In the name of God

S.U.M.S.

Department of Internal Medicine

Shiraz E-Medical Journal

Vol. 8, No. 1, January 2007

http://semj.sums.ac.ir/vol8/jan2007/pancreatitis.htm

Non-Occlusive Mesenteric Ischemia in Acute Pancreatitis.

Aminian A*, Shamimi K**, Moazami F**, Jalali M*, Mirsharifi R*.

*Assistant Professor, ** Associate Professor, Department of Surgery, Imam Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran.

Correspondence: Dr. Ali Aminian, Department of Surgery, Imam Hospital Complex, Tehran University of Medical sciences, Tehran, Iran, Telephone: +98 21 66937185, Email: aliaminian@doctor.com.

Received for Publication: November 7, 2006, Accepted for Publication: December 25, 2006.

Abstract:

Non-occlusive mesenteric ischemia (NOMI) has been defined as diffuse intestinal ischemia that often results in intestinal gangrene in the presence of a patent arterial trunk with vasospasm. A few cases of severe acute pancreatitis have been reported to be complicated by NOMI. Here we report a case of acute hemorrhagic biliary pancreatitis with intestinal gangrene due to NOMI. Clinicians who manage acute pancreatitis should consider NOMI as one of the severe complications of this condition.

Key Words: Pancreatitis, mesenteric ischemia, non-occlusive mesenteric ischemia (NOMI), intestinal gangrene, complication, vasospasm.

Introduction:

Acute pancreatitis is a potentially fatal disease and its severe form is often associated with pancreatic necrosis with higher mortality. Pancreatic necrosis may be the result of local vascular spasm and increased intravascular coagulability. Such complications can also develop in other organs if the inflammatory insult is sufficiently severe [(1)].

Non-occlusive mesenteric ischemia (NOMI), which is defined as diffuse intestinal ischemia in the presence of a patent arterial trunk with vasospasm, is usually diagnosed in critically ill patients. Progressive intestinal ischemia can lead to intestinal gangrene, sepsis and death from multiple organ failure (MOF) [(2)]. A few cases of acute pancreatitis have been reported to be complicated by NOMI.

Case Report:

A 61 years old smoker, hypertensive and diabetic lady was admitted with chief complaints of abdominal pain, nausea and vomiting for 3 days. On evaluation, she was dehydrated, tachycardic and drowsy but afebrile. There was generalized abdominal guarding and tenderness. Initial laboratory data revealed hemoconcentration, leukocytosis with shift to the left, pre-renal azotemia, metabolic acidosis and elevated creatine phosphokinase (CPK). After initial resuscitation, she was operated emergently. We encountered an acute hemorrhagic pancreatitis with intestinal ischemia from proximal of jejunum to cecum. Intra-venous bolus dose of heparin was administered and warm pads were put over small bowel. There were multiple small gallstones, but common bile duct was normal. Cholecystectomy was performed quickly. After several minutes, we found that both ends of small bowels were viable, so resection of gangrenous part from mid-jejunum to distal ileum with primary anastomosis was performed. There was no clot in the mesenteric vasculature. She was transferred to ICU, but postoperative course was complicated with sepsis, acute renal failure, acute respiratory distress syndrome (ARDS) and severe metabolic derangements. Second look operation was performed after 72 hours. The remained small bowel and anastomosis were normal, but the entire pancreas and retroperitoneum of lesser sac were diffusely necrotic. Pancreatic necrosectomy was performed. Despite aggressive supportive care, she was died of MOF 3 days later.

Histological examination of intestinal specimen showed extensive necrosis of the entire intestinal wall, consistent with NOMI (figure 1).

Discussion:

The true incidence of NOMI in patients with acute pancreatitis is unknown. Recently Takahashi et al. reviewed the intestinal and pancreatic slides of 48 autopsy cases of patients with acute pancreatitis to determine this incidence. They found that 13 (27%) of 48 autopsy cases of patients with acute pancreatitis were complicated by NOMI. The frequency of shock was significantly higher in patients with NOMI than in those without NOMI. The intestinal lesion was diffuse in many cases and gangrene was not an unusual finding.

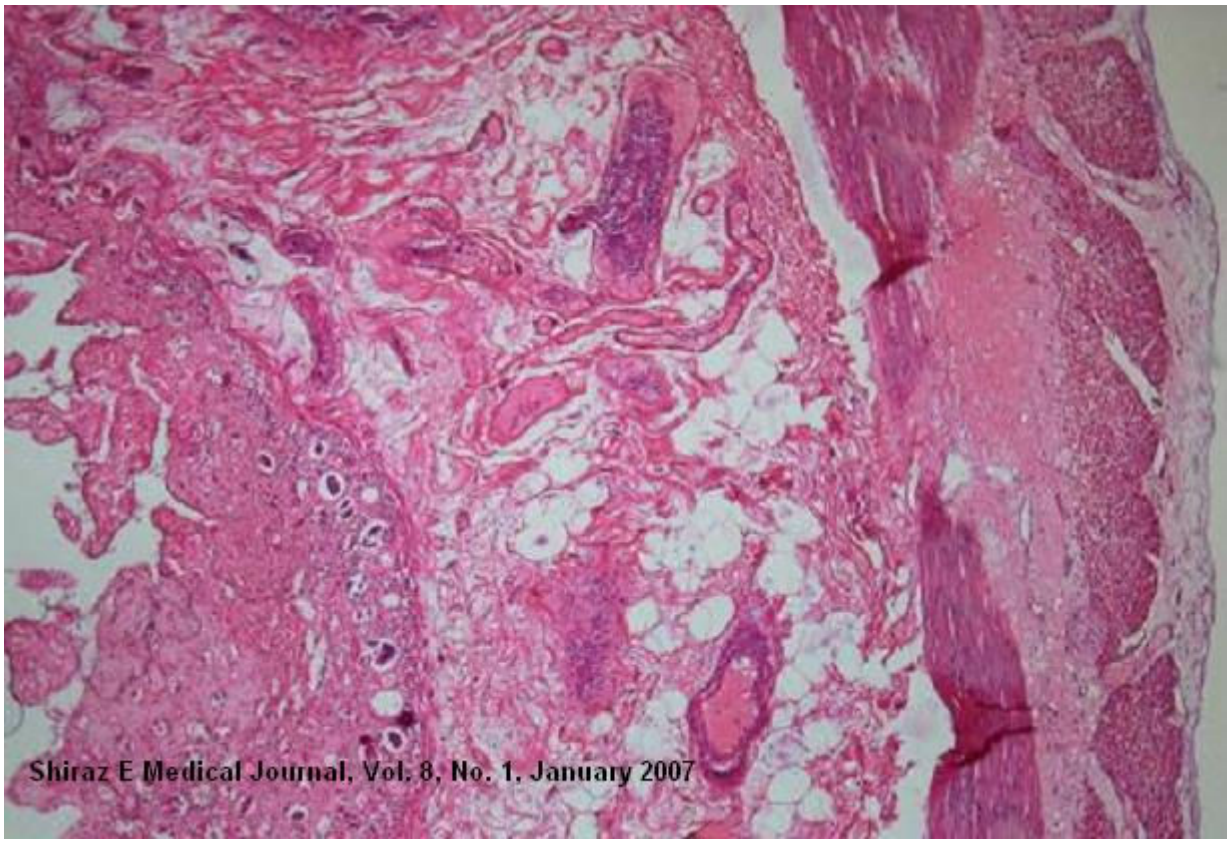

Figure 1- Microscopic appearance of NOMI-associated intestinal gangrene: extensive necrosis of the entire intestinal wall. HE stain.

They concluded that this complication is relatively frequent [3]. Two major mechanisms are proposed for NOMI. Most investigators believe that it is caused by mesenteric vasoconstriction, while others have postulated that it is a manifestation of DIC. Furthermore, shock, dehydration and increased abdominal pressure may also be associated with NOMI pathogenesis in pancreatitis patients [4].

Hirota M et al. investigated the nature of NOMI in acute pancreatitis. They found 8 cases of NOMI among 120 cases of acute pancreatitis. Five of 8 cases developed NOMI-associated intestinal gangrene quickly progressed and subsequently died of MOF. Plasma CPK and lactate levels were elevated significantly in patients with NOMI. These cases had mesenteric arterial spasms on angiography, which is typical finding for NOMI. They were able to inhibit the development of intestinal necrosis in some patients with spastic changes in the celiac and superior mesenteric arteries by performing continuous regional arterial infusion with nafamostat mesilate via both arteries. Local administration of nafamostat mesilate may suppress the enhanced coagulative activity and maintain microcirculation. They finally concluded that if both CPK and lactate are very high in the cases of

severe acute pancreatitis, angiography should be performed to check for the presence of NOMI or intestinal gangrene [5].

The standard treatment of NOMI without necrosis or perforation is primarily pharmacologic, with continuous selective infusion of vasodilator agents such as tolazoline, papaverine, or prostacyclin into the superior mesenteric artery [6]. Therefore administration of these vasodilators may also play a role in the management of NOMI occurred in the setting of acute pancreatitis.

In conclusion, clinicians who treat patients with acute pancreatitis should consider NOMI as one of the frequent and severe complications of this condition and if the plasma CPK and lactate levels are extremely high, NOMI should be suspected.

References:

1. Bradley EL, Murphy F, Furguson C. Prediction of pancreatic necrosis by dynamic pancreatography. Ann Surg 1989; 210: 495-503.
2. Howard TJ, Plaskon LA, Wiebke EA, et al. Non-occlusive mesenteric ischemia remains a diagnostic dilemma. Am J Surg 1996; 171: 405-408.
3. Takahashi Y, Fukushima J, Fukusato T, et al. Prevalence of ischemic enterocolitis in patients with acute pancreatitis. J Gastroenterol. 2005; 40(8): 827-832.
4. Bassiounty HS. Non-occlusive mesenteric ischemia. Surg Clin North Am 1997; 77: 319-326.
5. Hirota M, Inoue K, Kimura Y, et al. Non-occlusive mesenteric ischemia and its associated intestinal gangrene in acute pancreatitis. Pancreatology. 2003; 3(4): 316-322.
6. Lumsden AB, Lin PH, Bush RL, Chen C. Arterial disease. In: Brunicardi FC. Schwartz's principles of surgery. 8th edition. McGraw-Hill: 2005; p. 770-775.