Abstract: Acute thromboembolic occlusion of the superior mesenteric artery is a condition with an unfavorable prognosis. Treatment of this condition is focused on early diagnosis, surgical or intravascular restoration of blood flow to the ischemic intestine, surgical resection of the necrotic bowel and supportive intensive care. In this report, I describe a case of a 60-year-old man who developed a small bowel infarct because of an acute embolic occlusion of the superior mesenteric artery.

Keyword: superior mesenteric artery occlusion, Thromboembolic Occlusion, bowel gangrene, acute mesenteric ischemia.

Introduction
Acute thromboembolic occlusion of the superior mesenteric artery (SMA) is a condition with a serious prognosis. Acute mesenteric ischemia (AMI) is an uncommon occurrence and represents 0.1% of hospital admissions. Despite considerable advances in medical diagnosis and treatments over the past, mesenteric vascular occlusion still has a poor prognosis, with an in-hospital mortality rate of 59 to 93%. The high rate of mortality can be explained by the nonspecific signs and symptoms that characterize AMI. The classic teaching of "pain out of proportion to physical examination findings" is often seen during the early stage of ischemia when the abdomen is soft and not tender. Distention and severe tenderness with rebound guarding appear as a consequence of the bowel infarction. The serologic markers cannot aid in the diagnostic process because they are nonspecific (inorganic phosphate, lactic acid, aldolase, creatinine kinase, and alkaline phosphate). An elevated white blood cell (WBC) count (leukocytes measuring over 15,000 cells) is a common, but unspecified, finding. Treatment of this condition is focused on early diagnosis, surgical or intravascular restoration of blood flow to the ischemic intestine, surgical resection of the necrotic bowel, and supportive intensive care.

Case Presentation
A 60-year-old man presented in our emergency department with acute abdominal pain of 3 days duration associated with nausea, vomiting, and signs of intestinal occlusion. History of hematemesis, bloody diarrhea and oliguria was present. He is a known patient of coronary artery disease, CABG done 3 years before with pacemaker implanted. Aortic valve replacement was done in the same setting and the patient is on Tab Acitron 2mg OD, last INR value is 1.12. On examination Temperature was 102F, Pulse rate-94/min, Blood pressure 90/60mmhg, respiratory rate 26/min. Abdomen was distended with guarding and rebound tenderness present over epigastric, right hypochondrial and right iliac fossa. Altered blood was noted in ryles tube aspirate. Digital rectal examination showed dark black mucous discharge. Laboratory reports revealed Acute renal failure with Blood urea-86mg/dl, Sr creatinine-3.1, INR 1.2 (Hypercoagulopathy). Non contrast abdominal CT revealed Small bowel wall thickening, with free fluid in the abdomen. Atheromatous plaque was seen at the SMA origin. The finding was discussed with the radiologist. The condition of the patient was also told and he committed that it was an indirect sign of SMA thromboembolic occlusive disorder Patient was resuscitated with IV fluids and IV antibiotics. Inotropics and oxygen support was given. Patient was reviewed regarding the possibility of ischemic bowel and emergency laparotomy was planned. Laparotomy demonstrated gangrenous small bowel except for proximal 30 cm of jejunum. Caecum ascending colon, hepatic flexure were also gangrenous. Extensive small bowel resection was done. Complete peritoneal wash was done. End to side anastomosis was done with Jejunum and lateral 1/3rd of transverse colon. Suturing was not used for the entire procedure. Patient was kept in intensive care unit and managed with IV fluids, IV antibiotics, Oxygen support and Inotropics. Fresh blood transfusion, Total parentral nutrition and anticoagulants was given.

Thromboembolic Occlusion of Superior mesenteric artery-a case report
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The most common cause of AMI is SMAE, which represents 50% of the causes of AMI. These thromboembolic phenomena may be associated with prothrombotic disorders, such as protein C, protein S, and antithrombin III (AT III) deficiency.

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Flank drain was functioning. Blood pressure and SPO2 was constantly at the lower range and the patient expired on 8th post operative day.

Discussion
Acute mesenteric ischemia (AMI) accounts for 60% to 80% of all cases of mesenteric ischemia and has a mortality rate between 59% and 93%. AMI can be caused by arterial emboli, arterial or venous thrombosis and non-occlusive obstruction. In all causes, the sudden onset of intestinal hypoperfusion can lead to hypoxemia and intestinal hypoxia with irreversible bowel damage. Risk factors for developing AMI depend on the etiology: more often patients are older than 50 years and suffer from congestive heart failure, recent myocardial infarction, hypovolemia, hypotension or sepsis. Clinical presentation is nonspecific, often presenting as a sudden onset of severe abdominal pain, frequently out of proportion to what is found at physical examination. Accompanying symptoms and signs are nausea, vomiting and hypotension. The absence of specific signs upon physical examination can make the diagnosis of AMI very challenging and the clinical consequences of missed AMI can be catastrophic. A rapid diagnosis is the most important factor for prognosis, and urgent investigation of vessel patency should thus be done by abdominal CT. During the work-up, the patient should be closely monitored and stabilized. Once the diagnosis has been made, ischemic bowel segments should be resected and the blood flow restored as soon as possible. Depending on the etiology of the acute ischemia, the restoration of blood flow can be achieved either surgically, using intra- arterial vasodilatation, embolectomy or bypass, or via medical revascularization with, for example, intravenous heparin.

Chronic mesenteric ischemia (CMI) is a rare condition that accounts for only 5% of all intestinal ischemic events. As with AMI, clinical diagnosis is often difficult due to the vague symptomatology. Symptoms of CMI typically develop when there is a postprandial increase in blood flow demand. The most characteristic symptom is intestinal angina. This typical abdominal pain occurs within the first hour after ingestion of a meal. It is often located in the epigastric region or the mid-abdomen and is described by patients as dull and crampy. Diarrhea, nausea and vomiting are commonly present.

Conclusion
Mesenteric ischemia can be divided into acute and chronic ischemia, two separate entities, each with their own specific clinical presentation and diagnostic and therapeutic modalities. Diagnosis may be difficult due to the vague symptomatology and subtle physical signs. These diagnoses should therefore always be kept in mind in any patient with chronic postprandial abdominal pain in whom no other diagnosis can be made. The diagnosis should be confirmed by angiography. Management of intestinal ischemia consists of blood flow restoration through medical treatment or surgical management, depending on the etiology.

Reference:

fig.1 CT Picture showing Atheromatous Plaque at SMA origin
fig.2 Laparotomy Showing Gangrenous Bowel segments
fig.3 Gangrene involving the Hepatic Flexure
fig.4 Extensive Small Bowel Resection