Various complications of colon cancer may alter its presentation, which commonly include obstruction, perforation and bleeding. Only few cases of superior mesenteric artery occlusion (not caused by colon cancer) with discouraging clinical outcomes were reported elsewhere. Here, we report a case of superior mesenteric arterial occlusion caused by colon cancer, an extremely rare case never reported in English literature. Morbidity and mortality rates remain high because it frequently goes unrecognized until patients suffer adverse outcomes. It is important to keep this complication of SMA occlusion in mind when treating patients having colon cancer manifesting with acute abdomen.

Case Report

A 41 year-old man presented to the Emergency Department (ER) in the night of December 8, 2005, with a two-day history of epigastric pain, vomiting and abdominal distention. Prior to this presentation to ER, he had a three-month history of epigastralgia and anemia. He had been treated as gastritis and iron-deficiency anemia (IDA) by his physicians.

At ER, his temperature was 37.5 °C., pulse 156, and regular, respiratory rate 20, and blood pressure 175/79 mmHg supine. Initial laboratory investigations were as follows: Hemoglobin 5.6 g/dl; White Blood Cell 25,000 /ul; differential 94 neutrophils, 2 band forms, 2 monocytes, 2 lymphocytes; Serum sodium 133 mEq/l., Potassium 3.2 mEq/l, Chloride 99 mEq/l ; Blood Sugar 183 mg/100 mL; BUN 15 mg/100 mL. Arterial blood gas demonstrated metabolic acidosis. Chest x-ray showed no significant abnormalities. Plain abdominal films demonstrated distended air-filled loops of bowel. Bowel sound was hypoactive. There was no history of atrial fibrillation (Af). Emergency computed tomography (CT) of whole abdomen revealed an ill-defined mass lesion at transverse colon with annular stenosis and perifocal colonic wall thickening. Several enlarged regional lymph nodes were also noted. Proximal portion of superior mesenteric artery (SMA) was not well-visualized but compressed by surrounding tissues. (Figs. 1 and 2). Because no peritoneal sign was found at that time, the patient was kept at ER for further observation.
However, ten hours after CT scan, the patient developed diffuse peritonitis. On emergency consultation, there was severe tenderness over the entire abdomen. Severe rebound pain was present in all four quadrants, and there was voluntary guarding with rigidity on comprehensive physical examination. Bowel sounds were absent. Emergency colonoscopy revealed a tumor mass at proximal transverse colon with total obstruction. Biopsy was taken and several days later proven to be adenocarcinoma.

A presumptive diagnosis of transverse colon cancer with total luminal obstruction was made. On emergency exploration, the tumor was located at proximal transverse colon with severe adherence and fixation to adjacent pancreatic head, duodenum and stomach. It demonstrated unresectability of the primary tumor. Gangrenous change and transmural necrosis, in the distribution of SMA, extending from 50 cm below the ligament of Treitz to 8 cm proximal to the tumor with three hundred milliliters of yellowish turbid ascites were noted. The root of SMA was macroscopically invaded by the tumor. No pulses were present beyond the main trunk of SMA. Massive resection was performed from 50 cm below the ligament of Treitz to 6 cm proximal to the tumor (with the primary tumor left in place), followed by creation of end jejunostomy and colonic mucous fistula. The patient was left with 50 cm of small bowel and the distal two-third of his colon. The histopathological report revealed marked hemorrhagic infarction with focal neutrophilic infiltration.

After surgery, an echocardiogram was obtained and it excluded a cardiac source of embolic occlusion on SMA. The patient had been maintained on total parenteral nutrition for short bowel syndrome. Intermittent diarrhea was responsive to Cholestyramine and loperamide. Salvage chemotherapy and radiotherapy were also scheduled. He died of disease progression on July 10, 2006 (seven months following surgery).

**Discussion**

Many symptoms are non-specific for colon cancer and may not develop until later stages of the disease explain the high frequency of delayed diagnosis. Because of the tumor location, our patient initially presented with epigastralgia mimicking gastritis. This presentation made a delay in diagnosis for three months by his physician. Robinson et al. suggested that patients with a delayed diagnosis tended to have more advanced disease. This situation was also seen in our patient. The main reason for not early diagnosis is the common occurrence of bowel disturbances in
general population; consequently many physicians underestimate symptoms such as abdominal discomfort, pain or rectal bleeding might also be due to the presence of colon cancer. All patients with anorectal discomfort or abdominal symptoms lasting more than a few weeks should receive digital rectal exam, flexible sigmoidoscopy. Even colonoscopy should also be included if we consider that 30% to 40% of colorectal lesions are proximal to the splenic flexure.

Well-reported complicated presentations of colon cancer include obstruction, bleeding, and perforation, all of which are declining secondary to better awareness of patients and more sophisticated examination tools. Acute mesenteric ischemia (AMI) is a rare occurrence with numerous causes, the most common of which are cardiac arrhythmias, advanced age, low cardiac output states, generalized atherosclerosis, congestive heart failure, severe valvular heart disease, recent myocardial infarction, and intra-abdominal malignancy. Colon cancer manifesting with superior mesenteric artery occlusion is so extremely rare that no case was reported in the English literature until now.

The duration of symptoms prior to diagnosis was 2 days in our patient. In a fifteen-year review reported by Murray et al at the hospital of the University of Pennsylvania, the median time from the onset of symptoms to clinical diagnosis of SMA occlusion was 24 hours. The longest length of time recorded was 7 days. Incidence of the abdominal symptoms as reported by Liavag was abdominal pain in 100 percent, vomiting in 80 percent, diarrhea in 40 percent, bloody stool in 23 percent, and constipation in 33 percent. Our patient presented with abdominal pain and vomiting. This signifies tremendous variation in clinical features of the condition. The clinicians have difficulties in making the correct diagnosis at an early stage because there were no specific symptoms or clinical signs.

Laboratory evaluations usually reveal an increased in hemoglobin/hematocrit consistent with hemoconcentration. There is a marked leukocytosis with left shift. A metabolic acidosis is common with a persistent base deficit. In our patient, because he had chronic tumor bleeding, hemoglobin/hematocrit was decreased. Both marked leukocytosis with left shift and metabolic acidosis developed in our patient because he developed bowel necrosis on presentation. However, many of these laboratory abnormalities are not specific and do not develop until after bowel necrosis has occurred.

Completely normal plain abdominal radiographs have been reported in more than 25% of patients with mesenteric ischemia. Subtle signs of AMI on plain films include adynamic ileus and distended air-filled loops of bowel, which were also detected in our case. Pneumatosis of the bowel wall can be detected on plain films in advanced stages of ischemia, but this was not demonstrated in our patient.

CT of whole abdomen is usually obtained in many patients with abdominal pain of unknown cause. Focal or segmental bowel wall thickening can often be detected after a period of AMI. CT findings are nonspecific in 61% of patients and completely normal in 30% of patients. Specific signs of ischemic bowel on CT include intestinal pneumatosis, portal venous gas, or thumbprinting of the bowel wall. However, CT scan of our patient revealed no specific signs for ischemic bowel, and made early diagnosis somewhat difficult.

In spite of better insights into the pathophysiologies of AMI, these syndromes remain highly morbid events with reported mortality rates exceeding 60%. One of the reasons for high mortality associated with this type of vascular occlusion is the insidiousness of onset. Usually by the time the diagnosis is made, an irreversible gangrene of great length of small intestine and frequently colon has supervened. Better results depend on the early diagnosis and treatment.

In patients with high index of suspicion, mesenteric angiography should be performed as soon as possible. Thrombolytic therapy in patients with early stage of mesenteric embolus was suggested by some authors. Cases successfully managed by percutaneous angioplasty to dilate the occlusion site of SMA root were also found in literature. However, most cases of acute mesenteric occlusion need early surgical exploration. Arteriotomy with thrombectomy or some form of bypass could be considered for revascularization. If the small bowel is frankly nonviable, massive small bowel resection with life-long hyperalimentation is the only option, which
was also the condition of our case. Alternatively, patient preferences and family consultation may argue against this procedure and support simple abdominal closure with pain relief. In patients with long established gangrene, vascular reconstructive procedures are of no value. If bowel infarction is not profound, surgical revascularization should be performed. Frequently, ischemic small bowel can be restored if vascular flow is reestablished. Simple indicators of viability include visible peristalsis, a pink and normal color of the serosa, and palpation of the distribution of the SMA for arterial pulsations. Bowel continuity can be restored primarily or stoma exteriorized if the patient is unstable. In our case, because the patient was unstable, we created end-jejunostomy and colonic mucous fistula.

Even after massive resection of small intestine, a reasonably good health could be obtained as reported by Murray et al in 1969. In our case, because the patient had an un-resectable colonic tumor, he finally died of disease progression.

In conclusion, a high index of suspicion may decrease the delay in diagnosis of colon cancer. Delayed detection of colonic malignancy may lead to ominous complications. Morbidity and mortality rates remain high because it frequently goes unrecognized until patients suffer adverse outcomes. It is important to keep this complication of SMA occlusion in mind when treating patients having colon cancer manifesting with acute abdomen.

References


病例報告

上腸繫膜動脈阻塞：罕見之大腸癌表現

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大腸癌的延誤診斷可能會導致可怕的併發症。在這篇文章中，我們報導一個因大腸癌造成上腸繫膜動脈阻塞的罕見病例，甚至在英文文獻中亦沒有類似的記載。因為難以早期測知，所以此併發症的死亡率及罹病率相當高。當我們面對以急性腹症表現的大腸癌患者，應當要牢記此種併發症的可能性。

關鍵詞 大腸癌、急性腸繫膜缺血、上腸繫膜動脈阻塞。