$). Platelets may decrease for many reasons in patients with liver cirrhosis. Madthora asserted that 32% of patients with cirrhosis had thrombocytopenia <68,000 / mmc without concomitant splenomegaly. This could be explained by the low amount of thrombopoietin synthesized in the liver. Winkfield gives other explanations for this phenomenon that could be the presence of anti-platelet antibodies and platelet-associated immunoglobulins that can be found in serum of patients with cirrhosis. Thus, the use of platelet counts only as a non-invasive method of predicting the presence of esophageal varices can be misleading because thrombocytopenia cannot be attributed entirely to portal hypertension. Giannini says that the use of the platelet ratio / splenic longitudinal diameter overcomes this error by "normalizing" the total number of platelets, only taking into account those seized in the spleen.

Regarding spleen size, this study found that it was significantly higher in patients with esophageal varices (mean 14.7 cm) than in patients with cirrhosis without esophageal varices (mean 12.4 cm) ($p < 0.05$), so measuring longitudinal spleen diameter in ultrasound can be

considered a non-invasive indicator of predicting esophageal varices in patients with cirrhosis, as Mandal said.

In the present study, the ratio of "platelets / longitudinal spleen diameter" was significantly lower in patients with esophageal varices (mean 562), compared with patients without esophageal varices (mean 703), and the cut-off value the better was 651 (sensitivity = 89%, specificity = 87%). In other studies, higher cut-off values with increased specificity and sensitivity are mentioned. Agha mentions a cut-off value of 909 with a sensitivity of 100% and a specificity of 97.6%. Gianini mentions the same cut-off value of 909 with a sensitivity of 91.5% and a specificity of 67%. This difference in results could be attributed to a smaller lot. Chawla states that the ratio of "platelets / longitudinal splenic diameter" might not be adequate to completely replace the digestive endoscopy for esophageal varices screening.

In this study, hepatic rigidity was significantly increased in patients with esophageal varices compared to those without esophageal varices; from this point of view, the best cut-off value was 18.6 kPa, with a sensitivity of 92% and a specificity of 61%. Also, hepatic rigidity was significantly higher in patients with large esophageal varices compared to those with small esophageal varices; the best cut-off value for this differentiation was 27.4 kPa, with a sensitivity of 100% and a specificity of 72%.

These data are in agreement with the results reported by Sporea I, which identifies a similar group of 1000 patients. For the presence of esophageal varices, the optimum value of FibroScan was 31 kPa, and for the prediction of upper digestive hemorrhage, the value was 50.7 kPa after Lebrec. The larger the esophageal varices, the greater the risk of digestive haemorrhage, and the cut-off value of hepatic elastography predicting the risk of bleeding may be considered the same as the cut-off value predicting the presence of large esophageal varices. In addition, the study by Vizzutti  mentions a cut-off for predicting esophageal varices of 17.6 kPa, lower than that obtained in the current study, but the results may differ due to differences in demographics and batch characteristics study, as well as another type of FibroScan. Even Castera L mentions that hepatic elastography may be a valuable non-invasive tool in the diagnosis of liver cirrhosis, but cannot replace digestive endoscopy in esophageal varices screening.

An analysis of other non-invasive parameters for varicose detection in the current study involved the value of the "platelets / spleen size" ratio. By comparison, hepatic elastography has a significantly greater value than the "platelets / spleen size" ratio, which was previously confirmed by the Kazemi study.

**Discussions on the role of high-definition digestive endoscopy and virtual chromo-endoscopy in the diagnosis of portal hypertension complications, with particular reference to hypertensive portal gastropathy**.

After studying medical literature accessed through public databases, I found that there has not yet been a paper that discusses the role of i-scan examination in diagnosing PHG. There are, however, studies on the diagnostic accuracy of white light endoscopy. One such study, de Macedo's, reported a sensitivity of 4.35–52.17% and a specificity of 88.31–100.00% based on the Baveno criteria. There are also studies, Hancock's, Kodashima's, about the role of i-scan and other virtual chromoendoscopy techniques (NBI, FICE) in the diagnosis of various other upper digestive diseases (e.g., Barrett's esophagus, esophageal adenocarcinoma, atrophic gastritis, intestinal metaplasia, and early gastric cancer).

i-scan is a screening method that uses endoscopic image achieved in white light in real time and processing it using technical analysis software and changes in brightness per pixel by using three algorithms, surface enhancement (SE), contrast enhancement (CE), and tons enhancement (TE), and combining their results by means of endoscopic i-scan type 1 (especially useful for details of the mucosal surface), i-scan type 2, and i-scan type 3 (especially useful for details on the mucosal and submucosal vasculature). Switching from white light scanning to i-scan is done by pressing a button on the endoscope.

In the current study, we discussed each diagnostic criterion of PHG according to Baveno, calculating the diagnostic agreement between the two methods (white light endoscopy and i-scan endoscopy) on each Baveno criteria taken separately but also in general, the diagnostic agreement on PHG. Although both endoscopy in white light and i-scan endoscopy diagnosed PHG, 12% of patients who were initially considered without PHG, after viewing images with i-scan were classified as having PHG. So, i-scan endoscopy has a higher efficacy compared to classic endoscopy in terms of diagnosis of PHG.

The first Baveno criteria, the mosaic pattern, was reinterpreted in 16% of cases when used i-scan endoscopy which led to the diagnosis of PHG in some cases initially undiagnosed by white light, probably because i-scan 1 examination improves the visibility of the gastric mucosa due to increased contrast and thus increases the diagnostic performance of the criterion referring to mucosal gastric edema. This phenomenon was not observed when examining with i-scan 2 who had even lower accuracy in recognizing mucosal pattern in white light endoscopy.

Regarding the second Baveno criteria (presence and appearance of red spots), it was equally identified in white light and i-scan 1. However, using i-scan 2, 6% of patients who had

no red spots in white light endoscopy were diagnosed with red spots, and 12% of the cases were re-classified from isolated to confluent red spots. The values for the i-scan type 2 examination were slightly better than those for the white light examination probably because the i-scan type 2 increases the visibility of blood vessels in the gastric mucosa and the mucosal vascular ectasia appear as red spots. Although the improvement in the values of accuracy was not very distinct, the examiners saw better contrast and slightly more highlighted red points during examination with i-scan compared with white light.

The third Baveno criterion did not bring significant changes in the diagnosis of PHG when shifting from white light examination to i-scan examination, according to the data found in this study.

Regarding endoscopic diagnosis of PHG, a head-to-head comparison between i-scan technology and other methods of virtual chromoendoscopy is not available in the literature. Among the methods of virtual chromoendoscopy, NBI technology was introduced in endoscopic practice before i-scan technology.

With NBI technology, the device projects over the mucosa a white light with a wavelength band (415-540 nm) narrower than normal white light (400-700 nm), which makes the maximum absorption of the light to be at the structures that contain hemoglobin. Wong Kee Song LM says that this emphasizes the contrast to see vascular structures (capillary, venules) of the mucosa. In theory, this phenomenon could increase the accuracy of PHG diagnosis (based on the criterion of "red spots" from Baveno), but it remains questionable whether the accuracy of the details on the mucosal surface (those given by edema of the mucosa, namely the criterion of "mosaic pattern" of Baveno) it will be just as high. Of course, these are just some theoretical assumptions that need to be confirmed or denied by clinical studies. So far, there is only one study published in 2014, El Shazly YM's showing an increase of diagnostic accuracy of PHG using NBI technology**Eroare! Fără sursă de referință.**.

Finally, I want to discuss some flaws of this study: the relatively small number of patients that could have an impact on statistical significance of the results obtained, and the fact that we did not search for the presence or absence of Helicobacter pylori infection and gastric mucosal changes secondary to infection, a factor that could induce some confusion with PHG (regarding the mucosal edema and erythema). Another limitation of the study was the high prevalence of PHG (90%) in our study population, which can affect statistical calculations. In literature, according to Eleftheriadis the prevalence of PHG ranges from 4% to 98%.

**Discussions on the comparative study between non-invasive (CT angiography) and invasive (endoscopy / ecoendoscopy) methods in the assessment of port-systemic anastomoses and other manifestations of portal hypertension.**

Detection of deep varies (periosophageal / paraesophageal) is important because their presence correlates with the risk of recurrence after the ligation and the risk of rebleeding after bending. Thus, Sgouros's study shows that in the case of large varices, over 5 mm in diameter, the risk of recurrence after ligation is 93% vs 46% in the case of small varices (less than 5 mm); also the risk of re-bleeding is 43% vs 12%, respectively, in the case of deep varicose veins compared to the small ones.

Oesophageal varices were present in endoscopy in 75% (15/20) cases in which para esophageal veins were observed in CT angiography, while para esophageal veins were detected in CT angiography in 45% (20/44) of patients at which have been diagnosed with varices in endoscopy. Based on the presence of para esophageal veins in CT angiography, the sensitivity and specificity of CT angiography for the detection of esophageal varices was 34.1% (95% CI: 0.219-0.489) and 61.5% (95% CI: 0.355-0.823) respectively. No correlation was found between the endoscopic degree of esophageal varices and the diameter of the paraesophageal veins, Ayfle noted.

Approximately one third of patients with large gastric varices detected in the EUS are not diagnosed with EDS, and about a quarter of patients have only small varices according to Wiechowska-Kozłowska..

Sato says that Doppler screening in the EUS with measurement of portal vein velocity in the paraesophageal veins, gastric wall thickness and blood velocity in the azygous vein are all useful in predicting recurrent varicose bleeding.

**Discussions on personal findings on the role of CT in predicting the occurrence of higher digestive haemorrhage (UGIB) and hepatic encephalopathy (HE) in patients with portal hypertension (HTP)**

The risk of bleeding (upper gastrointestinal bleeding-UGIB) is predicted by the endoscopic size of the varicose veins. Hemorrhage is often predicted by the red color of the varicose veins (normal, they are white and opaque), the presence of cherry red spots on varicose veins. The results of my study show that the varicose diameter is a parameter associated with the UGIB risk. Patients with large esophageal varices (> 5 mm) identified in CT had upper gastrointestinl digestive haemorrhage after 6 months (5 patients in 25, 20%) and 12 months (8 patients out of 25, 32%), compared to patients without EV or with small EVs that did not have UGIB.

Samsouk demonstrated that the UGIB risk is higher in those with varicose veins ≥5 mm assessed in computer tomography, whereas those with varicose veins <3 mm at CT would have a low risk of haemorrhage and prevent invasive endoscopy for stratification of risk. Also, in my study, I have identified a positive correlation between the occurrence of hepatic encephalopathy and the presence of a rich collateral circulation.

After a search in international databases, I did not identify other studies or communications on this topic, but there were results regarding the correlation between the occurrence of hepatic encephalopathy and other parameters such as: portal vein diameter, antero-posterior right lobe diameter, the presence of hepatic nodularity. In this study, the author states that patients with a small diameter of the portal vein had a richer vein collateral circulation that correlated with the presence of hepatic encephalopathy, coming in support of the results identified in my study.

## CONCLUSIONS

There are very good, varied methods for assessing portal vein circulation. Unfortunately, many of them are based on invasive techniques such as hepatic venous pressure gradient (HVPG) and upper digestive endoscopy for varices. They have the advantage of being validated as gold-standard.

With the development of medical imaging technology, a non-invasive assessment can be performed. This can be done by using tools such as computer tomography, hepatic elastography, abdominal ultrasound, and combining biological algorithms that allow for optimal therapeutic decisions.

Doppler abdominal ultrasound and Doppler ultrasound is a valuable, rapid, non-invasive assessment method.

CT angiography is an important tool for investigating the portal venous system. Adding a portal acquisition with three-dimensional vascular reconstruction can enhance the surgeon's perception of potentially problematic anatomical variants, detailing the course of these torture vessels. This knowledge is of interest for major operations, such as liver transplantation, but also for the usual procedures where an anatomical variant can lead to significant bleeding. With this tool, the radiologist can significantly influence patient care and can alert a colleague about any potential danger.

The assessment of the venous portal system by abdominal CT is a non-invasive diagnostic method in relation to standard upper gastrointestinal endoscopy that can be performed for the screening and grading of esophageal varices in patients with cirrhosis. Concurrent diagnosis of other signs of portal hypertension does not help to discriminate between low-risk varicose veins and high-risk varices. Using abdominal CT as an initial screening method (for hepatocarcinoma, esophageal varices, other complications) may have a higher cost-benefit than superior digestive endoscopy for esophageal varices screening.

Portal hypertension may develop asymptomatic for a long time, but laboratory investigations and imaging tests may detect its presence. One of the most common clinical signs is splenomegaly, usually associated with thrombocytopenia (a phenomenon called hypersplenism).

Superior digestive endoscopy is considered the standard method for assessing esophageal and gastric varices (presence of varices and signs of risk: red spots). However it is invasive and, in rare cases, can lead to complications.

Ependoscopic ultrasound (EUS) and CT are more useful than endoscopy in detecting deep varices and have similar accuracy in the assessment of perisophageal and perigastric venous collaterals.

Although it is an invasive method, EUS brings important hemodynamic data into the assessment of upper gastrointestinal bleeding risk.

In recent years, there have been several studies in the literature that have dealt with the measurement of a correlation between hepatic rigidity, splenic rigidity (both determined by the transient elastography technique, FibrosScan), possibly combined with some clinical-biological parameters the bipolar spleen diameter or the number of peripheral blood platelets), on the one hand, and the existence of esophageal varices and their size, on the other.

Measurement of hepatic stiffness by Fibroscan is a non-invasive method which allows the identification of a large group of patients who are not eligible for screening for varices among patients with compensated liver cirrhosis with a low probability of esophageal varices and especially of large varices, limit endoscopic screening indications.

To date, literature is poor in communications about the use of non-invasive methods to predict the size of esophageal varices. Available studies were only able to identify esophageal varices. Using Fibroscan to determine the size of oesophageal varices may be very useful in following patients with cirrhosis to prevent the development of complications due to the presence of varicose veins with a high risk of haemorrhage.

In conclusion, measurement of hepatic stiffness by FibroScan is useful in identifying esophageal varices in patients with cirrhosis and has a higher diagnostic value than other non-invasive parameters in esophageal varicose vein prediction. This non-invasive method can help select patients who need to perform endoscopic screening for esophageal varices.

Upper digestive endoscopy with i-scan slightly increased the sensitivity of PHG diagnosis, diagnostic criteria (distinguishing mosaic edema and red spots) being more easily observed endoscopically with i-scan than in white light.

I-scan technology improves the quality of endoscopic imaging in terms of surface details (it can also identify cases of GPHT where the examination in white light did not reveal typical changes) and those related to submucosal vasculature.

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