NEUTROPENIC COLITIS

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Key-word: Colitis

Background: A 66-year-old-woman, undergoing chemotherapy for recently diagnosed acute myeloid leukemia developed increasing abdominal pain over a period of several days. Laboratory results showed, apart from severe neutropenia, increasing infection parameters.

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Work-up

Abdominal ultrasonography (Fig. 1) shows enlargement of the wall of the caecum with massively thickened and echogenic mucosa. There is very little peristalsis of the terminal ileum. A little pocket of free fluid is detected at the bottom of the caecum.

Contrast-enhanced CT of the abdomen (Fig. 2) demonstrates pathologically thickened colonic wall, extending from the caecal base until the mid portion of the transverse colon, clearly showing the, extent of the affected bowel. The caecal region of the colonic wall shows diminished contrast enhancement, indicative of ischemia.

Follow-up CT scan (Fig. 3) shows progression to necrosis, despite enforced medical treatment. Surgical intervention with resection of the affected intestinal segments was performed (Fig. 4).

Radiological diagnosis

In the setting of the pronounced neutropenic status of the patient and the suggestive imaging findings the diagnosis of neutropenic colitis (typhlitis) was made.

Discussion

Neutropenic enterocolitis (typhlitis) is an acute and life-threatening complication in the immune suppressed patient. It is an active, transmural inflammation, starting from the caecum and rapidly affecting the ascending colon and the terminal loops of the ileum. Due to the rapid course of the disease and the potentially dramatic outcome early recognition is of paramount importance.

With increased survival of patients with myeloproliferative disease, due to recent developments in immunosuppressive drug treatment and the introduction of bone marrow transplantation, complications related to decreased immunity have become more frequent.

Although infrequently encountered in general practice, incidence rates of up to 33% have been reported in pediatric patients with leukemia. Although the exact pathophysiology of neutropenic enterocolitis remains to be elucidated, neutropenia seems to play a crucial role. A possible etiological factor is mucosal degradation of the bowel, creating an entry point for bacteria. Caecal distention and altered bowel flora, caused by immunosuppressive and antibiotic agents, have also been proposed as possible factors. Prolonged mucosal and submucosal inflammation will progress to ischemia and bowel wall necrosis with resulting perforation and peritonitis.

In the differential diagnosis one should consider acute appendicitis, bowel ischemia, bacterial gastroenteritis and pseudomembranous colitis.

The clinical symptoms usually start 10-14 days after the start of immune suppression and mimic acute appendicitis. The patient complains of an intense, crampy abdominal pain, located in the right lower quadrant sometimes accompanied by nausea, vomiting and diarrhea. On physical examination the abdomen is distended and tender, especially in the right iliac fossa. There can be paucity of bowel sounds. If signs of shock are present the possibility of bacteraemia and sepsis must be considered.

Since plain radiography of the abdomen will at best show aspecific signs, the radiological work-up starts with ultrasonography of the abdomen. Ultrasonography is helpful in the diagnosis as well as in the follow-up, bowel wall thickening being the important sign. Wall thickness greater than 5 mm is associated with a significant mortality rate. A particularly ominous sign is a wall thickness greater than 10 mm, which has a reported mortality of 60%.

Contrast-enhanced CT is the golden standard for the detection of typhlitis. Bowel wall thickening, stranding of the mesentery and free fluid are signs of local inflammation. As progression occurs, enhancement of the bowel wall mucosa and a hypodense submucosa will indicate transmural inflammation. Finally necrosis follows, indicated by pneumatosis coli and absence of contrast enhancement. Free abdominal air (perforation) and portal venous gas are signs of advanced disease.

To our knowledge there is no universally accepted treatment protocol for neutropenic enterocolitis.

Both conservative medical treatment and surgery are to be considered, depending on the specific conditions. Conservative therapy includes bowel rest, broad spectrum antibiotics, close clinical monitoring with serial laboratory work-up and abdominal ultrasonography. Surgical intervention is indicated in case of clinical deterioration, onset of clinical signs of acute abdomen or radiological signs of perforation and/or abscess formation.

Since normal-appearing serosal surfaces may conceal mucosal breakdown and necrosis, wide resection margins are recommended.

Bibliography

