

## **Vesalius SCALpel™ : Portal hypertension** (see also: VIDs 165, 554, discussion 753)

25% of cirrhotics have varices

25-33% with varices will bleed at some point

25% mortality with variceal bleed

66% incidence of rebleeding without intervention

50% of pts with varices who bleed, bleed from other causes

normal portal pressure 8mm, higher 2/3 develop varices, 1/3 bleed > 12mm

varices bleed by rupture, not erosion

### **Location of blockage**

prehepatic: portal V, splenic V, tumor at porta

intrahepatic: cirrhosis (alcoholic, hepatitis, toxic injury)(polycystic liver and Caroli's may be complicated by portal hypertension)

posthepatic: Budd-Chiari

### **Encephalopathy**

cause unclear; nitrogenous compounds contribute

sepsis, constipation, dehydration, blood in gut

no diagnostic test (serum NH<sub>4</sub>, EEG non-diagnostic)

treat dehydration, decrease protein intake, cleanse gut (lactulose, induces diarrhea, does not decrease colonic flora), treat other causes

### **Ascites**

when sinusoidal pressure exceeds colloid oncotic pressure

induced by physiologic stress, IV fluids (decreased colloid oncotic press)

complications

spontaneous bacterial peritonitis

hepatorenal syndrome (extreme prerenal azotemia)

control:

medical therapy is treatment of choice

Na/H<sub>2</sub>O restriction, spironolactone (promotes Na diuresis), then give loop diuretic (lasix) if necessary

large volume paracentesis (several liters) is safe

replace albumin?

peritoneal-venous shunt

clog early with protein

sepsis within 6 weeks

increases chance of variceal bleeding (increases circulating volume)

contraindicated: bacterial peritonitis, uncontrolled coagulopathy, CHF

transjugular intrahepatic porta-systemic shunt (TIPS):

trade encephalopathy for ascites

doesn't always control, high occlusion rate and encephalopathy like shunt

last resort  
umbilical hernia with uncontrolled ascites 30% morbidity, 5% mortality, v 15 and 0 when ascites is controlled

## Bleeding

each bleeding admission carries a 25-50% mortality  
limit crystalloid, transfuse RBC, FFP, platelets (rarely, only if drops < 50K, mechanism: splenic engorgement and sequestration)  
maintain tissue perfusion, monitor urine output, don't overload (variceal pressure parallels CVP, increases bleeding)  
prior use of vasopressin: intense constriction all arterial beds, stopped 80% of bleeds risk peripheral, myocardial ischemia (NTG ameliorates latter)  
somatostatin/octreotide drug of choice, 85% success, less side effects, safer as effective as vasopressin  
50mcg bolus, then 50mcg/h X 24h after endoscopy  
EGD: 50% of UGI bleeds in cirrhotics are non-variceal  
varices source if active bleeding or stigmata and no other source  
start somatostatin as soon as possible after accurate Dx, not before scope  
with encephalopathy protect airway with intubation before scope  
Rx: banding treatment of choice v sclerosing, safer  
occasionally requires TIPS  
emergency shunt last resort  
support after bleeding stops: coag. correction, FFP, vit K, (platelets)  
hepatitis profile, angio if evaluation for transplant appropriate

## Child-Pugh (Gardner numeric score modification)

original classification to assess for portacaval shunt

	A	B	C
Bilirubin	<2	2-3	>3
albumin	>3.5	2.8-3.5	<2.8
ascites	none	controlled	uncontrolled
encephalopathy	none	minimal	advanced
nutrition	excellent	good	poor

point score: A=1, B=2, C=3 (15 max)

Class (total score): A 5-6, B 7-9, C 10-15

## Hepatic coma

decrease protein to 50g/d, control bleeding, lactulose cathartic (acidifies colon, decreases ammonia absorption)  
non-absorbable antibiotics: kanamycin, neomycin

## MELD (model for end-stage liver disease score)

developed for TIPS but general applicability for liver transplant, more precise than Child Cr, tot bili, INR, etiology of cirrhosis factors in score

## Definitive treatment

2/3 of bleeders will rebleed, most early, <6w

25% of bleeders will die on that admission

definitive treatment indicated with first bleed

beta block decreases bleeding by decreasing cardiac output

prophylaxis for varices that haven't bled

endoscopic Rx

banding treatment of choice, as effective as sclerosing with fewer complications,

lowest incidence of encephalopathy

sclerosing: multiple sessions, >60% rebleed, 1/3 fail, 30% complications (ulceration, stricture, perforation, fever, mediastinitis, CNS (embolization of sclerosant))

rarely done anymore

surgery

surgical treatment is rarely done anymore since the advent of endoscopic treatment, octreotide and TIPS

total shunt: 90% prevention of rebleed, 40% encephalopathy

selective: eg distal splenorenal, same mortality, effect, decreased encephalopathy

Childs class A, B candidates, not C, high mortality

low thrombosis (8%, technical error), promotes, does not decrease ascites

less rebleeding, encephalopathy than portacaval

distal splenorenal does not require splenectomy v central splenorenal

partial shunt: short, 8-10mm (larger, 16mm clot) straight supported PTFE

non-shunt: esophageal transection, variceal ligation, devascularization (done mostly outside US)

transplant: treatment for liver failure only, not bleeding

TIPS (transjugular intrahepatic portacaval shunt)

not indicated for prehepatic causes; polycystic liver or Caroli's (risk hemorrhage)

surgery superior to TIPS Child class A, B

99% successful technically, 9-50% complications, 3-13% mortality

30-40% encephalopathy (v 10-15% for distal splenorenal)

less rebleeding than Endoscopic banding

33-73% occlusion at 1y, 18% rebleed

US monitor every few months, dilate or restent

role: refractory bleeding (after emergency shunt)

bridge to transplant

Child class C

refractory ascites (trade for encephalopathy)

## **Splenic Vein thrombosis**

due to pancreatitis, cancer  
most do not result in varices  
if coronary vein joins splenic proximal to the thrombosis, may result in esophageal varices  
isolated gastric varices, < 10% bleed  
anticoagulation increases risk of bleed  
sclerosis and banding not effective for gastric varices  
routine EGD not indicated  
Rx with splenectomy if bleeding

## **Portal Vein thrombosis**

congenital cavernous transformation, neonatal omphalitis  
normal liver function with esophageal varices  
mostly extrahepatic portal vein thrombosis in children, liver spared  
excellent results with shunt  
Endoscopic Rx, distal splenorenal shunt (with normal liver function prevents encephalopathy)

## **Budd-Chiari (hepatic vein thrombosis)**

etiology:

coagulopathy: polycythemia, myeloproliferative, estrogen, disease, paroxysmal nocturnal hemoglobinuria, factor V Leiden, essential thrombocythosis, antiphospholipid syndr, protein C, S deficiency, antithrombin III deficiency  
IVC occlusion: right atrial myxoma, pericarditis, IVC membrane (most common cause in Asia: thrombolysis, angioplasty)  
liver mass  
hi dose chemotherapy  
(no association with right heart failure or cirrhosis)

presentation

abdominal pain, ascites, new onset hepatomegaly (liver congested, capsule stretches)

Dx: duplex US, CT, angio.

Rx: depends of status of liver, Bx

no central necrosis treat symptomatically  
lifelong anticoagulation for hypercoagulable state  
necrosis: do shunt (portacaval, mesoatrial) or transplant (in fulminant hepatic failure)  
75% long term transplant survival  
shunt effective in preserving liver function, controlling ascites, 85% symptomatic relief

50% of patients with portal hypertension and GI stoma develop parastomal varices: local measures, resite stoma