PERITONEAL CAVITY

The peritoneal cavity houses the abdominal viscera and is lined by a single layer of mesothelial cells; these cover the visceral and parietal surfaces and are supported by a thin layer of connective tissue to form the peritoneum. Here we discuss inflammatory, infectious, and neoplastic disorders of the peritoneal cavity and retroperitoneal space. Although they are less common than inflammatory and infectious processes, tumors can carry a grave prognosis and, thus, also deserve discussion.

Inflammatory Disease

Peritonitis may result from bacterial invasion or chemical irritation and is most often due to:

- Leakage of bile or pancreatic enzymes, which produces *sterile peritonitis*
- *Perforation or rupture of the biliary system* that evokes a highly irritating peritonitis, usually complicated by bacterial superinfection
- *Acute hemorrhagic pancreatitis* (Chapter 19), which is associated with leakage of pancreatic enzymes and fat necrosis. Damage to the bowel wall may allow bacteria to spread to the peritoneal cavity.
- *Foreign material*, including that introduced surgically (e.g., talc and sutures), that induces foreign body-type granulomas and fibrous scarring
- *Endometriosis*, which causes hemorrhage into the peritoneal cavity, where it acts as an irritant
- *Ruptured dermoid cysts* that release keratins and induce an intense granulomatous reaction
- *Perforation* of abdominal viscera.

Peritoneal Infection

Bacterial peritonitis occurs when bacteria from the gastrointestinal lumen are released into the abdominal cavity, most commonly following perforation. *E. coli*, streptococci, *S. aureus*, enterococci, and *C. perfringens* are implicated most often.

Spontaneous bacterial peritonitis develops in the absence of an obvious source of contamination. It is seen most often in patients with cirrhosis and ascites and less frequently in children with nephrotic syndrome.

MORPHOLOGY

The cellular inflammatory response is composed primarily of dense collections of neutrophils and fibrinopurulent debris that coat the viscera and abdominal wall. Serous or slightly turbid fluid begins to accumulate and becomes suppurative as infection progresses. Subhepatic and subdiaphragmatic abscesses may be formed. With the exception of tuberculous peritonitis, the reaction usually remains superficial.

Sclerosing Retroperitonitis

Sclerosing retroperitonitis, also known as idiopathic retroperitoneal fibrosis or Ormond disease, is characterized by dense fibrosis that may extend to involve the mesentery. Although the cause of sclerosing retroperitonitis is unknown, many cases are now thought to fall within the spectrum of IgG4-related sclerosing disease, an immunoinflammatory disorder that can lead to fibrosis in a wide variety of tissues. Because the process frequently compresses the ureters, this entity is described in more detail in Chapters 6 and 21.

Tumors

Primary malignant tumors arising from peritoneal lining are mesotheliomas that are similar to tumors of the pleura and pericardium. Peritoneal mesotheliomas are almost always associated with significant asbestos exposure. Rarely, primary benign and malignant soft-tissue tumors may also develop within the peritoneum and retroperitoneum. The most common of these is desmoplastic small round cell tumor. This is an aggressive tumor that occurs in children and young adults and bears resemblance to Ewing sarcoma and other small round cell tumors. It is characterized by a reciprocal translocation, t(11;22)(p13;q12) that results in the formation of a fusion gene involving *EWS* and *WT1* genes.

Secondary tumors may involve the peritoneum by direct spread or metastatic seeding, resulting in peritoneal carcinomatosis. Mucinous carcinomas, particularly those of the appendix may cause pseudomyxoma peritonei.

SUGGESTED READINGS

Congenital Abnormalities

Amin SC, Pappas C, Iyengar H, et al: Short bowel syndrome in the NICU. *Clin Perinatol* 40:53, 2013. *[A comprehensive discussion of the epidemiology and management of short bowel syndrome in neonates.]*

Kapur RP: Practical pathology and genetics of Hirschsprung's disease. *Semin Pediatr Surg* 18:212, 2009. *[A review of Hirschsprung disease etiology and diagnosis.]*

Peeters B, Benninga MA, Hennekam RC: Infantile hypertrophic pyloric stenosis–genetics and syndromes. *Nat Rev Gastroenterol Hepatol* 9:646, 2012. *[A review that focuses on disease mechanisms and clinical associations.]*

Esophagus

Esophageal Obstruction

Clarke JO, Pandolfino JE: Esophageal motor disorders: how to bridge the gap between advanced diagnostic tools and paucity of therapeutic modalities? *J Clin Gastroenterol* 46:442, 2012. *[A diagnosis-based discussion of esophageal motor disorders.]*

Richter JE, Boeckxstaens GE: Management of achalasia: surgery or pneumatic dilation. *Gut* 60:869, 2011. *[A therapy-focused discussion of the etiology, pathophysiology, and management of achalasia.]*

Esophagitis

Abonia JP, Rothenberg ME: Eosinophilic esophagitis: rapidly advancing insights. *Annu Rev Med* 63:421, 2012. *[Comprehensive review of pathobiology and treatment of eosinophilic esophagitis.]*