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# *Diagnostic value of portal blood velocity measurements in the assessment of the severity of liver cirrhosis*

Liver haemodynamics is characterized by dual venous and portal blood supply whose physiological variations are particulary evident during digestion (2). Portal haemodynamics vary in response to eating and other stimuli (7, 11). Any increase in portal venous pressure may be a risk factor for variceal bleeding in cirrhotic patients. Intrinsic regulation of hepatic blood flow is mediated only through the hepatic artery, because the liver is not able to directly regulate portal vein blood flow (12). Portal hypertension in its various forms is the most frequent and important circulatory alteration in chronic liver disease. Cirrhosis and portal hypertension affect the flow profile of the liver vasculature (3, 5, 8). In pathological conditions increased arterial blood flow accompanies the decreased portal flow (1, 9, 10, 14). Diminished postprandial portal hyperaemia has been demonstrated by echo-Doppler flowmetry in patients with chronic liver disease, but its diagnostic role is unclear. Recent reports regarding the validity and clinical relevance of Doppler flowmetry in measuring changes in postprandial portal blood flow are controversial (13, 14, 15). The purpose of our study was to investigate the effect of the meal stimulation on portal haemodynamic changes. This study represents an attempt to correlate portal haemodynamics with the severity of portal hypertension and hepatic failure.

## MATERIAL AND METHODS

We examined two groups of subjects: 20 patients with liver cirrhosis and 10 healthy controls. Patients with liver cirrhosis were divided into three groups according to Child-Pugh score: A, B and C. The diagnosis of liver cirrhosis was based on clinical (history and physical examination: jaundice, vascular spiders, dilated superficial vessels of abdomen, peripheral oedema, ascites), sonographic (hyperechogenity of the liver, reduction of peripheral vessel pattern of the liver, diameter of portal vein > 13mm, ascites, splenomegaly, visible collaterals), and laboratory criteria (hypoalbuminaemia, hyperbilirubinaemia, prolonged prothrombin time and other coagulation disorders, signs of hypersplenism). None of the patients had clinical or echocardiographic signs of failure of the right side of heart. Characteristics of patients and the relevant clinical data are shown in Table 1.

The control group consisted of 10 healthy volunteers (subjects). We measured the portal vein diameter, portal flow velocity (PBV), and portal volume flow with duplex Doppler spectral

and color ultrasonography. All Doppler procedures were done with commercialy available Doppler unit Logiq 500 (General Electric Medical Systems). Duplex Doppler ultrasonography was performed during both fasting state (an overnight fast) and 30 minutes after ingestion of a standard meal (60 kJ per kg of body weight). All patients and controls were examined in the supine position by a single investigator. Doppler findings were compared with the presence and size of oesophageal varices and Child-Pugh class.

Statistics. Statistical analysis was performed using the unpaired Student's t-test, the Mann- Whitney U-test, the $\chi^2$ test with Fisher's correction. The significance level was set at $p< 0.05$. Results are expressed as means ± SD.

Table 1. Characteristics of patients with liver cirrhosis and control group

| | Patients (n=20) | Controls (n=10) |
|---|---|---|
| Age* (years) | 53 ±14 | 43 ±12 |
| Range of age (years) | 34 - 75 | 22 - 65 |
| Sex: female<br>male | 7<br>13 | 3<br>7 |
| Etiology of liver disease:<br>- alcoholic<br>- HBV infection<br>- HCV infection<br>- HBV and HCV infection<br>- primary biliary cirrhosis<br>- unknown | <br>13<br>3<br>1<br>1<br>1<br>1 | |
| Child-Pugh classification<br>A<br>B<br>C | <br>6<br>10<br>4 | |
| Duration of liver disease (years)*<br>(range) | | |

* All data are expressed as mean ± SD

## RESULTS

No correlation was found between portal vein measurements and presence and size of varices. The portal vein flow volume did not differ in the presence of varices or ascites. The lowest values of the mean baseline portal flow velocity (PBV) (during fasting) were observed in cirrhotic patients class C (15.9 ± 4.2 cm/s vs class B 18.1 ± 7.8 cm/s vs class A 20.4 ± 8.6 cm/s vs controls 21.6 ± 3.8cm/s). There was a significant decrease in the portal flow velocity in patients with Child's C cirrhosis as compared to patients with class A and class B cirrhosis and control group. A similar situation appeared after our assessment of PBV values in the postprandial state (class C 16.5 ± 4.8cm/s vs class B 19.8 ± 9.1cm/s vs class A 22.4 ± 6.4cm/s vs controls 29.6 ± 10.6cm/s). There was no difference in the maximum inner diameter of the portal vein in cirrhotics and controls. The increment of PBV after a meal in patients with the Child-Pugh class C was significantly diminished compared to control group (0.6 ± 4.1cm/s vs 8.0 ± 5.2cm/s; $p< 0.05$). The results of our study are shown in Table 2.

Table 2. Portal blood velocity (cm/s) in patients with cirrhosis and healthy individuals (controls) *

| Study group | Portal baseline | Blood postprandial | Velocity increase |
|---|---|---|---|
| Controls (n= 10) | 21.6 ± 3.8 | 29.6 ± 10.6 | 8.0 ± 5.2 |
| Cirrhosis class A (n= 6) | 20.4 ± 8.6 | 22.4 ± 6.4 | 2.0 ± 7.1 |
| Cirrhosis class B (n= 10) | 18.1 ± 7.8 | 19.8 ± 9.1 | 1.7 ± 5.3 |
| Cirrhosis class C (n= 4) | 15.9 ± 4.2 | 16.5 ± 4.8 | 0.6 ± 4.1 |

* All data are expressed as mean ± SD

## DISCUSSION

Duplex doppler flowmetry gives non-invasive access to the portal system and permits the measurements of portal hemodynamics during pre- and postprandial states (3, 6, 15). It is a reliable technique to evaluate changes in portal hemodynamic parameters during a short period of time in patients with cirrhosis. Changes in architecture of the liver in cirrhosis and chronic active hepatitis affect hepatic vascular haemodynamics (3, 4, 8, 11). The previous studies showed that portal venous pressure increases in patients with cirrhosis after a meal and postprandial haemodynamic changes were maximum 30 min after the meal (4, 7, 13). Thus we assessed our patients at that interval of time. Food intake induced increases in flow parameters in all groups of patients and healthy individuals. These findings agree with previous reports that portal vein flow increases after all meals, especially after fat, but also after the control meal (7). It suggests that described hemodynamic changes may contribute to the triggering of the hemorrhagic episodes, because any increase in portal venous pressure in cirrhotic patients may be a risk factor for variceal bleeding. We observed that baseline portal velocity and postprandial hyperaemia tend to be lower in cirrhosis when compared to controls. Healthy volunteers and patients who suffer from liver cirrhosis Child's class C differ significantly with respect to their postprandial hyperaemia. Previous reports revealed that blunted increase in postprandial blood velocity was more dependent on Child-Pugh classification than on the grading of oesophageal varices (3, 10, 13) and our findings correspond to that. Results of the current study suggest that portal blood velocity may be a useful test for differentiating chronic liver disease. They indicate that portal flow significantly decreases in cirrhotic patients with worsening Child's grade of cirrhosis. Other authors also observed that severe liver failure was associated with lower portal velocity and flow; portal blood velocity was a more sensitive indicator for assessing portal hemodynamics (8, 11, 13). However, the portal vein velocity measurement alone are not useful parameter for discriminating patients with cirrhosis from healthy subjects. It was not useful in the diagnosis of early cirrhosis. Controls and patients with Child-Pugh's class A liver cirrhosis showed similar hemodynamic changes. Further studies are required to investigate a possible relationship between hemodynamic postprandial changes and severity of chronic liver disease.

## CONCLUSION

The postprandial portal blood velocity assessment is useful for evaluation of the severity of liver disease.

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## SUMMARY


The aim of our study was to check the significance of portal blood velocity /PBV/ in evaluation of the severity of liver disease. Duplex doppler sonography evaluation of PBV was performed in 20 patients (pts) with liver cirrhosis (7 women, 13 men, aged 34–75) and 10 pts with normal liver function (controls). We checked it during fasting and 30 minutes after a meal. The diagnosis of liver cirrhosis was based on clinical, laboratory and sonographic criteria. The severity of liver cirrhosis was classified according the Child– Pugh score as class A– 6 pts.; class B – 10 pts. and class C – 4pts. The lowest values of the mean baseline PBV (during fasting) were observed in cirrhotic pts class C (15.9 ± 4.2 cm/s vs class B 18,1 ± 7.8 cm/s vs class A 20.4 ± 8.6 cm/s vs controls 21.6 ± 3.8cm/s). A similar situation appeared after our evaluation of PBV values in the postprandial state (class C 16.5 ± 4.8cm/s vs class B 19.8 ± 9.1cm/s vs class A 22.4 ± 6.4cm/s vs controls 29.6 ± 10.6cm/s). The increment of PBV after a meal in pts with the Child-Pugh class C was significantly diminished compared to control group

(0.6 ± 4.1cm/s vs 8.0 ± 5.2cm/s; p< 0.05). We concluded that the postprandial portal blood velocity assessment is useful for evaluation of the severity of liver disease.

## Wartość diagnostyczna pomiarów szybkości przepływu krwi w żyle wrotnej w ocenie zaawansowania marskości wątroby

Celem badania było określenie znaczenia szybkości przepływu krwi w żyle wrotnej (PBV) w ocenie zaawansowania choroby wątroby. Pomiary PBV metodą ultrasonografii Duplex Doppler przeprowadzono u 20 pacjentów z marskością wątroby (7 kobiet, 13 mężczyzn w wieku 34–75 lat) oraz u 10 chorych z prawidłową funkcją wątroby (grupa kontrolna). PBV oceniano na czczo i 30 min. po posiłku. Marskość wątroby została zdiagnozowana w oparciu o kryteria kliniczne, laboratoryjne i ultrasonograficzne. Pacjentów podzielono na podstawie stopnia zaawansowania marskości wątroby, określonego wg skali Child–Pugh: klasa A–6 pts; klasa B–10 pts i klasa C–4 pts. Najniższe wartości PBV na czczo obserwowano w grupie pts z marskością klasy C (15,9 ± 4,2 cm/s vs class B 18,1 ± 7,8 cm/s vs class A 20,4 ± 8,6 cm/s vs controls 21,6 ± 3,8cm/s). Podobna sytuacja występowała w czasie oznaczania wartości PBV po posiłku (class C 16,5 ± 4,8cm/s vs class B 19,8 ± 9,1cm/s vs class A 22,4 ± 6,4cm/s vs controls 29,6 ± 10.6cm/s). Poposiłkowy wzrost PBV u chorych z marskością w klasie Child-Pugh C był istotnie statystycznie obniżony w porównaniu z wartością grupy kontrolnej (0,6 ± 4,1cm/s vs 8,0 ± 5,2cm/s; p < 0,05). Pomiar poposiłkowych zmian w szybkości przepływu krwi w żyle wrotnej jest przydatny w ocenie stopnia zaawansowania marskości wątroby.