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Case Report

Acute necrotizing enterocolitis due to sigmoid colon cancer: a rare entity case report

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ABSTRACT

Adult necrotizing enterocolitis and non-occlusive mesenteric ischemia are rare causes of acute abdomen. Necrotising enterocolitis usually affects children and only few cases have been described in adults especially in geriatric patient. We report a rare case of acute necrotizing colitis of a 61 years old male patient. X-ray abdomen led to bowel dilatation. We performed emergency laparotomy under general anaesthesia and revealed that the dilatation of intestines and colon was caused by obstructive colitis due to sigmoid tumour. The digestive system was expanded proximally and the mucosae was seen turning black from the sigmoid colon to 80 cm proximal to the terminal ileum, from the findings we concluded that the patient was suffering from acute necrosis without significant vessel occlusion. The occlusion from the tumour may increase intraluminal pressure that affect colonic blood flow, and in closed-loop colonic obstruction without perforation, mucosal ischemia may be the primary event leading to massive colonic gangrene. Necrotising enterocolitis usually affects children and only few cases have been described in adults especially in geriatric patient. We performed complete colectomy with 90 cm resection of terminal ileum and ileostomy to remove the necrotic colon during surgery. Hartmann procedure was performed to distal part of rectum. The surgery was successful and patient was stable after the surgery.

Keywords: Acute necrotizing enterocolitis, Sigmoid colon cancer, Obstructed colon cancer

INTRODUCTION

Colonic gangrene is one of the most dangerous complications of large bowel obstruction requiring emergency surgery. Until now, only a small number of studies have reported cases of acute necrotizing colitis associated with colon cancer. Almost all cases of necrotizing enterocolitis occur in the paediatric population. Only a few cases of necrotizing enterocolitis have been found in the adult population. Therefore, acute abdomen caused by necrotizing enterocolitis and non-occlusive mesenteric ischemia in adults is extremely rare. Establishing a preoperative diagnosis of this case is often

difficult. The diagnosis is made by finding diffuse ulceration and necrosis of the distal small intestine and large intestine. Unfortunately, surgical options in advanced cases are usually limited and the prognosis is often poor.[1,2]

In this case report, we present an adult patient diagnosed with acute necrotizing colitis and ileitis due to highly progressive sigmoid colon cancer.

CASE REPORT

A 61 years-old male patient was brought to emergency room by his family with acute abdominal pain, that was irresistible. A day before, patient was admitted to a private hospital due to the same symptom that occurred earlier that day but later he was discharged because the symptom got better after administration of pain killer intravenously.

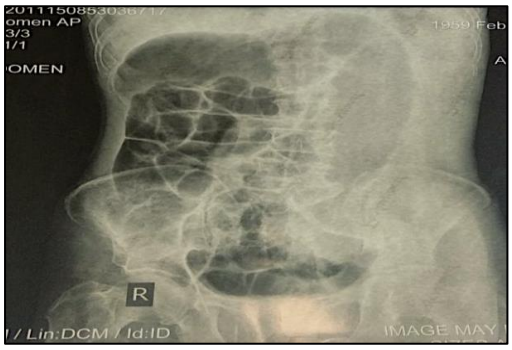

Figure 1: X-ray abdomen showed bowel dilatation.

The next day the pain was gradually worsening and patient could not resist it so his family decided to bring him to general hospital. From the physical examination we found that patient was fully conscious, with blood pressure 110/60, heart rate 105 times per minute, respiratory rate 22 times per minute, temperature was 37.3oC, and oxygen saturation 99%.

Figure 2: Intraoperative picture: stenosis at sigmoid colon.

Abdominal examination revealed that his abdomen was distended and tender, defans muscular was also shown. We performed several laboratory tests on patient and showed elevated leucocyte (18.86x103 /µl), haemoglobin was also higher than referral value (16.4 g/dL). X-ray abdomen showed bowel dilatation (Figure 1). From the physical examination and supportive examination, our tentative diagnosis was acute general peritonitis.

Figure 3: Acute necrosis at resected tissue.

Figure 4: White coloured tumour in the sigmoid colon.

We decided to perform emergency laparotomy under general anaesthesia and from the procedure we revealed that the dilatation of intestines and colon was caused by obstructive colitis due to sigmoid tumour. Intraoperative discovered serious stenosis of the colon sigmoid and proximal widening but no perforation was shown.

The digestive system was expanded proximally and was seen black from the sigmoid colon to 80 cm proximal to the terminal ileum, from the findings we were suggesting acute necrosis without significant vessel occlusion (Figure 3). We performed complete colectomy with 90 cm resection of terminal ileum and ileostomy during surgery. Hartmann procedure was performed to distal part of rectum.

Macroscopic findings demonstrated a white coloured tumour (8x5x2 cm) in the sigmoid colon that was causing colonic block (Figure 4). The bowel, from the sigmoid colon to 80 cm proximal to the terminal ileum, was obviously necrotized. Normal mucosa was seen between the necrotic injury and the tumour in the sigmoid colon, which was viable with a finding of obstructive colitis.

From microscopic examination we discovered tumour parenchyma was consisted of proliferated cells of anaplastic epithelial with morphologic core of nuclear-cytoplasmic ratio was increased. Histopathologic conclusion was grade II adenocarcinoma and was assessed as T2N0M0. Post-surgery evaluation, patient was stable and the next day after surgery patient was allowed to be moved from high intensive care unit to normal ward.

DISCUSSION

A colorectal carcinoma in situ includes an intraepithelial and an intramucosal carcinoma. They are defined as malignant cells that are confined to the basement membrane (intraepithelial carcinoma) and that have invaded into the mucosal lamina propria and have extended into, but not through, the muscularis mucosae (intramucosal carcinoma). A colorectal carcinoma in situ includes an intraepithelial and an intramucosal carcinoma. They are defined as malignant cells that are confined to the basement membrane (intraepithelial carcinoma) and that have invaded into the mucosal lamina propria and have extended into, but not through, the muscularis mucosae (intramucosal carcinoma).

Colorectal carcinoma is a malignant tumour of the large intestine, involving the colon, sigmoid, and rectum. Colorectal carcinoma in situ is classified into intraepithelial carcinoma and intramucosal carcinoma. An intraepithelial carcinoma is defined as malignant cells confined to the basement membrane. Meanwhile, in intramucosal carcinoma, malignant cells have invaded the lamina propria of the mucosa and have extended to the muscularis mucosa. One of the most common complications of colorectal cancer is intestinal obstruction and gastrointestinal tract where this obstruction can cause colitis.[3] Obstructive colitis is a non-specific inflammatory condition of the large intestine, including erosion and ulceration, which occurs in the proximal colon of the obstructive lesion. These obstructive lesions can be benign or malignant and may cause partial or complete obstructions.[4] In rare cases, obstructive colitis may become fulminant which is referred to as 'acute necrotizing colitis'. Acute necrotizing colitis is extensive colonic gangrene due to colonic obstruction. Cases of acute necrotizing colitis due to colon cancer are very rare. However, when these complications occur, they can be dangerous and require immediate surgery.[5,6]

Acute abdomen caused by necrotizing enterocolitis in adults is extremely rare. This is because most necrotizing enterocolitis occurs only in children. The diagnosis of necrotizing enterocolitis is characterized by the finding of diffuse ulceration and necrosis of the distal small intestine and large intestine. Thus, establishing a preoperative diagnosis of necrotizing enterocolitis is extremely difficult. Cases of advanced necrotizing enterocolitis have limited surgical options and tend to have a poor prognosis.[7]

Obstructive colitis due to colorectal carcinoma is extremely rare with an incidence of 0.3% to 3.1% of all colorectal carcinomas. The criteria for obstructive colitis are the presence of ulceroinflammatory lesions that are normal macroscopically and histologically located on the proximal and distal side of the obstruction site, a normal mucosa between the obstruction and ulceration sites, and a clear margin between them. Most necrotizing enterocolitis involves a single bowel segment (50%), but some cases may involve multiple segments. The terminal ileum is the most common site of necrotizing enterocolitis, followed by the large intestine. About 44% of necrotizing enterocolitis cases occur in both the large and small intestines. Pan-necrotizing enterocolitis or necrotizing enterocolitis totalis is fulminant necrotizing enterocolitis characterized by necrosis of more than 75% of the intestine. As many as 19% of all cases of necrotizing enterocolitis treated with surgery and necrotizing enterocolitis causing death were pan-necrotizing enterocolitis.[9]

In some obstructive colitis cases, few present with fulminating gangrene of the colon which is known as ‘necrotizing colitis’. The exact etiology, however, is yet to be determined. The increase of intraluminal pressure may affect the colonic blood flow, and in unperforated closed-loop colonic obstruction, mucosal ischemia may be the main event that leads to massive gangrene of the colon. One of the factors associated with this event is the proliferation of bacteria in the obstructed fecal. Meanwhile, hypoxia plays a major role by stimulating the germination of spores and bacterial growth, leading to rapid production of exotoxins that destroy and liquefy surrounding tissue, ultimately causing rapid spread of the disease.[9] A similar mechanism is found in the case of bowel necrosis due to non-occlusive mesenteric ischemia. In such cases, gut hypoperfusion and intestinal necrosis occur because of vasoconstriction, with bacterial translocation as the secondary event.[7]

Intestinal ischemia and bacterial infection have shown to be the major factors in the development of necrotizing colitis. Common organisms associated with necrotizing colitis are bacteria such as *Klebsiella, E. coli, Enterobacter, Pseudomonas, Clostridia spp.*, and *Staphylococcus epidermidis*, however, the exact etiology remains unknown. The most common location where necrotizing colitis occurs is the ‘watershed’ areas of the bowel, where the blood flow from the major mesenteric arteries overlaps.[10]

Prevention of necrotizing colitis or the disease progression to late stages which require surgery and/or bowel resection may be done through early diagnosis of gut ischemia and mucosal inflammation/ necrosis. Diagnosis of abnormalities and changes in the gastrointestinal tract or the size and shape of the lung and heart can be done through conventional radiography like abdominal or chest x-ray, respectively. Early detection of gut ischemia or necrotizing colitis may be conducted through experimental and clinical methods including serum hexosaminidase,

plasma amylin, serum cytosolic β-glucosidase activity, plasma pro- and anti-inflammatory cytokines, serum creatinine kinase isoenzymes, cerebro-splanchnic oxygenation ratio, GI tonometry, rectosigmoid pH monitoring, urinary EGF, D-lactate, or thromboxane, breath hydrogen, and MRI.[11]

Histological examination reveals distinctive pathological features such as intestinal necrosis which begins in the mucosa, without the obstruction of the mesenteric vessels. Management of necrotizing colitis can be achieved through medical and surgical attempts. Medical management aims to produce local vasodilatation whenever possible and surgical resection of the affected intestinal segment. In many cases, the diagnosis is made at exploratory laparotomy. Many cases are diagnosed during exploratory laparotomy. However, there are only limited surgical options for advanced cases. In case the patients survive after surgical resection, late complications including short bowel syndrome and malnutrition may occur.[7]

The gross appearance of necrotizing colitis throughout the surgery is fairly constant. The bowel is markedly distended with patchy areas of thinning. The typical red to grey serosa surfaces may be covered by a fibrinous exudate. In cases with frank gangrene, black or, in the most advanced cases, bland grey to white serosa due to the complete loss of perfusion can be observed. Gas collection is frequently observed in the subserosa. The mucosal surface may be ulcerated with wide areas of epithelial sloughing. If necrosis of the bowel is present, bloody peritoneal fluid may be seen, while brown and turbid fluid may be observed if perforation has occurred.8

CONCLUSION

The occurrence of obstructive colitis in cases of colorectal carcinoma is rare but troublesome with an incidence rate ranging from 0.3% to 3.1%. Some cases may even progress to idiopathic fulminant gangrene of the colon known as 'necrotizing colitis'. Surgical findings in acute necrotizing colitis may include a very distended bowel with areas of uneven thinning, a red to gray serous surface covered with a fibrinous exudate, and light gangrene with a black serous or, in the most severe case, pale gray to white serous, due to complete loss of perfusion. Another most common findings during surgery are the collection of subserous gas. In addition, it is common to find ulcerations with large areas of epithelial sloughing on the serous surface. When bowel necrosis and perforation occur, we may find bloody peritoneal fluid and turbid brown fluid. The clinical findings in our patient are consistent with the existing literature. During surgery, we found a dilated colon due to obstruction by the sigmoid tumor and blackish area from the sigmoid colon up to 80 cm proximal to the terminal ileum which means acute necrosis without significant vascular occlusion. Complete ileostomy and colectomy with 90 cm resection were performed in the terminal ileum, while the Hartmann procedure was performed on the distal rectum. The operation was successful and the patient was stable postoperatively. In conclusion, emergency surgery and intensive care are very important in saving patients' lives in cases of acute necrotizing colitis due to sigmoid colon cancer.

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