



MRSA PERITONITIS SECONDARY TO PERFORATED RECTOSIGMOID CARCINOMA WITH EXTENSIVE HEPATIC METASTASIS: A RARE SURGICAL EMERGENCY

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ABSTRACT

Introduction: MRSA peritonitis is documented in literature as being secondary to peritoneal dialysis but it is a lesser known cause of perforation peritonitis

Case presentation: we report a 72 year old male with rectosigmoid cancer perforation with MRSA peritonitis and his emergency surgical management

Conclusion: we conclude that though MRSA peritonitis in this patient followed a fulminant course which was exacerbated by his underlying disease process.

KEYWORDS : MRSA peritonitis, perforated Rectosigmoid carcinoma, Hepatic metastasis

INTRODUCTION:

Left sided colon cancers consist of those arising from the splenic flexure, descending colon, sigmoid colon, rectosigmoid junction and rectum. It is more common for left sided colon cancers to present with a polypoidal growth as compared to right sided cancers which are usually ulcerative in nature. Due to this reason, advanced left sided colorectal malignancy may present with features of intestinal obstruction. Perforation in such patients may be secondary to obstruction or due to the invasion of tumor into the bowel wall as seen in this case. This leads to localised or generalised faecal peritonitis and patients may present in the emergency setting with abdominal pain, guarding, rigidity, hypotension and septic shock. The poor nutritional status of the patient and the underlying inflammation due to advanced malignancy complicates the picture. The prognosis of such patients is usually dismal even after aggressive surgical and medical management. The organisms usually isolated in cultures of peritoneal fluid are those that form commensals of the gastrointestinal tract: *Bacterioides*, *Enterococcus* and *Escherichia*. Methicillin resistant *Staphylococcus aureus* (MRSA) is a gram positive bacteria first described in 1961 and has since then been increasing in prevalence in both healthcare and the community [1]. The *mecA* gene in MRSA encodes PBP2a a penicillin binding protein which is responsible for the methicillin resistance. MRSA is usually isolated from the nares and throat. Colonization increases the risk of infection [2]. It is an important cause of necrotising skin infections, endocarditis, osteomyelitis, deep seated abscesses and pneumonia. MRSA peritonitis, though well documented in literature is usually seen secondary to post surgical infections and long term peritoneal dialysis. MRSA is less commonly isolated in faecal peritonitis cultures. We report this case to demonstrate the fulminant nature of rectosigmoid carcinoma perforation with MRSA faecal peritonitis which is not normally a colonizing bacterium in the lower gastrointestinal tract.

Case Presentation

A 72 year old male patient presented to the emergency department with complaints of pain in abdomen which was gradually progressive since the past two months but had suddenly increased in severity over the last few hours. The patient was a known case of moderately differentiated adenocarcinoma of the rectum which was diagnosed on colonoscopic biopsy four months back when he had a history of progressive weight loss and fatigue. Since imaging done at that time revealed extensive metastasis to the liver, he was started on folinic acid, 5-fluorouracil and oxaliplatin (FOLFOX regimen). He had completed 8 cycles in the last four months. He smoked half a pack of cigarettes a day for a number of years and drank two alcoholic beverages every day. He had no family history of cancer.

On examination he had a pulse of 110 beats per minute, blood pressure 100/70mm Hg and a saturation of 98% on room air. His abdomen was distended with generalised tenderness and guarding. Per rectal examination was empty without staining of gloves.

A supine X ray film done on admission revealed air lining outer and inner surfaces of the bowel indicating a positive Rigler sign



Fig1: Rigler sign on abdomen X ray

Contrast enhanced computed tomography of the abdomen revealed pneumoperitoneum with peripherally enhancing peritoneum suggestive of peritonitis. Bowel wall appeared normal in caliber. Multiple variable sized peripherally enhancing centrally hypodense rounded lesions were seen scattered in both lobes of the liver.

The patient was shifted to the operation theatre and midline laparotomy was done. The peritoneal fluid obtained at the point of opening of the cavity was faecal stained and was sent for examination and culture sensitivity. Examination of bowel showed a perforation in the rectosigmoid junction of about 1cm diameter as shown in the image. There was significant induration around the lesion. There was also evidence of extensive metastasis to liver.



Fig 2. Rectosigmoid cancer perforation

Fig 3. Extensive hepatic metastasis

Since surgical resection with curative intent was not possible due to the extent of the lesion and continuity of bowel wall was maintained, the margins of the perforation were freshened and primary closure was done followed by a diversion transverse colostomy. Thorough saline washes and intraperitoneal antibiotic lavage were given and the abdomen closed after insertion of subhepatic and pelvic drains.

Peritoneal fluid examination revealed more than 2000 nucleated cells/cubic millimeter with 95% neutrophils and 5% lymphocytes

Culture and sensitivity of peritoneal fluid revealed following results

Table 1. Peritoneal fluid culture sensitivities

Organism detected: Methicillin Resistant <i>Staphylococcus aureus</i>	
Antibiotic	Sensitivity
Cephoxitin	Resistant
Ciprofloxacin	Resistant
Clindamycin	Sensitive
Gentamicin	Sensitive
Vancomycin	Sensitive

The patient was shifted to the intensive care unit post surgery where mechanical ventilation continued. Adequate fluid resuscitation was done and urine and drain outputs were charted. Blood products were infused as per requirement. He was started on combined gentamicin plus vancomycin therapy. Electrolytes were monitored every twelve hours. Noradrenaline and dopamine support continued in the postoperative period. Despite aggressive management, the patient succumbed to sepsis on the second postoperative day.

DISCUSSION

Advanced colorectal carcinoma is a rare cause of intestinal perforation. When this happens it represents size T4a of the tumor. The patient in our case had hepatic metastasis and his lymph node status could not be assessed in the emergency setting. Therefore his disease stage was T4aNxM1a by TNM classification and stage IVa by AJCC 8th edition classification. The five year survival rate of Stage IV colon cancer is 14% as compared to 91% for localised disease. Though the stage of presentation is the most important factor in the prognosis of colorectal carcinoma, the presence of perforation carries an adverse prognostic impact and is independently predictive of poorer survival. Such cancers are also more likely to show an aggressive histologic profile [3]. The other determinants of survival are the presence of extramural tumor deposits, lymphovascular or perineural invasion, histologic grade of differentiation, preoperative carcinoembryonic antigen (CEA), microsatellite instability (MSI) and RAS and BRAF mutations.

The College of American Pathologists specifies the use of a four tiered histologic grading system for Colorectal carcinoma based on the degree of gland formation.

Grade 1 : Well differentiated (>95 percent gland formation)
Grade 2 : Moderately differentiated (50 to 95 percent gland formation)
Grade 3 : Poorly differentiated (<50 percent gland formation)
Grade 4 : Undifferentiated (no gland or mucin formation; no squamous or neuroendocrine differentiation) [4]

The patient in this case had moderately differentiated carcinoma.

Peritonitis may occur either due to translocation of bowel microorganisms into peritoneal cavity as is the case in spontaneous bacterial peritonitis, by external entry as is seen in peritoneal dialysis associated peritonitis or as a result of direct entry into peritoneal cavity due to hollow viscus perforation as is seen in this case. The symptoms of peritonitis are abdominal pain, fever, nausea, vomiting and in severe cases hypotension. In cases that are complicated by ascites, a classic surgical abdomen with rigidity does not develop as ascites causes separation of parietal and visceral peritoneum. The usual organisms seen in peritoneal fluid cultures are those that form commensals of the gastrointestinal tract: *Bacteroides*, *Enterococcus faecalis*, *Escherichia coli*. MRSA peritonitis is documented in literature as being secondary to peritoneal dialysis contamination. As MRSA is not a usual gut commensal it is rare for it to be isolated in peritoneal cultures unless complicated by intraperitoneal abscesses, infected surface wounds post surgery or immunocompression and prolonged hospital stay.

Shawn M Vuong et al in 2011 presented a case report of MRSA peritonitis secondary to perforation of sigmoid diverticulitis [5]. There had not been any documented report regarding MRSA peritonitis secondary to diverticulitis prior to this study. Aditya Safaya et al reported two cases of MRSA cultured from peritoneal fluid after laparoscopic appendicectomies of two patients, one of which was perforated [6]. There has been no previous documentation of MRSA appendicitis before this 2018 study. Shorena Khetsuriani et al published a study in 2017 on rectal MRSA colonisation in colorectal cancer patients coming to a conclusion that they are more pathogenic than non-MRSA strains [7]. Hence the alteration in gut flora in this case

could be attributed to the patient's underlying disease process.

CONCLUSION:

MRSA peritonitis secondary to a perforated rectosigmoid carcinoma is a rare clinical entity. The robust nature of the infection combined with the advanced stage of the cancer with extensive hepatic metastasis contributed to the poor response of the patient to aggressive surgical and medical management. Though MRSA is not a usual gut commensal, alteration of bowel flora is seen in patients with colorectal carcinoma.

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