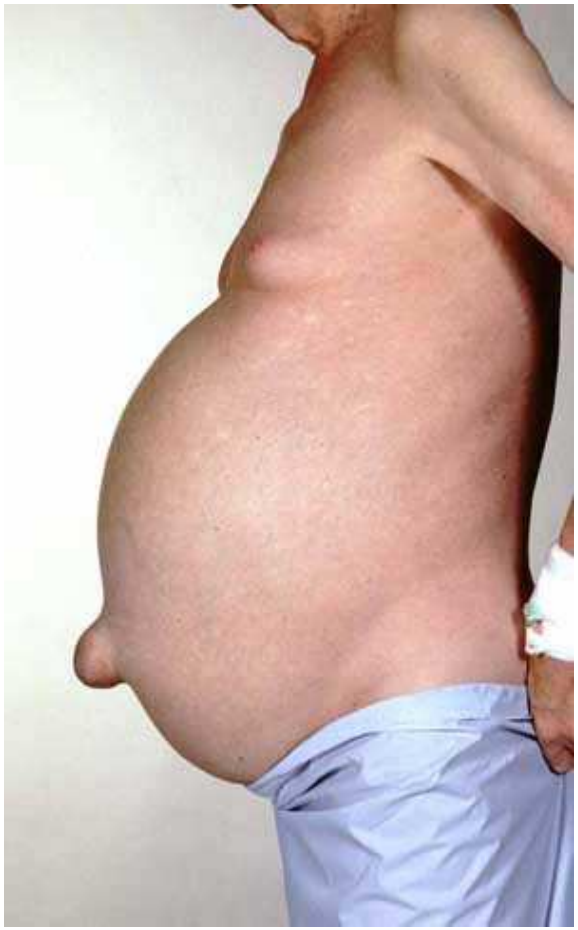
Approach to the patient with ascites

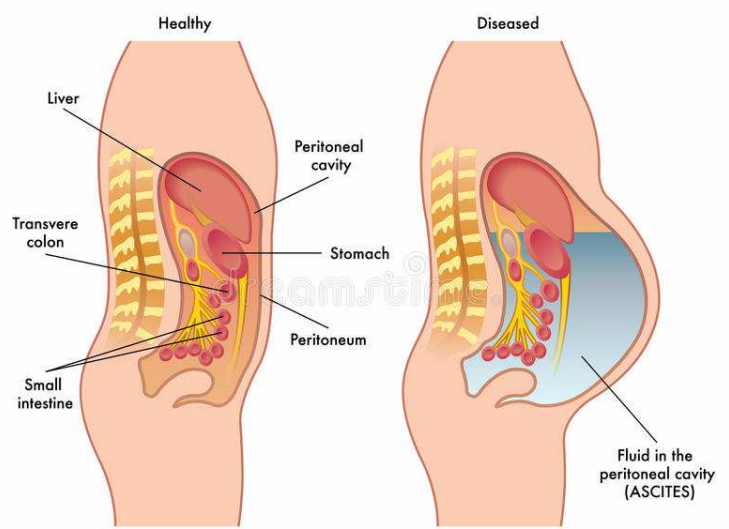
Prof. G. Zuliani



Ascites

Definition of ascites:

- ASCITES IS THE CONDITION OF PATHOLOGICAL ACCUMULATION OF FLUID INTO THE ABDOMINAL CAVITY

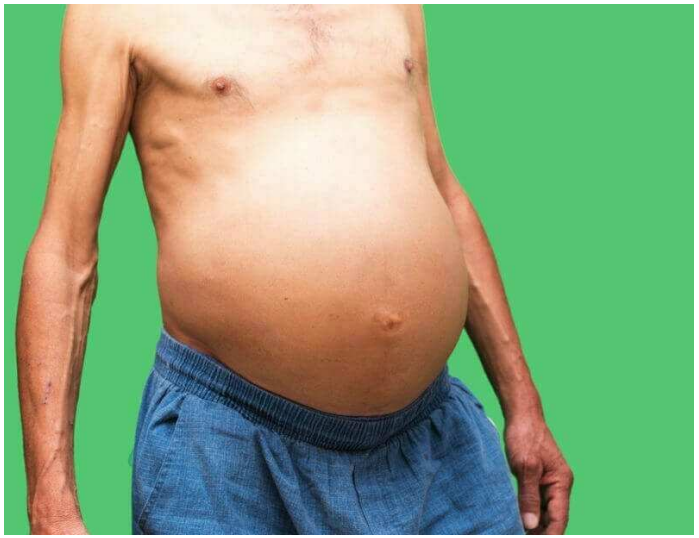


Ascites

CAUSES

Ascites can be broadly classified on the basis of:

1. **Normal peritoneum**
2. **Diseased peritoneum**

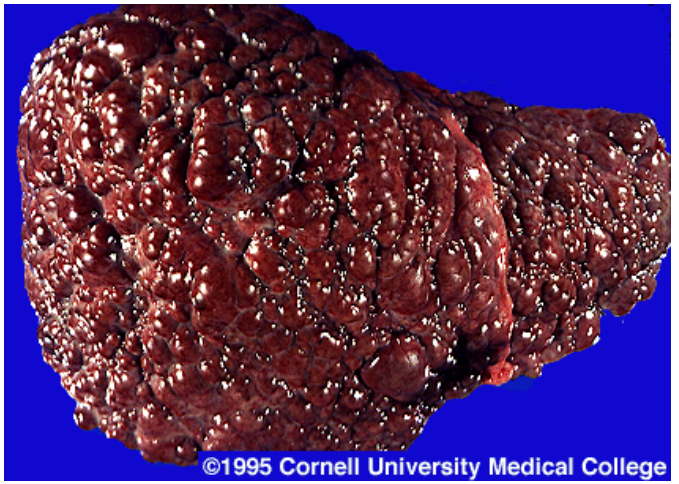


Ascites

1. NORMAL PERITONEUM

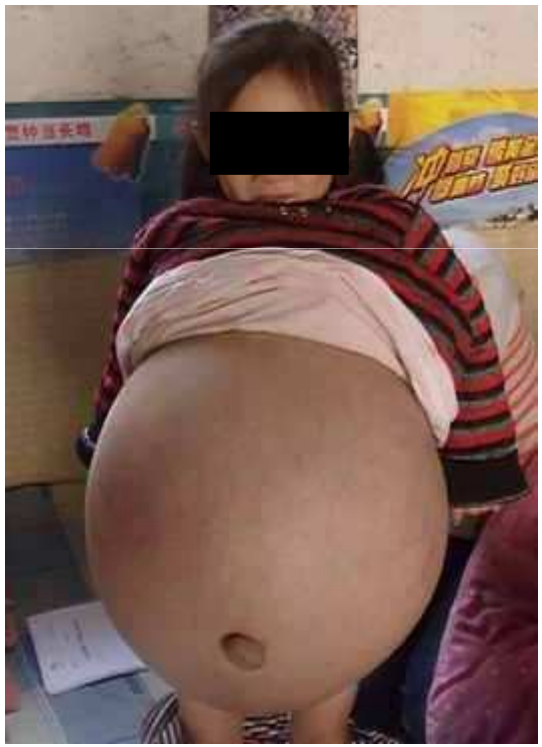
A. With portal hypertension:

- **Liver Cirrhosis**
- **Congestive Heart Failure**
- Alcoholic Hepatitis
- Fulminant Hepatic Failure
- Massive Hepatic Metastases
- Constrictive Pericarditis
- Budd-Chiari Syndrome
- Tricuspid Insufficiency



Budd-Chiari Syndrome

Budd-Chiari syndrome is a rare condition induced by ***thrombotic-nonthrombotic obstruction to hepatic venous outflow.*** Budd described it in 1845, and Chiari added the first pathologic description of a liver with "obliterating endophlebitis of the hepatic veins" in 1899.

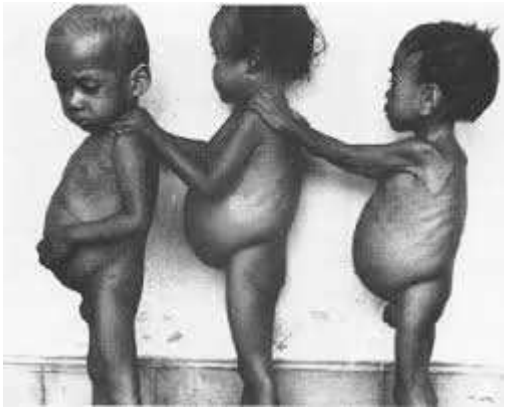


Hepatomegaly + ascites + abdominal pain characterize Budd-Chiari syndrome.

The syndrome most often occurs in patients with underlying thrombotic diathesis, including myeloproliferative disorders, such as ***polycythemia vera and paroxysmal nocturnal hemoglobinuria, pregnancy, tumors, chronic inflammatory diseases, clotting disorders, and infections.***

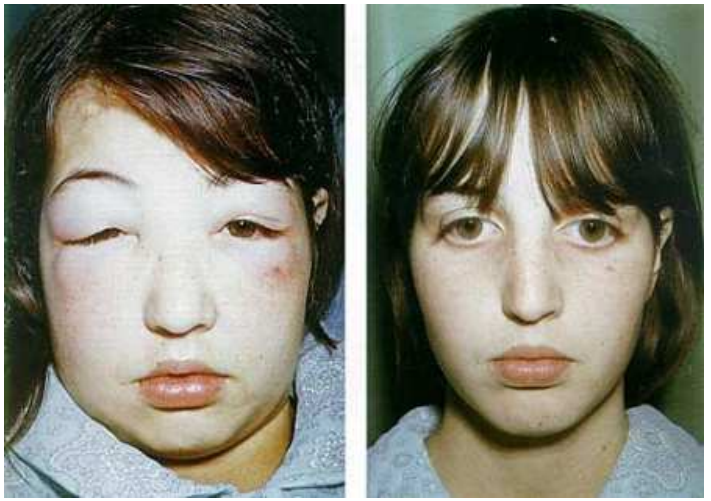
Ascites

1. NORMAL PERITONEUM



B. With Hypoalbuminemia:

- Nephrotic Syndrome
- Protein-losing enteropathy
- Severe malnutrition with anasarca



Ascites

1. NORMAL PERITONEUM

C. Miscellaneous conditions:

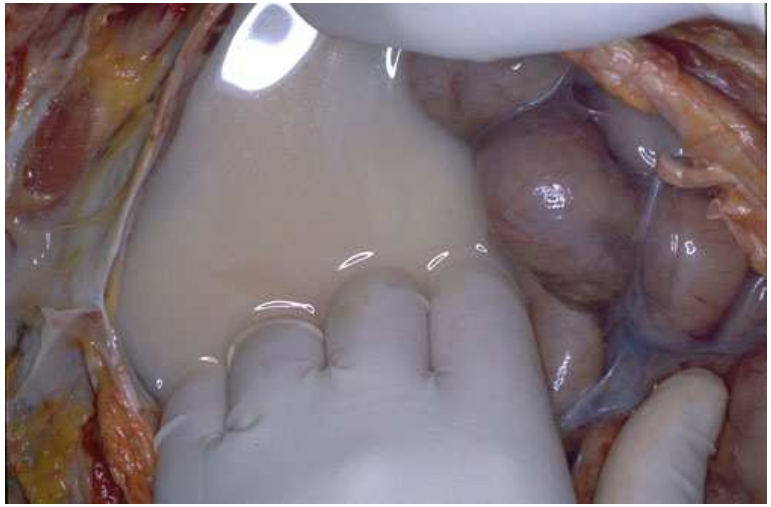
- Chylous ascites
- Pancreatic ascites
- Nephrogenic ascites
- Meig’s syndrome

Chylous ascites

Chylous ascites is the extravasation of milky chyle into the peritoneal cavity. This can occur de novo, but an existing clear ascitic fluid can turn chylous as a secondary event.

True chylous ascites is defined as the presence of ascitic fluid with **high triglyceride content,** usually **higher than 110 mg/dL.**

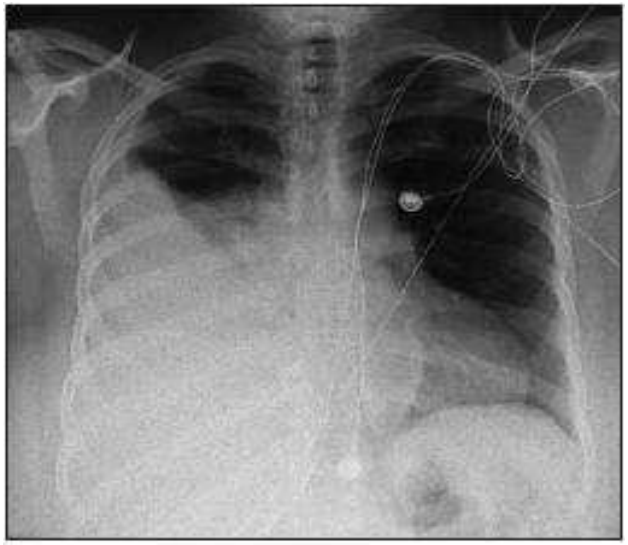
Multiple causes have been described, including the following:
Abdominal surgery, Abdominal trauma, Malignant neoplasms, Hepatoma, small bowel and retroperitoneal Lymphoma, small bowel Angiosarcoma, SBP, Cirrhosis (up to 0.5% of patients with cirrhosis may have chylous ascites), Pelvic irradiation, Peritoneal dialysis, Abdominal tuberculosis



Meig’s syndrome

Meigs' syndrome is the triad of: **ascites + pleural effusion + and benign ovarian tumor (fibroma).** It resolves after the resection of the tumor. For reasons unknown, the pleural effusion is classically on the right side.

It may mimic other conditions! Because of the presence of ascites and transudative pleural effusions, the differential diagnosis includes: liver failure (cirrhosis), congestive heart failure, renal diseases, and metastatic tumors to peritoneal surfaces.
These entities must be clinically excluded.



Ascites

2. DISEASED PERITONEUM

A. Infections:

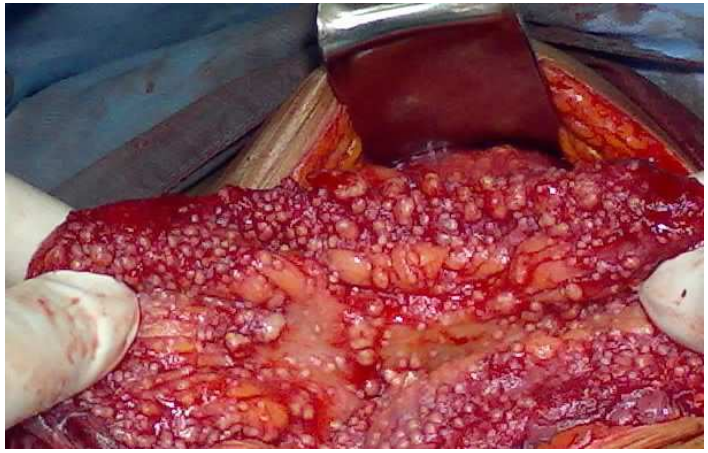
- Tuberculous Peritonitis
- Bacterial Peritonitis
- Fungal Peritonitis
- HIV associated peritonitis

Peritoneal tuberculosis

- ***Acute tuberculous peritonitis***
- ***Chronic tuberculous peritonitis***
  - ***Ascitic form***

    Insidious in onset, abdominal pain usualy absent, rolled up omentum infiltrated with tubercle may felt as a transverse solid mass
  - ***Encysted (loculated) form***
  - ***Fibrous form***

    Wide spread adhesions may cause coils of intestine matted together and distended, they may act as blind loop

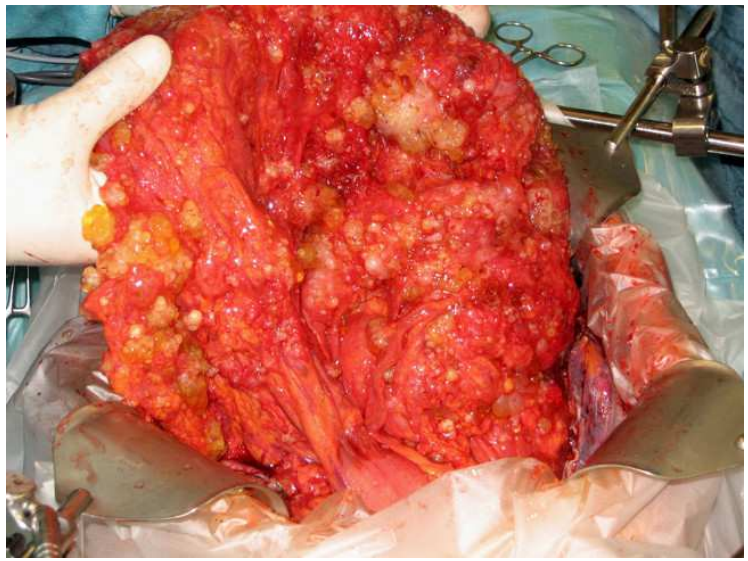


Ascites

2. DISEASED PERITONEUM

B. Malignant conditions:

- **Peritoneum Carcinomatosis**
- **Hepatocellular Carcinoma**
- Primary Mesothelioma
- Pseudomyxoma Peritonei



Ascites

2. DISEASED PERITONEUM

C. Other rare conditions:

- Granulomatous Peritonitis
- Vasculitis

Differential Diagnosis of Ascites

Cirrhosis
Alcoholic Hepatitis
Heart Failure
Cancer (peritoneal carcinomatosis, massive liver metastases, etc)
"Mixed" Ascites, i. e. Cirrhosis Plus Another Cause for Ascites
Pancreatitis
Nephrotic Syndrome
Tuberculous Peritonitis
Acute Liver Failure
Budd-Chiari Syndrome
Sinusoidal Obstruction Syndrome
Postoperative Lymphatic Leak
Myxedema

PATHOPHYSIOLOGY OF ASCITES

⬆ **HYDROSTATIC PRESSURE**

- CIRRHOSIS
- CHF
- CONSTRICTIVE PERICARDITIS

⬇ **OSMOTIC PRESSURE**

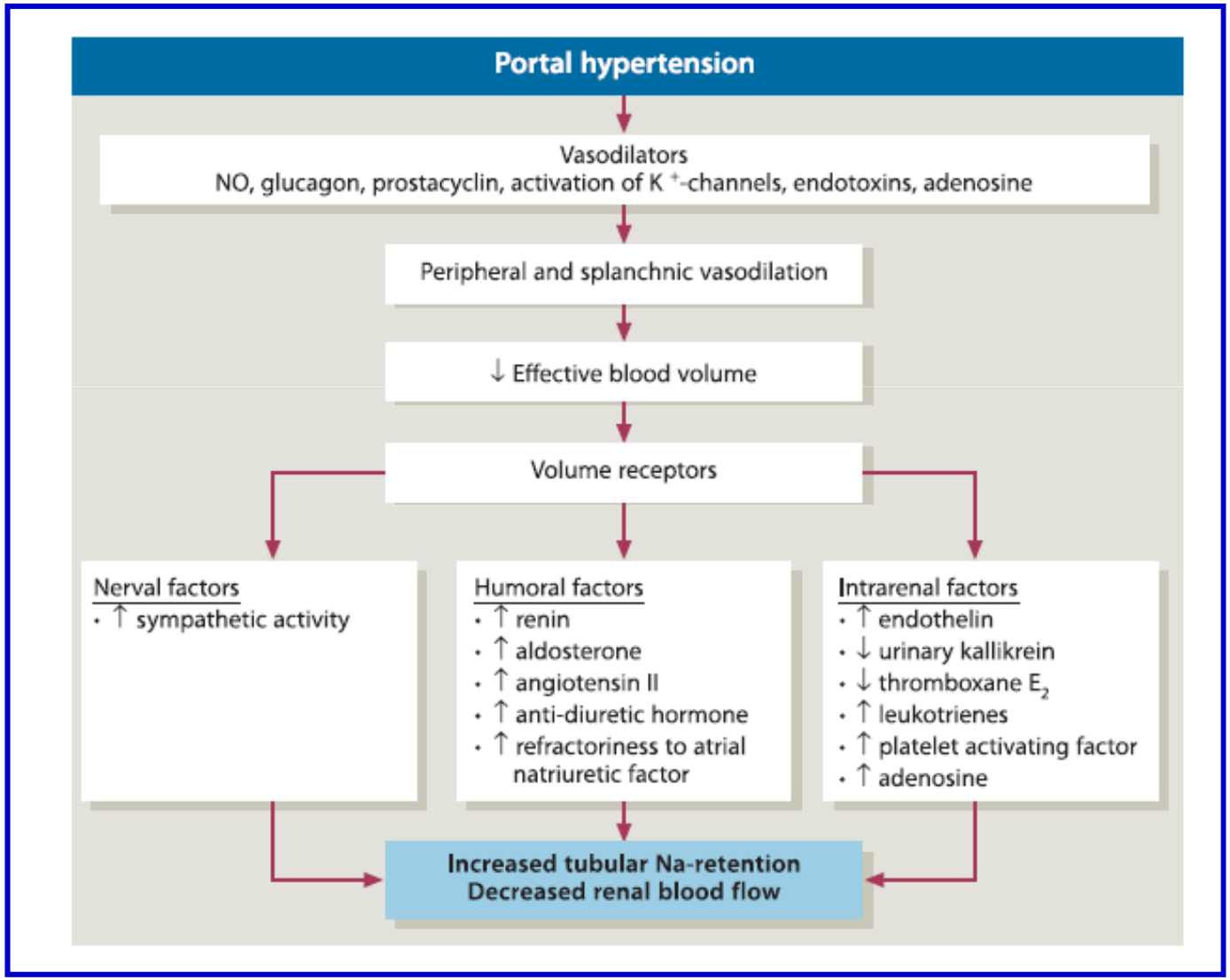
- NEPHROTIC SYNDROME
- MALNUTRITION, CIRRHOSIS
- PROTEIN LOSING ENTEROPATHY

FLUID PRODUCTION EXCEEDING RESORPTIVE CAPACITY

- INFECTIONS: TBC
- MALIGNANCY

JAMA

COMPLEX PATHOPHYSIOLOGY OF ASCITES



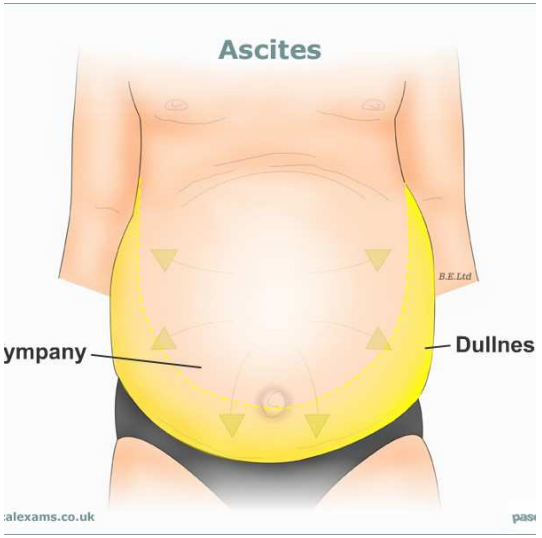
ASCITES CLINICAL FEATURES

PRESENTING COMPLAINTS:

- Abdominal distension
- Diffuse abdominal pain
- Bloated feeling of abdomen
- Dyspnea and sometimes orthopnea (due to elevation of diaphragms)
- Indigestion and heart burn (due to increased intra-abdominal pressure)

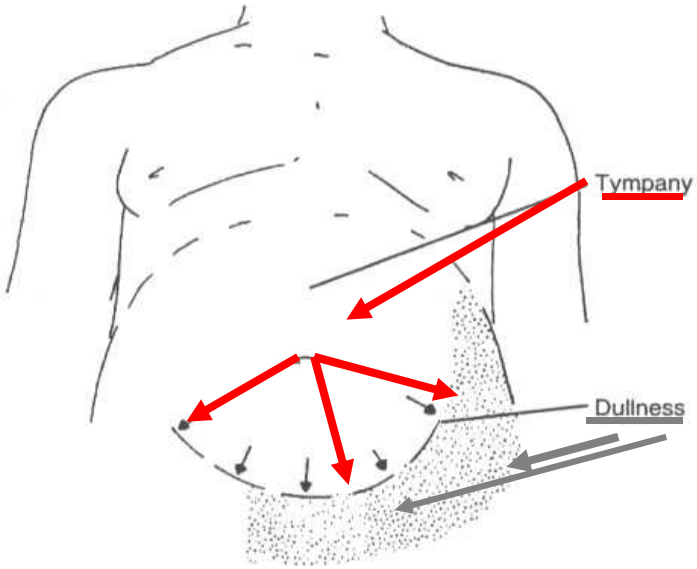
ASCITES CLINICAL FEATURES

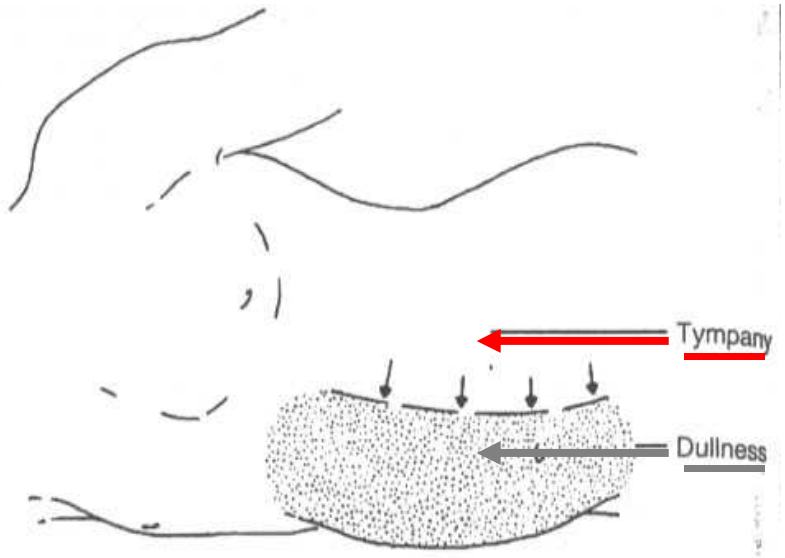
PHYSICAL EXAMINATION:



- Abdominal distension
- Fullness of flanks
- Umbilicus dilated and everted
- Diverticulation of recti muscles
- Distended abdominal veins
- Shifting dullness (esp. when >1000ml of fluid)
- Fluid thrill

SHIFTING DULLNESS





METHOD OF EXAMINATION

BEGIN BY PERCUSSING AT THE UMBILICUS AND MOVING TOWARD THE FLANKS. THE TRANSITION FROM AIR TO FLUID CAN BE IDENTIFIED WHEN THE PERCUSSION NOTE CHANGES FROM TYMPANIC TO DULL.

ROLL THE PATIENT ON THEIR SIDE AND PERCUSS AS BEFORE. THE AREA OF TYMPANY WILL SHIFT TOWARDS THE TOP AND THE AREA OF DULLNESS TOWARDS THE BOTTOM.

JAMA

ASCITES CLINICAL FEATURES

PHYSICAL EXAMINATION

- SIGNS OFTEN RELATED TO SECONDARY EFFECTS OF ASCITES:

- **Scrotal Edema**
- **Pleural effusion** (due to defect in the diaphragm and fluid pass into the pleural space - more often Right side)
- **Edema**
- **Cardiac apex shifted upward** (due to raised diaphragm)
- **Distended neck veins** (due to increased right atrial pressure)

ASCITES CLINICAL FEATURES

PHYSICAL EXAMINATION

- SIGNS RELATED TO THE CAUSE OF ASCITES:

- LIVER DISEASE:

Jaundice, Anemia, Palmar erythema, Spider angiomas,

Hepatosplenomegaly

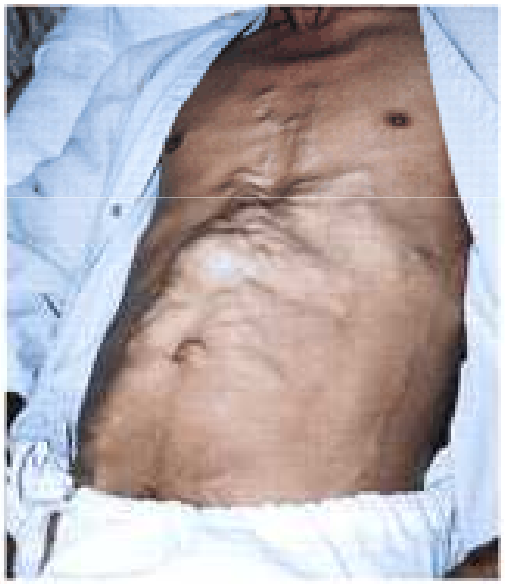
- CARDIAC DISEASE:

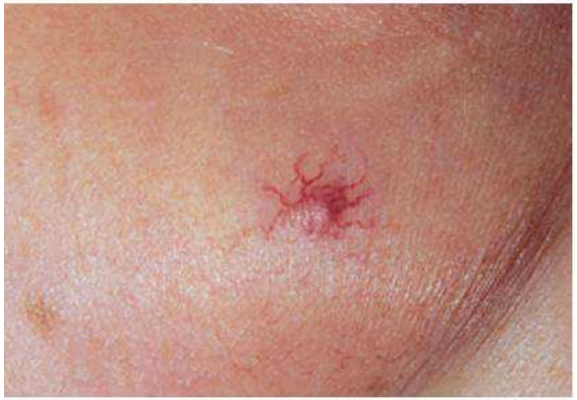
Elevated JVP

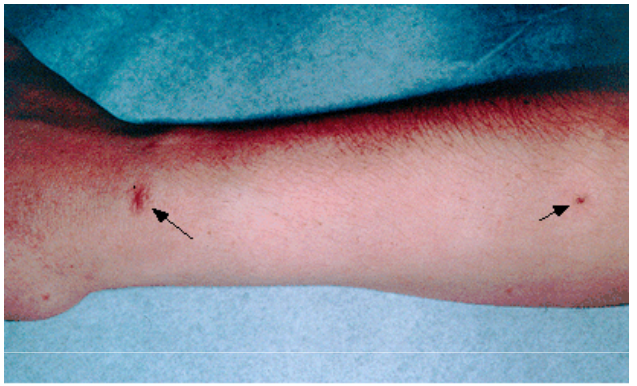
- NEPHROTIC SYNDROME:

Edema or Anasarca

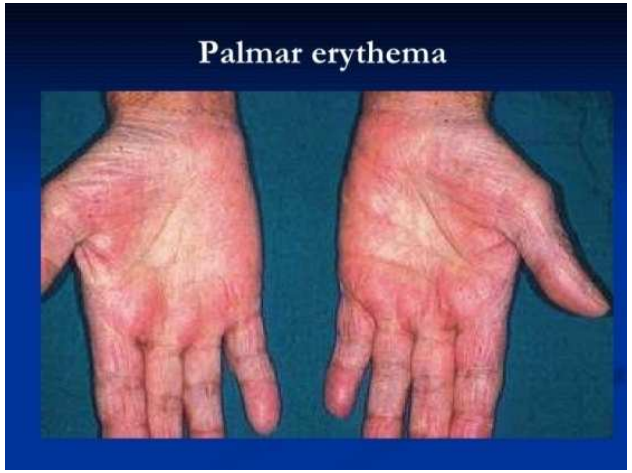
ASCITES CLINICAL FEATURES

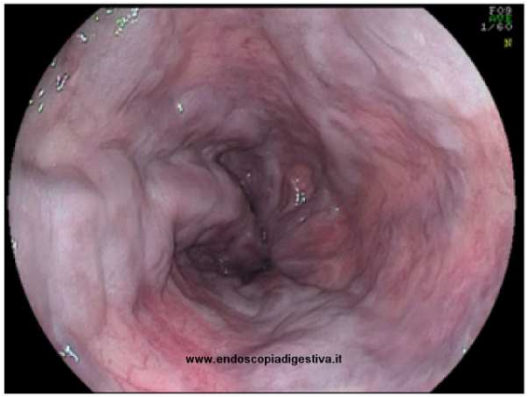




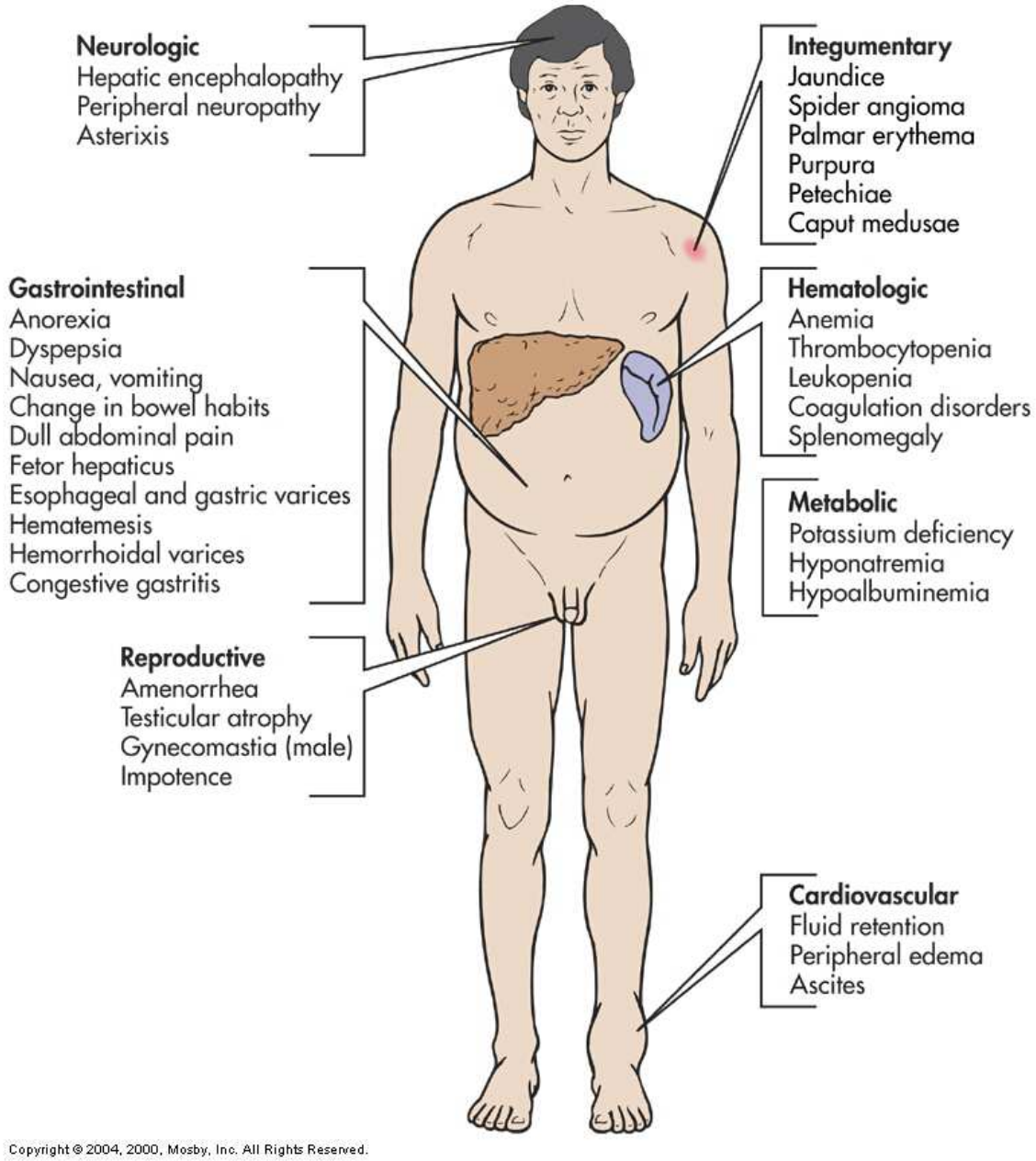


Spider angiomas This photograph shows two spider angiomas (spider telangiectasias) on the arm of a pregnant woman. A central feeding vessel, most easily seen in the lesion on the right, leads to other telangiectatic vessels, arranged in the shape of a spider, best appreciated in the lesion on the left. Pressure over the central vessel with the end of a paper clip or a glass slide causes the entire lesion to blanch. Similar lesions can be seen in patients with cirrhosis, and are most commonly seen on the upper chest, face, and back.





Manifestations of Liver Cirrhosis



Ascites: investigations

IMAGING STUDIES

CHEST AND ABDOMINAL Xray

- Detects ascites only is if >> 500ml fluid
- Elevated diaphragms
- Pleural effusion (right hepatic hydrothorax)
- Diffuse abdominal haziness

USG (ECO) of ABDOMEN

- Can detect as small as 5ml fluid
- Can identify the cause like liver cirrhosis

CT SCAN:

- Can identify the cause like malignancies

+ BASELINE INVESTIGATIONS LIKE BLOOD CP, LFTS, PT APTT

Paracentesis

Paracentesis

MANDATORY INDICATIONS:

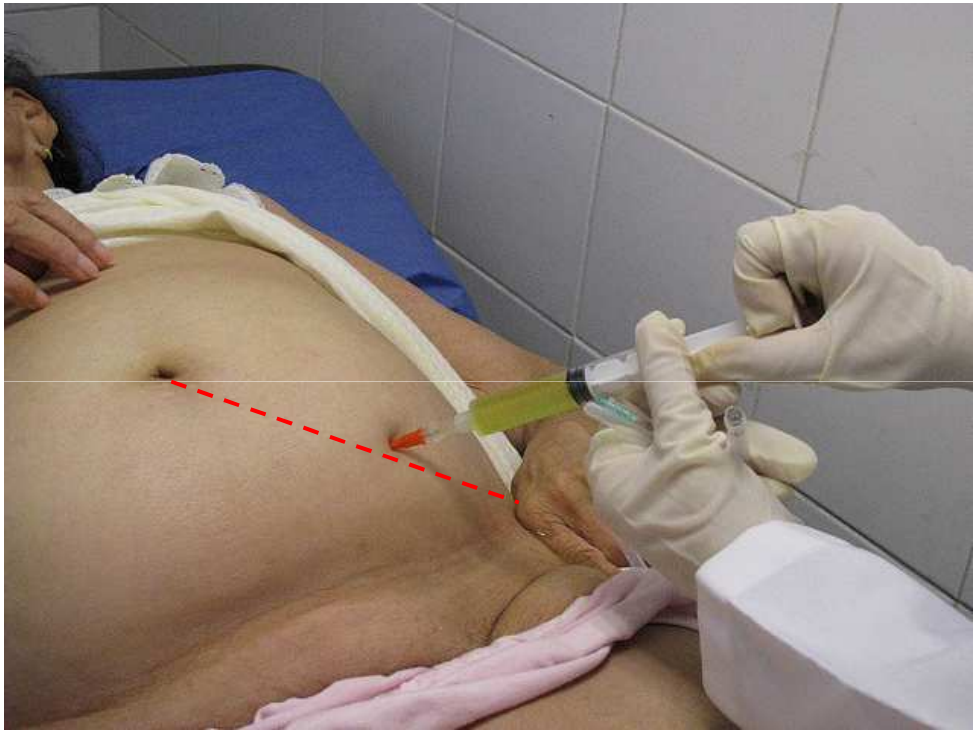
- New onset ascites !
- Surveillance at time of hospital admission
- Alleviation of discomfort / dyspnea
- If clinical deterioration: fever, pain, tenderness, mental status change, hypotension
- If suspicion of infection by labs: leukocytosis, acidosis, renal dysfunction

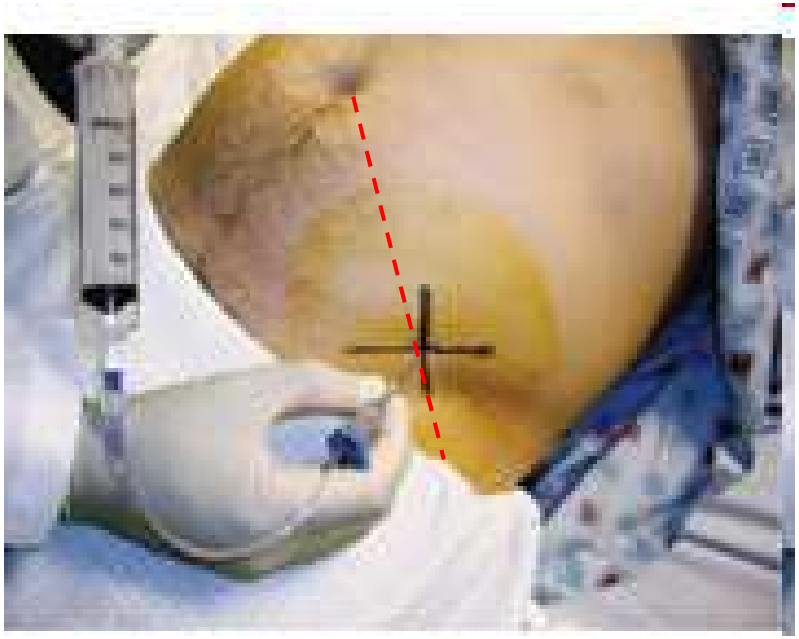
Paracentesis

ONLY FEW PRECAUTIONS:

- Severe coagulopathy or thrombocytopenia
- Pregnancy
- Organomegaly (Echo)
- Bowel obstruction
- Intra-abdominal adhesions
- Distended urinary bladder (Foley first)

Paracentesis





Recommendations

1. *Diagnostic abdominal paracentesis should be performed and ascitic fluid should be obtained from inpatients and outpatients with clinically apparent new-onset ascites. (Class I, Level C)*

2. *Since bleeding is sufficiently uncommon, the routine prophylactic use of fresh frozen plasma or platelets before paracentesis is not recommended. (Class III, Level C)*

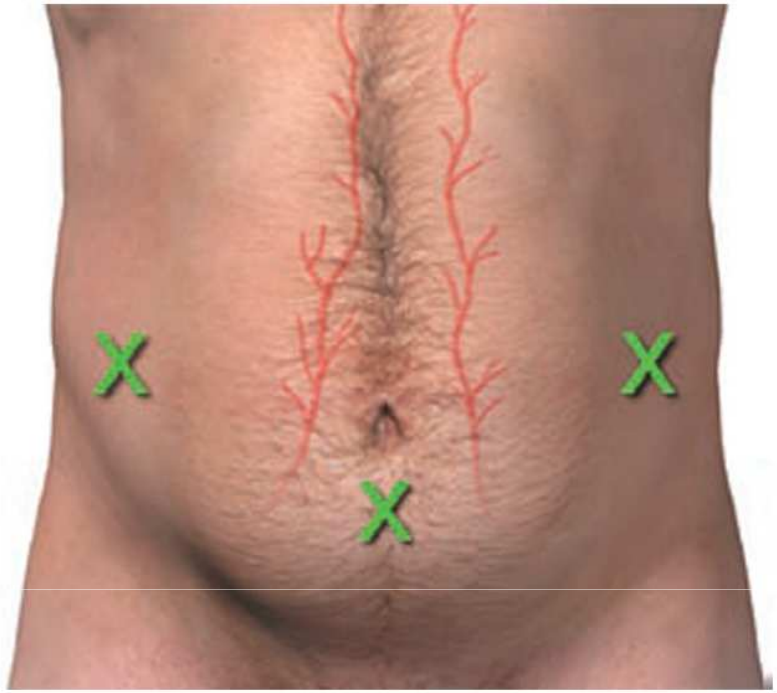


Fig. 1. Diagram of the abdomen showing the three usual sites for abdominal paracentesis. The author prefers the left lower quadrant site. Reproduced from Thomsen TW, Shaffer RW, White B, Setnik GS. Paracentesis. N Engl J Med 2006;355:e21 with permission from the Massachusetts Medical Society. Copyright (*2006) Massachusetts Medical Society. All rights reserved*.

Paracentesis

RARE COMPLICATIONS:

- Very large-volume paracentesis: circulatory dysfunction (but it is really rare)
- Persistent leakage, localized infection, abdominal wall hematoma (rare)
- Even more rare: hemorrhage (0.2% incidence), viscerous perforation, arterial puncture

Paracentesis

LAB EVALUATION:

Ascitic fluid should be always analyzed for:

- APPEARANCE
- CELL COUNT
- TOTAL PROTEINS
- **SAAG (SERUM ASCITIC ALBUMIN GRADIENT)**
- CYTOLOGY
- CULTURE

Recommendations

3. *The initial laboratory investigation of ascitic fluid should include an ascitic fluid cell count and differential, ascitic fluid total protein, and SAAG. (Class I, Level B)*

4. *If ascitic fluid infection is suspected, ascitic fluid should be cultured at the bedside in aerobic and anaerobic blood culture bottles prior to initiation of antibiotics. (Class I, Level B)*

5. *Other studies of ascitic fluid can be ordered based on the pretest probability of disease (Table 3). (Class IIa, Level C)*

6. *Testing serum for CA125 is not helpful in the differential diagnosis of ascites. Its use is not recommended in patients with ascites of any type. (Class III, Level B)*

Table 3. Ascitic Fluid Laboratory Data*

Routine	Optional (When There is Suspicion of Infection)	Unusual	Unhelpful
Cell count and differential	Culture in blood culture bottles	AFB smear and culture	pH
Albumin	Glucose	Cytology	Lactate
Total protein	Lactate dehydrogenase	Triglyceride	Cholesterol
	Amylase	Bilirubin	Fibronectin
	Gram's stain		Glycosaminoglycans

Abbreviation: AFB, acid-fast bacteria.
*Adapted from Runyon.[17] Reprinted with permission from Saunders Elsevier.

Paracentesis

LAB EVALUATION:

1. Is infection present ?

-Suspect Spontaneous Bacterial Peritonitis (SPB) if PMN elevated

- Empiric treatment with **Cefotaxime or Ceftriaxone**
- If on peritoneal dialysis: >100 WBC with >50% PMN
- Fluid cultures: 10mL per bottle

2. Is portal hypertension present ?

SAAG >/= 1.1g/dL

Paracentesis

LAB EVALUATION:

SAAG (Serum Ascitic Albumin Gradient)

The Difference between Serum Albumin and Ascitic fluid Albumin = Serum A – Ascites A

Is the best single test to differentiate between ascites due to portal hypertension and non-portal hypertension

- **When SAAG >1.1g/dl:** strongly suggest portal hypertension
- **When SAAG < 1.1g/dl:** non portal hypertensive causes

Accuracy more than 97%

Paracentesis

SAAG (Serum Ascitic Albumin Gradient)

Serum-Ascites Albumin Gradient (SAAG)	
High Gradient (≥ 1.1 g/dl)	**Low Gradient (< 1.1 g/dl)**
Cirrhosis	Peritoneal carcinomatosis
Alcoholic hepatitis	Peritoneal tuberculosis
Cardiac failure	Pancreatic ascites
Massive liver metastases	Biliary ascites
Fulminant hepatic failure	Nephrotic syndrome
Budd-Chiari syndrome	Serositis
Portal-vein thrombosis	Bowel obstruction or infarction
Veno-occlusive disease	
Fatty liver of pregnancy	
Myxedema	
"Mixed" ascites	

NEJM

Paracentesis: other test

Table 1. Additional Analyses of Ascitic Fluid.*

Test and Ascitic-Fluid Container	Comments
Tube without additives	
Total protein	Values ≥1 g/dl suggest secondary peritonitis instead of SBP
Lactate dehydrogenase	Values greater than the upper limit of normal for serum suggest secondary peritonitis instead of SBP
Glucose	Values <50 mg/dl suggest secondary peritonitis instead of SBP
Carcinoembryonic antigen	Values >5 ng/ml suggest hollow viscus perforation
Alkaline phosphatase	Values >240 U/liter suggest hollow viscus perforation
Amylase	Values markedly elevated (often >2000 U/liter or five times serum levels) in patients with pancreatic ascites or hollow viscus perforation
Triglyceride	Values >200 mg/dl suggest chylous ascites
Syringe or evacuated container	
Cytology	Sensitivity increased if three samples submitted and promptly evaluated
Mycobacterial culture	Sensitivity only 50%

* Data are from Runyon.[1,10] SBP denotes spontaneous bacterial peritonitis.

NEJM

Paracentesis: sample appearance

Causes of ascites divided according to the type of ascitic fluid

Straw-coloured
Malignancy (most common cause)
Cirrhosis
Infective
- Tuberculosis
- Following intra-abdominal perforation – any bacteria may be found (e.g. *E. coli*)
- Spontaneous in cirrhotics

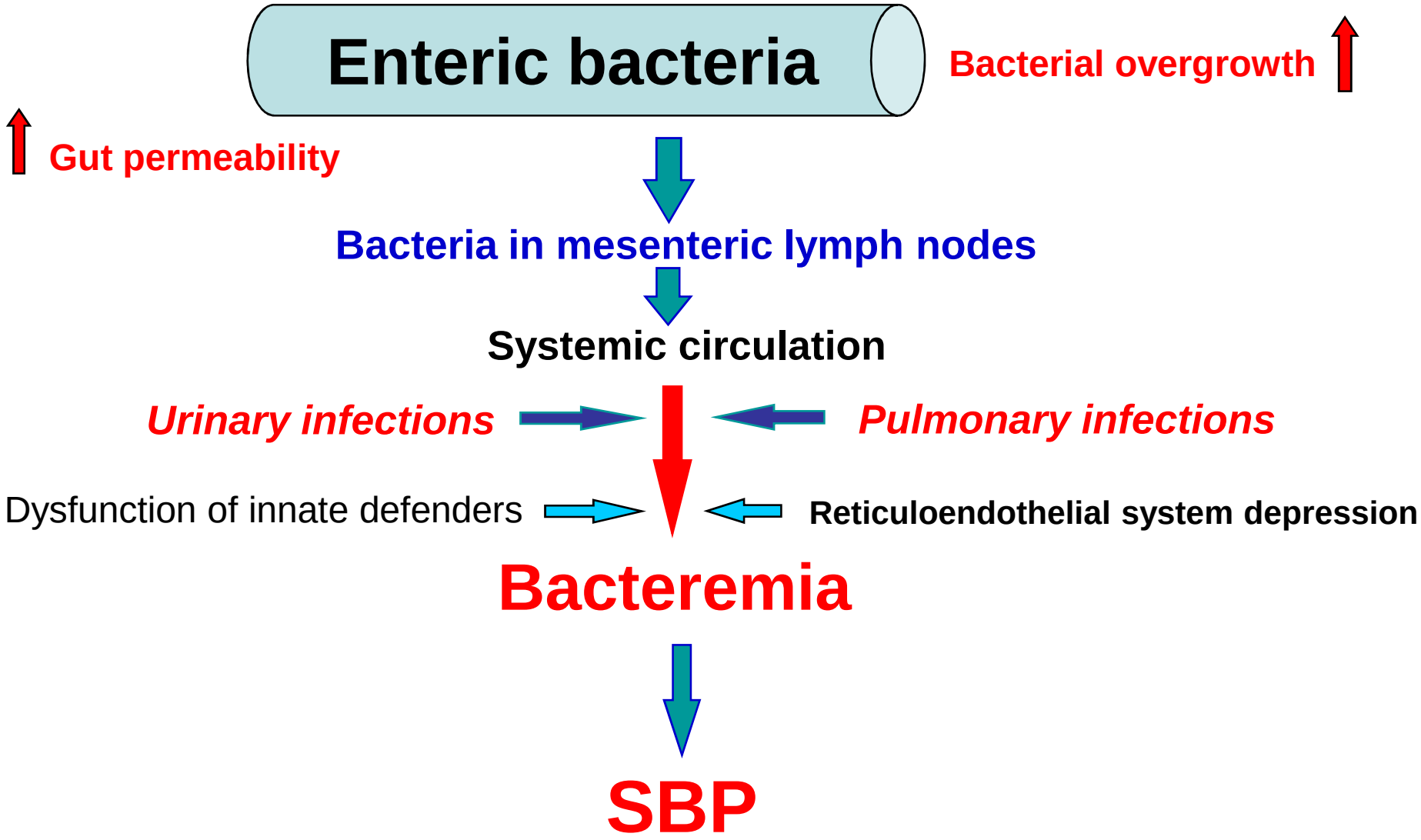
Hepatic vein obstruction (Budd–Chiari syndrome) – protein level high in fluid
Chronic pancreatitis
Congestive cardiac failure
Constrictive pericarditis
Meigs' syndrome (ovarian tumour)
Hypoproteinaemia, (e.g. nephrotic syndrome)

Chylous
Obstruction of main lymphatic duct (e.g. by carcinoma) – chylomicrons are present
Cirrhosis

Haemorrhagic
Malignancy
Ruptured ectopic pregnancy
Abdominal trauma
Acute pancreatitis

Spontaneous Bacterial Peritonitis - SPB

Infections and cirrhosis pathophysiology



Paracentesis

LAB EVALUATION: **CELL COUNT**

- **WBC < 500/mm3 and NEUTROPHILS < 250/mm3: NORMAL**

- **NEUTROPHILS > 250/microL: suggests SBP**

- **LYMPHOCYTES PREDOMINANCE:** ABDOMINAL
- TBC OR MALIGNANCY

Diagnosis of SBP

Diagnosis of SBP must be based on the PMN count

PMN > 250 /mm^3

- Gram stain positive in only a few cases (< 10 %)
- Culture into blood culture bottles positive in 50-70%

- *Main mortality predictive factor: renal function*

Follo et al. Hepatology

Treatment of SBP: antibiotic

Authors	Treatments	N	Cure %	Survival %
Félisart 85	Tobra/Ampi	36	56	61
	Cefotaxime 2g x 6	37	85 *	**73**
Rimola 95	Cefotaxime 2 g x 4	71	77	**69**
	Cefotaxime 2g bid	72	79	**79**
Navasa 96	Ofloxacin 0.4 g x 2 PO	64	84	81
	Cefotaxime 2 g x 4	69	85	81
Terg 00	Ciprofloxacin IV	40	76	77
	Ciprofloxacin 2d IV then PO	40	78	77
Sort 99	**Cefotaxime 2g x 4**	63	94	**71**
	Cefotaxime and Albumin	63	98	**90****
Ricart 00	Amox/clavu 1g tid IV then PO	24	87	87
	Cefotaxime 1g x 4	24	83	**79**

*** p<0.02 ; ** p =0.01**

Treatment of SBP: other

SBP: PMN > 250/ mm^3

- **Cefotaxime or Amoxicillin/Clavulanic acid**
- **Prevention of renal insufficiency**
  → Avoid aminoglycosides, NSAIDs, and large volume paracentesis
  → Baseline BUN elevation ⇒ **Albumin**
- Assess response to treatment (48 hours)
- Uncomplicated SBP: oral therapy with quinolones or amoxicillin-clavulanic acid

One year survival: 30 to 40%

– Secondary prophylaxis
– Evaluation for liver transplantation

Treatment of ascites in chronic liver disease

Non Diuretic therapy

- ***Renal sodium retention*** is the phenomenon primarily responsible for fluid retention and ascites formation.
- Strict ***bed rest*** is not indicated (but it will reduce plasma aldosterone anyway!)
- ***Fluid restriction*** is not strictly indicated unless serum sodium is <120 mmol/L.
- Fluid loss and weight changes are directly related to sodium balance.
- ***Dietary sodium should be restricted, usually to < 90 mmol/day.***

GUIDELINES

Guidelines on the management of ascites in cirrhosis

K P Moore, G P Aithal

Recommendation

- Dietary salt should be restricted to a no-added salt diet of 90 mmol salt/day (5.2 g salt/day). (Level of evidence: 2b; recommendation: B.)

Non Diuretic therapy

- Measurements of urinary sodium excretion is useful.
- A major goal of treatment is to increase urinary sodium excretion to ***> 80 mmol/day***.
- Only 10-15% of patients spontaneously excrete > 80 mmol/ day.

Diuretic therapy

Initial oral diuretic therapy consists of single morning doses of **Spironolactone** 100mg, or spironolactone 100mg + **Furosemide** 40 mg.

If weight loss and natriuresis are inadequate, the dose of spironolactone mono-therapy is increased to 200 mg daily, and if necessary to 3-400 mg daily, or the doses of both furosemide and spironolactone are increased simultaneously, ***maintaining the 2.5 ratio between the two doses.***

GUIDELINES

Guidelines on the management of ascites in cirrhosis

K P Moore, G P Aithal

Recommendations

- Firstline treatment of ascites should be spironolactone alone, increasing from 100 mg/day to a dose of 400 mg/day.
- If this fails to resolve ascites, frusemide should be added in a dose of up to 160 mg/day, but this should be done with careful biochemical and clinical monitoring.

(Level of evidence: 1a; recommendation: A.)

Diuretic therapy

• Spironolactone monotherapy may suffice if fluid overload is minimal and is more effective than furosemide monotherapy.

• The onset of action may not appear except after few days.

• Tolerance of spironolactone may be reduced in the presence of parenchymal renal disease.

Diuretic therapy

- Amiloride and Furosemide may be temporarily withheld if hypokalemia occurs.
- **When edema is present there is NO limit to daily weight loss.**
- When edema has resolved, maximum daily weight loss should be about **0.5kg/day** to avoid ↑ uremia due to intravascular volume depletion (pre-renal syndrome).
- Diuretic sensitive patients should not be treated with serial large-volume paracenteses.

Indications to stop diuretics

- P-S Encephalopathy
- Serum sodium <120mmol/L despite fluid restriction
- Clinically significant complications of diuretics
- Hyperkalemia and metabolic acidosis (for spironolactone)

Long-term albumin administration in decompensated cirrhosis (ANSWER): an open-label randomised trial

*Paolo Caraceni, Oliviero Riggio, Paolo Angeli, Carlo Alessandria, Sergio Neri, Francesco G Foschi, Fabio Levantesi, Aldo Airoldi, Sergio Boccia, Gianluca Svegliati-Baroni, Stefano Fagiuoli, Roberto G Romanelli, Raffaele Cozzolongo, Vito Di Marco, Vincenzo Sangiovanni, Filomena Morisco, Pierluigi Toniutto, Annalisa Tortora, Rosanna De Marco, Mario Angelico, Irene Cacciola, Gianfranco Elia, Alessandro Federico, Sara Massironi, Riccardo Guarisco, Alessandra Galioto, Giorgio Ballardini, Maria Rendina, Silvia Nardelli, Salvatore Piano, Chiara Elia, Loredana Prestianni, Federica Mirici Cappa, Lucia Cesarini, Loredana Simone, Chiara Pasquale, Marta Cavallin, Alida Andrealli, Federica Fidone, Matteo Ruggeri, Andrea Roncadori, Maurizio Baldassarre, Manuel Tufoni, Giacomo Zaccherini, Mauro Bernardi, for the ANSWER Study Investigators**

Summary

Background Evidence is scarce on the efficacy of long-term human albumin (HA) administration in patients with decompensated cirrhosis. The human Albumin for the treatmeNt of aScites in patients With hEpatic ciRrhosis (ANSWER) study was designed to clarify this issue.

Methods We did an investigator-initiated multicentre randomised, parallel, open-label, pragmatic trial in 33 academic and non-academic Italian hospitals. We randomly assigned patients with cirrhosis and uncomplicated ascites who were treated with anti-aldosteronic drugs (≥200 mg/day) and furosemide (≥25 mg/day) to receive either standard medical treatment (SMT) or SMT plus HA (40 g twice weekly for 2 weeks, and then 40 g weekly) for up to 18 months. The primary endpoint was 18-month mortality, evaluated as difference of events and analysis of survival time in patients included in the modified intention-to-treat and per-protocol populations. This study is registered with EudraCT, number 2008–000625–19, and ClinicalTrials.gov, number NCT01288794.

Findings From April 2, 2011, to May 27, 2015, 440 patients were randomly assigned and 431 were included in the modified intention-to-treat analysis. 38 of 218 patients died in the SMT plus HA group and 46 of 213 in the SMT group. Overall 18-month survival was significantly higher in the SMT plus HA than in the SMT group (Kaplan-Meier estimates 77% *vs* 66%; p=0·028), resulting in a 38% reduction in the mortality hazard ratio (0·62 [95% CI 0·40–0·95]). 46 (22%) patients in the SMT group and 49 (22%) in the SMT plus HA group had grade 3–4 non-liver related adverse events.

Interpretation In this trial, long-term HA administration prolongs overall survival and might act as a disease modifying treatment in patients with decompensated cirrhosis.

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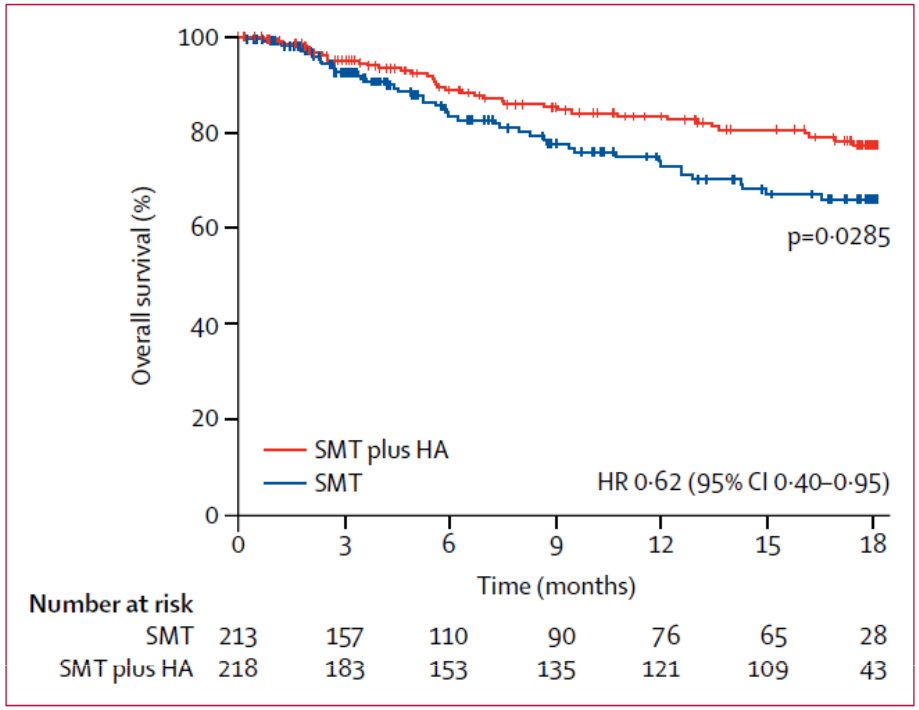

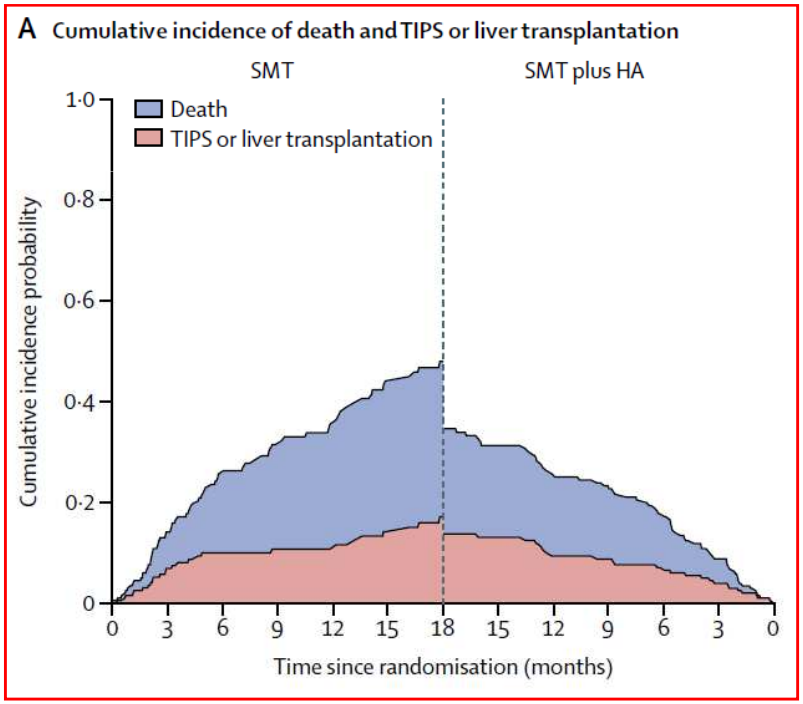

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Treatment of refractory ascites

Indications of **failure** of diuretic therapy include; 1. minimal or no weight loss, and 2. inadequate urinary sodium excretion (<80 mmol/day).

Recommendations

- Firstline treatment of ascites should be spironolactone alone, increasing from 100 mg/day to a dose of 400 mg/day.
- If this fails to resolve ascites, frusemide should be added in a dose of up to 160 mg/day, but this should be done with careful biochemical and clinical monitoring.

(Level of evidence: 1a; recommendation: A.)

Large volume paracentesis

- Large volume paracentesis is not the first line therapy
- If tense ascites is causing clinically symptoms, a single large volume paracentesis (4-8L) can be performed without affecting hemodynamics, and without necessity of concomitant colloid infusion, as initial treatment to relieve the symptoms.
- If the paracentesis is >6L, IV infusion of **Albumin** (6-8g for L of fluid removed) or **colloids** (e.g. *Emagel 125 ml for each L of fluid removed*) is recommended.
- Dietary sodium restriction and diuretic therapy are instituted.

Serial large-volume paracentesis

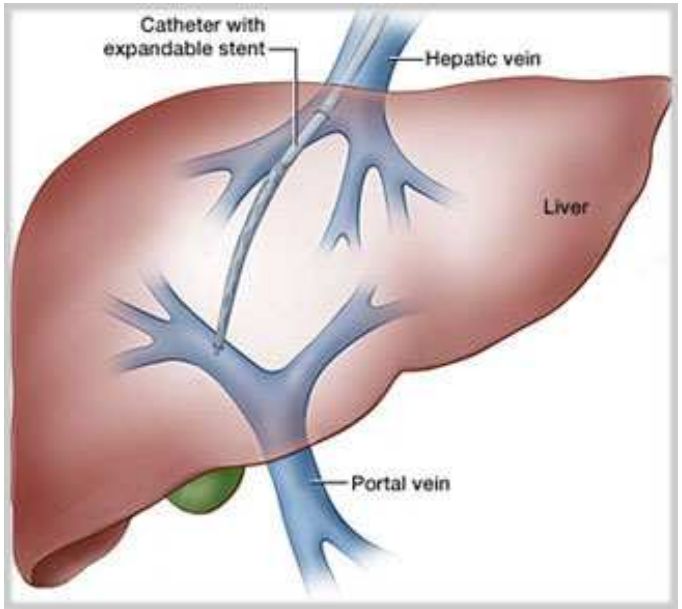
- Serial large-volume paracentesis (6-12L) is safe and effective in controlling refractory ascites.
- In patients with no urinary sodium excretion and dietary intake of ***88 mmol*** sodium daily, the required frequency is about one every 2 weeks.
- Indeed, the frequency is influenced by the degree of compliance with the low sodium diet. The sodium content of ascites fluid is about ***130 mmol/L***. Thus, a 6L paracentesis removes 780 mmol sodium (in non-urinary losses) retained over a period of about 10 days; a 10 L paracentesis removes the sodium retained over approximately 15 days (***1300 mmol***) ***(88 x 15=1320)***

Serial large-volume paracentesis

- Patients with urinary sodium excretion greater than zero should require paracenteses less frequently.
- ***Patients requiring paracentesis of 10 L more frequently than every 2 weeks are not complying with the low sodium diet!***
- IV colloid replacement is recommended immediately following a large-volume paracentesis (>6L) to minimize intravascular hypovolemia, activation of vasoconstrictor and antinatriuretic systems, and impairment of renal function.

Trans-jugular intrahepatic portal systemic stent shunt (TIPSS)

- A **TIPS** is a side-to-side portal systemic shunt placed by an interventional radiologist.
- TIPS is an effective treatment for patients with **refractory ascites.** The incidence of encephalopathy is (not necessarily) increased and survival may be better than in patients treated with serial large-volume paracenteses.
- TIPS is associated with suppression of antinatriuretic systems, and an improvement in renal response to diuretics.



Treatment Options for Patients with Cirrhosis and Ascites

First-Line

- Cessation of alcohol use, when present
- Sodium restricted diet and diet education
- Dual diuretics, usually spironolactone and furosemide, orally with single daily dosing
- Discontinue non-steroidal anti-inflammatory drugs
- Evaluation for liver transplantation

Second-Line

- Discontinue beta blockers, angiotensin converting enzyme inhibitors, and angiotensin receptor blockers
- Consider adding midodrine especially in the profoundly hypotensive patient
- Serial therapeutic paracenteses
- Evaluation for liver transplantation
- Transjugular intrahepatic portasystemic stent-shunt (TIPS)

Third-Line

- Peritoneovenous shunt