Review paper

Common peritoneal disorders: what the physician should know

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Abstract

Diseases affecting the peritoneum are diverse, ranging from reactive to neoplastic. The abdomen with its peritoneal cavity is part of the body in which diseases are usually thought to be diagnosed and treated almost exclusively by surgeons. However, it is often the general physician who is faced with the initial diagnosis of many of the common diseases of the region, the diagnosis of which may be very difficult. In addition, many chronic lesions that may require surgical treatment may first be seen by the general physician or gastroenterologist. For these reasons, some of the common peritoneal disorders, their presentation, and treatment are reviewed.

Anatomy and physiology

The peritoneal cavity is a mesothelial-lined closed sac, which is invaginated from behind by most of the intra-abdominal organs, which also have a peritoneal lining on at least 2 or 3 surfaces as well as on each side of the vascular pedicles. In the gut these pedicles perform a line of attachment to the back of the bowel known as the mesentery. The sac contains a small amount of fluid, which allows the organs to move freely without friction. Under normal circumstances the sac is completely empty in the male except for lubricating fluid, but in women the ovum shed at ovulation passes across the cavity to the tubes. The parietal peritoneum that lines the abdominal wall is sensitive to pain and is innervated by the somatic nervous system whereas the visceral peritoneum, which lines the intra-abdominal organs, has vague, non-specific/localized sensory innervations with pain referring to the midline origin of the gut. Visceral sensation and pain from the abdominal and pelvic organs travel back to the spinal cord along with efferent autonomic nerves. The peritoneum has a large surface (2 m^2) that is almost equivalent to the total body surface area. Its semi-permeable membrane allows rapid 2-way passive fluid transport of water and most solutes, and the lymphatics in the diaphragm actively absorb bacteria, fluids, and deformable particles as large as leucocytes. In the normal peritoneum there is rapid movement of fluids, bacteria, and leucocytes along well-defined pathways around the peritoneum, through the diaphragmatic lymphatics, to the mediastinal lymphatic, and thence to the thoracic duct. This fibrinolytic activity of the peritoneum is lost during injury, and peritoneal resistance to infection relies upon localization rather than dispersal of a contaminant. The omentum 'abdominal policeman' and the intraperitoneal viscera have a remarkable ability to confine infection as seen, for example, in acute appendicitis, perforated duodenal ulcer/diverticulum [1]. Generalised peritonitis will occur when there is failure of localization for the following reasons: a) a rapid contamination that does not permit localization, such as in a perforated colon/anastomotic leak, b) persistent or repeated contamination that overwhelms an attempt to overcome it, or c) a localized abscess that continues to expand and ruptures into the peritoneal cavity, e.g. appendix, diverticular abscess [2].

Peritonitis

Acute bacterial peritonitis

Most cases of infective peritonitis are secondary to diseases of intra-abdominal organs. Most commonly, enteric organisms enter the peritoneal sac by passing

through the wall of a diseased organ or by perforation of a hollow viscus. The commoner underlying disorders are as follows: necrosis of bowel due to obstruction, infarction, or a neoplasm; inflammatory disease such as appendicitis, diverticulitis, fulminant ulcerative colitis; and perforation of a peptic ulcer (initially sterile/chemical peritonitis) or intestinal typhoid lesion [2]. Less commonly, peritonitis may occur in the absence of an intra-abdominal lesion, i.e. primary or spontaneous bacterial peritonitis (SBP), usually through a haematogenous spread of infection [3]. Ascites in patients with cirrhosis or nephrotic syndrome are prone to primary peritonitis. *Escherichia coli* (E-coli) is the commonest infecting organism, but pneumococcus or streptococcus may be implicated. SBP also occurs in people who are on peritoneal dialysis for kidney failure, which may be due to immunological defects of the peritoneum or bacterial translocation in uraemia. Bacterial contamination of the peritoneal cavity leads to the production of an inflammatory exudate, which spreads through the peritoneum leading to paralytic ileus [2] and reflex spasm of the abdominal wall muscles. If the condition is not treated early it leads to volume depletion, shock, oliguria, and occasionally acute tubular necrosis. Many patients show the characteristic clinical picture of abdominal pain, guarding with rebound tenderness, vomiting, absent bowel sounds, tachycardia, hypotension, and pyrexia. It should be noted that these clinical findings may not always be present, particularly in the elderly in whom the abdominal findings may be minimal, and a rising pulse rate or falling blood pressure may be the only clues to the diagnosis [2, 4, 5]. Delay in diagnosis of the acute abdomen is common in infants because the classical signs and symptoms may be absent or obtainable, and perforation is common because host defences including the omentum are not fully developed [4, 5]. Similarly, patients who have been receiving corticosteroids may show very few of the classical signs of peritonitis. The peritoneum is often able to 'wall off' an inflammatory process and restrict the peritonitis area of the abdomen over the inflamed or ruptured viscus. A complicated abdominal infection extends beyond the hollow viscus of origin of the peritoneal space and is associated either with peritonitis or abscess formation. The diagnosis is usually made on the clinical findings together with the history of a preceding abdominal inflammatory disorder. An erect chest X-ray will reveal air under the diaphragm in 70% of patients, and a plain X-ray of the abdomen may show air in the peritoneal cavity [2]. Because infection, inadequate tissue perfusion, and a persistent inflammatory state are the most important risk factors for the development of multiple organ failure it seems logical that initial therapeutic efforts should be directed at their early treatment or prevention (early goal-directed therapy). The patient's general condition should be restored as rapidly as possible using intravenous fluid replacement, and fresh frozen plasma or fresh blood may be required if there is severe volume depletion on a background of leaky capillaries [6]. A nasogastric tube should be passed, and a broad-spectrum antibiotic regimen using ceftriaxone and metronidazole for anaerobes should be started. Adequate analgesia is necessary, but it should be used with care in the early stages of the disease because of the danger of masking the diagnosis. As soon as the patient is fit, surgical exploration should be carried out with appropriate source control of sepsis [7]. It is generally futile to attempt to drain an anastomosis or the general peritoneal cavity because an enterocutaneous fistula may ensue. The adhesions that occur in the healing process of the anastomosis or general peritoneal cavity will attract the peritoneal drain (foreign body), which may physically damage the anastomosis or small bowel. Also, an anastomotic leak will occur if the gaining of extra blood supply from adhesions to adjacent vascular structures is impeded [8]. The condition of Curtis-Fitz-Hugh syndrome, when transperitoneal spread of pelvic inflammatory disease produces right upper quadrant pain due to perihepatic adhesions, is now well recognized, and care must be taken to distinguish this from acute biliary conditions [9].

Tuberculous peritonitis

Unlike acute bacterial peritonitis, the symptoms of tuberculous peritonitis are usually insidious and consist of fever with night sweats, anorexia, malaise, and weight loss. In about 70% of cases there is abdominal distension due to ascites, and sometimes a mass can be palpated following an ascitic tap. The diagnosis is made by examination of the peritoneal fluid for its cellular content, biopsy of the peritoneum, or by culture of ascitic fluid or biopsy material. The most common treatment is isoniazid in combination with rifampicin, pyrazinamide, and ethambutol. Surgery is indicated if there is associated intractable intestinal obstruction or perforation [10].

Fungal and parasitic peritonitis

These rare forms of peritonitis present a similar clinical picture to tuberculous peritonitis. Fungal peritonitis usually occurs in patients who are immunosuppressed, particularly those on corticosteroids. Cases of peritonitis due to *Candida albicans* infection are successfully treated with caspofungin, micafungin, fluconazole, or amphotericin B as an alternative regimen, especially in patients with CAPD (continuous ambulatory peritone-

al dialysis), as well as 5-flurocytosine given intraperitoneally or intravenously. Cryptococcal infections have also been reported in immunosuppressed patients or patients on CAPD [11]. Peritoneal schistosomiasis has been observed occasionally, often simulating a malignant disease [12].

Starch-granulomatous peritonitis

Until the early 1950s, talc powder, used to reduce stickiness of surgical rubber gloves after sterilization, caused a granulomatous peritonitis, which sometimes resulted in adhesions, delayed healing, infection, and the formation of faecal fistulae. Since then, a combination of starch and magnesium oxide has been used for treating surgical gloves, but even this mixture can occasionally cause a chronic granulomatous peritonitis. The condition is characterized by postoperative fever, pain, and intestinal obstruction, which may persist for a considerable time. At laparotomy there may be extensive adhesions with loculate fluid cavities, and the peritoneal lining is studded with granulomata. Starch peritonitis should be treated conservatively [13].

Familial paroxysmal peritonitis

Familial paroxysmal peritonitis is a condition that occurs mainly in ethnic groups from the Mediterranean area, is part of the syndrome of familial polyserositis or familial Mediterranean fever. It presents the most urgent diagnostic problem, to be differentiated from common acute abdominal infections, acute intermittent porphyria, and relapsing pancreatitis. Prognosis as to life is generally favourable, but treatment is generally unsatisfactory [14].

Subphrenic abscess

This condition usually arises secondary to a source of infection in the abdomen, especially a perforated appendix or diverticulum, cholecystitis, perforated peptic ulcer, pancreatitis, or as an extension of a hepatic abscess. Occasionally, there may be no obvious source of infection in the abdominal cavity. The clinical picture is usually characterized by malaise, fever, pain either in the region of the abscess or referred to the shoulder tip (same phrenic nerve innervation [C3, 4, 5]), and polymorphonuclear leukocytosis. In most cases there are minimal physical signs, but there may be localized abdominal tenderness and a small pleural effusion on the affected side. Erect chest or plain X-ray of the abdomen may show gas under the diaphragm, and on screening there may be impaired movement of the diaphragm. The diagnosis may be facilitated by ultrasound examination, computed tomography (CT) scanning, or occasionally by gallium-67 scanning. Treatment is by surgical drainage together with appropriate antibiotic therapy [15].

Ascites

Several mechanisms may lead to accumulation of fluid in the peritoneal cavity. First, there may be an increase in the permeability of the peritoneal capillaries due to inflammatory or neoplastic disease of the peritoneum, or of the organs that it covers. Secondly, there may be diminished plasma osmotic colloid pressure due to hypoalbuminaemia such as occurs with the nephrotic syndrome, protein-losing enteropathy, liver disease, or malnutrition. Thirdly, ascites may develop if there is an elevation of the hydrostatic pressure in the hepatic sinusoids. This occurs in patients with cirrhosis, hepatic congestion due to heart failure or constrictive pericarditis, or with inferior vena cava or hepatic vein obstruction (Budd-Chiari syndrome). It may also result from portal vein occlusion. In patients with cirrhosis, or in other conditions in which there is an alteration in plasma volume or renal perfusion, ascites may be further aggravated by retention of sodium and water and the plasma volume is expanded. As the underlying liver disease progresses, splanchnic arteriolar vasodilatation is increased and can no longer be compensated for because the retained fluid is sequestered in the peritoneal space [16]. Finally, ascites may occur in patients with myxoedema or benign ovarian tumours (Meigs syndrome), the mechanism of which is unknown [17]. Meigs syndrome cannot be diagnosed until the patient has surgery. Hence, it is essential to rule out alternative diagnoses before contemplating abdominal surgery. The important differential diagnoses include ovarian carcinoma, cirrhosis of liver, other cancers (gastrointestinal, lung), congestive heart failure, nephritic syndrome, tuberculosis, pseudo Meigs syndrome (ascites and pleural effusion in patient with pelvic or abdominal benign or malignant tumour), pseudo-pseudo-Meigs syndrome (ascites, pleural effusion, elevated serum CA125 in a patient with systemic lupus erythematosus). Meigs syndrome is a benign condition, and early detection and intervention result in a good prognosis with permanent resolution of the ascites and pleural effusion [17]. Ascitic fluid is usually classified as either being an ‘exudate’, i.e. with a protein composition of 2.5 g/dl or more, or as a ‘transudate’ with a lower protein content. In fact, this is not useful in 56% of patients [16]. Occasionally, ‘transudative conditions’ such as cirrhosis and congestive cardiac failure are associated with relatively high protein levels. Similarly, malignant ascites may sometimes have a low amount of protein. A better classification measures the ascitic protein level and subtracts it from the serum albumin

level, resulting in the serum-ascites gradient (transudate is > 1.1 g/dl; exudates is < 1.1 g/dl) [16]. Based on this measurement, Runyon has classified the causes of ascites as follows in Tables I and II. Ascitic fluid is characterized by an increase in abdominal girth associated with shifting dullness and fluid thrill. A plain abdominal X-ray may show an opaque appearance. The diagnosis is confirmed by paracentesis in which the protein concentration and cellular composition of the fluid is analysed. A blood-stained fluid usually indicates neoplastic disease, often confirmed by cytological examination. A high polymorphonuclear leukocyte (PMN) count usually reflects an underlying inflammatory disease, although it is found occasionally in malignant disease. A high lymphocyte count is suggestive of tuberculous peritonitis. The fluid should always be cultured for routine organisms and, when appropriate, for acid-fast bacillaceae. An elevated amylase level is indicative of pancreatic disease. A reduced sugar level (< 60 mg/100 ml) occurs quite commonly in neoplastic disease. Malignant effusions may be controlled temporarily by the use of intraperitoneal antitumour agents but peritoneal carcinomatosis is amenable to hyperthermic intraperitoneal chemotherapy (HIPEC) [18]. Ascites from other causes is managed by treating the underlying disease. Refractory ascites is defined as the group of patients who fail to achieve negative sodium balance or develop complications of medical therapy. This constitutes 10% of all cases of ascites, and the treatment options include repeated therapeutic paracentesis, peritoneovenous shunting, transjugular intrahepatic portosystemic shunt (TIPS), extracorporeal ultrafiltration of ascitic fluid with reinfusion, and liver transplantation [16].

Chylous ascites is the condition that occurs due to the presence of lymph lipoproteins and chylomicrons in the peritoneal cavity. It is diagnosed by finding ascites characterized by a white, turbid fluid on aspiration. Because not all turbid fluid is chylous, the diagnosis must be confirmed by analysis of the fluid for neutral fat. It must be distinguished from pseudochylous ascites, in which there is a turbid fluid due to the presence of cell debris; this is usually associated with neoplasia [19]. In adults, chylous ascites are usually caused by lymphatic obstruction due to lymphoma. An acute form has been described after trauma or rupture of a chylous cyst. In children, the condition may be associated with intestinal lymphangiectasia resulting in lymph leakage into the small bowel lumen and consequent protein-losing enteropathy leading to lymphopaenia, hypoalbuminaemia, and hypogammaglobulinaemia. Recurrent chylous ascites together with chylous pleural effusions occur in lymphangioleiomyomatosis, a rare, slowly progressive, low-grade, metastasizing neoplasm affecting young females. The condition is diagnosed by exclusion of other chylous effusions and by characteristic biopsy and lymphangiogram appearances. There are anecdotal reports suggesting the condition may respond to oophorectomy and anti-oestrogen treatment (tamoxifen) [20]. Urine ascites may occur as urine accumulating in the peritoneal cavity after trauma or in infants with congenital obstructive lesions of the urinary tract [21].

Tumours of the peritoneum

Most tumours in the peritoneal cavity are due to secondary metastases from carcinomas arising in the

Table I. Ruyon's classification of causes of ascites

Related to portal hypertension (gradient > 1.1 g/dl)	Unrelated to portal hypertension (gradient < 1.1 g/dl)
Cirrhosis	Peritoneal carcinomatosis
Alcoholic hepatitis	TB
Cardiac ascites	Pancreatic ascites
Massive liver metastaseis	Biliary ascites
Fulminant hepatic failure	Nephrotic syndrome
Budd-Chiari syndrome	Serositis in connective tissue diseases
Portal vein thrombosis	
Veno-occlusive disease	
Fatty liver of pregnancy	
Myxoedema	

Table II. Main ascitic fluid findings following a diagnostic paracentesis

Condition	Albumin [g/dl]	WBCs/ml	RBCs/ml	Ascites-albumin gradient [g/dl]
Cirrhosis	< 2.5	300–500	< 1000	> 1.1
Hepatoma	< 2.5	300–500	1000–50000	> 1.1
Carcinomatosis	> 2.5	Variable	Usually < 1000	< 1.1
Cardiogenic	> 2.5–3.0	300–500	< 1000	> 1.1

breast, the lung, the colon, the stomach, the pancreas, the uterus, the ovary, and the liver. It is a late-stage manifestation and often leaves patients with only palliative treatment. For colorectal peritoneal carcinomatosis, the median survival is approximately 5 months, and palliative systemic therapy may extend this to approximately 12 months. The median survival with cytoreduction surgery with hyperthermic intraperitoneal chemotherapy (HIPEC) has been reported to range from 22 to 63 months with a 5-year survival of 40–51% in selected patients with limited tumour burden [18]. Rarely a primary malignant tumour may occur. The commonest of these is a primary mesothelioma [22]. It is an interesting tumour, which may occur in the pleura or peritoneal cavity. Most cases involve males working in the textile industry, as brake-liners in the motor car industry, or as insulators in the building trade, and on board ships where asbestos was in the past widely used. If the patient has been exposed to the asbestos for a considerable length of time, in 60–80% of cases there will be evidence of underlying pulmonary asbestosis, 17% have the tumour in the pleura, and 30% develop primary peritoneal mesotheliomata. Only 23% of women with previous asbestos exposure will develop the disease, and younger patients have been found to occasionally develop the disease with no history of exposure. Malignant peritoneal mesothelioma commonly presents with diffuse, extensive spread throughout the abdomen, with rare metastatic spread beyond the abdominal cavity. However, due to its rarity and nonspecific symptoms, it is usually diagnosed late when the disease burden is extensive. The distinction between malignant mesothelioma and other malignant neoplasia diffusely involving the peritoneum is important for proper patient treatment. Extra-ovarian peritoneal serous papillary carcinoma is a rare, primary, multicentric peritoneal tumour that is morphologically identical to ovarian serous carcinoma of equivalent grade but can spare or minimally involve the ovaries. The amount of residual disease is an important prognostic determining factor in primary papillary serous carcinoma of the peritoneum because there was good outcome following debulking to no macroscopic disease and platin-based chemotherapy [23]. Peritoneal serous papillary carcinoma is a rare primary tumour of the peritoneum. It is associated with high protein ascites and lower abdominal pain. Macroscopically, the parietal and visceral peritoneum is studded with prominent white nodules up to 5 mm in diameter, and multiple biopsies will reveal a tubulopapillary serous adenocarcinoma that responds to platin-based chemotherapy. The diagnosis is established after exclusion of metastatic peritoneal carcinomatosis (especially ovarian cancer) and malignant mesothelioma [24]. Retroperitoneal lymph nodes are a common site of metastases in primary peritoneal carcinoma (PPC). Pelvic nodes are involved in 53% of cases, while para-aortic nodes are involved in 24% of cases. Nodal involvements are poor prognostic factors with 5-year overall survival of 63% vs. 25% in node-positive vs. -negative cases. However, patients with primary ovarian cancer have a higher rate of positive para-aortic nodes (58%) [24]. The rationale for pelvic (PND) or para-aortic (PAND) lymphadenectomy is oncologically logical for locoregional control, optimal cytoreduction in producing microscopic residual disease and removing grossly positive nodes in patients with residual disease of less than 1 cm. Patients with residual disease after primary surgery with lymphadenectomy have a median survival of 5.8 years with microscopic disease, 3.2 years with < 1 cm macroscopic residual disease, and 1.3 years with > 1 cm macroscopic residual disease [25]. The rare condition of ***Pseudomyxoma peritonei*** is caused by the rupture of a cystadenocarcinoma of the appendix or colon with seeding of these mucin-producing cells across the peritoneal surface. Clinically, the condition presents with enlargement of the abdomen, which is found to contain the characteristic fluid. Even in female patients with synchronous appendiceal and ovarian mucinous tumours, it appears that the appendiceal tumour is often the primary source of the malignancy. An important challenge may be the distinction of localized, acellular collections of mucin (which can be observed in primary ovarian mucinous neoplasms) from disseminated mucin deposits containing mucinous epithelium (more in keeping with an appendiceal lesion). Thus, extensive sampling, frozen section, immunohistochemical work-up, and correlation with clinical findings are important. The surgeon should be aware of the importance of resecting the appendix even if it is grossly normal in these situations. Patients with pseudomyxoma peritonei benefit from debulking procedures, and recent works show the benefits of intraperitoneal chemotherapy. Peritoneal dissemination from appendiceal tumours usually occurs in the absence of lymph nodes, and liver metastases and appendix cancer is usually of low biologic aggressiveness with minimal invasiveness and thus greater penetration by intraperitoneal chemotherapy. A few cases secondary to ovarian teratoma or fibromata have been recorded. Although carcinoid tumours often arise from the bowel and from the neuroendocrine cells within Meckel's diverticulum, they can also originate from the mesentery and peritoneum. However, peritoneal carcinomatosis from carcinoid tumours is uncommon because the tumours are of the non-epithelial type. Therefore, unlike colon and ovarian tumours and mesothelioma, carcinoid tumours with isolated peritoneal recurrences are uncommon.

Retroperitoneal fibrosis

Since the report of Ormond (1948) [26], there have been various aspects of this interesting condition, which consists of a marked fibrosis in the tissue of the retroperitoneal space. It follows an initial non-suppurative inflammation characterized by infiltration of lymphocytes, mononuclear cells, and plasma cells. Later collagen is deposited in association with a marked vascular change, and finally the fibrosis becomes massive with dense vascular collagen deposition. Considerable fibrosis may be associated with radiotherapy, tumours (particularly carcinoids), chronic infection, leakage of intestinal contents, urine, or blood, chronic lymphangitis, and trauma. The most interesting relationship is with the administration of drugs such as methysergide and β-adrenoreceptor antagonists. The disease can occur at any age but most commonly during middle age. Pain occurs commonly in the early stages followed by anorexia, fatigue, malaise, loss of weight, and occasionally oedema. In the late stages the symptoms result from obstruction of the urinary tract. Less commonly, fibrosis may involve the duodenum, small intestine, or colon, and gastrointestinal symptoms may dominate the clinical picture due to obstruction. More frequently, the gastrointestinal symptoms caused by uraemia and hypertension from obstructive uropathy may be severe. The possibility of the disease should be kept in mind when abdominal symptoms and weight loss are associated with a raised erythrocyte sedimentation rate (ESR). Confirmation is usually made after investigation of the renal tract radiologically or at operation when biopsies should be taken to exclude malignant fibrosis. Treatment is surgical and is aimed at relieving the obstruction of the renal vessels, urinary tract, intestines, or peripheral arteries, which are rarely involved. Corticosteroids are often used post-operatively after uterolysis in an attempt to prevent further fibrosis [26, 27].

Retroperitoneal sarcomas

Retroperitoneal sarcomas are a rare type of malignancy, accounting for 15–20% of all soft-tissue sarcomas and about 1–2% of all solid malignancies. They arise in mesenchymal tissues such as fat (liposarcoma), muscle (leiomyosarcoma), blood vessels, nerve sheaths, and fibrous tissue. The peak incidence is in the 5th decade of life, and the risk factors include previous radiation, inherited genetic disorders such as naevoid basal cell carcinoma syndrome (Gorlin syndrome,) familial adenoposis syndrome (Gardner's syndrome), Li-Fraumeni syndrome, tuberous sclerosis (Bourneville disease), and neurofibromatosis type 1 (von Recklinghausen's disease). Being located in the retroperitoneal space, they grow indolently to a massive size with potential involvement of adjacent organs, which may limit surgical or other therapeutic interventions. The exclusion of pathologies arising from all the retroperitoneal organs and vascular structures will lead to its diagnosis. Needle tumour biopsy is indicated to elucidate the histological type of tumour and thus the appropriate treatment. Surgical excision with negative margins (Ro) is the only chance of potential cure, and it increases the overall survival. A multidisciplinary team management is important. Because these tumours usually recur locally and do not usually metastasize, there is benefit from neo/adjuvant radiotherapy. Retroperitoneal liposarcoma frequently recurs within 2 years of the initial surgical resection. Thus, a shorter follow-up interval with CT scan or magnetic resonance imaging (MRI) would be helpful in early detection of recurrence. For metastases, systemic anticancer therapy may be guided by the sarcoma subtype [28, 29].

Diseases of the omentum

Lesions arising primarily in the omentum are rare, although the organ is frequently involved with inflammatory or neoplastic disease in the abdominal cavity. Metastatic tumours and cysts may occasionally be the cause of abdominal masses. A rare but interesting lesion is the idiopathic segmental infarction of the omentum. The condition may occur in children who develop a progressive peritoneal irritation usually misdiagnosed as appendicitis, but it has been reported at any age. Characteristic laparotomy findings are of wedge-shaped infarction along the right inferior border of the greater omentum. The cause remains obscure, but the condition may be associated with torsion of the omentum. CT is the most commonly used imaging modality in the assessment of omental or peritoneal disease, and optimal bowel contrast opacification is critical [30].

Conclusions

Disorders of the peritoneum are fairly common. They include peritonitis, cancer, and complications from peritoneal dialysis, for which the understanding of the pathophysiological mechanisms are important in the management. Secondary (metastatic) involvement of the peritoneum is common among malignant lesions and should always be considered and included in the differential of primary Mullerian or mesothelial tumours. When encountering peritoneal disease, a detailed patient history, risk factors, laboratory studies, and radiographic evaluation will aid in the diagnosis.

Conflict of interest

The author declares no conflict of interest.

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