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Hepatic perfusion index in portal hypertension of cirrhotic and non-cirrhotic aetiologies

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Summary

The hepatic perfusion index (HPI), the ratio of hepatic arterial to total liver blood flow, was determined by radionuclide angiography in 28 subjects with normal livers and 62 patients with portal hypertension of various aetiologies. The latter group comprised 50 patients with cirrhosis (14 Child class A, 20 Child class B, 16 Child class C) and 12 patients with non-cirrhotic portal hypertension (7 non-cirrhotic portal fibrosis, 5 extrahepatic portal venous obstruction). The mean (± S.D.) HPI was significantly higher among the patients with cirrhosis (Child class A, 53.9 ± 18.1; Child class B, 65.6 ± 29.4; Child class C, 78.6 ± 33.5) and non-cirrhotic portal hypertension (54.9 ± 17.7) compared with the subjects with normal livers (35.6 ± 10.5). The patients with non-cirrhotic portal hypertension had a mean value similar to that of the cirrhotic patients in Child class A. A higher HPI was associated with worsening liver status. We conclude, therefore, that the HPI will be high in portal hypertension irrespective of aetiology and a rise in the HPI may indicate a deterioration in the condition of the liver.

Introduction

The normal liver receives about 20–25% of its blood supply from the hepatic artery and the rest via the portal vein. Portal venous flow increases in some patients with gross splenomegaly, but in virtually all types of liver disease portal blood flow is reduced. The measurement of the ratio of hepatic arterial to total liver blood flow may be useful in assessing the severity of liver disease [1] and sequential measurements may indicate progression of the disease or its response to treatment [2].

A number of methods for measuring liver blood flow have been developed, each of which has its disadvantages. For example, clearance techniques which depend upon hepatocyte extraction require hepatic vein catheterization. Indicator dilution techniques and electromagnetic flowmeters measure the total volumetric flow through the liver but not the effective or nutritive flow, which is the clinically significant factor. And ultrasound is handicapped by non-linear flow and turbulence within the vessels.

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An ideal technique would measure both total and effective (nutritive) liver perfusion and would be safe, simple to perform, reproducible and non-invasive [3]. Radionuclide methods fulfil most of these requirements. This approach does not measure total liver blood flow but estimates the relative proportion of hepatic arterial to total liver inflow, termed the hepatic perfusion index (HPI) [4]. The HPI has been shown to change in the presence of chronic liver disease and hepatic metastases [1,5].

In this study, we performed radionuclide angiography on subjects with normal livers and patients with portal hypertension of various aetiologies using $^{99}Tc^{m}$-phytate, the radiopharmaceutical routinely used by us for liver scans.

Methods

The 28 individuals with normal livers were referred to us in an effort to rule out liver involvement in the presence of hepatic disease. Their liver function tests and ultrasonography results were normal, as were their static radionuclide scans. The 62 patients with portal hypertension of various aetiologies were categorized on the

basis of clinical findings, liver function test results, ultrasonography and histology. Based on their Child–Pugh score, the 50 patients with cirrhosis were classified as Child class A, B or C.

All of the subjects were required to refrain from eating for a period of 8 h before the procedure in order to avoid any increase in portal flow related to the consumption of food. A large field-of-view scintillation camera (Apex SP4, Elscint), equipped with a low-energy, general-purpose, parallel-hole collimator (APC3, Elscint) and interfaced to a dedicated 32 bit computer, was used in this study. With the patient in a supine position, anterior images of the liver, spleen and kidneys were obtained. A rapid bolus of 185 MBq (5 mCi) $^{99}Tc^{m}$-phytate was injected into an antecubital vein. The images were acquired for 60 s at a rate of one frame per second into a 64×64 matrix.

All of the frames in each study were summated to form composite images. From these composite images, regions of interest (ROIs) were drawn for the liver (avoiding the bases of the lungs, right kidney, aorta and inferior vena cava) and the right kidney. Time–activity curves were then generated from these two ROIs. If the bolus was adequate (half-rise time of renal curve = 8 s or less), the average slope of the liver curve was calculated for two 8 s periods immediately before and after the peak of the renal curve. These are considered to be proportional to the hepatic arterial and portal venous inflow, respectively [4, 5].

The HPI was defined as:

$$HPI = G_1/(G_1 + G_2) \times 100$$

where G_1 is the gradient (or slope) of the arterial phase and G_2 is the gradient (or slope) of the portal phase [6].

Results

The HPI was significantly higher among the patients with portal hypertension compared with the subjects with normal livers (Table 1). In patients with cirrhosis, the index rose progressively with worsening liver function. Patients with non-cirrhotic portal hypertension had a mean value similar to that of the Child class A cirrhotic patients. Regarding the patients with non-cirrhotic portal hypertension, there was no difference between those with non-cirrhotic portal fibrosis ($n = 7$) and those with extrahepatic portal venous obstruction ($n = 5$).

Table 1. Hepatic perfusion index in the normal and disease states.

Diagnosis	*Hepatic perfusion index*	
	Mean ± S.D.	*Median (range)*
Normal liver ($n = 28$)	35.6 ± 10.5	35.6 (18–55)
Cirrhosis of the liver		
Child A ($n = 14$)	53.9 ± 18.1	50.0 (30–95)[a]
Child B ($n = 20$)	65.6 ± 29.4	63.5 (23–120)[a]
Child C ($n = 16$)	78.6 ± 33.5	90.0 (16–128)[b]
Non-cirrhotic portal hypertension ($n = 12$)[c]	54.9 ± 17.7	57.0 (32–76)[a]

[a]$P < 0.001$ compared with normal liver; [b]$P < 0.01$ compared with Child class A cirrhosis.
[c]Includes non-cirrhotic portal fibrosis ($n = 7$) and extrahepatic portal venous obstruction ($n = 5$).

Discussion

The relative contributions of the hepatic artery and portal vein to total liver blood flow may be affected by physiological, pathological and pharmacological factors. Among patients with portal hypertension of various aetiologies, portal venous inflow is decreased; thus the hepatic arterial/portal venous inflow ratio may be considerably disturbed. This may have metabolic consequences, considering the nutritive role of portal venous blood. In addition, the drugs employed in the management of portal hypertension may affect portal venous flow, with variable effects. Consequently, there is a need for a simple, accurate and non-invasive method to determine the hepatic arterial and portal venous contributions to total blood flow in patients with chronic liver disease.

We performed radionuclide angiography on subjects with normal livers and patients with portal hypertension of various aetiologies and found the HPI to be significantly higher in the patients with portal hypertension. Among the patients with portal hypertension, those with relatively well-preserved liver function had a lower index (i.e. patients with non-cirrhotic portal hypertension and Child class A cirrhosis).

Some of the HPI values recorded, particularly among the patients with Child class C cirrhosis, were 100+. This is because the slope of the portal phase of the hepatic curve is affected by the simultaneous washout of arterial tracer flow. When portal blood flow is markedly decreased, this may lead to underestimation. In severely reduced portal flow, the upslope of the portal phase may even be replaced by a downslope, leading to HPI values of 100+. Similar observations have been made elsewhere [4, 5].

Although the HPI has been determined in patients with cirrhosis of the liver [e.g. 6], there are few data on changes in non-cirrhotic causes of portal hypertension. This study has shown that alterations in the HPI occur irrespective of the aetiology of the portal hypertension and reflect the degree of liver damage.

Conclusion

The HPI is generally higher among patients with portal hypertension compared with normal subjects. Among the former group, the HPI reflects indirectly the degree of liver dysfunction or architectural distortion, though it may not differentiate between non-cirrhotic and early cirrhotic disease. The HPI, therefore, may be used not only as a diagnostic tool, but also as a prognostic tool based on the extent of current damage.

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