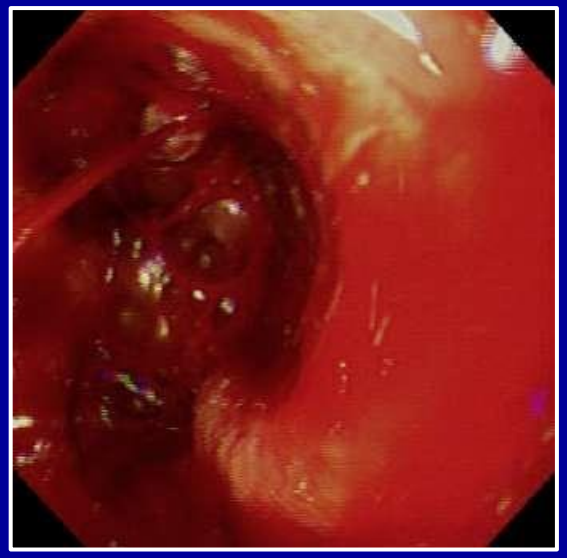
Approach to Hematemesis and gastrointestinal bleeding



Clinical Presentation of GI bleeding

- **Hematemesis**
  - Vomiting of fresh or old blood
  - Proximal to Treitz ligament
  - Bright red blood = significant bleeding
  - Coffee ground emesis = no active bleeding
- **Melena**
  - Passage of black & foul-smelling stools
  - Usually upper source – may be right colon
- **Hematochezia**
  - Passage of bright red blood from rectum
  - If brisk & significant → UGI source
- **Occult bleeeding**
  - Bleeding not apparent to patient
  - May lead to dyspnea, AP & even MI

Assessing the severity of bleeding

First step

Bleeding severity	Vital Signs	Blood loss (%)
Minor	Normal	< 10 %
Moderate	Postural (Orthostatic hypotension)	10 – 20 %
Massive	Shock (Resting hypotension)	20 – 25 %

Resuscitation

Proportional to bleeding severity

- 2 large-bore IV catheters: Normal saline – Ringer lactate
- Oxygen by nasal cannula or facemask
- Monitoring of vital signs & urine output
- **Blood Transfusion**: Ht raised to Elderly: **30 %**
  Young: **20 – 25 %**
  PHT: **27 – 28 %**
- **Fresh frozen plasma & platelet transfusion**
  If transfusion of **> 10 units** of packed red blood cells

History

• **Elderly**	Diverticula - Angiodysplasia - Cancer
• **Young**	Peptic ulcer – Varices – Esophagitis
• **< 30 years**	Meckel diverticula
• **Previous bleeding**	Bleeding from similar causes
• **Aortic surgery**	Aortoenteric fistula
• **Known liver disease**	Esophageal or gastric varices
• **NSAIDs**	
• **Retching**	Mallory-Weiss tear
• **Non GI sources**	Especially from nasopharynx

Physical examination

• **PHT**	Spider naevi – caput medusa …
• **Acanthosis nigricans**	Underlying cancer
• **Pigemnted lip lesions**	Peutz-Jeghers syndrome
• **Cutaneous lesions**	Neurofibromatosis
• **Purpura**	Henoch-Schonlein purpura
• **Splenomegaly**	PHT - portal vein thrombosis
• **Telangiectasia**	Osler-Weber-Rendu disease

Spider Naevi

Central arteriole

Blanch if occluded with pinhead

SVC Chest above nipple

Face

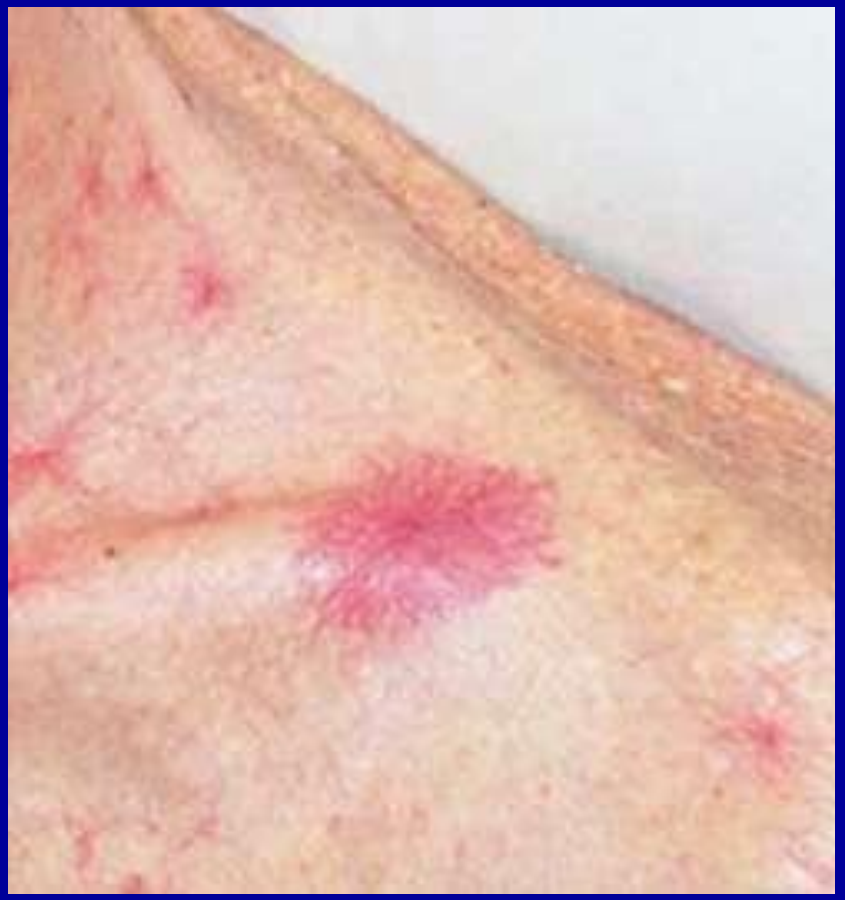
Arms

Hands

DD Childhood

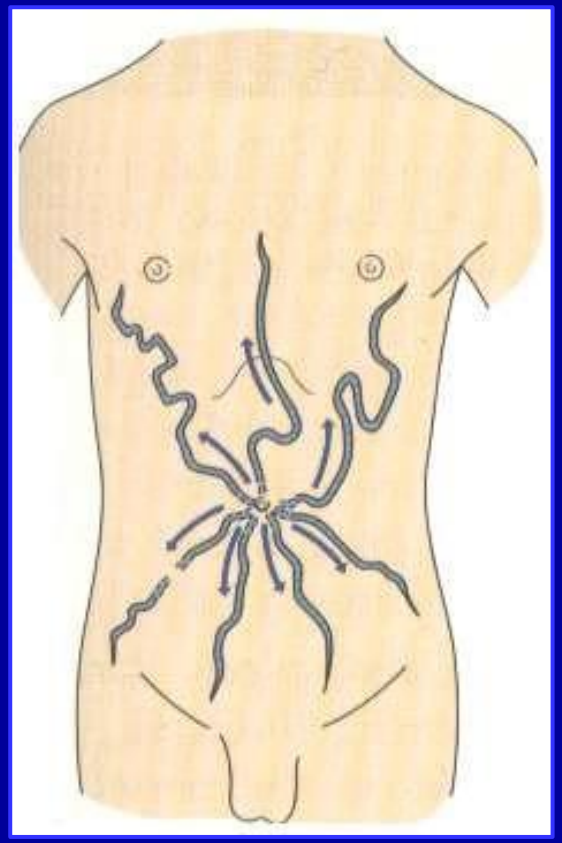
Pregnancy

Chronic liver disease

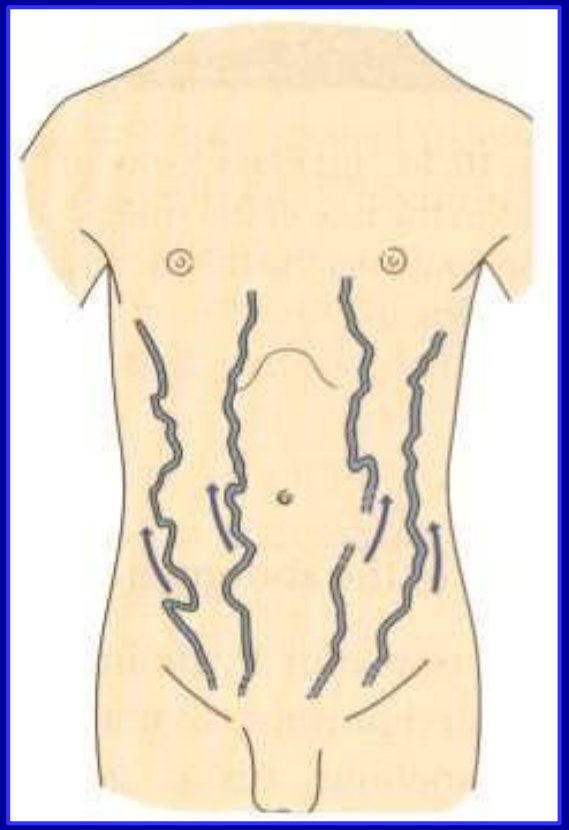


Direction of blood flow in anterior abdominal wall

PV obstruction

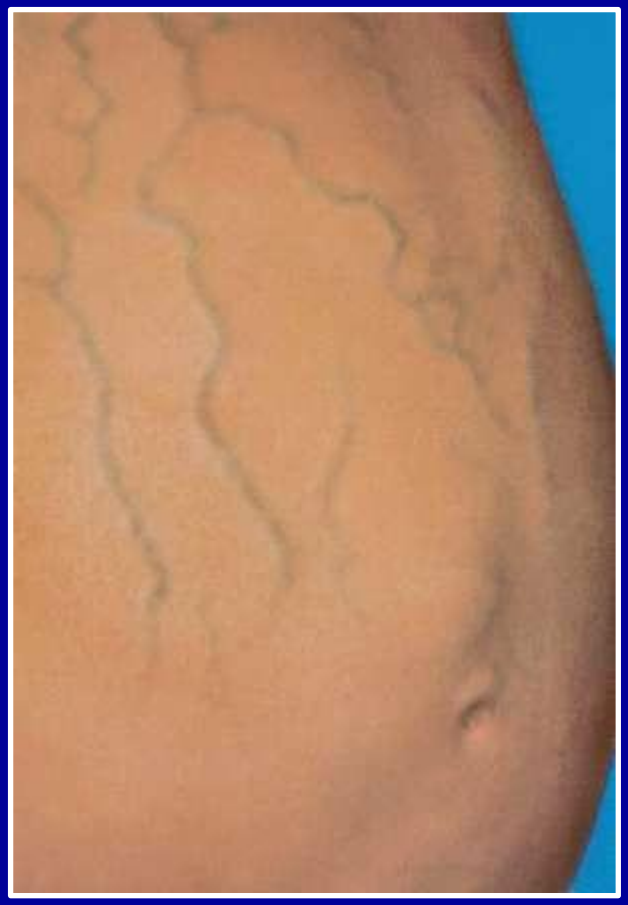


IVC obstruction



S Sherlock & J Dooley. Diseases of the Liver & Biliary System – 2002.

Collateral circulation

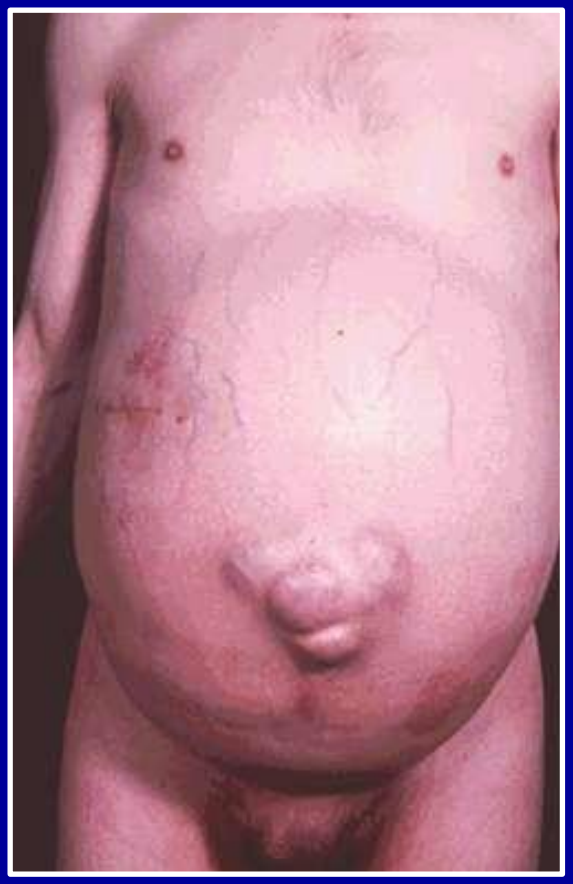


Vein dilatation & tortuosity in abdominal wall

of a cirrhotic patient suffering from ascites & jaundice

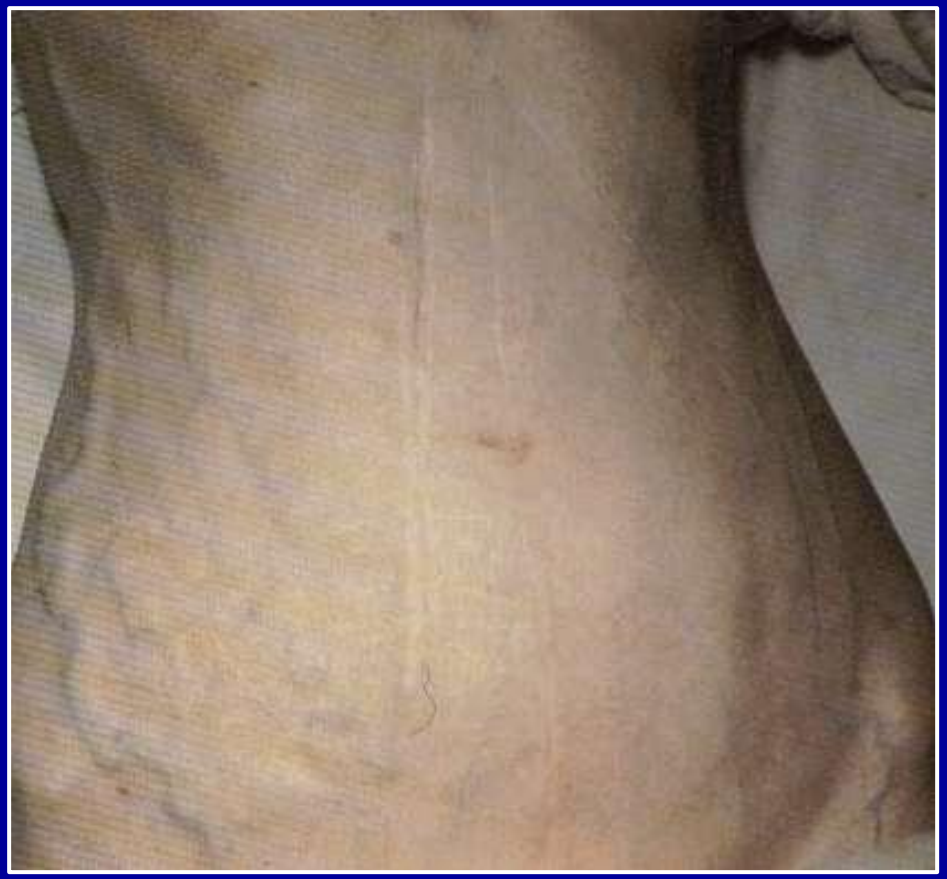
Caput Medusa

Portal hypertension



Seen much less frequently

Occlusion of the IVC



Gynecomastia in cirrhosis

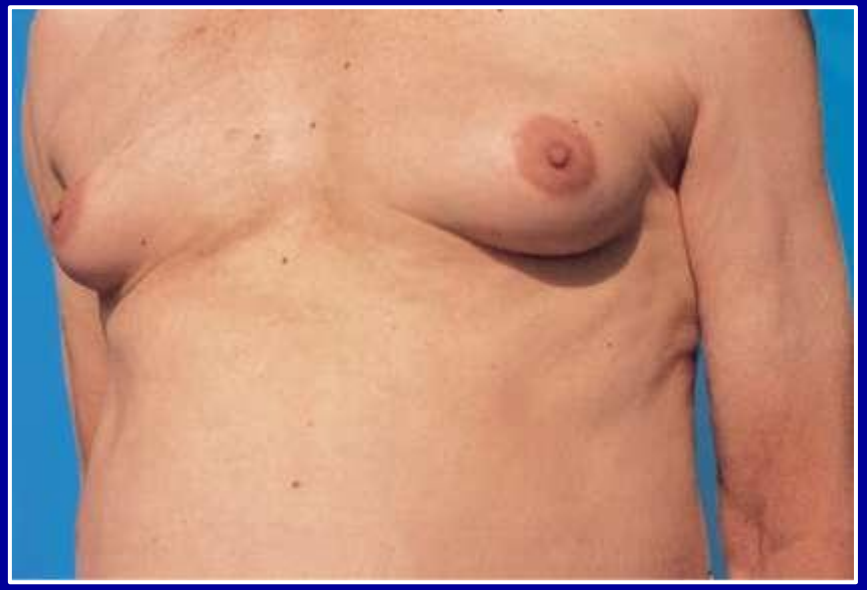
Seen in cirrhotic males

Spironolactone is frequent cause

Absent hair body

Associated diminished libido

Associated testicular atrophy



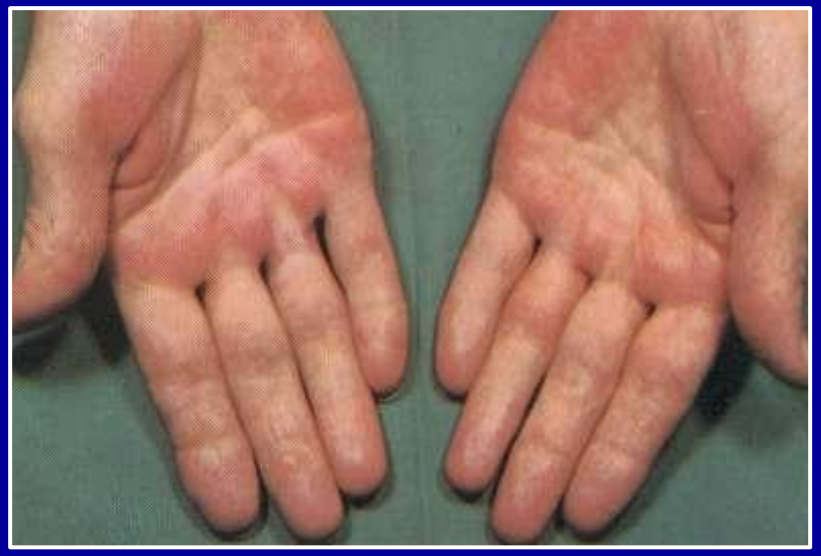
Palmar erythema

Exagereted red flushing of palms

Fades on pressure

Specially Thenar eminence
Hypothenar eminence
Bases of fingers

DD Pregnancy
Thyrotoxicosis
Bronchial carcinoma
Genetically determined



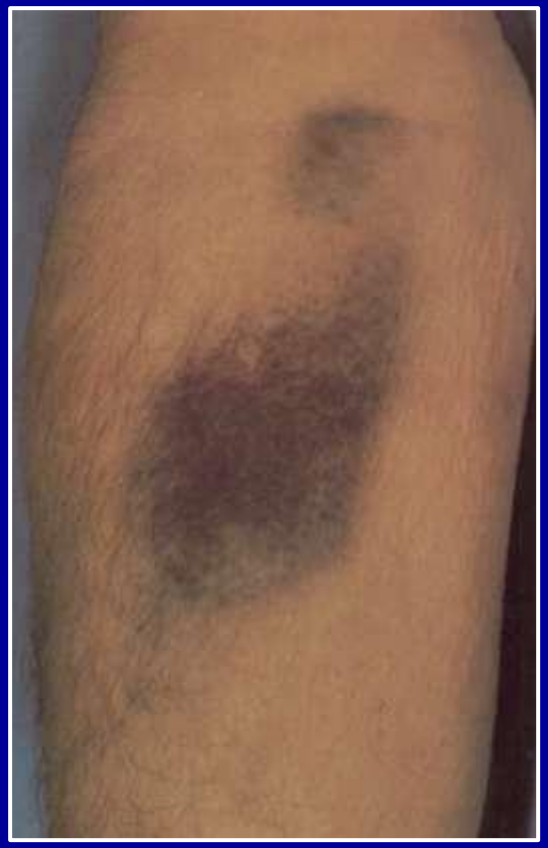
White nails

- Congenital
- Cirrhosis:
  Present in most patients
  Due to hypoalbuminemia

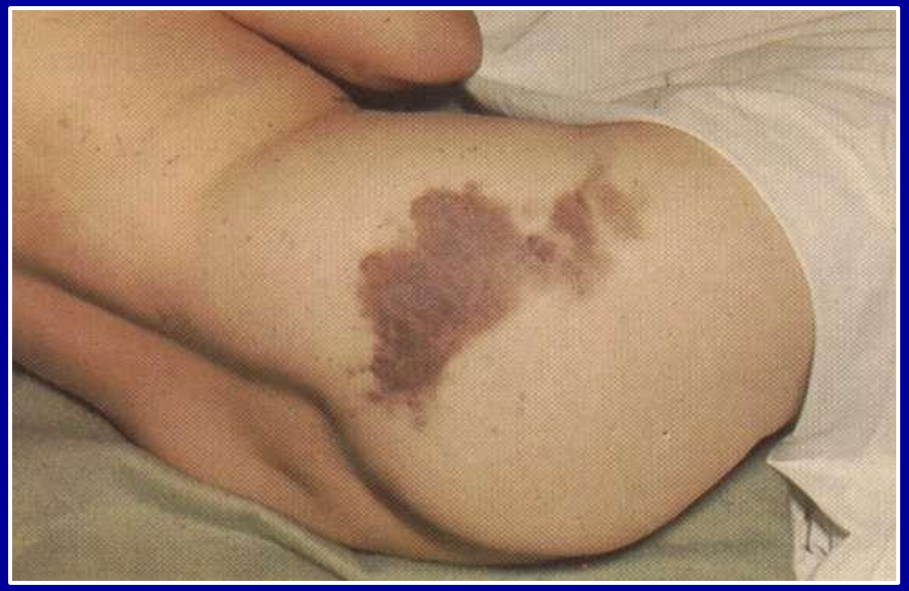
Bruising

Clotting disorder

Around venepuncture site



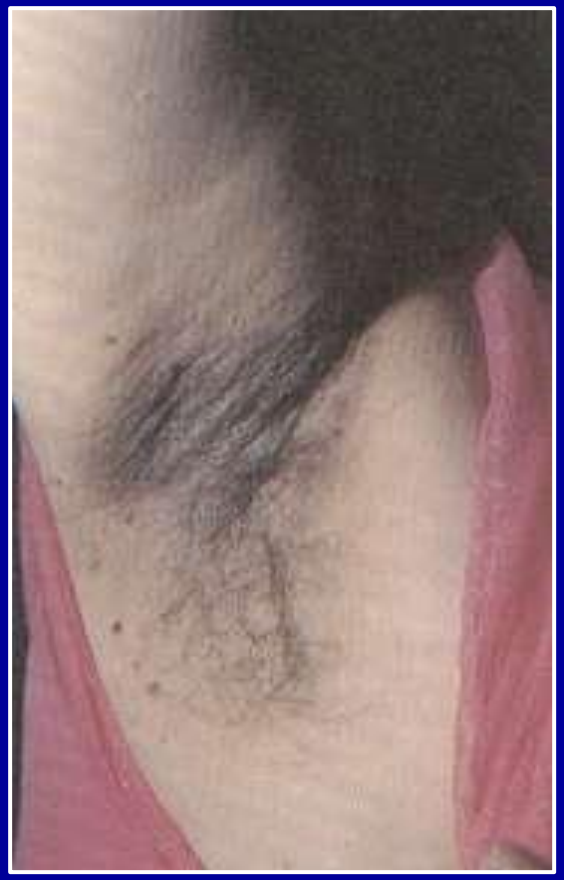
From intramuscular injection



Acanthosis nigricans

Pigmentation of
- Axilla
- Groins
- Angles of mouth
- Hands

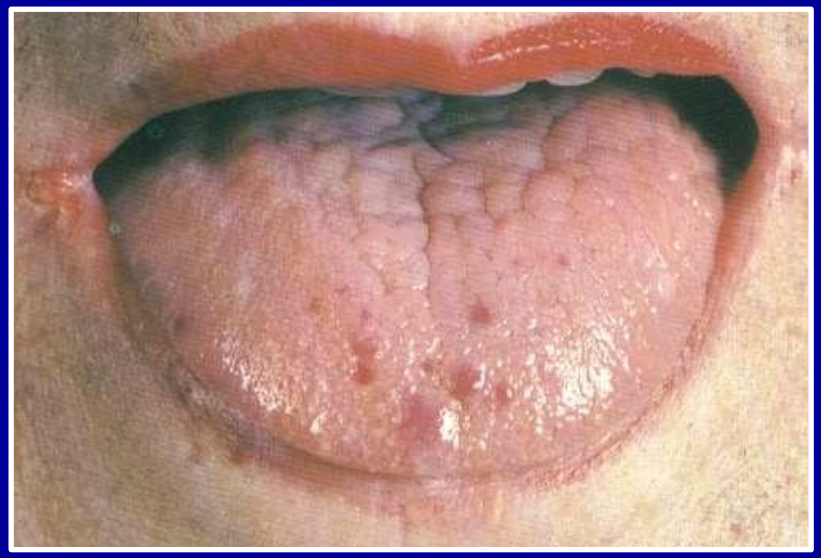
Malignant disease
- Gastric carcinoma
- Pancreatic carcinoma
- Bronchial carcinoma



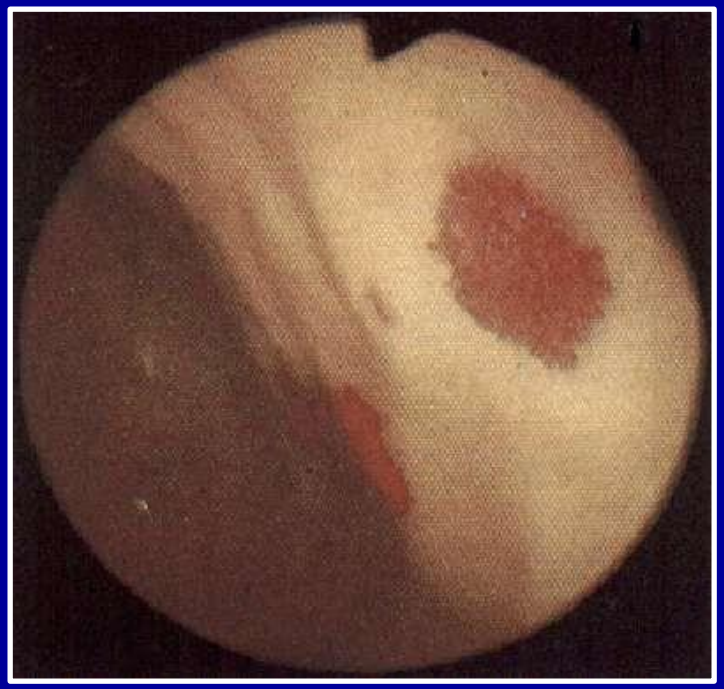
Hereditary telangiectasia

Rendu-Osler-Weber disease

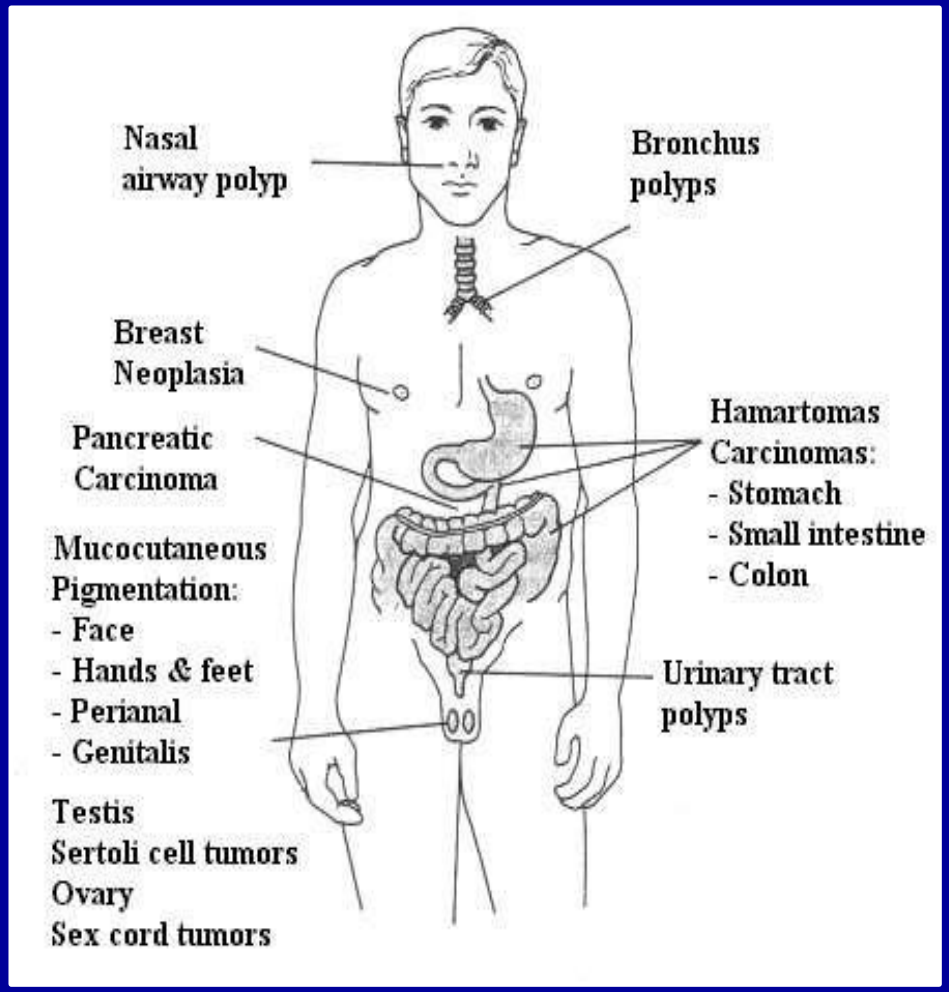
Tongue

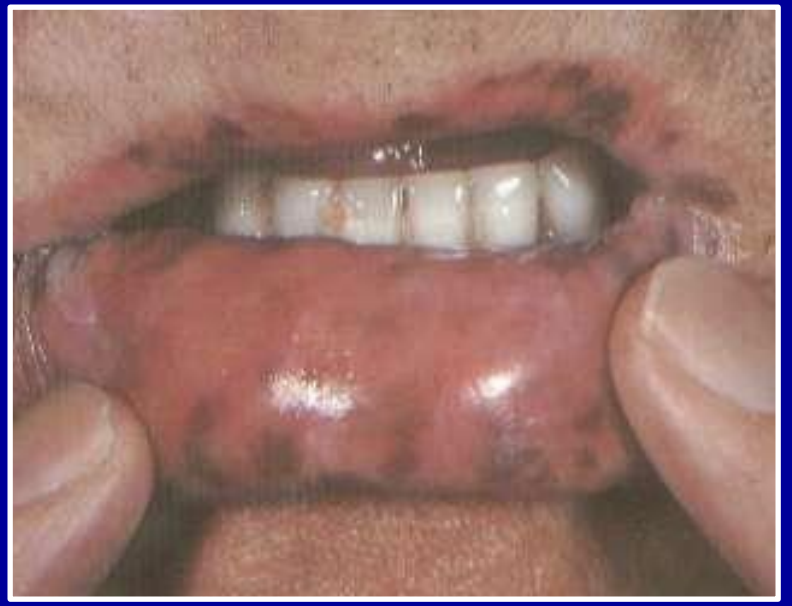


Stomach



Peutz-Jeghers Syndrome



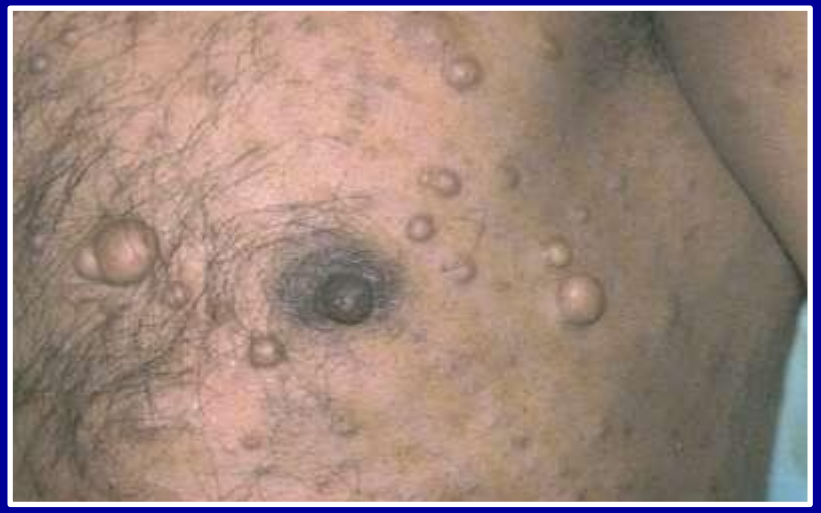


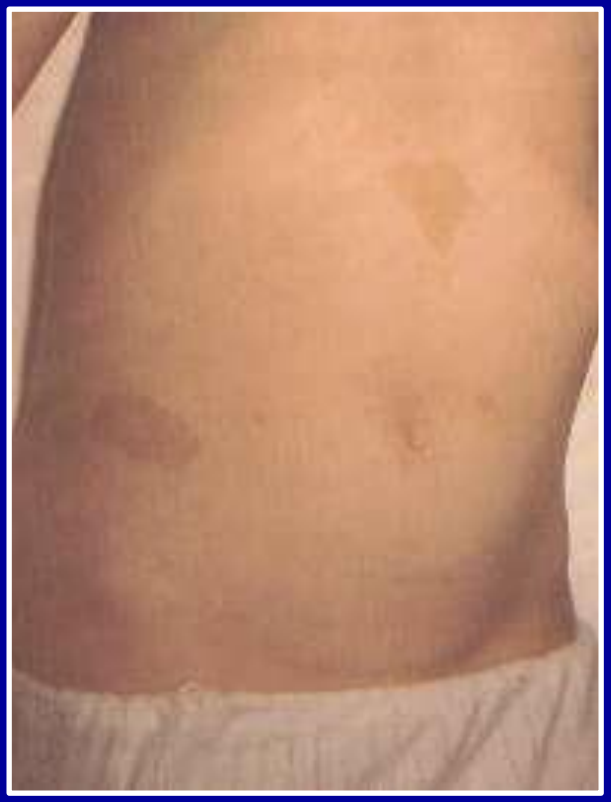
Neurofirmatosis

"von Recklinghausen's Disease"

"Café au lait" spots

Neurofibromas



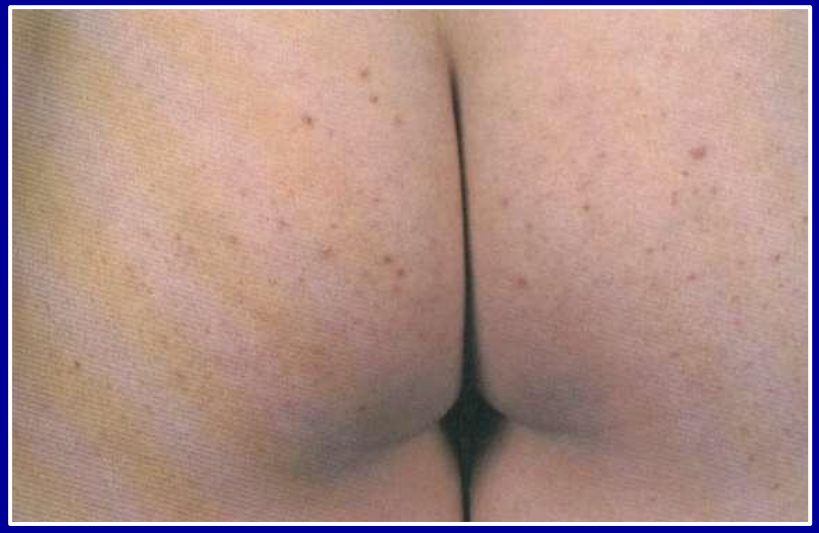


Henoch-Scholein purpura

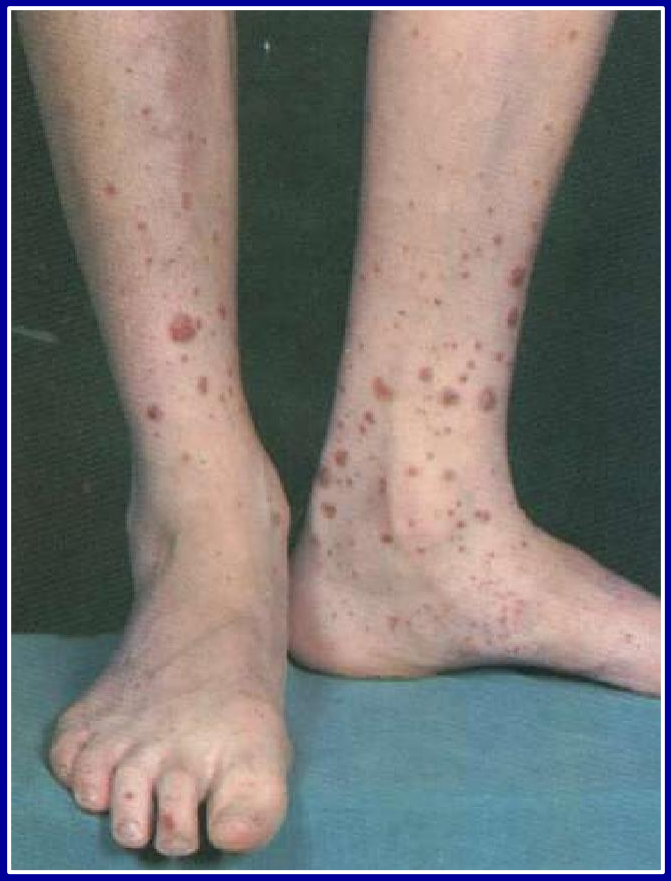
Age	Prepubertal boys (6 m – 6 years) Can occurs in adults
Tetrad	Purpuric rash: feet – buttocks – legs Colicky abdominal pain - bloody diarrhea Arthralgia Glomerulonephritis
Prognosis	Self-limited
Complications	Rapidly progressive renal failure GI hemorrhage

Henoch-Scholein Purpura

Buttocks



Extenseor surfaces of legs



Thyphoid fever

Rose spots

Frequency: 10 – 90 %

During second week

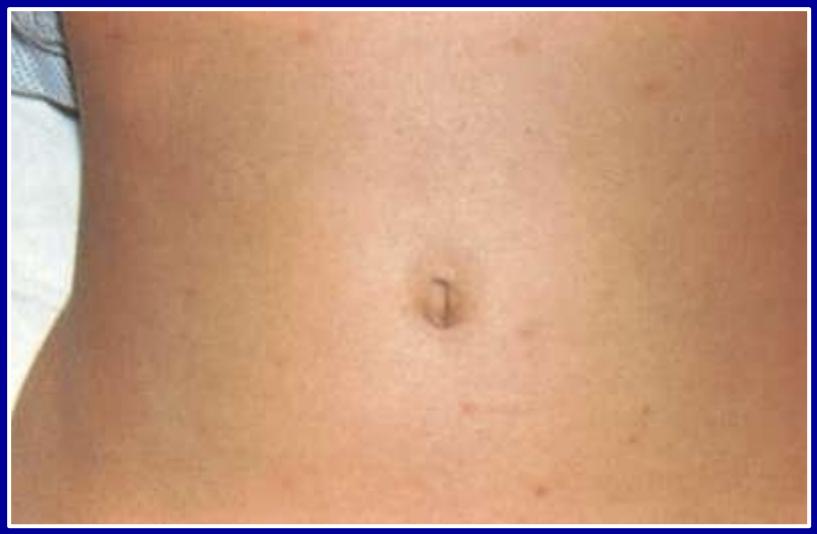
Erythematous macules (2 – 4 mm)

Upper abdomen & anterior thorax

Occur in small numbers

Blanch on pressure

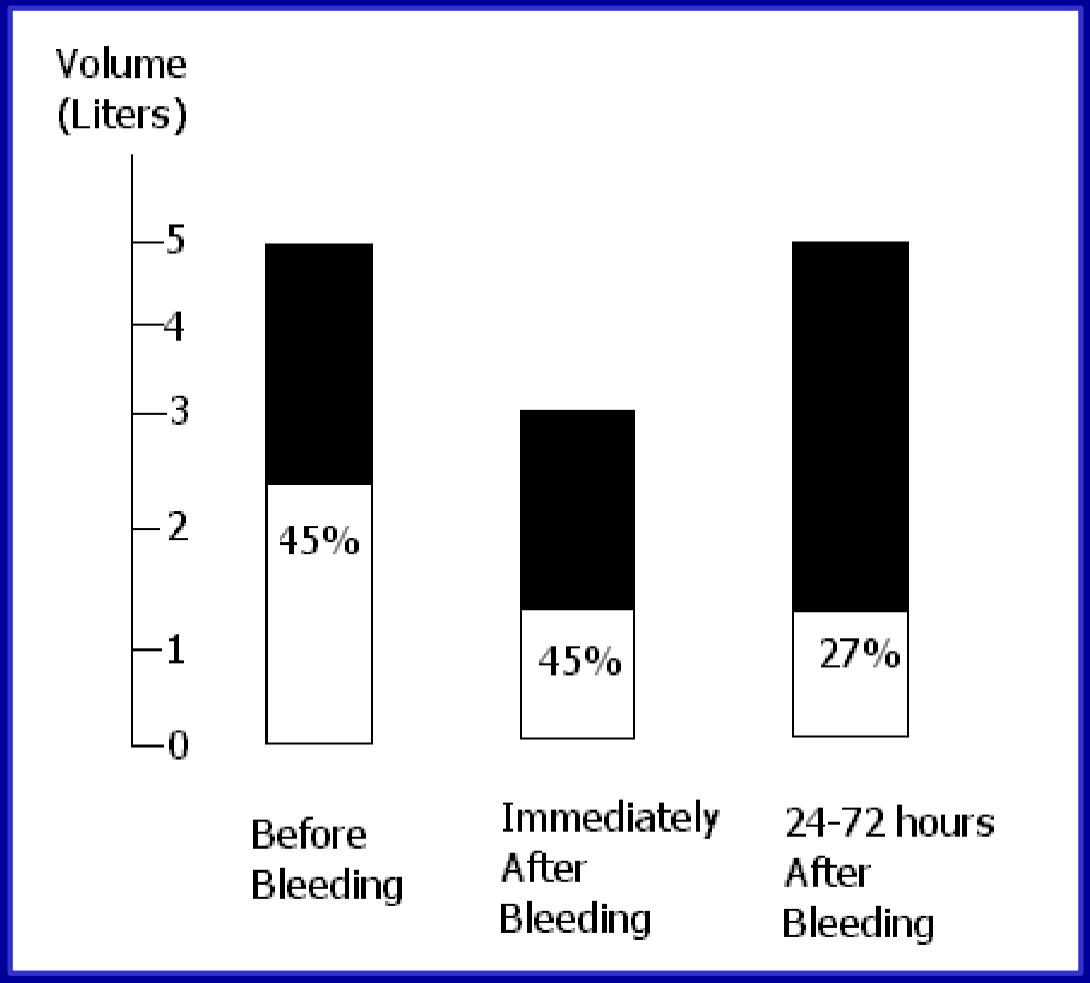
Lasts 2 – 3 days



Laboratory evaluation

- **Hematocrit** May not reflect blood loss accurately
- **Elevated BUN** Not correlated to creatinine level
  Breakdown of blood proteins to urea
  Mild reduction of GFR
- **Iron deficiency anemia**
- **Low MCV**
- **Low ferritin level**

Hematocrit values before & after bleeding



Diagnostic test in GI bleeding

- **Upper GI endoscopy**
- **Colonoscopy**
- **Small bowel endoscopy**
- **Capsule endoscopy & double balloon enteroscopy**
- **Barium radiograph**
- **Radionuclide imaging**
- **Angiography**
- **Miscellaneous tests: abdominal US or CT**

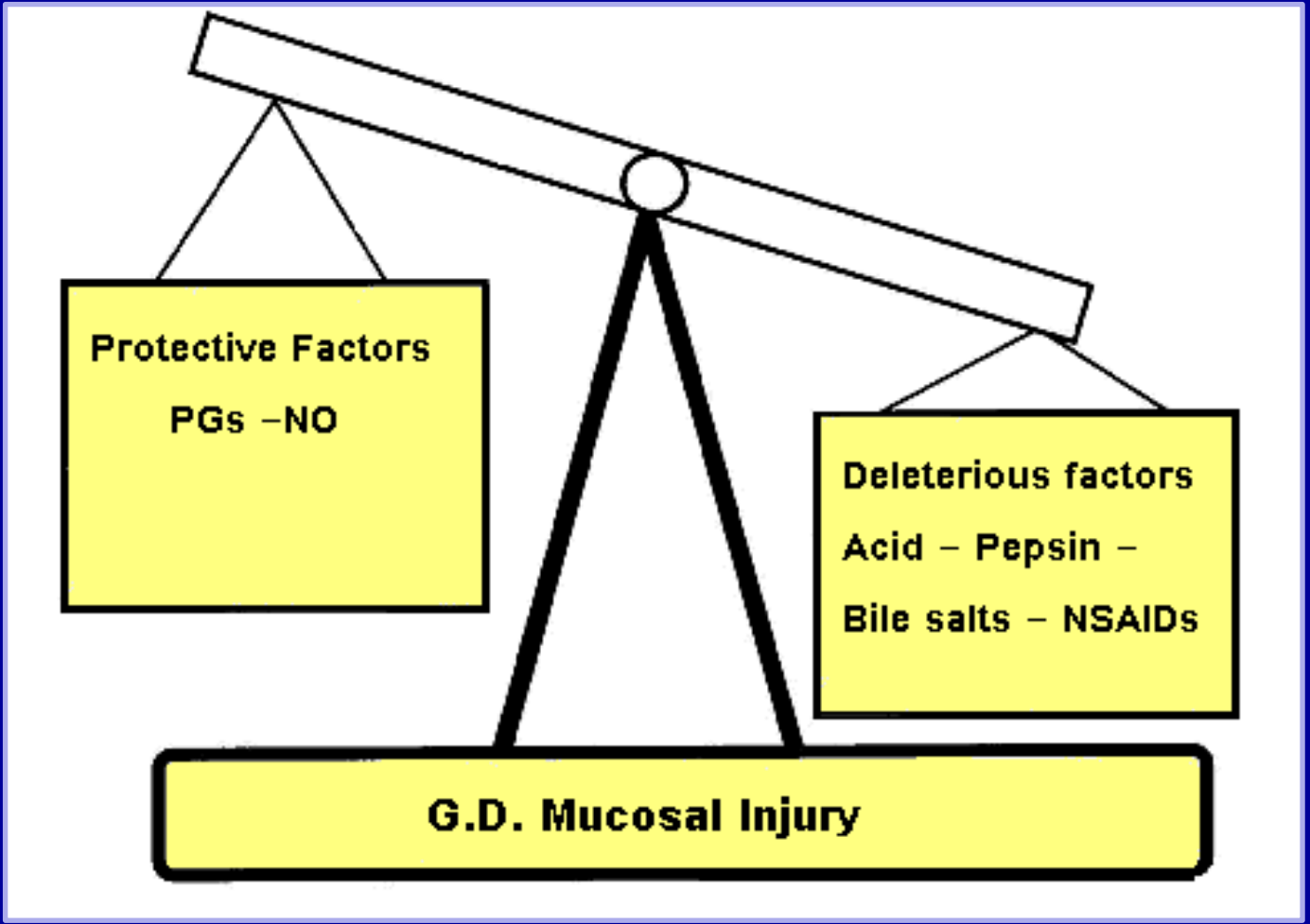
Causes of UGI bleeding

Common	Less Frequent	Rare
Peptic ulcer	Dieulafoy's lesion	Esophageal ulcer
Varices	Vascular ectasia	Erosive duodenitis
Mallory-Weiss	Watermelon stomach	Hemobilia
	Gastric varices	Crohn's disease
	Neoplasia	Aorto-enteric fistula
	Esophagitis	

Causes & associations of PU

Common forms of PU (95%)	Uncommon forms of P U (5%)
HP-associated	Acid hypersecretion :ZES – mastocytosis
NSAID-associated	Other infections: HSV type 1 – CMV
Stress ulcer	Duod obstruction: bands-annular pancreas
	Radiation-induced lesions
	Chemotherapy-induced lesions
	Idiopathic

Sleisenger & Fordtran's Gastrointestinal & Liver Disease -1998


Protective Factors
PGs –NO
Deleterious factors
Acid – Pepsin –
Bile salts – NSAIDs
G.D. Mucosal Injury

Predisposing factors to bleeding PU

- **Acid** Most prominent factor
- Helicobacter pylori
- NSAIDs
- Biphosphnate alendronate
- Chronic pulmonary disease
- Cirrhosis
- Anticoagulants
- Ethanol

Bleeding peptic ulcer

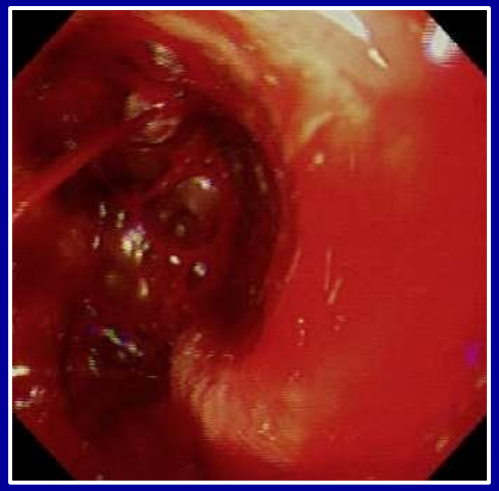
- Most frequent cause of UGI bleeding (**50%**)
- Especially high on gastric lesser curvature or postero-inferior wall of duodenal bulb
- Most ulcer bleeding is self-limited (**80%**)

Forrest's classification for PU bleeding

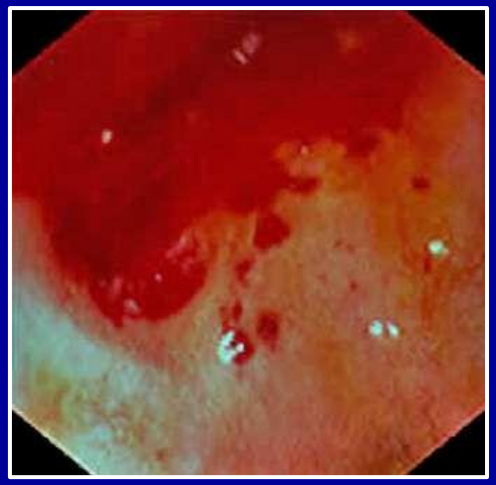
Stage	Characteristics	Rebleeding
I a	Jet arterial bleeding	90 %
Ib	Oozing	50 %
IIa	Visible Vessel	25 - 30 %
IIb	Adherent clot	10 - 20%
IIc	Black spot in ulcer crater	7 - 10%
III	Clean base ulcer	3 - 5 %

Forrest's classification for PU bleeding

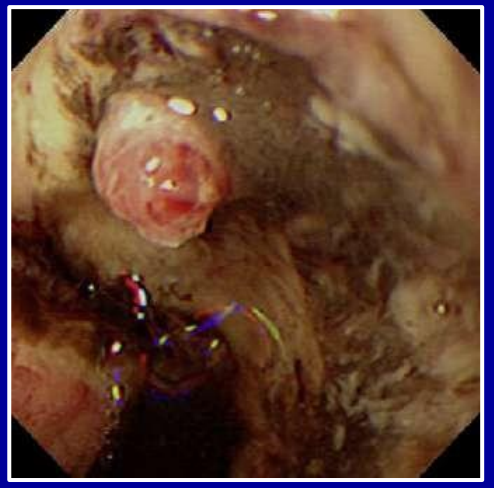
I-a (arterial jet)



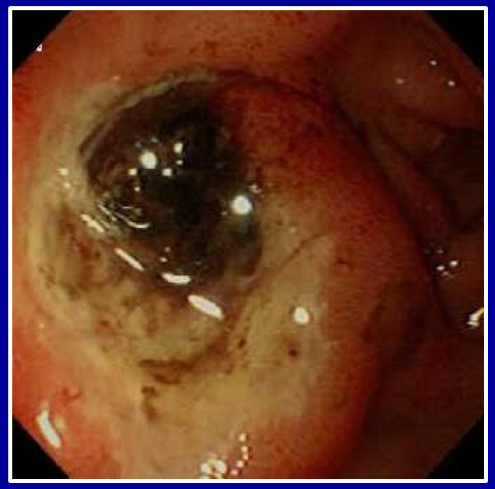
I-b (oozing)



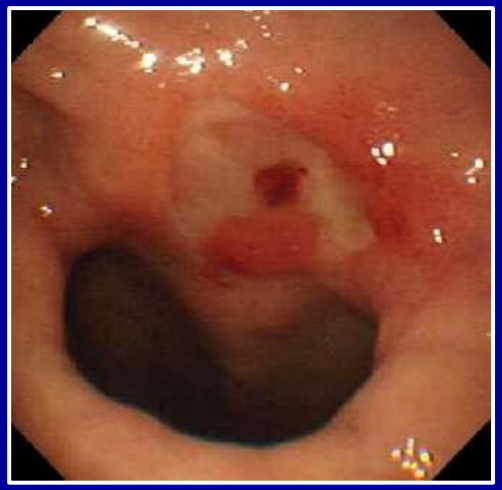
II-a (visible vessel)



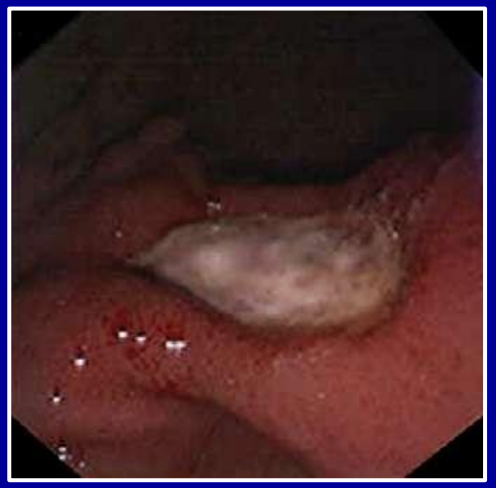
II-b (adherent clot)



II-c (black spot)



III (clean base)



GI side effects of NSAIDs

Organ	Side Effects
Esophagus	Esophagitis – Ulcer – Stricture
Stomach & duodenum	Subepithelial hemorrhage – Erosion – Ulcer
Small Intestine	Ulcers – Strictures – NSAID enteropathy
Colon	No pre-existing colonic disease: Ulcerations – Stricture – Diaphragm – Colitis Pre-existing colonic disease: ↑ Complications of diverticular disease Activate IBD
Ano-rectum	Inflammation – Ulcer – Stricture

GI safety of non-selective NSAIDs

RR of different NSAIDs could differ 10-fold

Lowest risk	Ibuprofen * Diclofenac	
Moderate risk	Indomethacin Naproxen Sulindac Aspirin	
Highest risk	Azapropazone Tolmetin Ketoprofen Piroxicam	} Longer half-time

* Risk at higher doses (> 1.5 –2.4 g/d) comparable to others *NSAIDs*

Br Med J 1996 ; 312 : 1563 – 1566.

Prevention of GI toxicity

due to NSAIDs

Patients at increased risk for NSAIDs CV toxicity

High risk	Patients with risk factors for CV disease often receive prophylactic aspirin Arbitrarily defined as requirement for low-dose aspirin for prevention of serious CV events
Low risk	No risk factors

Patients at increased risk for NSAIDs GI toxicity

High risk	1. History of complicated ulcer especially recent 2. Multiple (> 2 risk factors)
Moderate risk (1 – 2 risk factors)	1. Age > 65 years 2. High dose NSAID therapy 3. Previous history of uncomplicated ulcer 4. Concurrent use of aspirin 5. Concurrent use of corticosteroids 6. Concurrent use of anticoagulants
Low risk	No risk factors

HP is independent & additive risk factor & addressed separately

ACG guidelines for prevention of NSAID-related ulcer complications .
Am J Gastroenterol 2009 ; 104: 728 – 738.

Prevention of NSAID-related ulcer complications

	Low GI risk	*Moderate GI risk*	*High GI risk*
Low CV risk	NSAID alone (least ulcerogenic at lowest dose)	NSAID + PPI/misoprostol	Alternative therapy or Coxibs + PPI/misoprostol
High CV risk	Naproxen + PPI/misoprostol	Naproxen + PPI/misoprostol	Avoid NSAIDs & coxibs Use alternative therapy

Naproxen may have some cardioprotective properties

Patients with ulcer history: search for HP & if present eradicated

ACG guidelines for prevention of NSAID-related ulcer complications.
Am J Gastroenterol 2009 ; 104: 728 – 738.

Treatment of bleeding PU

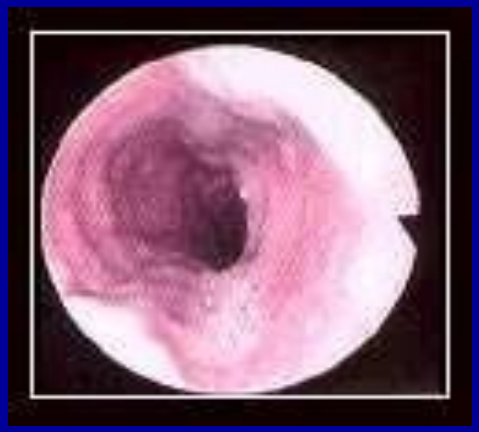
- **Pharmacological**
  - PPI 80 mg IV bolus
  - 8mg / hr / 72 hours IV infusion
- **Endoscopic**
  - Injection (epinephrine 1/10.000)
  - Monopolar coagulation
  - Bipolar coagulation
  - Heater probe
  - Hemoclips
  - Argon plasma coagulation
- **Surgical**
  - When endoscopic treatment fails

Summary of therapy of bleeding PU

- Patients must be adequately **resuscitated**
- UGI endoscopy is the primary diagnostic modality
- **Intubation** if severe bleeding or altered mental status
- Endoscopic therapy indicated in high risk lesions
  Combine 2 methods of endoscopic treatment
- IV PPI should be used in high risk patients

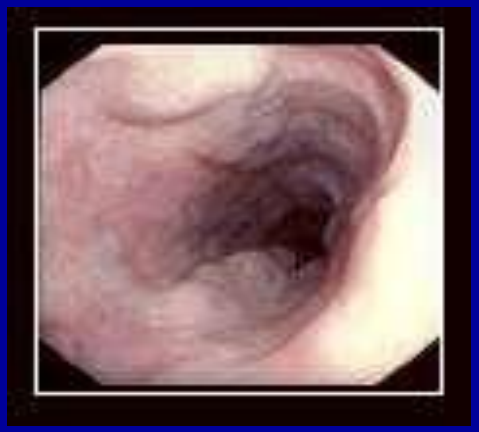
Classification of esophageal varices

Grade 1
Small



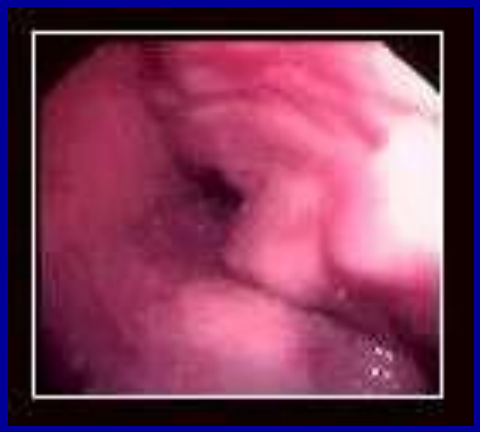
Minimally elevated veins above surface

Grade 2
Medium



Tortuous veins occupying < 1/3 of esophageal lumen

Grade 3
Large



Occupying > 1/3 of esophageal lumen

AASLD practice guidelines: prevention & management of gastroesophageal varices. Hepatology 2007 ; 46 : 922 – 938.

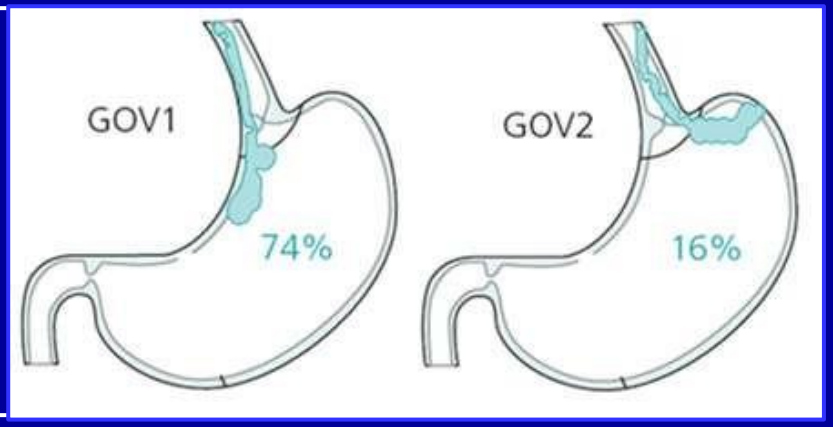
New classification of esophageal varices

- **Small Varices:** **< 5 mm**
- **Large Varices:** **> 5 mm**

Classification of gastric varices

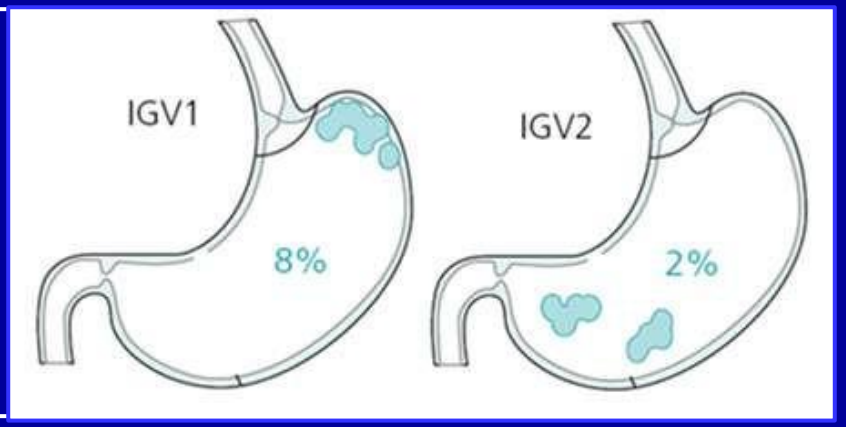
Gastro-Oesophageal Varices

Type I	Along lesser curve
Type II	To gastric fundus



Isolated Gastric Varices

Type I	Fundal
Type II	Ectopic



Yamada T et all. Yamada's textbook of gastroenterology. Blackwell Publishing, West Sussex, UK, 5th edition, 2009.

Predictive factors for risk of bleeding

North Italian Endoscopic Club Index

• **Variceal size**	Best predictor of bleeding
• **Severity of liver disease**	Expressed by Child-Pugh
• **Red signs**	On the varices

NIEC. N Engl J Med 1988 ; 319 : 983 – 989.

Child-Pugh score

Category	1	2	3
Bilirubin (mg/dl)	< 2	2 - 3	> 3
Albumin (g/l)	> 35	28 – 35	< 28
Ascites	Absent	Mild- Moderate	Severe
Encephalopathy	0	I – II	III – IV
INR	< 1.7 (70%)	1.7 – 2.3 (40 – 70%)	> 2.3 (< 40%)

Class A: 5 – 6 **Class B: 7 – 9** **Class C: 10 – 15**

MELD Score

$$0.957 \times \text{Log}_e(\text{creatinine mg/dL}) + 0.378 \times \text{Log}_e(\text{bilirubin mg/dL}) + 1.120 \times \text{Log}_e(\text{INR}) + 0.643$$

Multiply score by 10 & round to nearest whole number

Laboratory values < 1.0 are set to 1.0

Maximum creatinine within MELD score: 4.0 mg/dl

Dialysis twice/week prior to creatinine test: creatinine 4.0 mg/dl

* 0.643 for etiology to make score comparable to previous published data

Interpretation of MELD score

Score	3 month mortality
≥ 40	100%
30 – 39	83%
20 – 29	76%
10 – 19	27%
< 10	4%

The maximum score given for MELD is 40

All values > 40 are given a score of 40

www.unos.org/resources/MeldPeldCalculator

Treatment of acute variceal bleeding

Recommendations - 1

- Best approach is combined use of:
  - **Pharmacological agent** started from admission &
  - **Endoscopic procedure**
- **Terlipressin & somatostatin** preferable if available
  Octreotide, vasopressin + nitroglycerin may be used
- Drug therapy maintained for at least **48 h**
  5 day therapy recommended to prevent early rebleeding

Treatment of acute variceal bleeding

Recommendations - 2

- **Bleeding EV**

  Band ligation is the endoscopic treatment of choice

  Sclerotherapy may be used

- **Bleeding GV**

  Obturation with **cyanoacrylate**

- **TIPS**

  Rescue procedure if medical & endoscopic tt fails

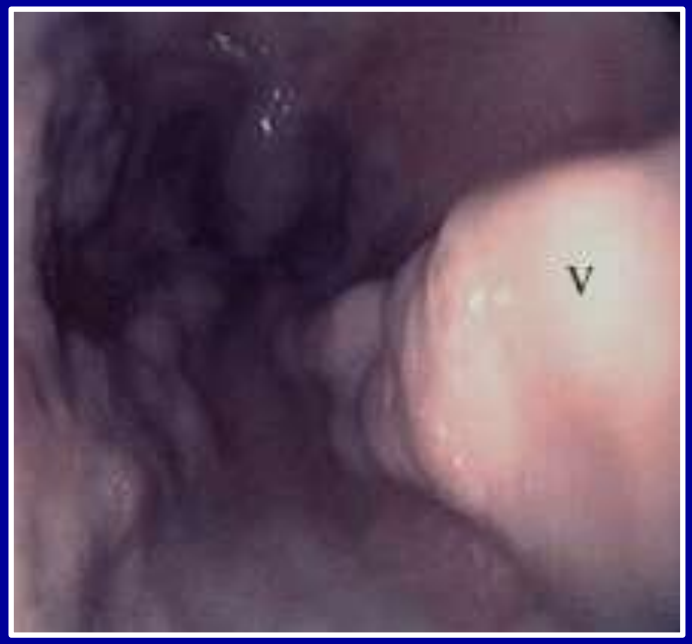
  Bleeding from GV may require earlier decision for TIPS

Treatment of acute variceal bleeding

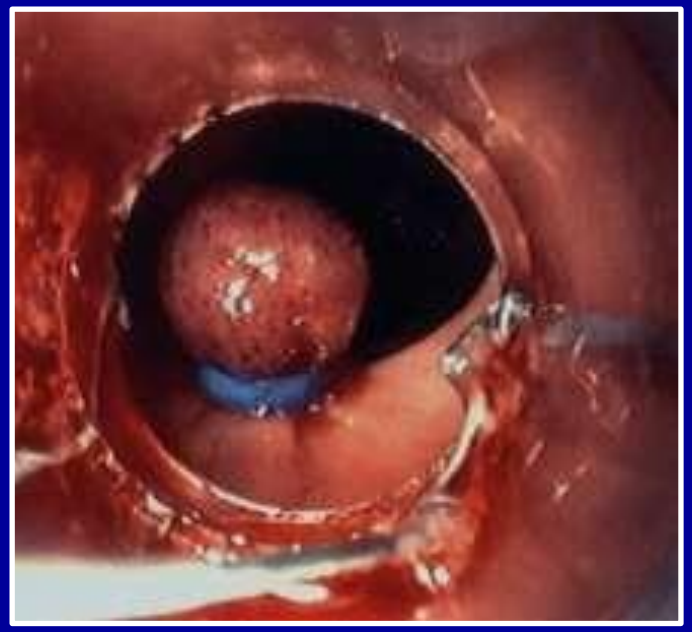
Recommendations - 3

- **Shunt surgery**
  Mesocaval graft shunts or traditional portacaval shunts may be an alternative to TIPS in Child A patients
- **Blood transfusion**
  Done cautiously using packed red cells (Ht: 25 – 28 %)
  Plasma expanders to maintain hemodynamic stability
- **Prophylaxis of infection**
  Given to all patients (norfloxacin 400 mg /12 hours)

Esophageal varices

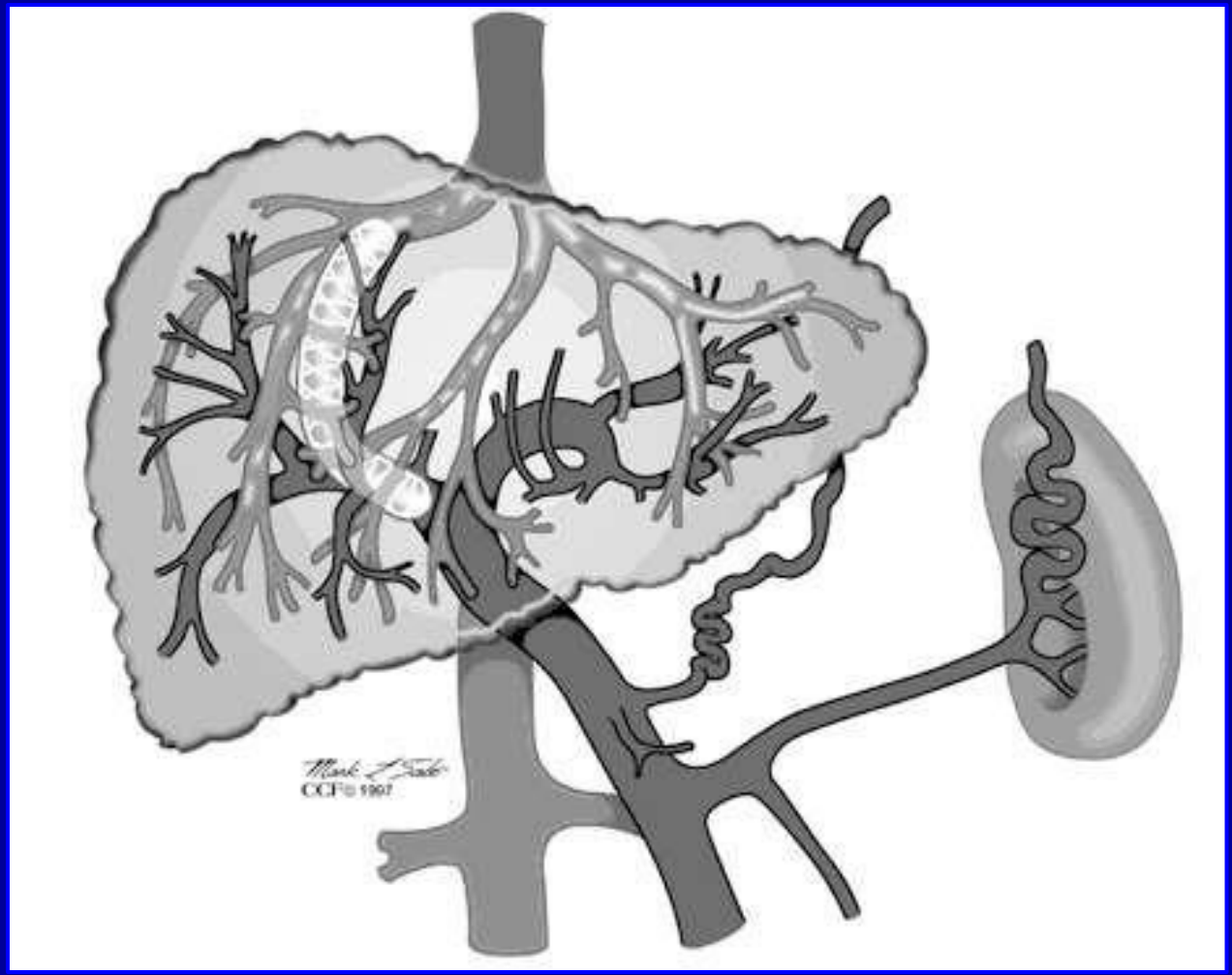


Endoscopic view of esophageal varices



Varix endoscopically ligated with a band

TIPS



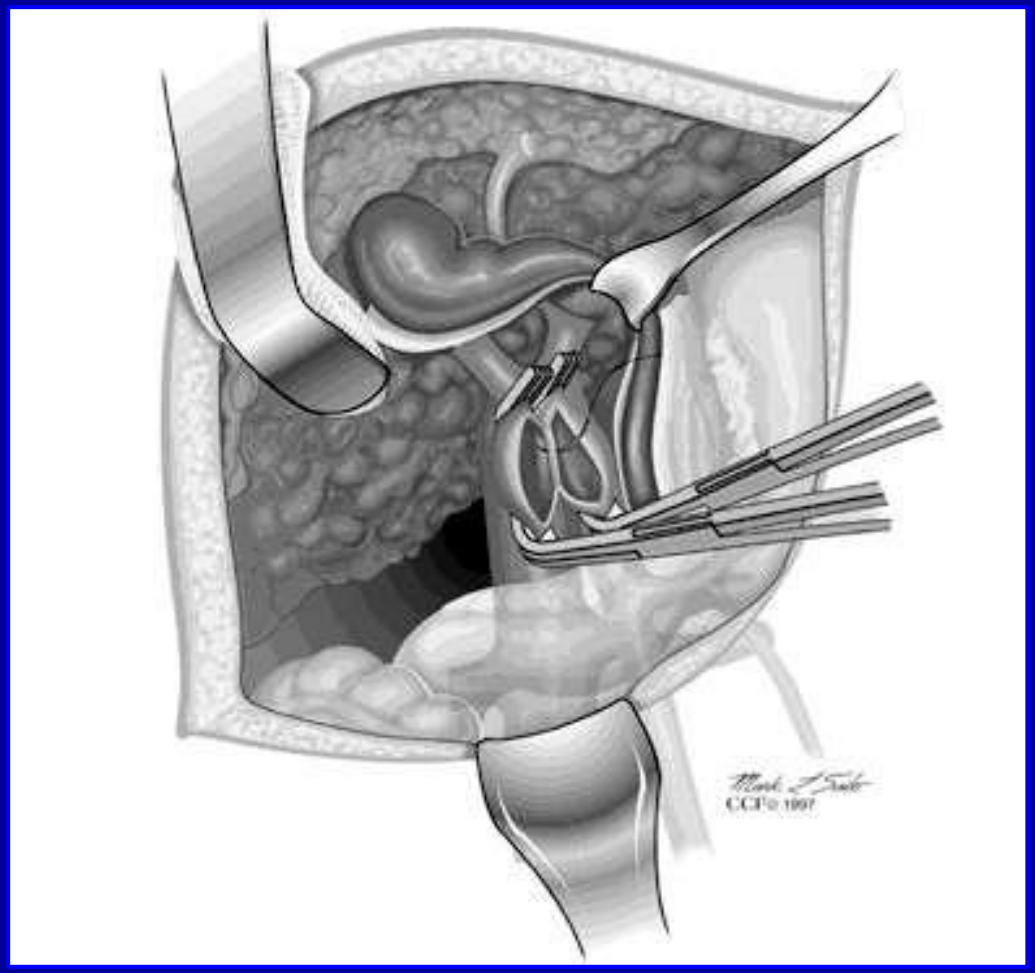
Transjugular Intrahepatic Portosystemic Shunt

Technique	Metallic stent between branch of PV & HV Under sedation with local anesthesia US guidance essential during the procedure Time of procedure: 1 – 2 hours Difficult (skilled interventional radiologist)
Indications	Control of bleeding from EV or GV Medical & endoscopic tt given before TIPS
Results	Bleeding control 90 %
Mortality	< 1 %

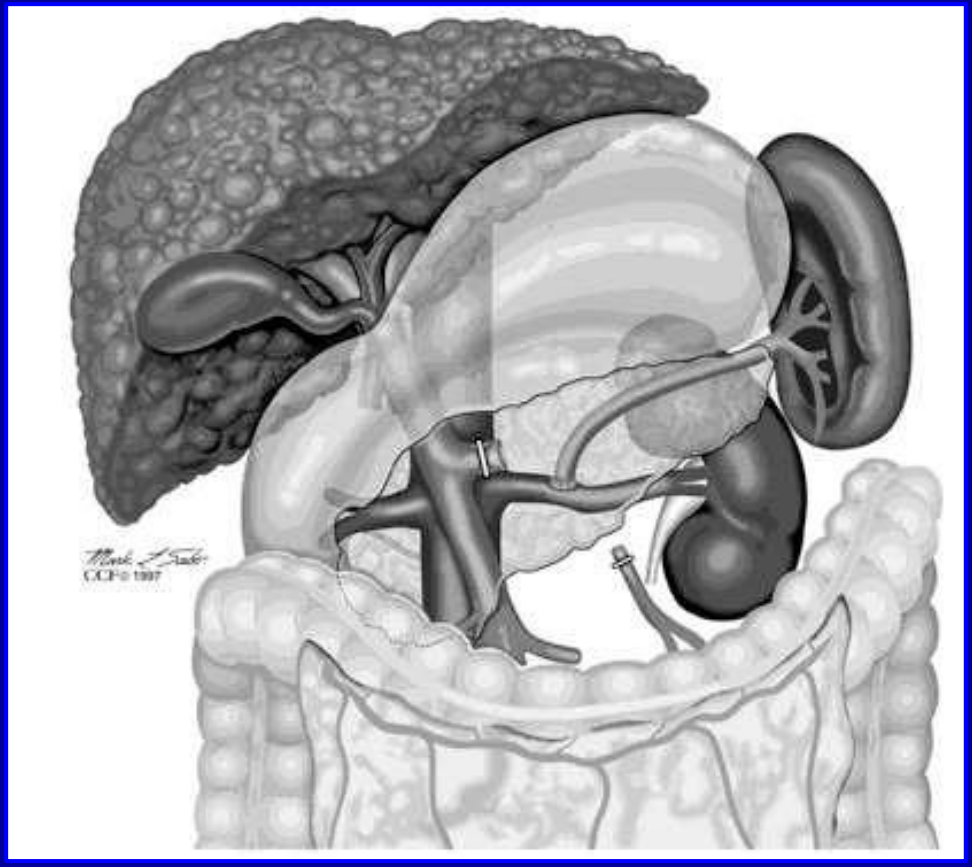
General results of surgical shunts

Bleeding	Prevented or at least decreased Varices disappear in 6 – 12 months
Complications	Post-operative jaundice Increase cardiac output & failure
Hepatic encephalopathy	May be transient Chronic changes in 30 – 40 % Increase with the size of shunt More common in older patients
Mortality	**5 %** in good-risk patients **50 %** in poor-risk patients

Side-to side porto-caval shunt



Distal spleno-renal shunt



Veins feeding varices ligated: coronary-rt gastric-rt gastroepiploic

Spleen is preserved

Distal spleno-renal shunt

Mortality similar to non-selective shunts

Hepatic encephalopathy similar to non-selective shunts

Better results in non-alcoholic patients & in gastric varices

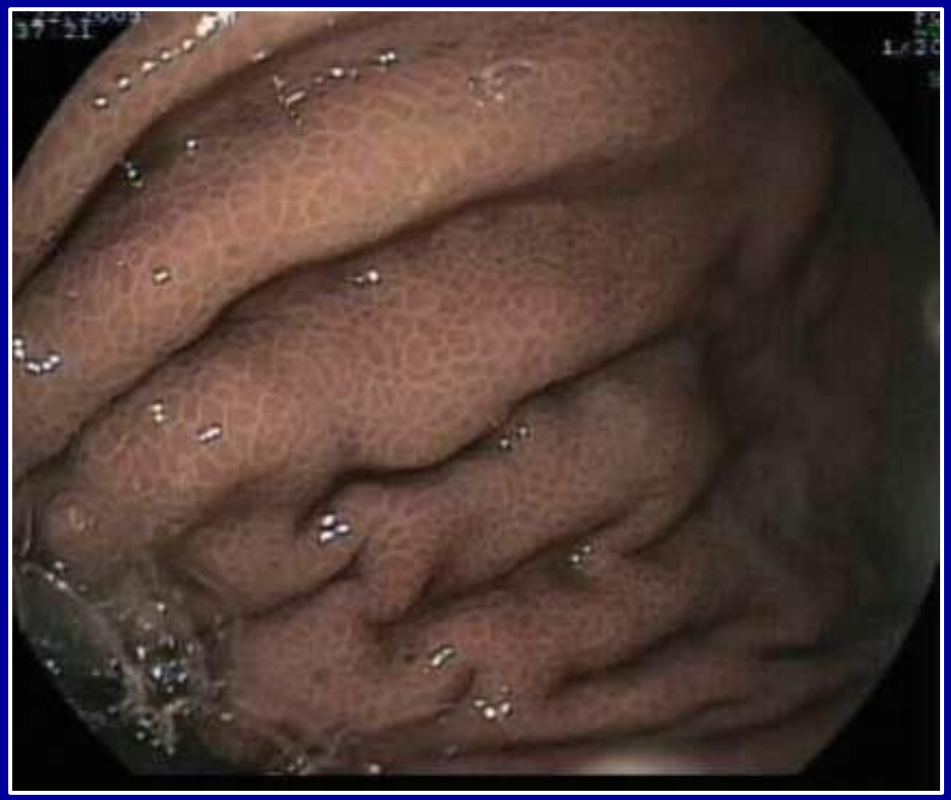
Does not interfere with subsequent liver transplant

Technically difficult (fewer surgeons willing to perform it)

Causes of bleeding in PHT

- Esophageal varices
- Gastric varices
- Ectopic varices
- Portal hypertensive gastropathy

Portal gastropathy



Mosaic-like mucosal pattern

Snake-skin appearance

Endoscopic images of PHT gastropathy

New Italian Endoscopic Club

- **Mosaic-like mucosal pattern** (snake-skin appearance)
- **Red point lesions**
  Small (<1 mm), red, flat, point-like marks
- **Cherry-red spots**
  Large (>2 mm), round, red-colored, protruding lesions
- **Black–brown spots**
  Irregular black & brown flat spots not fading upon washing
  Might represent intramucosal hemorrhage

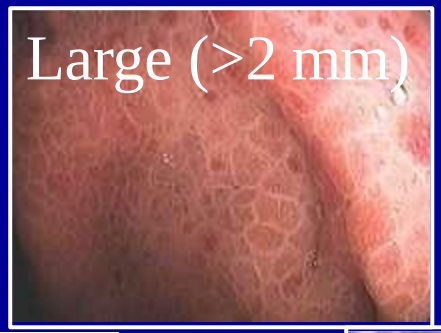
Primignani M et al. Gastroenterology 2000 ; 119 : 181 – 187.

PHT gastropathy – Four main findings

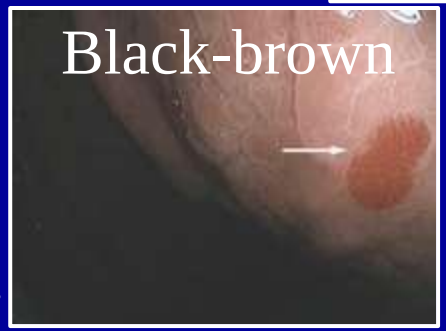
Mosaic-like pattern
Snake-skin appearance

Red point lesions

Cherry-red spots



Black–brown spots



Gastroenterology 2000;119:181-187.

Mallory-Weiss syndrome

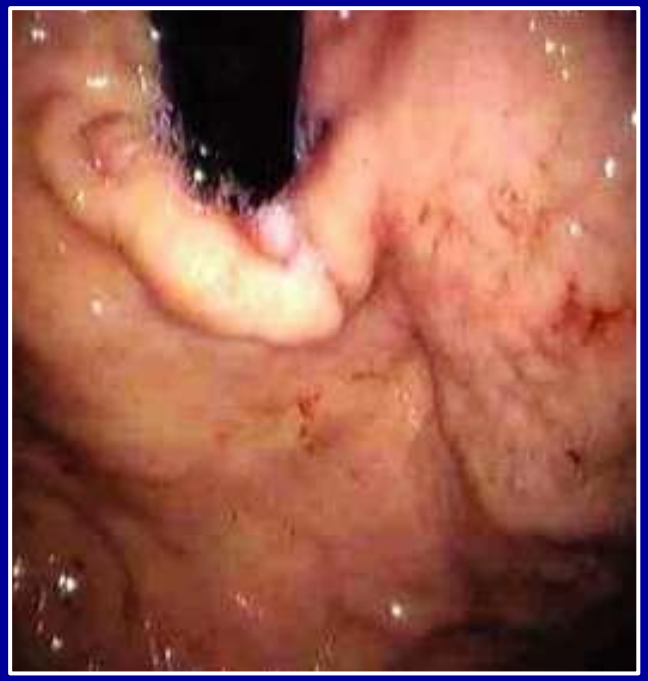
5- 10 % of UGI bleeding

Typically in gastric mucosa

Stop spontaneously in 80-90%

Not bleeding: discharge promptly

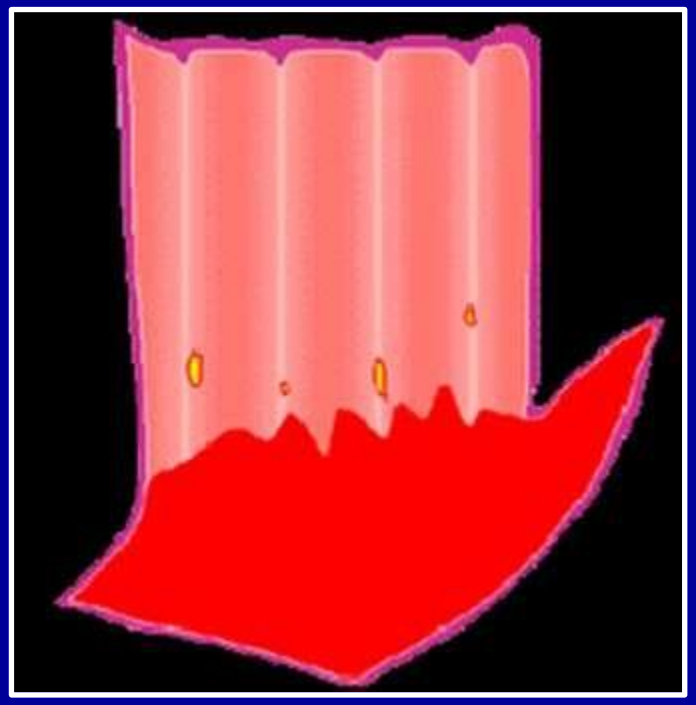
Active bleeding: injection – banding

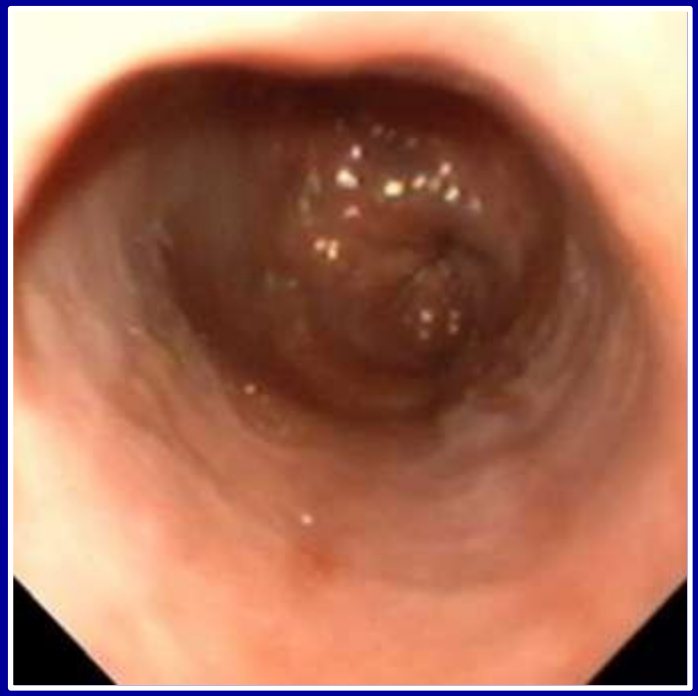


Retroflexed view

LA classification system of esophagitis

Grade A

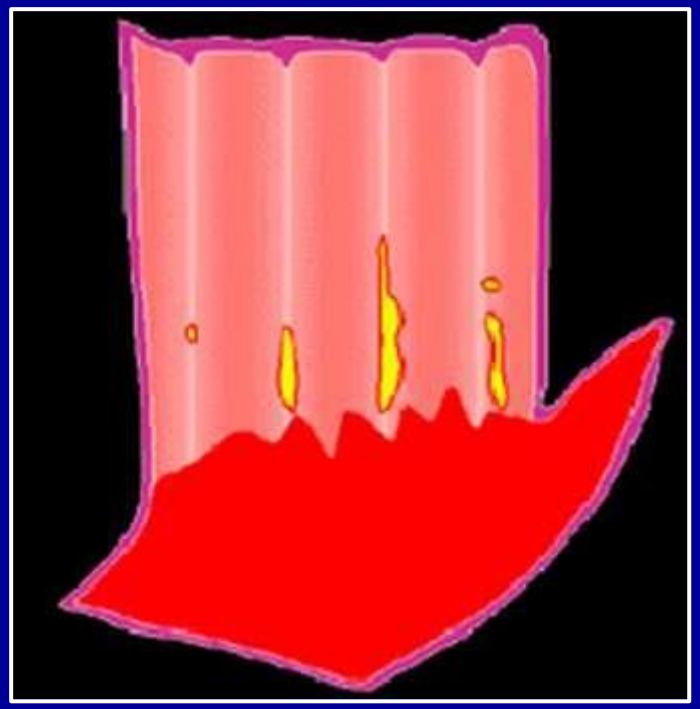


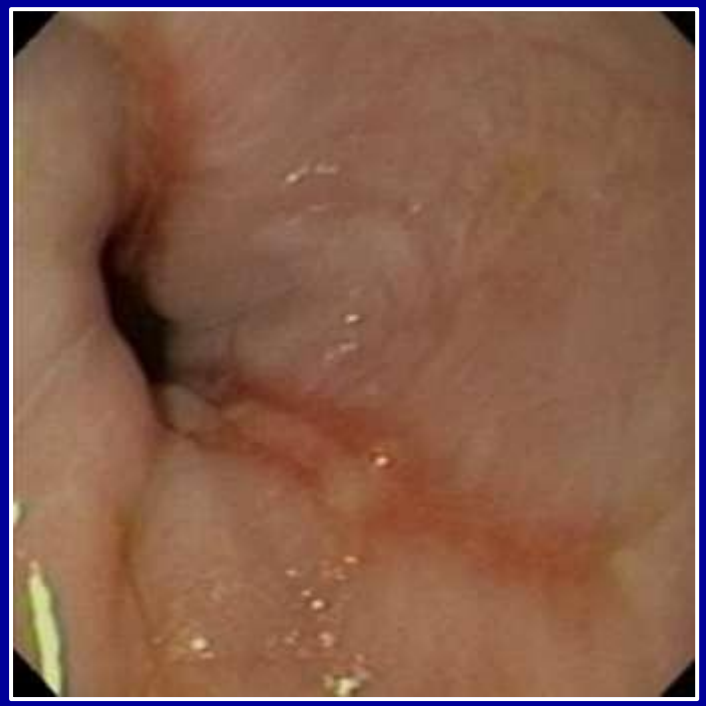


One (or more) mucosal break, no longer than 5 mm, that does not extend between tops of 2 mucosal folds

LA classification system of esophagitis

Grade B

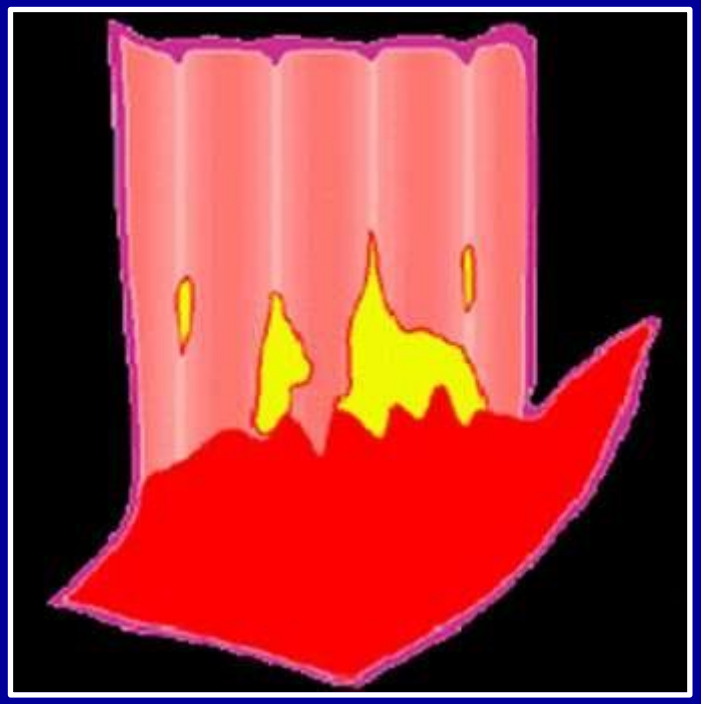


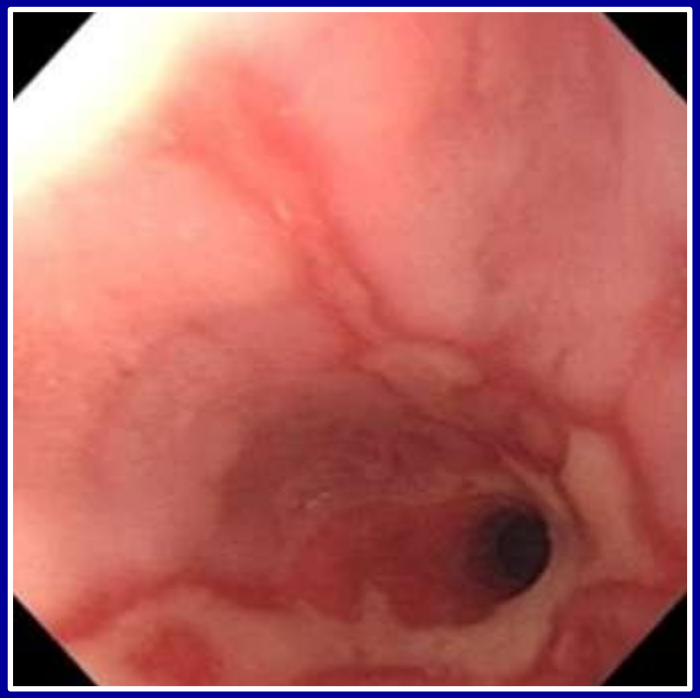


One (or more) mucosal break, more than 5 mm long, that does not extend between tops of two mucosal folds

LA classification system of esophagitis

Grade C

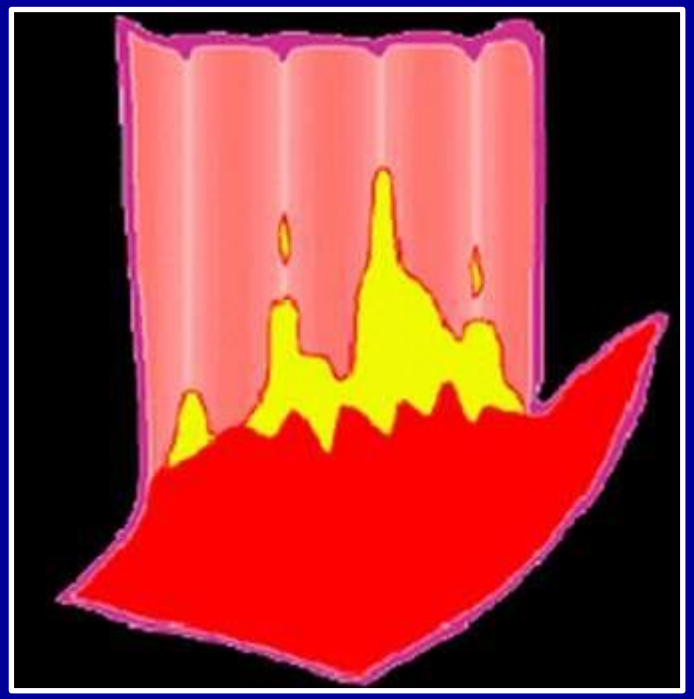


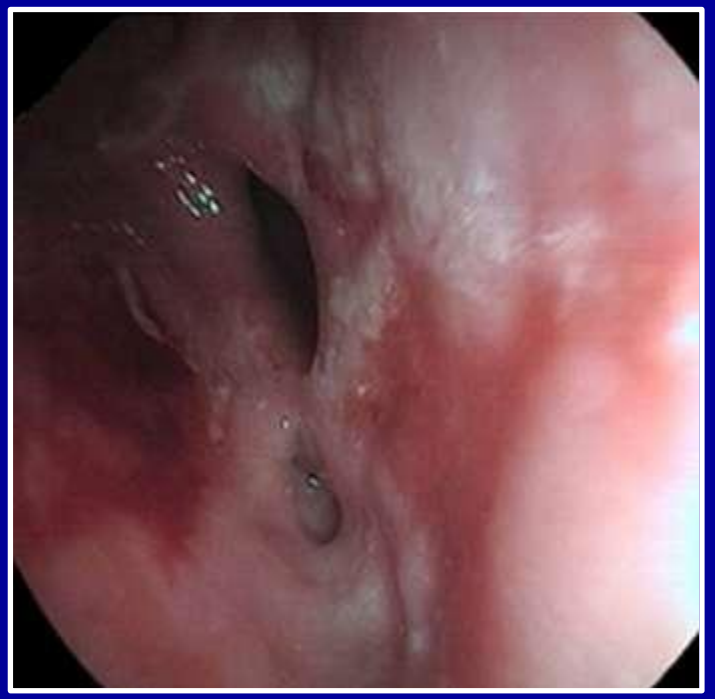


One (or more) mucosal break continuous between tops of > 2 mucosal folds, but which involves < 75% of circumference

LA classification system of esophagitis

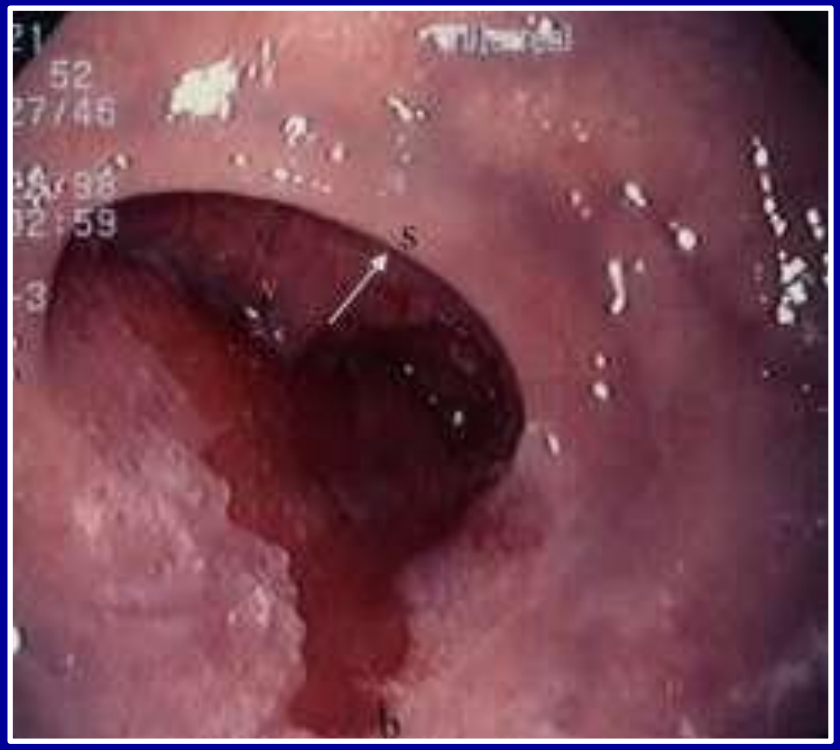
Grade D





One (or more) mucosal break that involves at least 75% of the esophageal circumference

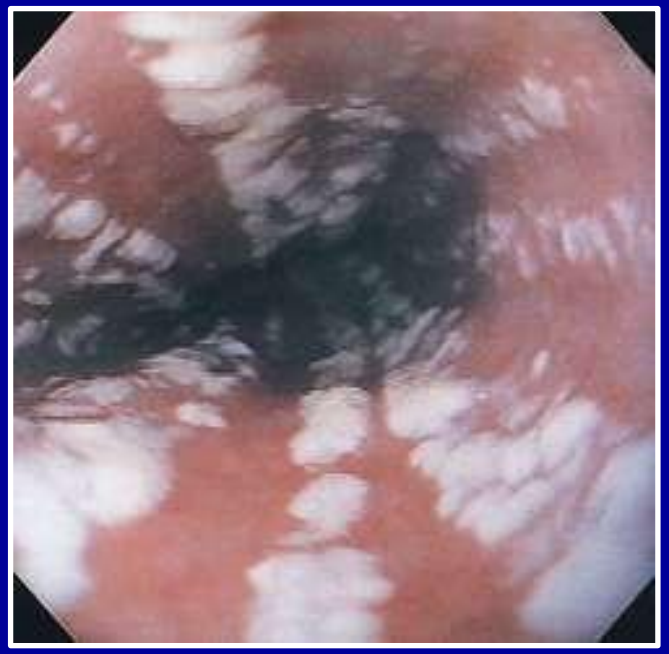
Barrett's esophagus



Endoscopic view of distal esophagus from a patient with GERD

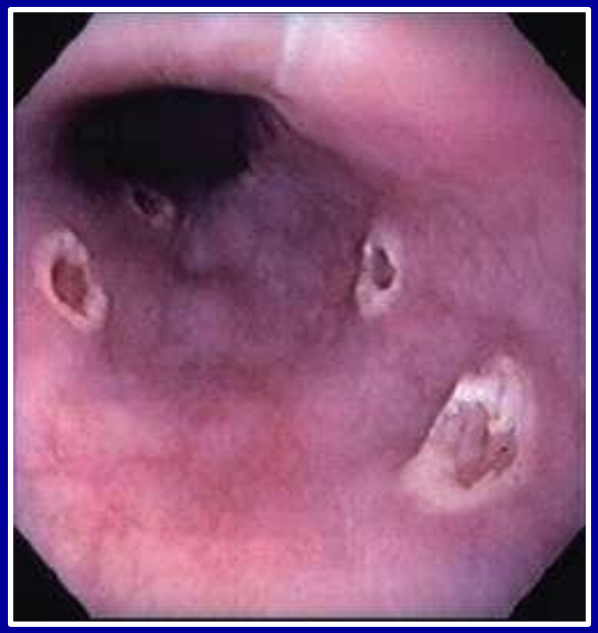
Tongue of Barrett's mucosa (b) & Schatzki's ring(s) (arrow)

Esophageal candidiasis



Multiple small white plaques of Candida seen on background of abnormally reddened esophageal mucosa

Herpes Simplex in the esophagus



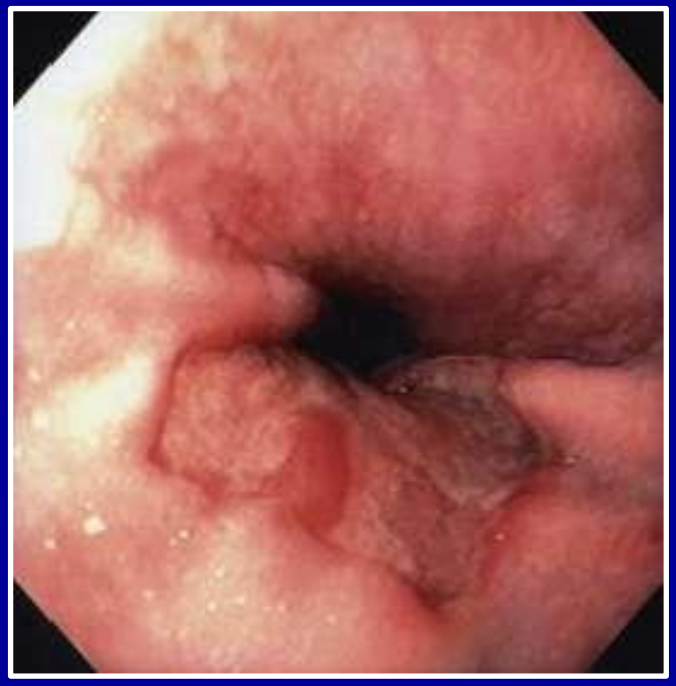
Small volcano-like ulcers due to HSV

Appearance not diagnostic of HSV infection

It could be due to drug-induced lesion (K supplement)

Presence of vesicles in mucosa virtually diagnostic of HSV

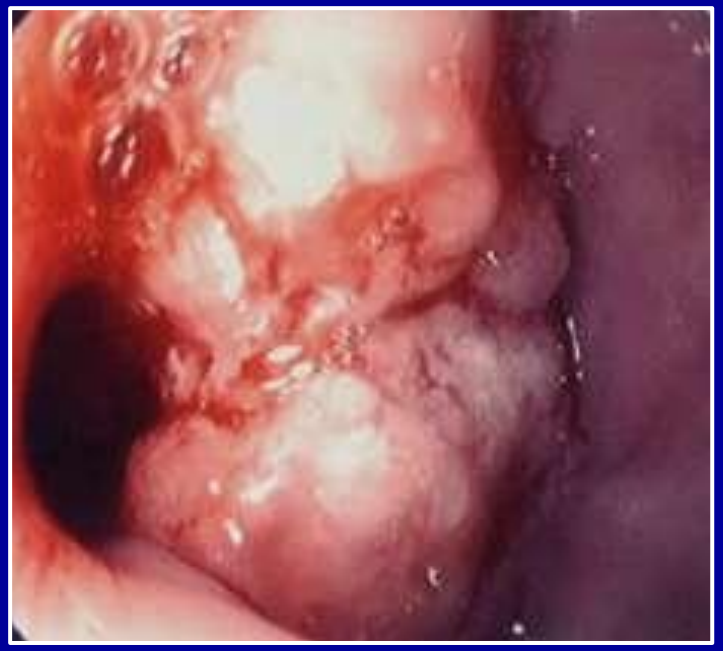
CMV esophagitis



Solitary deep well-circumscribed ulcer

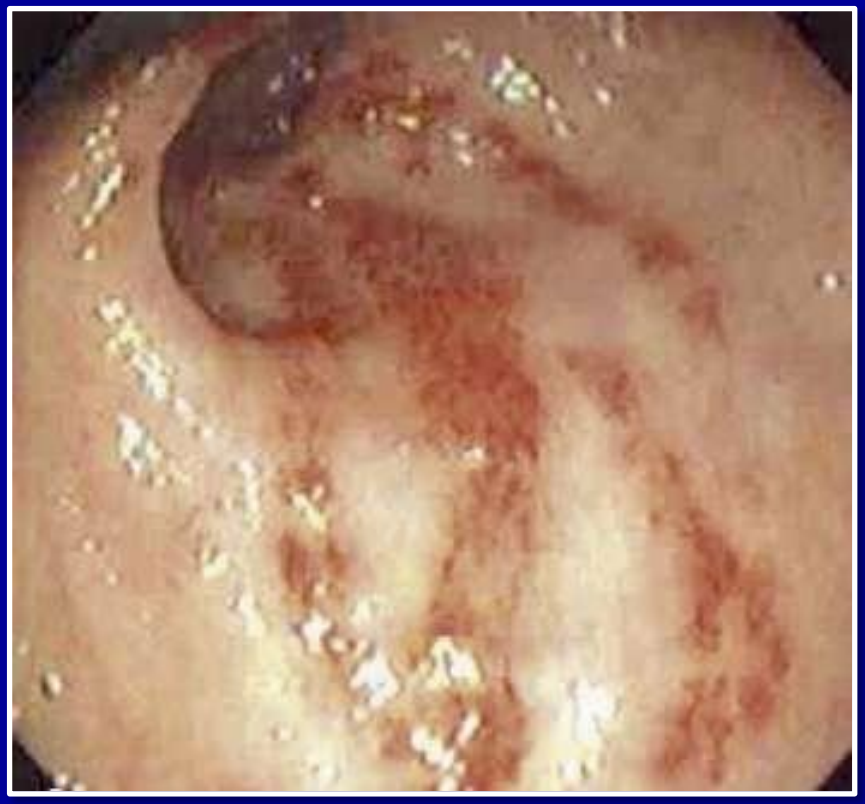
at gastroesophageal junction

Cancer of gastroesophageal junction



Large malignant mass at GE junction

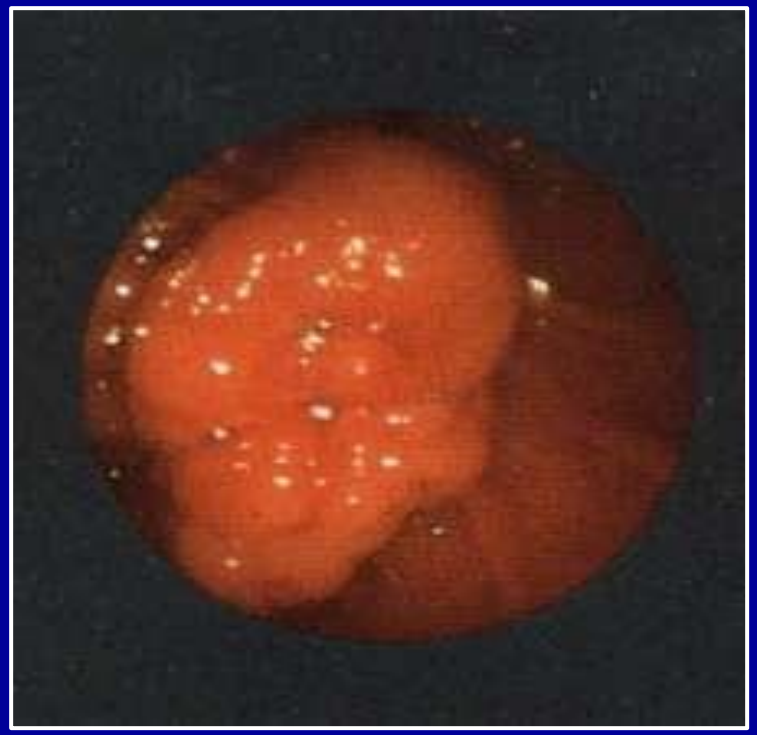
Watermelon stomach



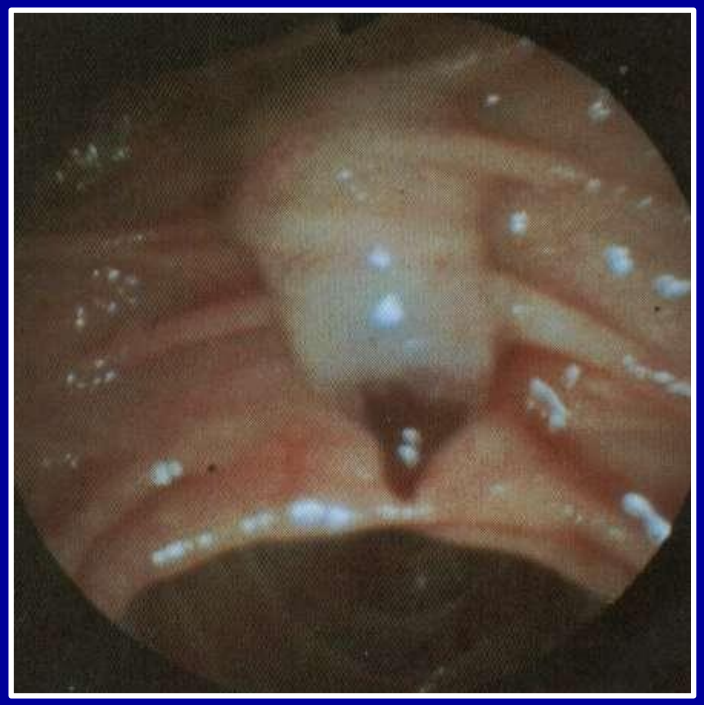
Gastrointest Endosc 2005; 61 : 631 - 633.

Ampulloma

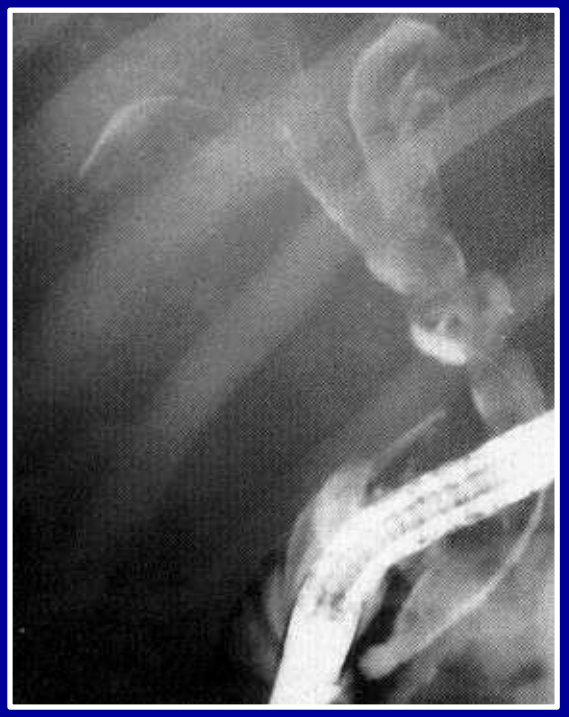
Endoscopic view



Hemobilia



Blood clot protruding from the ampulla

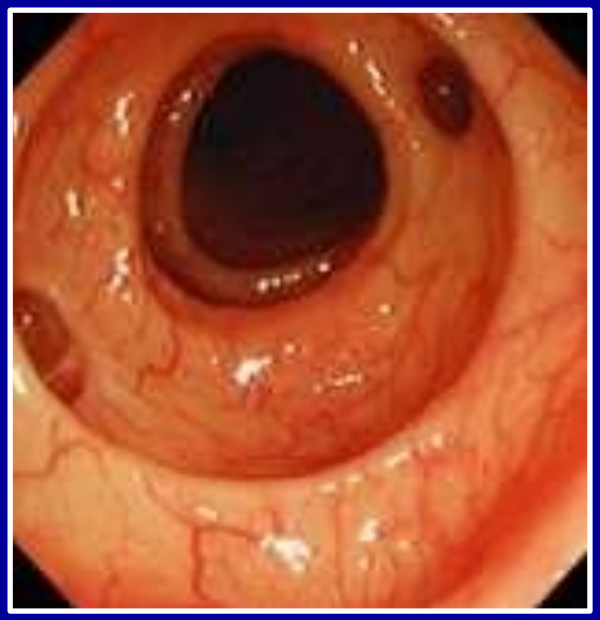


Corresponding ERCP

Causes of lower GI bleeding

Common	Less Frequent	Rare
Diverticula	Neoplasia	Dieulafoy's lesion
Vascular ectasia	IBD	Colonic ulceration
	Colitis: ischemia – radiation	Rectal varices
	Hemorrhoids	
	Small bowel source	
	UGI source	

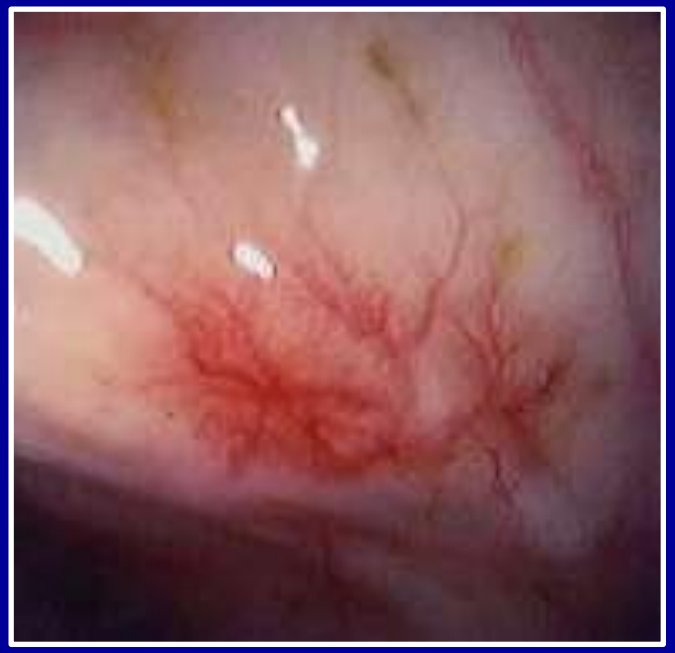
Diverticular disease of the colon



Wide-mouthed openings to diverticula are present

They were seen throughout the sigmoid colon in this patient

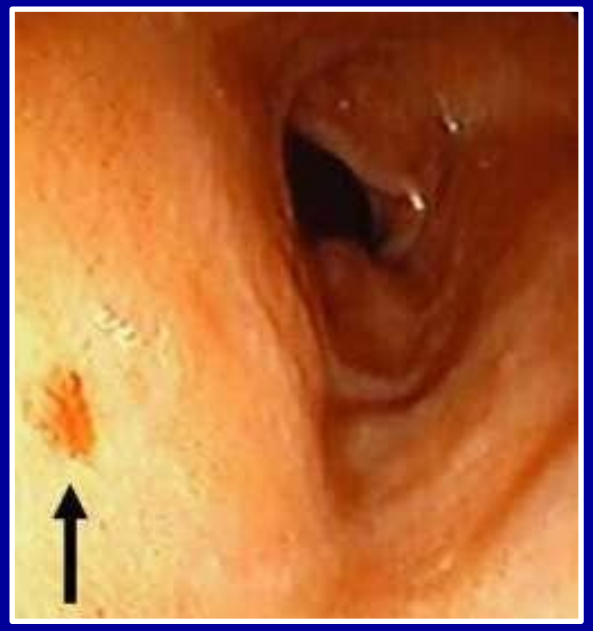
Mucosal telangiectasia of the colon



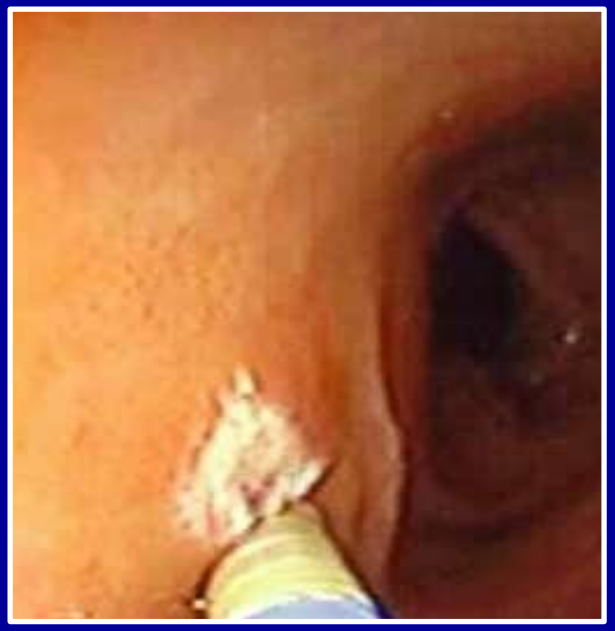
The patient presented with hematochezia

The lesion was subsequently cauterized endoscopically

Telangiectasia

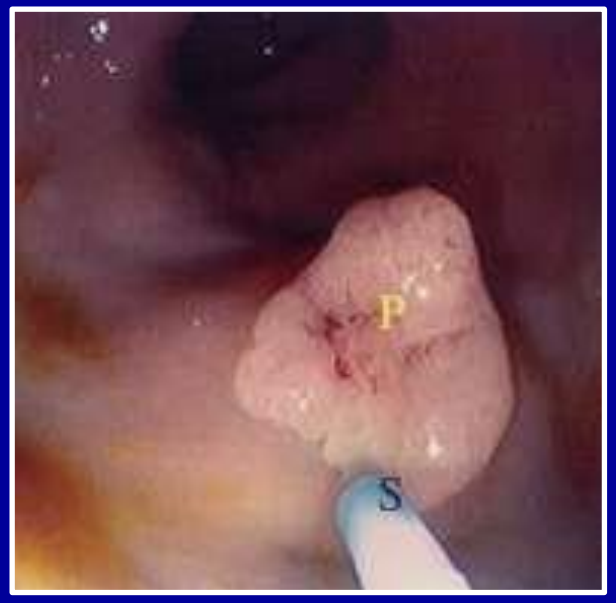


Telangiectasia in duodenum in patient with microcytic anemia



Treatment with **APC** (Argon Plasma Coagulation)

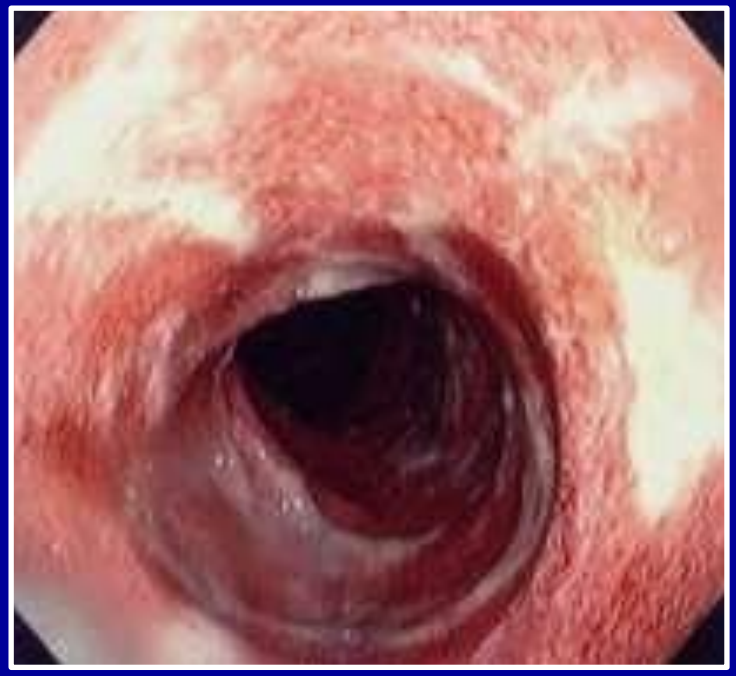
Endoscopic polypectomy



Snare passed through endoscope & positioned around polyp (P)

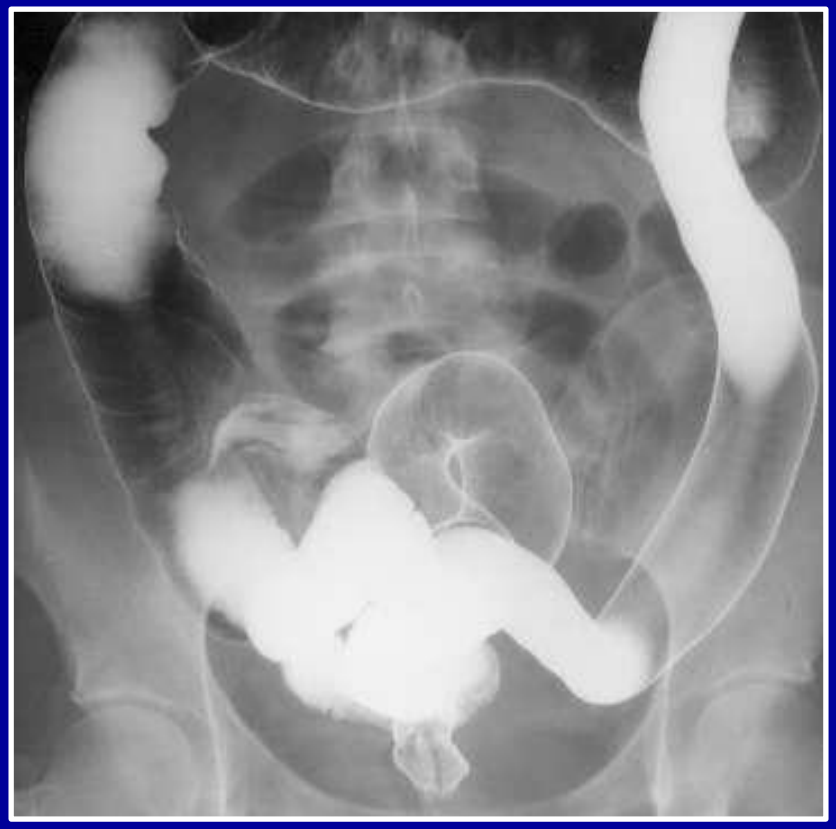
Cautery applied & polyp resected leaving clean mucosal defect

Ulcerative colitis



Colonic mucosa in a patient with idiopathic ulcerative colitis, showing a friable mucosa, extensive ulceration, and exudates.

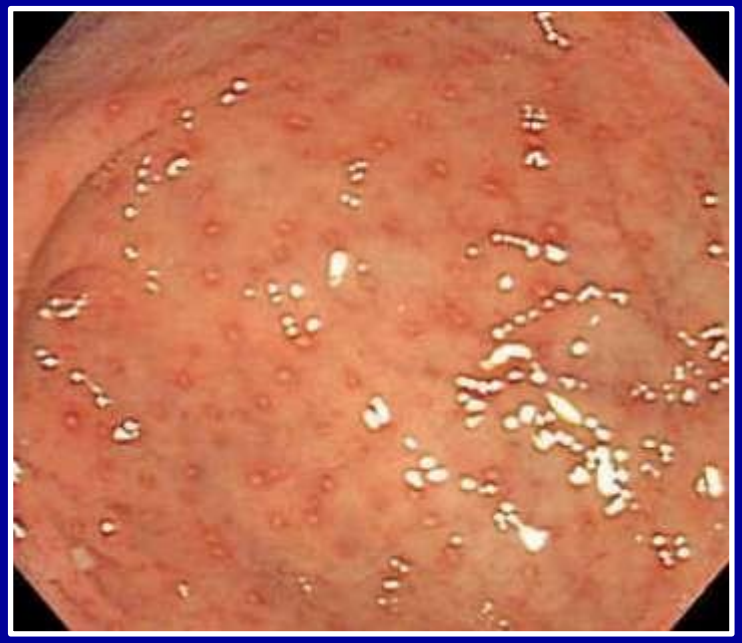
Ulcerative colitis



Air contrast barium enema demonstrating luminal narrowing

& loss of haustra in sigmoid & descending colon in UC

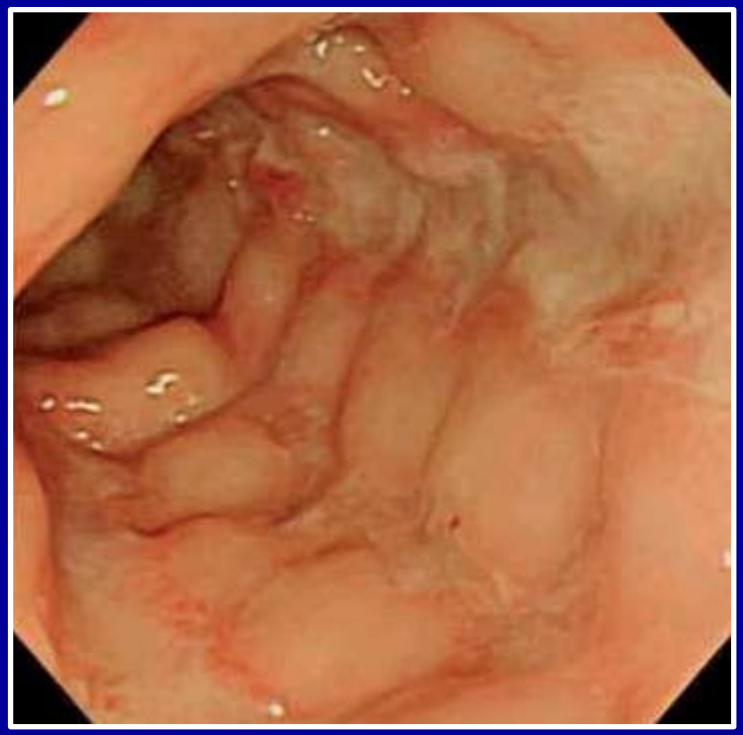
Crohn's disease



Aphthous ulcers in the rectum in a patient with Crohn's disease

Lee YJ et al. Endoscopy 2006; 38 : 592 – 597.

Crohn’s disease



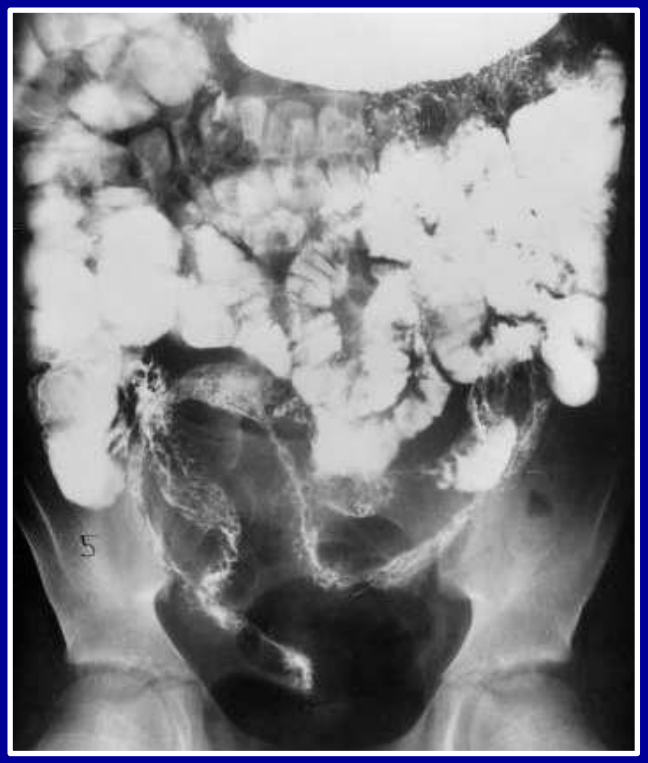
Longitudinal ulcers & cobblestone appearance

in a patient with Crohn’s disease

Lee YJ et al. Endoscopy 2006; 38 : 592 – 597.

Crohn's disease of the ileum

Small bowel follow-through in ileal Crohn's disease

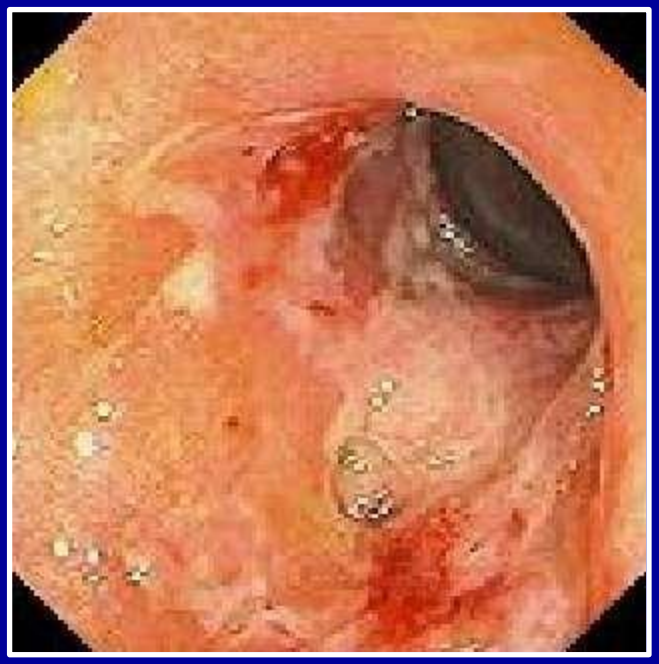


Luminal narrowing

Mucosal ulceration

Separation of barium-filled loops (thickening of bowel wall)

NSAIDs-induced colitis

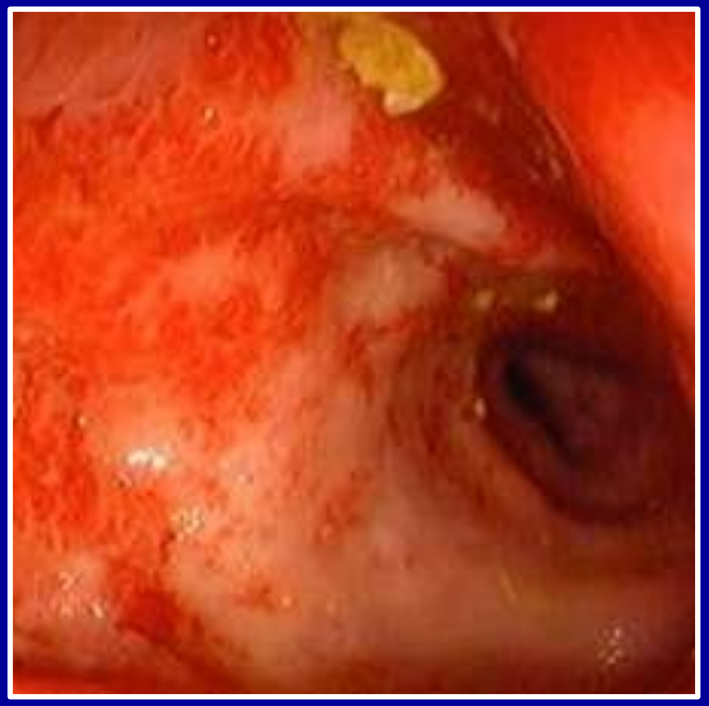


Endoscopically nonspecific findings

Histologically nonspecific

DD: infections, IBD, ischemia, vasculitis

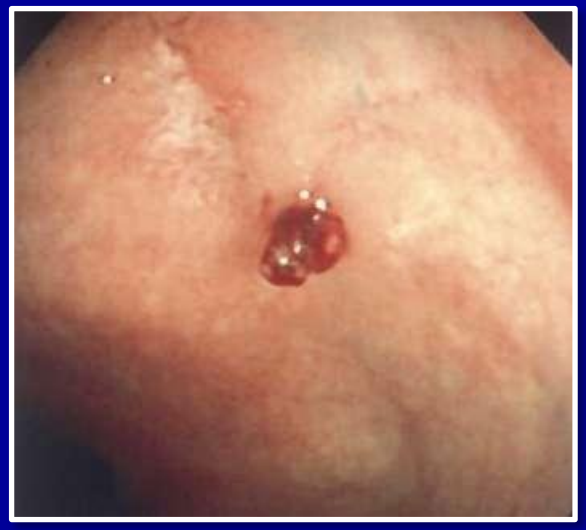
Radiation proctitis



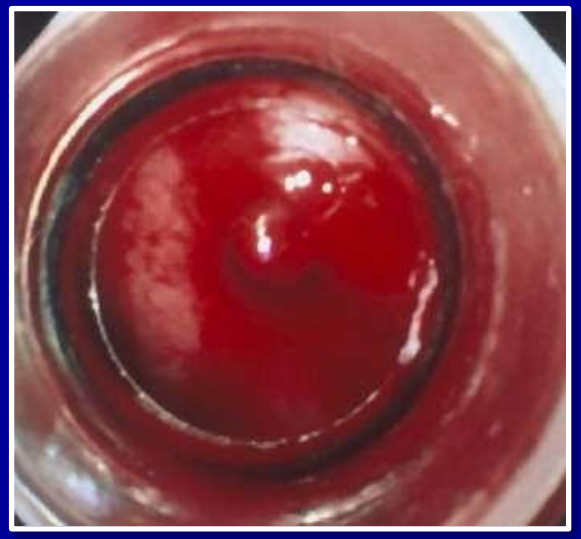
Radiation proctitis in a patient with hematochezia

Extensive neovascularization of the mucosa

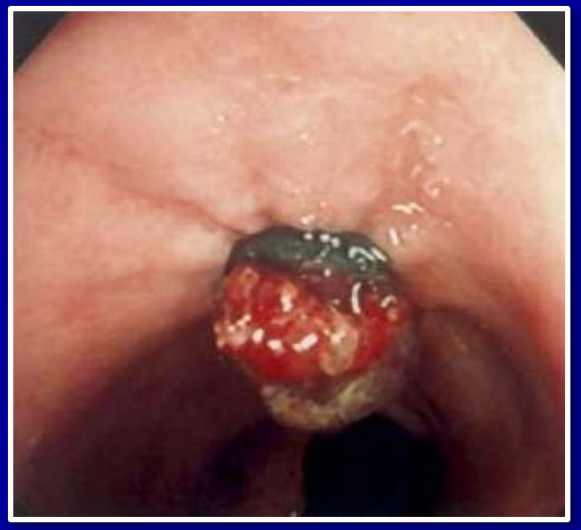
Rectal Dieulafoy's lesion



Endoscopic appearance



During ligation



After ligation

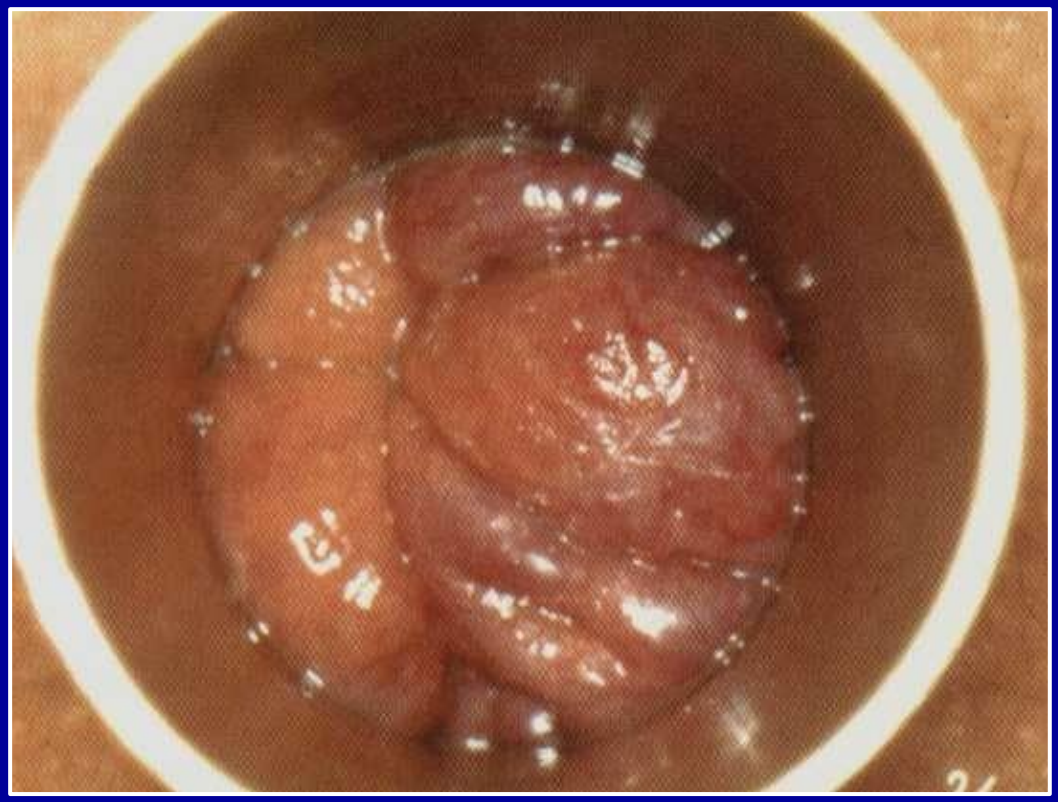
Gastrointest Endosc 2004 ; 60 : 796.

Classification of hemorrhoids

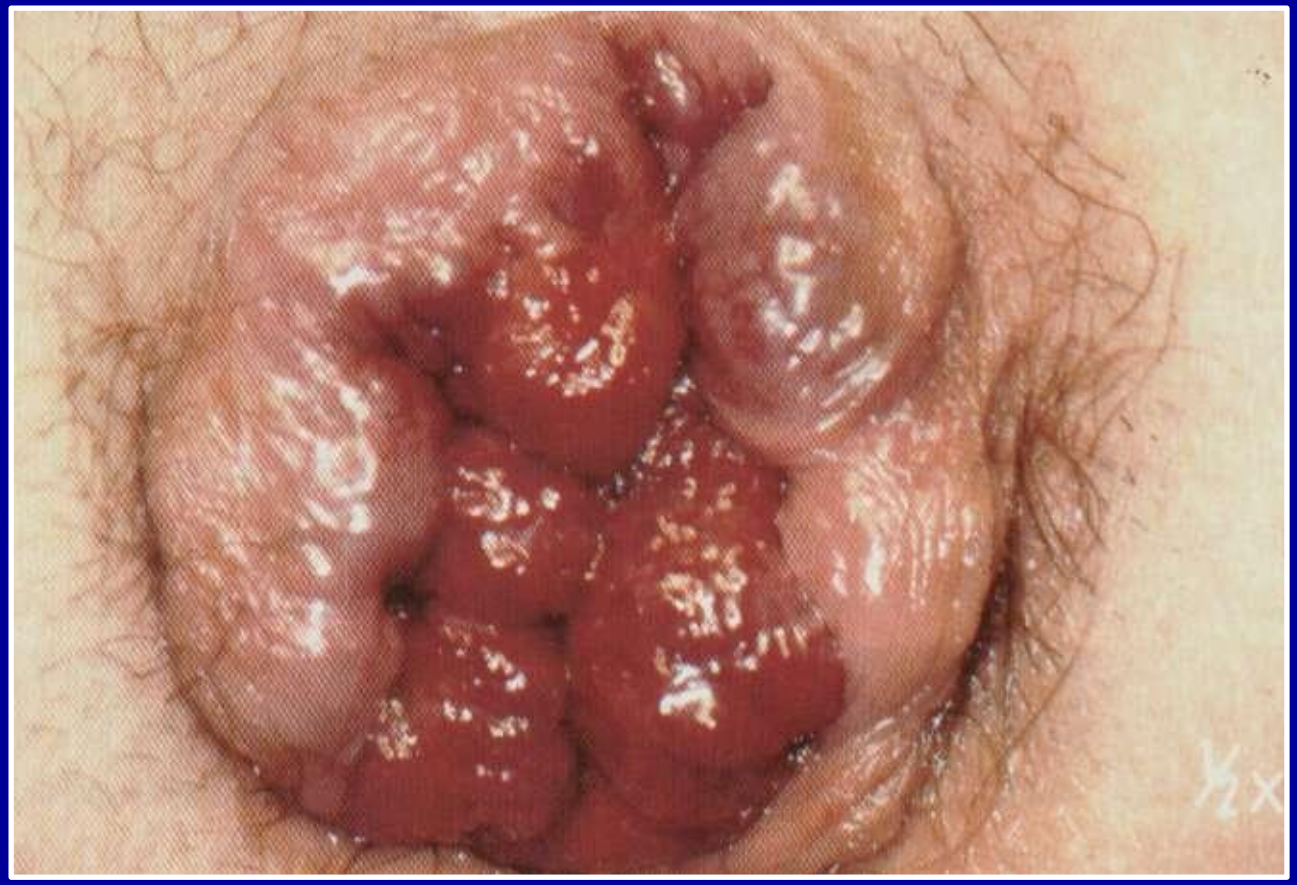
Degree	Description
First degree	Project a short way into anal canal Only symptom is bleeding
Second degree	Prolapse during defecation Reduce spontaneously
Third degree	Must be reduced manually
Fourth degree	Irreducible

Internal hemorroids

Seen with the proctoscope



Prolapse of 3 mains hemorrhoidal piles



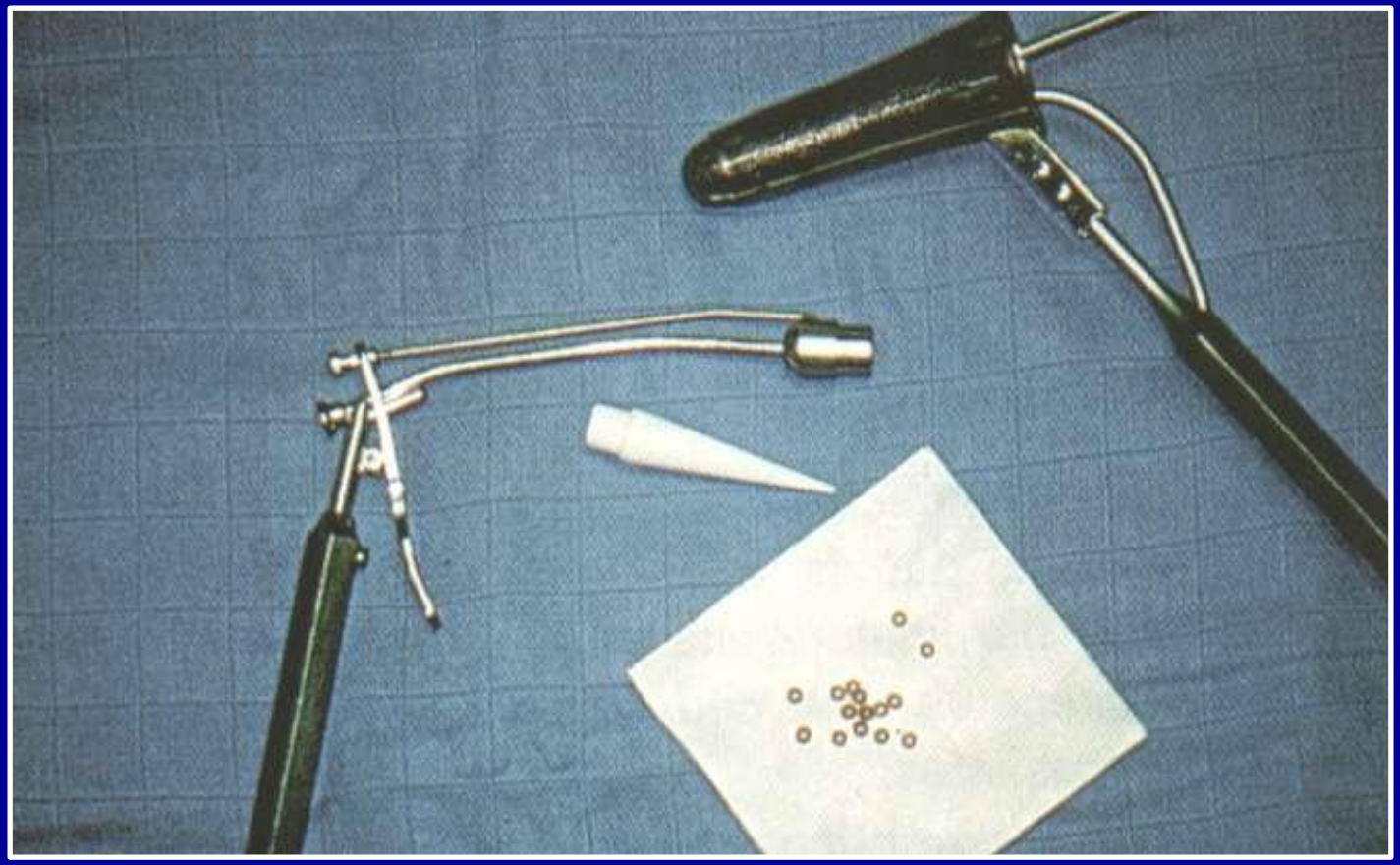
Preferences for treatment of hemorrhoids

Degree or Grade	Treatment
1	Sclerosing injections Infrared coagulation
2	Infrared coagulation Rubber band ligation
3	Rubber band ligation
4	Hemorrhoidectomy

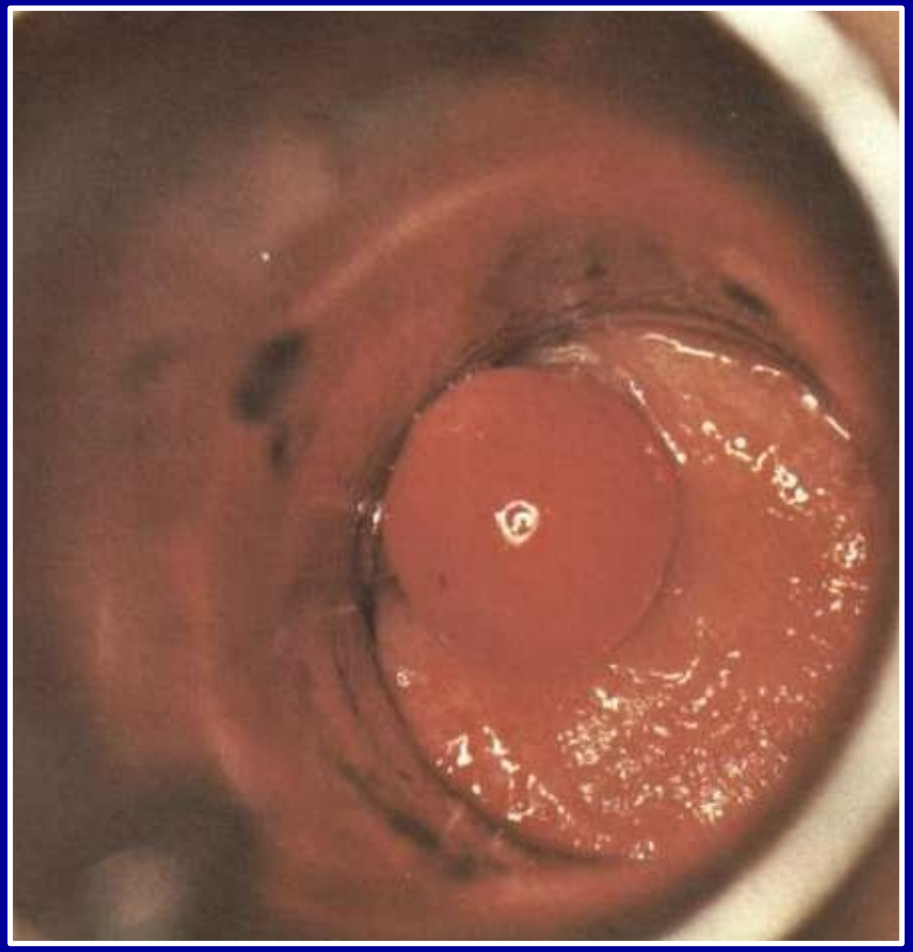
Sclerosing injection

Infrared photocoagulation

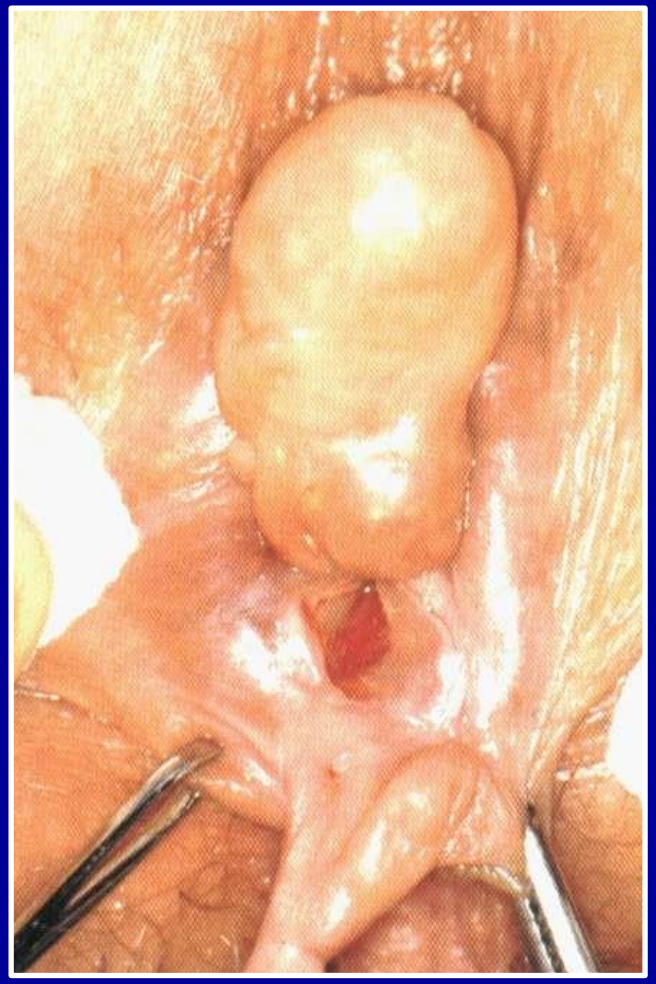
Rubber band ligation



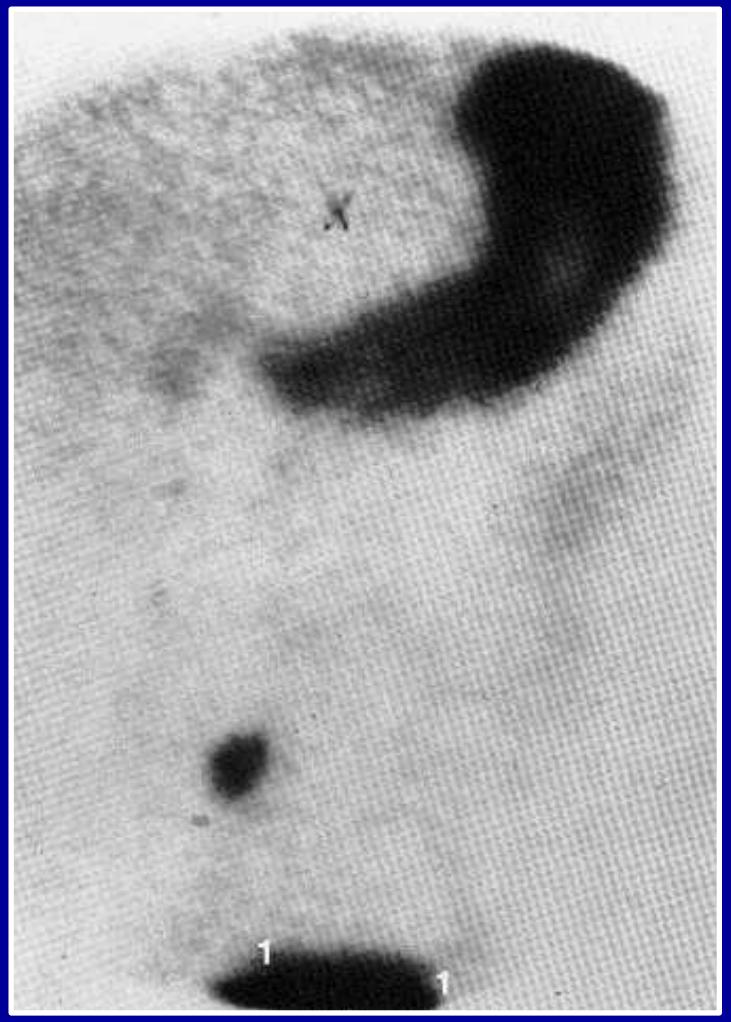
Rubber band ligation



Anal fissure



Meckel's divertculum



Isotope scan with Tc^{99m}

Approach to lower GI bleeding

- Less common than UGI bleeding
- Usually less hemodynamicaly significant
- Most common cause of severe bleeding: **diverticula**
- Most common cause of minor bleeding: **hemorrhoids**
- Controversial best diagnostic approach if severe:
  Urgent colonoscopy – RBC scintigraphy – angiography

Thank You