Learning objectives

To illustrate various pathways of portosystemic communication on patients with portal hypertension, to describe them as they appear on CT scans, to classify and try to determine their significance in the pathological context.

Background

PORTAL HYPERTENSION

Normal pressure in the portal system varies between 5 and 10 mmHg. Normal hepatic blood flow of 550-900 ml/min (~ 25 % of cardiac output) passes through portal system (2/3) and through hepatic artery (1/3) \(^1\). Portal hypertension is defined as portal venous pressure greater than 10 mmHg.

Two important factors exist in the pathophysiology of portal hypertension\(^2\):

1. Vascular resistance and
2. Blood flow.

The relation between these two factors and portal pressure is defined by the following formula:

\[ P = FR \]

Where P- pressure gradient through the portal venous system, F- volume of blood flowing through the portal venous system. R- resistance to the flow. Changes in either F or R affect the pressure. In most types of portal hypertension, both the blood flow and the resistance are altered.

Increased portal resistance

The initial factor is the increase in vascular resistance in portal blood flow from a combination of deposition of collagen in the spaces of Disse and hepatocyte swelling. Each of them increases the sinusoidal pressure and causes relative resistance to sinusoidal flow.
Portal vascular resistance can be defined by the Poiseuille law, which states that, \( R = \frac{8hL}{pr^4} \) where:

- \( h \) is the viscosity of blood, related to the hematocrit
- \( L \) is the length of blood vessel which are relatively constant
- \( r \) is the radius of blood vessel

Changes in portal vascular resistance are determined primarily by the blood vessel radius. Because portal vascular resistance is indirectly proportional to the four power of the vessel radius, small decreases in the vessel radius cause large increases in portal vascular resistance and, therefore, in portal blood pressure.

Liver disease is responsible for decrease in portal vascular radius due to hepatic architectural disorder and dynamic component (active contraction of myofibroblast, activated stellated cells and vascular smooth muscle cells of intrahepatic veins).

**Increase in portal blood flow**

The second factor that contributes to the pathogenesis of portal hypertension is the increase in blood flow in portal veins due to splanchnic arteriolar vasodilatation caused by an excessive release of endogenous vasodilators. The increase in portal blood flow aggravates the increase in portal pressure and contributes to existence of portal hypertension despite the formation of an extensive network of portosystemic collaterals that may divert as much as 80% of portal blood flow.

**Formation of varices**

Varices form when the hepatic venous pressure gradient exceeds 10 mmHg. The hypertensive portal vein is decompressed by diverting up to 90% of portal flow through portosystemic collaterals and resulting in enlargement of these vessels.

**CLASSIFICATION OF CAUSES OF PORTAL HYPERTENSION**[^1]

<p>| Dynamic /hyperkinetic portal hypertension | Congenital, traumatic, neoplastic arteriportal fistula |
| Increased portal prehepatic resistance | Portal vein thrombosis, oral contraceptives, coagulopathy, neoplastic |</p>
<table>
<thead>
<tr>
<th>Portal vein compression</th>
<th>invasion, pancreatitis, neonatal omphalitis</th>
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<tr>
<td>Tumor, trauma, lymphadenopathy, portal phlebosclerosis, pancreatic pseudocyst</td>
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<tr>
<td>Intrahepatic presinusoidal (obstruction of portal venules)</td>
<td>Congenital hepatic fibrosis</td>
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<td>Idiopathic noncirrhotic fibrosis</td>
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<td>Primary biliary cirrhosis</td>
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<td>#1-antytripsin deficiency</td>
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<td>wilson disease</td>
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<td>sarcoid liver disease</td>
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<td>toxic fibrosis</td>
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<td>reticuloendotheliosis</td>
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<td>myelofibrosis</td>
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<td>Schistosomiasis</td>
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<td>Cystic fibrosis</td>
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<tr>
<td>Chronic malaria</td>
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<td>Hepatitis</td>
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<td>Sickle cell disease</td>
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<tr>
<td>Cirrhosis: Laennec cirrhosis, postnecrotic cirrhosis from hepatitis</td>
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Portal Vein

The portal vein is about 7 to 8 cm in length and carries the visceral blood to the liver, where it ramifies following the segmental pattern, reaching the sinusoids, from which the blood again converges to drain into the inferior vena cava through the hepatic veins. The portal vein results from the confluence of the splenic vein and the superior mesenteric vein. Besides the splenic and the superior mesenteric veins, other tributaries are the left gastric, right gastric, the paraumbilical, and cystic veins.

Splenic Vein

The splenic vein is one of the two larger tributaries of the portal vein, and its confluence with the superior mesenteric vein actually forms the portal vein. Tributaries: short gastric veins, left gastroepiploic vein, pancreatic vein.

Inferior Mesenteric Vein

The inferior mesenteric vein drains the rectum, the sigmoid colon and the left colon, joining the splenic vein distally, close to the confluence with the inferior mesenteric vein. Occasionally it ends at the union of the splenic and superior mesenteric vein, and sometimes at the superior mesenteric vein itself. Tributaries, superior rectal vein, sigmoid veins, left colic vein.

Superior Mesenteric Vein

The superior mesenteric vein is the largest tributary to the portal vein. It drains the small intestine, cecum, and ascending and transverse parts of the colon, carrying the blood to enter the portal circulation. The superior mesenteric vein is formed by the union of the tributaries from the terminal ileum, the cecum, and the appendix, receiving several other tributaries along its length.
ANASTOMOSES BETWEEN THE PORTAL AND SYSTEMIC CIRCULATION

In portal vein obstruction or portal vein hypertension due to liver disease, anastomoses between the portal vein and systemic veins may develop, carrying portal blood into the systemic circulation\[^3\].

There are four main groups of portal systemic collaterals.

**Group I** - protective mucosal epithelium adjoins absorptive epithelium.

Group I (A) At the cardia of the stomach, where the left gastric vein and short gastric veins of the portal system anastomoses with the intercostal, diaphragm-esophageal, and azygos tributaries, veins of the caval system, creating esophageal and gastric fundus varices.

Group I (B) At the anal canal the superior rectal (hemorrhoidal) vein, tributary of the inferior mesenteric vein (portal system), anastomoses with the middle and inferior rectal (hemorrhoidal) veins of the inferior vena cava system, creating hemorrhoids.

**Group II** - In the falciform ligament through the paraumbilical veins, vestiges of the umbilical circulation of fetal life. The enlargement of these connections, in the presence of portal hypertension, may produce varices of veins radiating from the umbilicus, the caput medusae (part of the Cruveilhier-Baumgarten syndrome). The remaining umbilical vein does not rechannel within the ligamentum teres.

**Group III** - the abdominal organs are in contact with retroperitoneal tissues or adherent to the abdominal wall (intercostal veins, lumbar veins). Includes veins from the liver to the diaphragm (veins of Sappey), veins in the lienorenal ligament and omentum, lumbar veins (veins of Retzius), and veins developed in adhesions and scars of previous surgeries.

**Group IV** - Connections between the portal system and the left renal vein. This may be through communications directly from the splenic vein or via diaphragmatic, pancreatic, left adrenal, gonadal, or gastric veins.

**Other Collaterals**

The communications from the gastroesophageal collaterals, retroperitoneal, and venous systems of the abdomen eventually reach the superior vena cava via the azygos or hemiazygos systems. Very rarely a patent ductus venosus connects the left branch of the portal vein to the inferior vena cava. In cases of extrahepatic portal venous obstruction,
additional collaterals develop toward the liver, entering the liver through the portal vein in the porta hepatis. These collaterals include the veins at the hilum, *venae comitantes* of the portal vein and hepatic arteries, veins in the suspensory ligaments of the liver, unnamed veins around the gallbladder, and diaphragmatic and omental veins.

**Spontaneous portosystemic shunts** *(Fig. 1 on page 22)*

1. connection to superior vena cava
   - esophageal varices (between subepithelial and submucosal veins) supplied by anterior branch of left gastric vein
   - paraesophageal varices supplied by posterior branch of coronary (left gastric) vein draining into azygos and hemiazygos venules and vertebral plexus. They are not connected to esophageal varices

2 connection to pulmonary circulation
   - gastropulmonary shunt (between gastric or esophageal vein and left pericardiophrenic or inferior pulmonary veins)

3 retrograde mesenteric flow
   - veins of Retzius (anastomoses between portal vein and inferior vena cava)
   ileocolic veins - right gonadal vein - IVC
   pancreaticoduodenal vein - IVC
   proximal small left branches of superior mesenteric vein - left gonadal vein - left renal vein
   ileocolic veins - directly into IVC

4 retroperitoneal collaterals
   - splenorenal or splenoadrenorenal shunts
   - gastrorenal shunt
   - mesenterorenal shunt (between superior mesenteric vein and right renal vein)
   - mesenterogonadal shunt (between ileocoloc vein and right testicular vein)
   - splenocaval shunt (between splenic vein and left hypogastric vein)

5 intrahepatic shunt (portal vein to hepatic vein)
Spontaneous portosystemic Shunts

<table>
<thead>
<tr>
<th>Type of varices</th>
<th>Frequency (%)</th>
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<tr>
<td>Coronary venous</td>
<td>80-86</td>
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<tr>
<td>Esophageal</td>
<td>45-65</td>
</tr>
<tr>
<td>Paraumbilical</td>
<td>10-43</td>
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<tr>
<td>Abdominal wall</td>
<td>30</td>
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<tr>
<td>Perisplenic</td>
<td>30</td>
</tr>
<tr>
<td>Retrogastric/gastric</td>
<td>2-27</td>
</tr>
<tr>
<td>Paraesophageal</td>
<td>22</td>
</tr>
<tr>
<td>Omental</td>
<td>20</td>
</tr>
<tr>
<td>Retroperitoneal-paravertebral</td>
<td>18</td>
</tr>
<tr>
<td>Mesenteric</td>
<td>10</td>
</tr>
<tr>
<td>Splenorenal</td>
<td>10</td>
</tr>
<tr>
<td>Gastrorenal</td>
<td>7</td>
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</tbody>
</table>

CT APPEARANCES OF VARICES IN PORTAL HIPERTENSION

On CT scan varices appear as well defined, round, tubular, or serpentine structures, that are smooth, have homogeneous attenuation, and enhance with contrast material to the same degree as adjacent vessels \(^4\). Varices are usually easy to detect on CT scan because are often surrounded by the fat in retroperitoneum, greater or lesser omentum and mesentery. Because without contrast they mimic pathological masses as adenopathy or tumors as well as bowel loops it is necessary to inject contrast.

Coronary venous collateral vessels

Are the most common seen portosystemic collaterals pathways in portal hypertension with a frequency of 80-86%. They are the most important collateral in cranially directed hepatofugal flow.

Coronary and short gastric venous collaterals are usually drained via coexisting esophageal and paraesophageal varices and occasionally via spontaneous gastrorenal or splenorenal shunts.
These appear as vascular channels in the triangular fatty tissue between the medial wall of upper gastric body and posterior margin of left hepatic lobe, the lesser omentum. (Fig. 2 on page 23)

**Fig.**: Dilated vessels in the triangular fatty tissue between the medial wall of upper gastric body and posterior margin of left hepatic lobe (black arrows).

**References:** Arcadia Medical Center, Iasi / Romania 2010
A left gastric vein larger than 5-6 mm or multiple dilated veins 4-6 mm in diameter in the lesser omentum on CT scan is considered abnormal and is an indicator in portal hypertension [4]. (Fig. 3 on page 24).

Fig.: Patient with cirrhosis and portal hypertension. Dilated coronary vein (white arrows)
References: Arcadia Medical Center, Iasi / Romania

Usually, coronary venous collaterals are associated with esophageal and paraesophageal varices and occasionally can associate retrogastric varices.

Gastric varices

Fed by left gastric vein or gastroepiploic vein gastric varices drain via esophageal or parasophageal veins and further into azygos or hemiazygos vein system. They appearance on CT scan as multiple round or tubular areas of increased attenuation in the posterior or postero-medial aspect of gastric fundus, near the cardia [4].
Esophageal and paraesophageal varices

Esophageal varices are the most common source of gastrointestinal hemorrhage.

Esophageal varices are formed by dilated subepithelial and submucosal vein and the dilated accompanying veins of vagus nerves that lie in the adventitia outside of *tunica muscularis* and are usually supplied by the anterior branch of the left gastric vein.

**Fig.**: Dilated submucosal veins prominent in gastric lumen, near the cardia (arrows).

**References:** Arcadia Medical Center, Iasi / Romania 2010
The size of the varices depends on the degree of portal hypertension and the presence of other portosystemic collaterals.[5]

Fig.: The CT appearances of esophageal varices are as intraluminal protrusion with scalloped margins (arrows).

References: Arcadia Medical Center, Iasi / Romania 2010

Paraesophageal varices are collateral vessels in the posterior mediastinum beyond the esophageal wall, supplied by posterior branch of left gastric vein draining into azygos and hemiazygos venules and vertebral plexus. They are situated outside the wall of the
esophagus in the mediastinum, parallel with the esophageal veins at the distance of 1-2 cm without direct communication with them. They are not connected to esophageal varices.

![Image: CT scan showing paraesophageal varices]

**Fig.**: Paraesophageal varices (white double arrows) appear as well defined, round, tubular, or serpentine structures situated outside the wall of the esophagus in the mediastinum.

**References:** Arcadia Medical Center, Iasi / Romania 2010

Endoscopy is the most reliable diagnostic procedure for detecting oesophageal varices. CT is able to detect paraesophageal varices and esophageal collateral vessels.

**Paraumbilical venous collateral vessels and abdominal wall varices**

The normal falciform ligament contains 1-3 paraumbilical veins. They are normally tiny and collapsed, and due to portal hypertension the number and caliber of this vein increase.

They arise from left portal vein and accompany the falciform ligament downwards to the umbilicus. On CT scans they appear as nodular or serpentine enhancing structures usually more than 3 mm diameter.
Abdominal wall varices appear as prominent veins radiating from the umbilicus. Usually they are one or two. When they are in large number the condition are named caput medusae, but this is rare. These collaterals are connected with superior and inferior epigastric veins and are fed by the paraumbilical and omental veins. The vast majority of paraumbilical flow return to the systemic circulation via one of the inferior epigastric vein (Fig 8 on page 29).

**Fig.**: Dilated paraumbilical veins anterior to left hepatic lobe (black arrows) arising from left portal vein (white arrow).

**References**: Arcadia Medical Center, Iasi / Romania 2010
Fig.: Abdominal wall varices radiating from the umbilicus. Connection with superior epigastric veins (A and C) inferior epigastric veins (D) and multiple vessels forming caput medusae (B).

References: Arcadia Medical Center, Iasi / Romania 2010

The abdominal wall varices are easily identified on CT scans because they are surrounded by fatty subcutaneous tissue.

Perisplenic varices

They appear as dilated veins antero-inferior to the spleen or superiorly and posteriorly and may extend to the left kidney. May communicate with retrogastric varices or dilated inferior frenic veins (Fig. 9 on page 30).
Fig.: Large perisplenic varices (white arrows) and dilated splenic vein (black arrows).

References: Arcadia Medical Center, Iasi / Romania 2010

Mesenteric varices

Appear as dilated or tortuous branches of mesenteric vein in the depth of mesentery.

These varices communicate with systemic veins via the retro-peritoneal and mesenteric venous plexus (Fig. 10 on page 31)
**Fig.**: Multiple round areas of increased attenuation in the depth of mesentery.  

**References:** Arcadia Medical Center, Iasi / Romania 2010

**Omental varices**

The CT appearances of omental varices are as numerous tortuous areas of increased attenuation in the greater omentum (**Fig.11** on page 32).
Fig.: Dilated vessels in the great omentum (white arrows)

References: Arcadia Medical Center, Iasi / Romania 2010

**Splenorenal or splenoadrenorenal shunts**

Are seen as large, tortuous veins between splenic and left renal hilus that drain into left renal vein and further into inferior vena cava. They can decompress the high pressure portal flow and produce a low portosystemic pressure gradient (Fig. 12).
**Fig.**: Direct spleno-renal shunt (A and B) and indirect shunt via perisplenic and left perirenal space vessels (C and D).

**References:** Arcadia Medical Center, Iasi / Romania 2010

**Transhepatic portosystemic shunts**

Transhepatic portosystemic shunts are classified as two types on the basis of the draining vein:

- in the hepatic venous type, the intrahepatic portal vein communicates with the hepatic vein in or on the surface of the liver (**Fig. 13** on page 33).
- in the systemic venous type, the intrahepatic portal vein runs toward the outside of the liver and communicates with the systemic veins (Fig. 14 on page 34).

**Fig.**: Intrahepatic portosystemic shunt between anterior branch of portal vein and right hepatic vein afluente (white arrow)

**References:** Arcadia Medical Center, Iasi/Romania 2010

**Fig.**: Branch of intrahepatic right portal vein communicates via perirenal collaterals with right renal vein and drains the portal flow into inferior vena cava.

**References:** Arcadia Medical Center, Iasi / Romania 2010

**Venous collaterals between inferior mesenteric vein and inferior vena cava** (Fig. 15 on page 34).

It is possible to have a direct connection through multiple collaterals that divert portal flow between inferior mesenteric vein and inferior vena cava.
**Fig.**: Dilated round vessels (arrows) joining inferior mesenteric vein with inferior vena cava.

**References**: Arcadia Medical Center, Iasi / Romania 2010

**Anal canal varices**

At the anal canal the superior rectal (hemorrhoidal) vein, tributary of the inferior mesenteric vein (portal system), anastomoses with the middle and inferior rectal
(hemorrhoidal) veins of the inferior vena cava system, creating hemorrhoids (Fig. 16 on page 35).

Fig.: Internal hemorrhoids (A) and perirectal venous collateral circulation (B and C).

References: Arcadia Medical Centre, Iasi / Romania 2010

Images for this section:
### Fig. 1: Spontaneous portosystemic shunts

<table>
<thead>
<tr>
<th>Superior vena cava</th>
<th>Pulmonary circulation</th>
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<tr>
<td>submucosal</td>
<td>Left cardiofrenic</td>
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<tr>
<td>subepithelial</td>
<td>Inferior pulmonary vein</td>
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<tr>
<td>azygos</td>
<td>Gastric veins</td>
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<td>hemiazygos</td>
<td>Esophageal veins</td>
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<td>Esophageal varices</td>
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<td>Paraoesophageal varices</td>
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<td>Anterior branch</td>
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<td>Posterior branch</td>
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<td>Left gastric vein</td>
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**Intrahepatic shunts**

- Splenic vein
  - Renal vein
  - Adrenal veins
  - Left hypogastric vein
- SMV
  - Left renal vein
  - Right renal vein
- Ileocolic
  - Right testicular vein
- Gastric vein
  - Renal vein
  - Ileocolic veins

**Veins of Retzius**

- Ileocolic vein
- Right gonadal vein
- Pancreaticoduodenal vein
- Proximal branches of SMV
- Left renal vein
- IVC

**Portal system**

- Splenorenal shunt
  - Splenocaval shunt
  - Mesenteric renal shunt
  - Mesenteric gonadal shunt
  - Gastrorenal shunt
- Retroperitoneal collaterals
Fig. 2: Dilated vessels in the triangular fatty tissue between the medial wall of upper gastric body and posterior margin of left hepatic lobe (black arrows).
Fig. 3: Patient with cirrhosis and portal hypertension. Dilated coronary vein (white arrows)
Fig. 4: Dilated submucosal veins prominent in gastric lumen, near the cardia (arrows).
Fig. 5: The CT appearances of esophageal varices are as intraluminal protrusion with scalloped margins (arrows).
Fig. 6: Paraesophageal varices (white double arrows) appear as well defined, round, tubular, or serpentine structures situated outside the wall of the esophagus in the mediastinum.
**Fig. 7:** Dilated paraumbilical veins anterior to left hepatic lobe (black arrows) arising from left portal vein (white arrow).
Fig. 8: Abdominal wall varices radiating from the umbilicus. Connection with superior epigastric veins (A and C) inferior epigastric veins (D) and multiple vessels forming caput medusae (B).
Fig. 9: Large perisplenic varices (white arrows) and dilated splenic vein (black arrows).
**Fig. 10:** Multiple round areas of increased attenuation in the depth of mesentery.

**Fig. 11:** Dilated vessels in the great omentum (white arrows)
Fig. 12: Direct spleno-renal shunt (A and B) and indirect shunt via perisplenic and left perirenal space vessels (C and D).
**Fig. 13:** Intrahepatic portosystemic shunt between anterior branch of portal vein and right hepatic vein affluent (white arrow)

**Fig. 14:** Branch of intrahepatic right portal vein communicates via perirenal collaterals with right renal vein and drains the portal flow into inferior vena cava.
Fig. 15: Dilated round vessels (arrows) joining inferior mesenteric vein with inferior vena cava.
Fig. 16: Internal hemorrhoids (A) and perirectal venous collateral circulation (B and C).
Conclusion

Computed tomography is capable of accurately demonstration of many of the varied manifestations of portal hypertension.

The portosystemic collateral channels that can develop in portal hypertension secondary to cirrhosis are numerous and may vary in appearance. It is important for the radiologist to be familiar with aspects which may be present on cirrhotic patient, describe them and try to identify the dominant diversion paths.

Personal Information

References