Hepatofugal Collaterals in Advanced Liver Cirrhosis: Identification with CT Portography

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Portal hypertension is a common syndrome that is characterized by pathologic increase in portal venous pressure and by the formation of portal systemic collaterals that shunt part of portal venous blood to the systemic circulation. Knowledge of hemodynamic circulation and portal venous collateral pathways is important for clinical physicians. Multi-detector row computed tomography (CT) offers important advantages over conventional imaging methods in the evaluation of the portal venous vasculature because of faster scanning and thinner collimation. These advances allow excellent visualization of the portal veins and hepatofugal collateral channels. Three-dimensional CT portography may also have technical potential to generate excellent image for volumetric reconstructions and hemodynamic evaluation of the portal vein and its collaterals. From January to December 2004, 148 patients with liver cirrhosis were enrolled for this study and hepatofugal collaterals were identified in 112 patients. Three-dimensional CT portography provided comprehensive mapping of vascular compromise and collateral formation and therefore, improved medical treatment quality.

Key words: Computed tomography (CT); portography; Hepatofugal collaterals; Liver cirrhosis; Portal hypertension

Cirrhosis is an end stage of chronic diffuse liver disease which is characterized by alternation of the normal liver architecture into abnormal nodules of liver cells surrounded by fibrosis. The fibrosing and regenerative process may affect the intrahepatic vasculature and hemodynamics which may in turn participate in portal hypertension. Portal hypertension is defined as portal pressure higher than 10 mmHg [1]. Portal hypertension is a common syndrome that is characterized by a pathologic increase in portal venous pressure and by the formation of portosystemic collaterals that shunt part of portal venous blood to the low pressure systemic circulation. Regulation of splanchnic blood flow and portal venous flow is modulated by many complicated factors. Detection of spontaneous hepatofugal collateral portosystemic shunt is clinically important for diagnosis of portal hypertension and predicting prognosis in patients with liver cirrhosis because this hemodynamic pattern is more frequent in patients with advanced liver cirrhosis. Portal venous pressure may be measured directly by invasive catheterization of the portal vein or indirectly by hepatic vein catheterization [2-5]. Depending on the involved segments of the portal venous system, demonstration of hepatofugal flow is also possible at angiography after injection of contrast medium into the hepatic, superior mesenteric or splenic artery. With advent of Doppler ultrasonography, main portal vein hepatofugal flow is usually detected by Doppler ultrasound without difficulty [6]. In recent years, with more readily available of multi-detector row computed tomography (CT) in medical practice which allows entire assessment of the portal venous system and its collaterals, abdominal CT angiography is gaining increasing acceptance as a minimally invasive technique for imaging portal vascular system [7-11]. The increased speed and narrow collimation of multi-detector row CT, together with technical improvement of intravenous injection of contrast medium, CT angiography really generates...
Hepatofugal collaterals in cirrhosis

excellent images for volumetric reconstructions and hemodynamic evaluation of the portal veins and its collaterals. The purpose of this study is to assess patterns of spontaneous collateral pathways of the portal venous system in advanced liver cirrhosis patients with portal hypertension as demonstrated by three-dimensional CT angiography and advanced image processing.

CT Angiographic Scanning and Postprocessing Techniques

Between January and December 2004, 148 patients with hepatitis-B or -C liver cirrhosis were enrolled for this study. Exclusion criteria included (1) history of hepatectic surgery, (2) many tumors occupied the majority of liver, especially around the porta hepatis, (3) patients with portal vein tumor thrombosis, (4) history of transcatheter arterial chemoembolization for hepatocellular carcinoma and (5) portal or hepatic vein was not well depicted by contrast enhancement. The main purpose of CT examination was to evaluate the pathways of hepatofugal portosystemic collaterals in portal venous phase CT portography.

All CT images were performed on a sixteen channel Siemens Sensation 16 CT scanner (Siemens Sensation 16; Siemens AG, Germany). The scanning range was planned starting from the distal esophagus and ending at the low pole of the kidneys. Plain CT was performed before administration of contrast medium. Dynamic CT was performed during inspiratory breath-hold following administration of 100 ml of a non-ionic iodinated contrast medium (Ultravist 370; Schering, Berlin, Germany; 300 mg iodine/ml) with injection rate of 3ml/s using an automated injector (CT9000, LF, USA). Arterial phase imaging of the liver is initiated 25-30s after the initiation of contrast medium injection. Portal venous phase images are acquired after a delay of 60s from start of the injection and equilibrium phase images were taken 120s later. Technical parameters for the scanning were listed as follows: Scanning time was 0.75s/rotation; detector collimation was 1.5mm and slice thickness was 2.0mm; table feed was 5.0mm per rotation (Pitch 2); X-ray at 200mA and 120kVp; reconstruction interval was 1.25mm.

For three-dimensional (3D) reconstruction, all CT datasets were transferred to a Siemens Leonado Workstation. Source image of the three-dimensional CT angiography were obtained from the volumetric data obtained during portal venous phase. The 3D volume dataset was then manipulated by using various orientations and cut planes to best demonstrate the vascular anatomy of the portal vein and its tributaries. Maximum intensity projection was a preferred algorithm for creating vascular maps. The processing was performed by a CT-special technologist and the radiologist. All images of the three dimensional reconstructions were stored in the hard disk memory of the workstation and hospital PACS (Picture archiving and communication system) for image analysis. All images of the CT angiography were reviewed on workstation by the radiologist.

Normal Portal Vein

The liver has duplicated blood supply, deriving from hepatic artery and portal vein. The hepatic artery is normally originates from the celiac trunk. Variations in the arterial system are present in 40% of individuals. However, variations in the portal vein system are less common [12].

The main portal vein is formed by the union of the splenic and superior mesenteric veins. The portal venous system comprise all veins of abdominal part

Figure 1. Normal portal and hepatic veins. The right portal vein courses horizontally and divides into anterior and posterior branches. The left portal vein ascends to the left intersegmental fissure, where it divides into branches supplying the medial and lateral segments of the left lobe of liver. The middle hepatic vein runs the superior margin between right and left lobes. The left hepatic vein courses between the medial and lateral segments of the left lobe. The right hepatic vein courses obliquely between and anterior and posterior segments.
Hepatofugal collaterals in cirrhosis

of the digestive organs including lower esophagus, stomach, small and large intestine, biliary tract, pancreas and spleen. Tributaries of the portal vein are the splenic vein, superior mesenteric vein, inferior mesenteric vein, left gastric vein, right gastric vein, pancreaticoduodenal vein and cystic vein. The main portal vein courses toward right and superiorly in the hepatoduodenal ligament, along with the hepatic artery and common bile duct, anterior to the foramen of Winslow. At porta hepatitis, portal vein divides into the right and left branches. The right portal vein courses horizontally before bifurcating into anterior and posterior branches. The left portal vein ascends anterior to the caudate lobe before it courses ventrally to the left intersegmental fissure, where it divides into branches supplying the medial and lateral segments of the left lobe of liver. Venous blood drained from the liver by the hepatic vein system, which consists of the right, middle and left hepatic veins. All these veins drain into the inferior vena cava at the level of the diaphragm (Fig. 1). Rarely, a separate inferior right hepatic vein drains directly into the inferior vena cava caudally. The diameter of the main portal vein in normal individuals ranges from 0.64 to 1.21 cm. Diameter of the superior mesenteric vein is smaller than portal vein. Normally, the diameter of the right portal vein is larger than that of left portal vein. The caliber of coronary (left gastric) vein smaller than 5mm on CT image is considered normal [13].

Portal hypertension develops when the resistance of blood flow within portal vein system increases and/or portal blood flow increases.

**Figure 2.** Portal hypertension without collaterals: Early stage portal hypertension. The main portal vein is relatively large. The left and right portal veins are short and their branches are small. There is no hepatofugal collateral vessels can be identified. The spleen is large. Arteriosclerosis with calcified plaques is present in the wall of aorta, splenic artery and superior mesenteric artery. MPV: main portal vein. SMV: superior mesenteric vein. SV: splenic vein.

**Figure 3.** Portal hypertension with engorgement of mesenteric veins. **a.** The gastroepiploic vein (thick arrow) and mid-colic veins (thin arrow) and **b.** mesenteric veins (M) are markedly enlarged.
A portal vein larger than 1.3cm in diameter is a specific sign for portal hypertension [13]. Portal vein enlargement may be while a characteristic of portal hypertension in the appropriate clinical setting (Fig. 2), however the diameter of main portal vein may be normal or may even diminish as blood flow is diverted to opening collateral veins. On CT scans, portosystemic collaterals appear as tortuous, tubular, or round soft tissue masses that enhanced after intravenous contrast medium administration. Enlargement of gastroepiploic, middle colonic or mesenteric veins may be seen on the CT images (Fig. 3). One third of the patients with liver cirrhosis and portal hypertension present colonic wall thickening. This finding is related to radiological features and clinical consequences of portal hypertension [14].

**Portal Hypertension with Hepatofugal Collaterals**

CT was excellent in demonstrating portal hypertension with its attendant varices. Varices or portosystemic collateral channels appeared as tortuous, dilated tubular structures in esophageal, gastric, paraumbilical, retroperitoneal, periduodenal or intrahepatic region. Of the 148 patients with liver cirrhosis, Portal hypertension with hepatofugal collaterals was identified in 112 patients. Frequency of major collateral channels in this study was showed in Table 1.

### Table 1. Frequency of hepatofugal collateral channels in 112 patients with portal hypertension

<table>
<thead>
<tr>
<th>Collateral channel</th>
<th>Percentage</th>
</tr>
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<tbody>
<tr>
<td>1. Esophageal/paraesophageal</td>
<td>58.9</td>
</tr>
<tr>
<td>2. Gastric varices</td>
<td>24.1</td>
</tr>
<tr>
<td>3. Splenorenal shunt</td>
<td>16.1</td>
</tr>
<tr>
<td>4. Mesenteric or omental varices</td>
<td>5.4</td>
</tr>
<tr>
<td>5. Retroperitoneal varices</td>
<td>5.4</td>
</tr>
<tr>
<td>6. Pancreaticoduodenal varices</td>
<td>3.6</td>
</tr>
<tr>
<td>7. Carvenous transformation of portal vein</td>
<td>2.7</td>
</tr>
<tr>
<td>8. Recanalized paraumbilical vein</td>
<td>32.2</td>
</tr>
<tr>
<td>9. Intrahepatic portosystemic shunt</td>
<td>3.6</td>
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**1. Esophageal and Paraesophageal Varices**

Coronary venous collateral vessels are the most common collateral route of portosystemic shunt in our series. The coronary vein was seen in the region between posterior margin of left hepatic lobe and medial wall of gastric body on CT image. The diameter of coronary vein larger than 5mm is considered as an indicator of portal hypertension with coronary venous varices. The venous blood is usually divert to esophageal and/or paraesophageal varices and finally into azygos system and occasionally diverts to the posterior gastric vein.

Reverse flow of the left gastric vein usually
contributes to the formation of esophageal and/or paraesophageal varices. Unfortunately, esophageal various veins are the most common bleeding source of cirrhotic patients with gastrointestinal bleeding [15-17]. It is a serious complication and life-threatening problem. In addition to coronary vein, the posterior gastric veins may also feed the esophageal varix. Esophageal varices are frequently associated with paraesophageal varices. On CT scans, esophageal varices appear as enlarged, tortuous veins situated in the wall of lower esophagus (Fig. 4). Esophageal wall is thick. Paraesophageal varices situate outside the esophageal wall in the posterior mediastinum (Fig. 5). Blood from the esophageal varices usually drains into the left subclavian vein and/or brachiocephalic vein, while the blood of the paraesophageal varices commonly drain into the azygos vein (Fig. 5b). The esophageal or paraesophageal varix may rarely drain into the inferior vena cava (Fig. 4b). The frequency of esophageal varices in our series is 56%.

2. Gastric and Perigastric Varices

The left, posterior or short gastric vein may significantly enlarge in patients with portal hypertension. These enlarged veins not only contribute in the formation of esophageal varices, but also forms submucosal gastric varices within the fundus of stomach or perigastric areas (Fig. 6) and, the three-dimensional CT angiography provides excellent visualization of submucosal gastric fundal varices and perigastric varices (Fig. 16) as well as afferent and efferent veins. The gastric fundal varices drain commonly into the esophageal or paraesophageal veins. It may also drain into left renal vein by way of gastrorenal shunt or it may...
Hepatofugal collaterals in cirrhosis

6 drain cephaladly into left inferior phrenic vein via pericardiophrenic vein (Fig. 7). The superior mesenteric venous blood may occasionally flow into the left gastric vein and then to left renal vein directly via a gastrorenal shunt (Fig. 8). We found gastric varices in 24.1% of our patients. Gastric varices are the second common site of rupture of portosystemic collateral vessels.

3. Splenorenal Shunt

Varices form around the splenic hilum usually
Hepatofugal collaterals in cirrhosis

flow into the left renal vein directly via a splenorenal shunt (Fig. 9). The splenic venous blood may also drain into left suprarenal vein then the left renal vein via splenoadrenorenal shunt (Fig. 10). Dilatation of the left renal vein and superior portion of the inferior vena cava may be seen in this circumstance.

4. Mesenteric and Omental Varices

Mesenteric and omental varices may be seen in patients with portal hypertension as small but numerous veins in the mesentery (Fig. 11) and greater omentum (Fig. 12). They may communicate with perisplenic varices or varices in the retroperitoneum. The mesenteric varices may drain into the inferior vena cava through the dilated right gonadal vein. Awareness of a dilated gonadal vein in patient with portal hypertension may be helpful in consider the possibility of mesenteric varices [18]. The inferior mesenteric vein may divert caudally to the rectal venous plexuses and flows into the internal iliac vein around the rectum.

5. Retroperitoneal Varices

Varices may rarely occur in the retropancreatic, perisplenic (Fig. 13) and perirenal area (Fig. 14). Varices in these locations may communicate with retrogastric varices or inferior phrenic vein. Varices may also seen in paravertebral area (Fig. 16). They may drain directly into the inferior vena cava.

6. Duodenal Varices

Duodenal varices resulting from intrahepatic portal hypertension is rather uncommon, but do occur. The varix transverse the duodenum and is present in the submucosal layer of the posterior wall (Fig. 15, 16). The afferent vessel is superior or inferior pancreaticoduodenal vein originating in the portal vein trunk or superior mesenteric vein. The efferent vein drains into the azygos or hemiazygos vein.

7. Cavernous Transformation of the Portal vein

Communication among the right gastric, pancreatoduodenal and cholecystic venous branches
Hepatofugal collaterals in cirrhosis

Figure 11. Mesenteric varices. A 60-year-old male with HCV-related liver cirrhosis. Contrast-enhanced CT in portal phase, right anterior oblique projection, shows multiple tortuous mesenteric varices (long arrow). The mesenteric varical veins finally drain into hemiazygos or azygos vein. Recanalized paraumbilical veins are also present (short arrows).

Figure 12. Omental varices. Numerous omental varices are present as a venous network. The vessels communicate with perisplenic veins.

Figure 13. Retroperitoneal varices. Dilated perisplenic venous plexuses forming a perisplenic varices (white arrow).

Figure 14. Retroperitoneal varices. CT portography shows retroperitoneal varices in left side pararenal space (short arrow). The varicous vein may drain into azygos vein. Note the azygos vein is large (long arrow).

around the bile duct forms the parabiliary venous system. These veins usually joint the main trunk or major branches of portal venous system but occasionally enter the liver directly around the porta hepatic. When the main portal vein is occluded, portal to portal collateral by way of parabiliary venous system may occur (Fig. 17).

8. Recanalized paraumbilical vein
Paraumbilical veins consist of small veins around the falciform ligament that drain the venous
Hepatofugal collaterals in cirrhosis

Blood from the anterior part of the abdominal wall directly into the liver. These veins are divided into three subgroups: The superior and inferior veins of Sappey and the vein of Burow. The superior vein of Sappey drains the upper portion of the falciform ligament and medial part of the diaphragm and enters peripheral portal branches of the left hepatic lobe. It also comminutes with branches of the superior epigastric or internal thoracic veins. The inferior vein of Sappey drains the lower portion of the falciform ligament and enters peripheral portal branches of the left hepatic lobe. It descends along the round ligament and communicates with branches of inferior epigastric veins around the navel. The vein of Burrow also communicates with branches of inferior epigastric veins around the navel. However, it does not enter the liver directly but terminates in the middle portion of the collapsed umbilical vein although some small branches are present between it and the inferior vein of Sappey [19, 20]. The flow through these vessels may be reversed in cirrhotic livers or at portal hypertension, causing the vessels to serve as an efferent flow tract from the liver. Figure 18 and figure 19 showed recanalized paraumbilical veins communicate with the left portal vein and run downward and subcutaneously toward the umbilicus, where it anastomosis with the epigastric and hypogastric vein (Fig. 18). The epigastric vein may communicate with pericardial vein. The hypogastric vein runs finally to the external iliac vein (Fig. 19).

Rarely we detect multiple engorged left portal veins run through the segment 3 hepatic parenchyma to the epigastric vein around the surface of the liver (Fig. 20).

Figure 15. Duodenal varices. An engorged tortuous vein is seen over posterior wall of the third portion of the duodenum (white arrow).

Figure 16. Duodenal varices. Coronal reconstruction CT image in another patient reveals paraduodenal varices (black arrow), paravertebral varices (thick white arrow) and paragastric varices (thin white arrow). IVC: inferior vena cava. d: duodenum. s: stomach.

Figure 17. Cavernous transformation of the portal vein. Parabiliary venous collateral channels. The main portal vein is small, however parabiliary veins are markedly dilated. Pancreaticoduodenal veins drains to segment 4 of liver by way of parabiliary veins (white arrow).
9. Intrahepatic Portosystemic Shunts

An anastomosis between the portal and hepatic veins may be congenital or acquired. Communication of a large portal vein and large hepatic vein can spontaneously occur in the patients with cirrhosis with portal hypertension through it is a rather rare condition. CT images can clearly show an aneurysmal dilatation of the portal vein. The communicated hepatic vein is well enhanced with contrast medium in portal phase CT image (Fig. 21).

CONCLUSIONS

Portal hypertension is defined as a portal pressure higher than 10 mmHg. Both increase the sinusoidal resistance to sinusoidal flow and increase portal venous blood flow may result increased pressure in the portal venous system. Patients with increased portal venous pressures can have a reversal flow in the portal system, changing from hepatopetal to hepatofugal.

Portal systemic shunting in portal hypertension can be associated with hepatofugal flow in the main portal vein, intrahepatic branches or extrahepatic tributaries, depending on the location of the shunt and the associated hemodynamic disturbances. Knowledge of these collateral pathways is important in both diagnosis and treatment of the patients.
Multi-detector row computed tomography of the portal venous system combined with multiplanar reconstructions and high fidelity volume rendering can provide comprehensive mapping of vascular compromise and collateral formation therefore, improves medical treatment quality.

REFERENCES


Figure 21. Intrahepatic portosystemic shunt (IPSS). a. Coronal projection CT portography. A single shunt occurs between the right portal vein and the middle hepatic vein. A round aneurysmal dilatation of the portal vein (black arrow) and early enhancement of prominently dilated hepatic vein (white thick arrows) are noted. b. CT portography in right anterior oblique view. PV: right portal vein. HV: middle hepatic vein.
Hepatofugal collaterals in cirrhosis

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嚴重肝硬化離肝性門靜脈側枝通路：
以CT門靜脈圖確認

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重度肝硬化病人病程緩慢但合併發生門靜脈高血壓症。病人門靜脈血壓增高，向肝性血行漸減，久之，曲張之側枝循環靜脈於是形成。在臨床上，醫師事先能確認病人之門靜脈血行狀況及側枝靜脈圖譜，對於治療計畫之擬定有極大的幫助。近年來，多切面電腦斷層攝影配合靜脈注射對比劑技術，對重度肝硬化病人提供品質極為優異的門靜脈及其側枝靜脈圖譜。自2004年元月至同年12月，本院148位肝硬化病人，利用16切面電腦斷層攝影機施行CT門靜脈攝影檢查，其中112人顯示為重度肝硬化合併門靜脈高血壓症性離肝性靜脈側枝循環。研究顯示它確實提供全方位品質之側枝循環靜脈圖譜。對於側枝靜脈之確認以及治療方向之選擇，提供極為珍貴和正確的資料。

關鍵詞：電腦斷層；門靜脈攝影；門靜脈側枝通路；門靜脈高血壓症；肝硬化症