Ultrasound of portal hypertension
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Portal Hypertension
Common and less common causes:
  Cirrhosis
  Cavernous transformation
  Budd-Chiari Syndrome

Portal hypertension
- Causes classified according to:
  - Primary increased resistance
    - Prehepatic
    - Intrahepatic
  - Prehepatic
    - Primary increased flow

Primary increased resistance
- Prehepatic
  - PV/SV thrombosis, extrinsic compression of PV
- Intrahepatic
  - Presinusoidal*
    - Schistosomiasis, sarcoidosis, myeloproliferative diseases, PBC/PSC, toxins
  - Sinusoidal/mixed
    - Alcoholic cirrhosis, cirrhosis 2° chronic hepatitis B/C, cryptogenic cirrhosis
  - Post sinusoidal
    - VOD, HV obstruction (Budd-Chiari)
  - Post hepatic
    - Constrictive pericarditis, IVC web, severe right heart failure, tricuspid regurgitation

Early in course → progress to sinusoidal*
Primary increased flow
- Arteriportal fistula
  - Congenital, post-traumatic, erosion of HA aneurysm into portal vein
- Splenomegaly
- Myelofibrosis

Pathogenesis of portal hypertension
(model: alcoholic cirrhosis)
- Architectural derangement
  - Fibrosis, regenerative nodules
  - Sinusoidal changes
  - Hepatocyte enlargement from alcohol-induced protein and fat deposition
  - Compression of sinusoids

Vasoactive factors
- Vasoconstrictors
  - Endothelin, Thromboxane 2
    - ↑ production in cirrhosis
- Vasodilators
  - Nitric oxide
    - ↓ production in cirrhosis
- Imbalance contributes to portal hypertension

Duplex/color Doppler - portal vein
- Hepatofugal flow (main, right, or left)
  - Slow, bidirectional flow (alternating hepatopetal and hepatofugal)
  - Static blood flow

Ralls, Radiology 1990; 155:517-525

Hepatofugal flow

Hepatofugal flow - RPV
Duplex/color Doppler - hepatic artery
- Enlarged, tortuous
- High peak systolic and diastolic velocities
- Hepatic arterial buffer response
- Advanced cirrhosis with decreased portal flow

Duplex/color Doppler - hepatic veins
- Monophasic
  - Loose normal phasicity
  - ↑ resistance to transmission of right atrial pressures 2° focal hepatic vein stenoses

Lorenz, JUM 1996; 15:313-316
Portosystemic collaterals
- Tributary collaterals
- Normally existing vessels
  - Coronary (L gastric), short gastrics, SMV, IMV

Normal coronary vein

Coronary veins

Coronary veins

Short gastric veins
Splenic vein thrombosis

Portosystemic collaterals
- Developed collaterals
- Normally closed channels
  - Spleno-retroperitoneal, paraumbilical, spleno-renal

Short gastric veins

Recanalized paraumb. vein

Splenorenal shunt

Splenoretroperitoneal shunt
**Cavernous transformation**

- Formation of portoportal collaterals in the setting of acquired, benign PV thrombosis
- Unusual in setting of cirrhosis
- Unusual in presence of neoplasm
- Causes pre-hepatic portal hypertension

- Develops 6-20 days after thrombosis
- Origin of collateral vessels
  - Paracholedochal (adjacent) and epicholedochal (intramural) veins
  - Pancreatico-duodenal veins originating at pancreatic head
  - Vasa vasorum of PV wall
  - Recanalized channels in PV

- Duplex/color Doppler - portal vein
  - Non-visualization
  - Multiple periportal collateral vessels
    - Intrahepatic extension around thrombosed PV branches
    - Portal vein-like waveform

- Two types of venous collaterals
  - Portosystemic collaterals
    - Left gastric, perisplenic veins
  - Portoportal collaterals
    - Intrahepatic
    - Pericholecystic
    - Shunt blood into a patent right portal vein branch

*DeGaetano, AJR 1995; 165:1151-1155*  
*Walser, Radiology 2011; 258:146-153*
Cavernous transformation

Budd Chiari syndrome
- Intrahepatic HV thrombosis
- Hypercoagulable states
  - Myeloproliferative disorder, antiphospholipid syndrome, factor V leiden mutation, PNH, protein S/C deficiencies, BCP
  - Causes post-sinusoidal portal hypertension

- IVC thrombosis (hepatic portion)
  - Idiopathic (most common)
  - Poor standard of living, inf.? 
  - Diaphragm motion → injury
  - Hypercoagulable state
- Secondary
  - Extrinsic compression
  - Abscess, hydatid cyst
  - Invasion by neoplasm
  - HCC, RCC, adrenal carcinoma

Okuda, Hepatology 1998; 28:1191-1198

Duplex/color Doppler - hepatic veins
- Fulminant/acute
  - Visible thrombus
  - Obstructive or nonobstructive
  - No collateral vessels
- Subacute
  - Collateral vessels
- Chronic
  - Nonvisualization of hepatic veins
  - Collateral vessels


Collateral vessels
- Intrahepatic
  - Occluded HV → patent HV
  - Occluded HV → right inferior HV
  - Occluded HV → collateral
    - Tortuous, comma-shaped
    - Flow reversal
    - Monophasic waveform
    - Caudate veins → IVC

- Subcapsular
  - L inferior phrenic vein → SVC
- Extrahepatic
  - IVC → azygos system
  - Left renal vein → hemiazygos
  - Left renal vein → L inferior phrenic vein → SVC
  - Superficial abdominal wall
    - Inferior epigastric vein → subclavian vein → SVC

Cho, AJR 1996; 167:1163-1167
US plays an important role in the evaluation of the patient with portal hypertension. Findings can have a major influence on patient management.