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UPDATES ON HEPATIC ASCITES

Essay
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Master Degree in Tropical Medicine

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Summary

Ascites means pathological fluid accumulation within the peritoneal cavity.

The International Ascites Club classifies ascites according to severity:

- **Grade 1 (mild):** Ascites is only detectable by ultrasound examination.
- **Grade 2 (moderate):** Ascites causing moderate symmetrical distension of the abdomen.
- **Grade 3 (large):** Ascites causing marked abdominal distension.

There are many classifications for causes of ascites depending on different bases:

I. Ascites with normal peritoneum and diseased peritoneum.
II. Transudative and exudative ascites according to ascitic fluid total protein.
III. High and low serum - ascites albumin gradient (SAAG) according to the serum - ascites albumin gradient (SAAG).

Diagnosis of hepatic ascites:

- General evaluation:

  - Complete history and physical examination
  - Standard hematology ,coagulation ,liver tests and α – fetoprotein
  - Abdominal ultrasonography and Doppler flow
  - Evaluation of renal function

- Abdominal paracentesis with appropriate ascitic fluid analysis: The most rapid and cost-effective method of diagnosing the cause of ascites, with measurement of ascitic fluid albumin or protein, ascitic fluid neutrophils count, ascitic culture and ascitic fluid amylase. Ascitic fluid cytology should be requested when there is a clinical suspicion of underlying malignancy.

Conventional therapies used for the treatment of ascites include sodium restriction (<88 mmol/d), diuretics and large volume paracentesis (LVP) (>5 L). The most effective diuretic combination is that of a potassium-sparing, distal-acting diuretic (e.g., spironolactone) with a loop diuretic (e.g., furosemide). LVP provides rapid resolution of symptoms with minimal complications and is well tolerated by most patients.

Complications of hepatic ascites:

- **Ascitic fluid infection:**

Ascitic fluid infection can be classified into five categories based on PMN count, ascitic culture results and presence or absence of a surgical source of infection:

1- Culture-negative neutrocytic ascites (CNNA)

2- Monomicrobial non-neutrocytic bacterascites (MNB)

3- Polymicrobial bacterascites

4- Secondary bacterial peritonitis

5- Spontaneous bacterial peritonitis (SBP)

The management of ascitic fluid infection is complex and not just a matter of empirical therapy. Important issues include:

1. Identification of the underlying organism
2. Choice of safe and appropriate antibiotics
3. Duration of antibiotic therapy
4. Preservation of renal function and treatment of renal dysfunction
5. Antibiotic prophylaxis

- **Hepatorenal syndrome:**

  Currently recommended diagnostic criteria:

- Presence of cirrhosis with ascites.
- Serum creatinine >1.5 mg/dL.
- Failure of serum creatinine to improve to < 1.5 mg/dL after cessation of all diuretics +volume expansion with intravenous albumin 1g/kg body weight/day up to maximum of 100 g/d for at least 48 h.
- HRS can be diagnosed in the presence of infection if shock is absent.
- No current or recent use of nephrotoxic drugs.

  No evidence of parenchymal kidney disease:

- proteinuria <500 mg/d
- RBCs in the urine sediment <50/high power field
- Normal renal ultrasonography.

Treatment of HRS include:

- General measures
- Specific measure:
  - Vasoconstrictors: Terlipressin or Noradrenaline or Midodrine plus Octreotide
  - Transjugular intrahepatic portosystemic shunts
  - Renal replacement therapy
  - Liver transplantation

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Definition and Classification of Hepatic Ascites

Definition of ascites:

Ascites means pathological fluid accumulation within the peritoneal cavity. The word "ascites" itself is derived from the Greek word "askos" which means a bag or sack ***(Reynolds, 2000).***

Ascites is defined as the presence of more than 25 ml of fluid in the peritoneal cavity. The normal hepatosplanchnic lymph production is approximately 1 ml/min. In patients with cirrhosis, this rate may increase up to 10 ml/min .When the production of lymphatic fluid exceeds the lymphatic transport capacity, ascites develops ***(Henriksen and Møller, 2005).***

Classification of ascites:

The International Ascites Club classifies ascites according to severity, complication, and response to diuretic treatment.

I. Classification of ascites according to severity:

Grade 1 (mild): Ascites is only detectable by ultrasound examination.

Grade 2 (moderate): Ascites causing moderate symmetrical distension of the abdomen.

Grade 3 (large): Ascites causing marked abdominal distension.

(Arroyo et al., 1996).

II. **Classification of ascites according to complication:** Ascites is classified into complicated and uncomplicated ascites ***(Arroyo et al., 1996).***

III. **Classification of ascites according to response to diuretic therapy:** Ascites can be classified into responding to diuretic therapy and non responding to diuretic therapy which is defined as refractory ascites.

Refractory ascites:

Ascites that cannot be mobilized or early recurrence of ascites which cannot be satisfactorily prevented by medical therapy. The term refractory ascites includes the following two subtypes:

- **Diuretic resistant ascites:** Ascites that is refractory to dietary sodium restriction and intensive diuretic treatment (spironolactone 400 mg/day and furosemide 160 mg/day for at least one week, and a salt restricted diet of less than 90 mmol/day (5.2 g of salt /day)***(Moore and Aithal, 2006).***

- **Diuretic intractable ascites:** Ascites that is refractory to therapy due to the development of complications that prevent the use of an effective diuretic dosage ***(Moore and Aithal, 2006).***

Aetiology of Hepatic Ascites

Introduction:

Many diseases can lead to the accumulation of fluid within the peritoneal cavity. The most common cause of ascites is cirrhosis, which accounts for 80% of cases; peritoneal malignancy (e.g., peritoneal metastases from GI tumors or ovarian cancer), heart failure, and peritoneal tuberculosis account for another 15% of cases ***(Hwangbo et al., 2007).***

There are many classifications for causes of ascites depending on different bases:

I. Ascites with normal peritoneum and diseased peritoneum.
II. Transudative and exudative ascites according to ascitic fluid total protein.
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I. Ascites with normal peritoneum and diseased peritoneum:

A) Causes of ascites with normal peritoneum:

1) Portal hypertension:

- Cirrhosis.
- Hepatic fibrosis.
- Congestive heart failure.
- Budd-Chiari syndrome.
- Portal vein occlusion.
- Constrictive pericarditis.

2) Hypoalbuminemia:

- Nephrotic syndrome.
- Protein energy malnutrition.
- Protein losing enteropathy.

3) Miscellaneous condition:

- Chylous ascites.
- Pancreatic ascites.
- Bile ascites.
- Nephrogenic ascites.
- Myxoedema.
- Urine ascites.

(Hwangbo et al., 2007).

B) Causes of ascites with diseased peritoneum:

1) Infection:

- Bacterial peritonitis.
- Tuberculous peritonitis.
- Fungal peritonitis.
- Parasitic diseases.

2) Malignancy.

3) Other causes:

- Familial Mediterranean fever.
- Vasculitis.
- Granulomatous peritonitis.

(Hwangbo et al., 2007).

II. **Transudative and exudative ascites according to ascitic fluid total protein:**

Before the 1980, the ascitic fluid total protein concentration was used to classify ascites as either exudative or transudative ***(Runyon, 2010).***

Low protein ascites with total protein concentration of less than 2.5 g per deciliter is called transudative ascites and usually occurs with portal hypertension or hypoalbuminaemia. A higher protein ascites with total protein concentration of more than 2.5 g per deciliter is called exudative ascites and is usually associated

with tuberculosis, malignancy, pancreatitis, etc. However, a total protein concentration of greater than 2.5 g per deciliter has recently been shown to have an accuracy of only 56 % in detecting an exudate ***(Sood, 2008).***

Unfortunately, this classification does not work well in ascitic fluid, and these terms as applied to ascitic fluid were never carefully defined or validated as this classification is problematic. The protein concentration in ascitic fluid in the setting of cirrhosis is determined almost entirely by the serum protein concentration and portal pressure. A patient with cirrhosis and a relatively high serum protein concentration will have a relatively high ascitic fluid protein concentration. Because of this relationship, almost 20% of ascitic samples in patients with cirrhosis will have a protein concentration greater than 2.5 g/dL ***(Runyon, 2010).***

Therefore, the exudate/transudate method of classification of ascites places many patients with cirrhosis and ascites and all patients with cardiac ascites in the exudates category and many patients with malignant ascites and essentially all patients with spontaneously infected ascites in the transudate category.Clearly, this method of classification is not useful ***(Runyon, 2010).***

III. High and low serum – ascites albumin gradient (SAAG) :

Classification of ascites based on the serum – ascites albumin gradient (SAAG) has replaced the exudate–transudate concept and provides a reliable tool to determine whether ascites can be attributed to portal hypertension or has another aetiology **Table(1)** ***(Saadeh and Davis , 2004).***

The SAAG is based on oncotic hydrostatic balance. Portal hypertension results in an abnormally high hydrostatic pressure gradient between the portal bed and ascitic fluid. A similarly large difference must exist between ascitic fluid and intravascular oncotic forces. Albumin exerts greater oncotic force per gram than that exerted by other proteins. Therefore, the difference between the serum and ascitic fluid albumin concentrations correlates directly with portal pressure ***(Runyon, 2010).***

The SAAG is calculated by subtracting the ascitic fluid albumin concentration from the serum albumin concentration obtained on the same day ***(Saadeh and Davis, 2004).***

If the SAAG is >1.1 g/dL, the patient has portal hypertension, with approximately 97% accuracy ***(Poonwala et al., 2000).*** Patients who have portal hypertension plus a second cause for ascites formation also have a SAAG >1.1 g/dL ***(Runyon, 2009)***.Also high gradient ascites (≥1.1 g/dL) present