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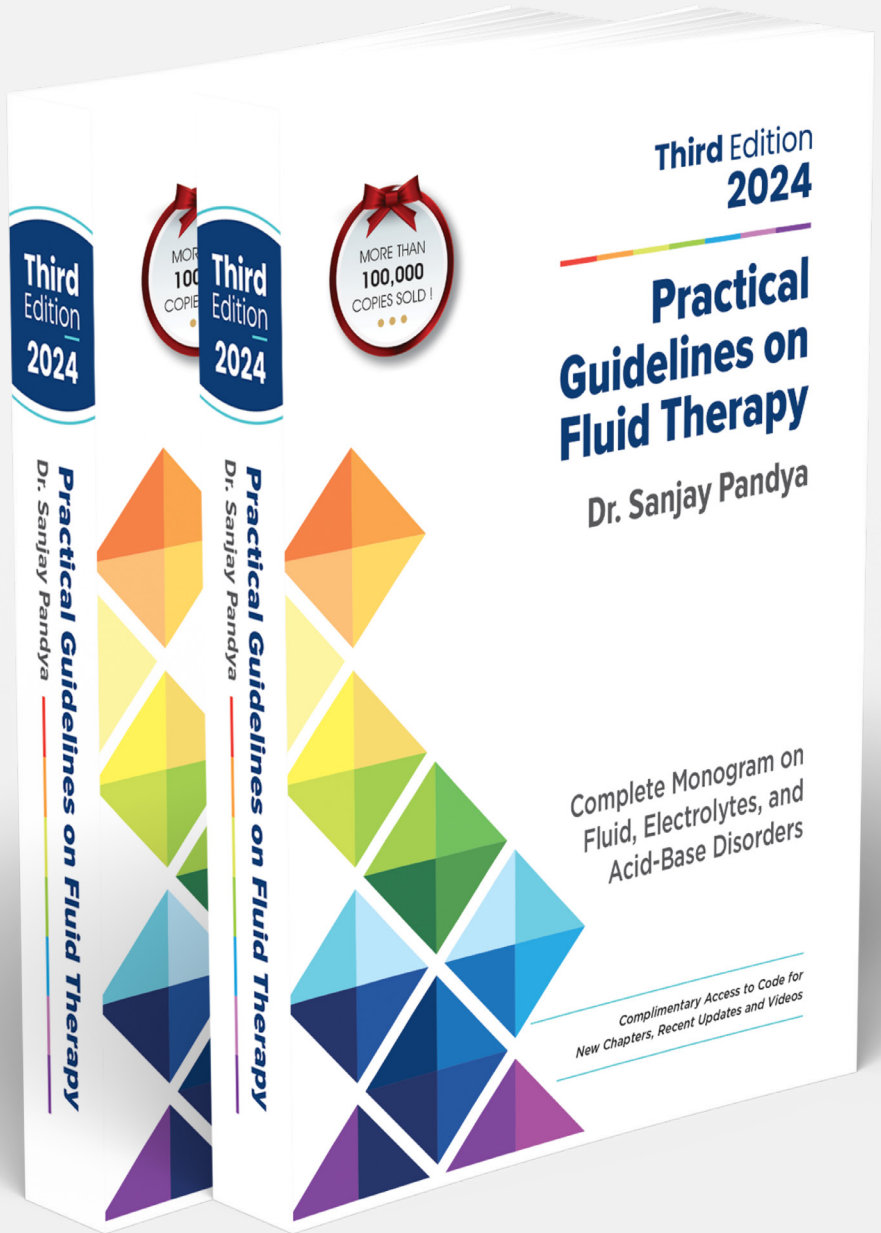
Chapter 35:
Ascites in Cirrhosis



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35 Ascites in Cirrhosis

The term ascites is defined as a pathologic fluid accumulation in the peritoneal cavity. The most common cause of ascites is decompensated cirrhosis which accounts for about 80% of the cases.

In patients with cirrhosis, ascites is the most common complication, which suggests the progression of diseases from a compensated to a decompensated stage [1]. About 60% of patients with compensated cirrhosis develop ascites within 10 years of its diagnosis [2]. Development of ascites in cirrhotic patients carries a poor prognosis as the five-year survival rate is significantly low in decompensated cirrhosis with ascites compared with compensated cirrhosis (about 30% vs. 80%) [1, 3].

Refractory ascites (RA) is defined as ascites that cannot be mobilized or recurs after large volume paracentesis (LVP) despite dietary sodium restriction and diuretic therapy. Refractory ascites is one of the most serious signs of the decompensated stage in liver cirrhosis which is associated with poor survival [4].

PATHOPHYSIOLOGY

In cirrhosis of the liver, the development of ascites is determined by four major factors: portal hypertension, splanchnic vasodilatation, renal sodium and water retention, and hypoalbuminemia.

A. Portal hypertension: The development of intrahepatic portal hypertension plays a crucial role in ascites formation. The normal value of portal pressure (pressure gradient between the hepatic and portal vein) is approximately <5 mm Hg. However, a portal pressure of usually >12 mmHg is required for ascites to develop. Without elevated portal pressure, ascites or edema do not occur.

Portal hypertension occurs in liver cirrhosis because of both increased resistance to portal blood flow (increase in intrahepatic resistance due to distortion of the hepatic vascular architecture) and increased portal venous inflow (due to splanchnic vasodilatation). This increased portal hypertension increases hydrostatic

pressure within the hepatic sinusoids, causing excessive fluid transudation into the peritoneal space, leading to ascites.

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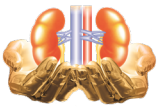
KidneyEducation 

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