Intraperitoneal collections

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Ascites Peritoneal fluid is constantly secreted and absorbed. Accumu lation of peritoneal fluid, termed ascites, occurs when there is excess production or reduced absorption. Production of large volumes of a protein-rich fluid occurs in peritonitis and car cinomatosis peritonei. Reduced absorption occurs when capillary pressure is increased as a result of generalised water retention, cardiac failure, constrictive pericarditis or vena cava obstruction. Capillary pressure is also raised selectively in the portal venous system in the Budd–Chiari syndrome, hepatic cirrhosis or extrahepatic portal venous obstruction. Plasma colloid osmotic pressure may be lowered in patients with reduced nutritional intake, diminished intestinal absorption, abnormal protein losses or defective protein synthesis, such as occurs in cirrhosis. Peritoneal lymphatic drainage may be impaired, resulting in the accumulation of protein-rich fluid. Harry H LeVeen , 1915–1997, Professor of Surgery , University of South Carolina, Columbia, SC, USA. George Budd , 1808–1882, Professor of Medicine, King’s College Hospital, London, UK. Hans Chiari , 1851–1916, Professor of Pathological Anatomy , Strasbourg, Germany (Strasbourg was returned to France in 1918 at the end of the First World War). Caput Medusa (head of Medusa) , in Greek mythology depicted as having venomous snakes instead of hair. Friedel Pick , 1867–1926, physician, Prague, the former Czechoslovakia, described this disease in 1896. Joe Vincent Meigs , 1892–1963, Professor of Gynecology , Harvard University Medical School, Boston, MA, USA. due to portal venous hypertension secondary to fibrosis of the intrahepatic venous bed. In the Budd–Chiari syndrome (see Chapter 69), thrombosis of hepatic veins leads to obstruction of venous outflow fr om the liver and hence from the mesen - teric domain in general. Alternative routes of venous drainage may open up. One such route involves the vestigial umbilical vein at the base of the falcif orm ligament. V enous drainage via this route may reach the systemic venous drainage at the umbi - licus. This is termed a portosystemic shunt and has a charac - teristic clinical appearance (involving veins) at the umbilicus (caput medusae). Congestive heart failure increases pressure in the vena cava and resistance to the venous outflow from the liver. In this set - ting, ascitic fluid is light yellow and has a low specific gravity and low protein concentration (<25 /uni00A0 g/L). In constrictive peri - carditis there is a diminished capacity of the right heart. This leads to simultaneous peritoneal and pleural e ff usions due to engorgement of the venae cavae (Pick’s disease). Ascites occurring in peritoneal metastases is due to e xces - sive exudation of fluid and lymphatic blockage. T he fluid is dark yellow and frequently blood stained. The specific grav - ity and protein content (>25 /uni00A0 g/L) are high. Rarely , ascites and pleural e ff usion are associa ted with solid fibromas of the ovary (Meigs’ syndrome). These e ff usions disappear when the tumour is excised. - Summary box 65.7 Causes of ascites /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Ascites normally becomes clinically recognisable when greater than 1.5 litres of fluid is apparent (although greater volumes may be required in obese patients). The abdomen is distended evenly with fullness of the flanks, which are dull to percussion. Usually , shifting dullness is present but, when there is a very large accumulation of fluid, this sign is absent. In such cases, flicking the abdominal wall produces a

characteristic

Transudates (protein <25 /uni00A0 g/L) Exudates (protein >25 /uni00A0 g/L) Low plasma protein Peritoneal malignancy concentrations Tuberculous peritonitis Malnutrition Budd–Chiari syndrome (hepatic vein occlusion or Nephrotic syndrome thrombosis) Protein-losing enteropathy Pancreatic ascites High central venous pressure Chylous ascites Congestive cardiac failure Meigs’ syndrome Portal hypertension Portal vein thrombosis Cirrhosis

reliable clinical sign. In women, ascites must be di ff erentiated from an enormous ovarian cyst. Investigations The aims are identification of ascites and determination of the underlying cause. Liver function tests (LFTs), cardiac function, ultrasonography and/or CT scanning (Figure 65.9) may help diagnose aetiology , e.g. carcinomatosis or liver disease. Ascitic aspiration or tap under imaging guidance helps minimise the risk of visceral injury . It can be both diagnostic and therapeutic. After the bladder has been emptied, puncture of the peritoneum is carried out under local anaesthetic using a moderately sized trocar and cannula. A peritoneal drain may be inserted at the time. In cases where the e ff usion is caused b y cardiac failure, fluid must be evacuated slowly . Fluid is sent for microscopy/cytology , culture, including mycobacteria (see Tuber culus peritonitis above), and analysis of protein content and amylase. Unless other measures are taken the fluid soon accumulates and repeated tappings remove valuable protein. Management Management aims to address any reversible primary pathol ogy (following which the ascites resolves) or symptom-based management of the ascites itself. If portal venous pressure is raised, it may be possible to lower it by tr eatment of the primary condition or by transjugular intrahepatic portosys temic shunt or transjugular intrahepatic portosystemic stent shunting (commonly abbreviated as TIPS or TIPSS). Dietary sodium restriction to 200 /uni00A0 mg/day may be helpful, but diuretics are usually required (combination of spironolac tone and furosemide). For patients failing to respond to such measures, therapeutic needle paracentesis can be performed. Serial large volume paracentesis (4–6 /uni00A0 L/day and up to 8 litres in one session) can be performed safely with colloid replace ment and can be perf ormed in patients with cirrhosis and deranged clotting. Guidelines recommend albumin replace ment after paracentesis to reduce complications. It may also be possible to leave an indwelling external drain f or smaller volume home paracentesis.

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