



MIRACULUS OUTCOME OF NON-OCCLUSIVE MESENTERIC ISCHAEMIA (NOMI) IN CARDIOGENIC SHOCK- A CASE REPORT

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ABSTRACT

Introduction: Non-occlusive mesenteric ischemia (NOMI) is an acute mesenteric circulatory disorder which includes all types of mesenteric ischemia without any blockage of blood vessels. It is usually seen in elderly age group with risk factors of previous myocardial infarction, congestive cardiac failure and post-cardiac surgery, renal disease, vasopressor use, aortic insufficiency. NOMI is an infrequent cause of acute mesenteric ischemia with atypical symptoms & constitutes 20-30% cases of acute mesenteric ischemia (AMI). The mortality rate is generally high, at least 50%, being the deadliest type of acute mesenteric ischemia (AMI). A high index of suspicion is required. Treatment is based on correction of the underlying cause, supportive therapy, vasodilation and surgery. Presenting a challenging case of an elderly patient with Diabetes Mellitus for 15 years in cardiogenic shock with NOMI. Colonoscopy was diagnostic of ischemic colitis. Patient was managed conservatively with successful outcome **Conclusion:** NOMI is a rare disease with a difficult diagnosis because of the unspecific symptomatology. Early recognition with high suspicion of index is necessary. Case highlights the challenges in the diagnosis and the multiple difficulties during management. Treatment with supportive therapy, vasodilator drugs and surgery if needed can significantly improve prognosis & lower mortality.

KEYWORDS : Non-occlusive mesenteric ischemia (NOMI), Acute mesenteric ischemia (AMI), colonoscopy, conservative management

INTRODUCTION:

Ischemic colitis is the most common form of gastrointestinal ischemia. [1]

AMI may be occlusive (most common) or nonocclusive (NOMI). Nonocclusive mesenteric ischemia (NOMI) occurs mostly due to vasoconstriction of mesenteric arteries. NOMI was first described in patients with heart failure. The majority of cases involve spasm of branches of the superior mesenteric artery (SMA). Early diagnosis is based upon a high index of clinical suspicion in patients with risk factors. [2]

The occurrence of intestinal ischemia depends upon the adequacy of systemic perfusion and collateral circulation, the number of mesenteric vessels affected and the duration of the ischemic insult. The intestine compensates for a 75 percent acute reduction in mesenteric blood flow for 12 hours without substantial injury, in part. [3]

NOMI occurs in approximately 20% of cases of AMI. Patients with NOMI typically suffer from severe illness, like cardiac failure. It may be precipitated by sepsis. Hypovolemia and the use of vasoconstrictive agents may precipitate NOMI. [4]

The clinical symptoms of excruciating abdominal pain with an unrevealing abdominal exam is classic for early AMI. [5]

If there are signs of peritonitis, there is likely irreversible intestinal ischemia with bowel necrosis. In a study of AMI, 95% of patients presented with abdominal pain, 44% with nausea, 35% with vomiting, 35% with diarrhea, and 16% with blood per rectum. [6]

Recommendations with Strong evidence of 2017 WSES World Congress, May 2017 in Campinas, Brazil. [7]

Severe abdominal pain out of proportion to physical examination findings should be assumed to be AMI unless proved otherwise.

Clinical symptoms help to differentiate AMI as mesenteric arterial emboli, mesenteric arterial thrombosis, NOMI or mesenteric venous thrombosis.

Conventional plain X-ray films have limited diagnostic value.

No laboratory studies can accurately identify the ischemic or necrotic bowel.

Computed tomography angiography (CTA) should be performed as soon as possible

Non-occlusive mesenteric ischemia (NOMI) should be suspected in critically ill patients with abdominal pain or distension requiring vasopressor support and multi-organ dysfunction.

With diagnosis, fluid resuscitation should be started immediately to enhance visceral perfusion. Electrolyte abnormalities should be corrected. Nasogastric decompression should be initiated.

Broad-spectrum antibiotics should be started immediately.

Prompt laparotomy should be done for patients with overt peritonitis.

When NOMI is suspected, the underlying cause should be corrected wherever possible. Mesenteric perfusion should be improved. Infarcted bowel should be resected promptly. Extensive ischemia was defined as digestive ischemia involving more than one digestive segment. Only undisputed mesenteric ischemia diagnosed by surgical or endoscopic explorations were classified as definite NOMI. Presenting a case report of a patient with Non-Occlusive Mesenteric Ischemia (NOMI) who was managed conservatively.

Case Report:

A 60 year male, with Diabetes Mellitus for 15 years, was admitted to our hospital after primary treatment from another hospital. Patient came with an acute onset of breathlessness, cough and orthopnea for 4 days. It increased on the day of admission. Patient was in cardiogenic shock on admission. He required 3 inotropes & Bi-pap ventilation. ECG showed Left Bundle Branch Block (LBBB), troponin I positive. Hemoglobin 11.7 g/dl, WBC 6400/mm. Platelet count 3 lacs/mm, Blood Urea 48 mg, Serum creatinine 2.1 mg/dl, Serum Na 133, Serum K 5.1. USG Abdomen- Liver normal, Right kidney normal, Left kidney small size with proximal & mid hydronephrosis & hydroureter,? Ureteric calculus present? Chronic obstructive uropathy. Echo showed ejection fraction 20-25%, inferobasal

& posterobasal thinning and gross hypokinesia, moderate PH 60, moderate MR, IVC 2.2cm, <50% collapsibility, jerky septal movement consistent with LBBB.

Keeping the diagnosis as Acute Coronary Syndrome with Ischemic Cardiomyopathy with Cardiogenic Shock with Acute Renal Failure (ARF), treatment was started.

Over 4 days, the patient was off Bi-pap, Inotropes were stopped & was recovering from ARF.

On day 6 Coronary Angiography was done which showed double vessel disease and was advised PTCA. Post CAG had one fever spike 101deg F. Urin c/s D3 no growth. Central line & foleys removed. Cultures sent.

On day 7 patient had loose motions 5 times, no fever. Creatinine 2.4, hemogram normal.

On day 8 again had loose motions 6 times, no fever. So antibiotic Augmentin was stopped. Central line & foley's catheter tip with blood & urine cultures came negative. PTCA to OM and RCA done.

Day 10 Patient continued to get loose motions 4 to 5 times daily but had no Malena. Had abdominal pain on left side with Malena once. patient had mild abdominal distention. So, inj enoxaparin & tab Ecosprin were stopped. ticagrelor continued. He was having intermittent hiccups also. His platelets dropped to 1 lac & leucocyte count 21,000, Sodium 126. His Dengue test came positive. Fluids and antibiotics up titrated.

On the 11th day, the patient's condition further deteriorated. He had convulsion & was obtunded. His loose motions continued 4 times but no Malena. He had no fever but blood pressure fell down to 86/50 mm Hg. WBC count further increased, Sr Na 122, Blood Urea 150mg/dl & Sr Cr was 4.2 mg/dl. Urine output was significantly decreased. Urgent CT brain was done which was normal. CT abdomen showed left hydroureter & hydronephrosis. Diffuse circumferential wall thickening is noted in ascending, transverse & descending colon with diffuse submucosal edema with wall stratification. Xray abdomen showed dilated transverse colon. Opinion of Gastroenterologist, Urologist & Neurologist was taken. Diagnosis was? Pyelonephritis? Dengue related? Ischemic bowel with contrast induced nephropathy. Patient catheterized and all cultures repeated.



Figure No 1. CT Abdomen

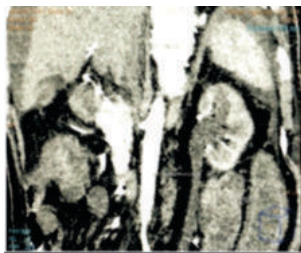


Figure No. 2 CT Abdomen- Left Hydronephrosis

Over next few days till day 15 the patient's condition improved neurologically but was remaining drowsy. His loose motions (greenish) continued and had no fever. His hemodynamics were stable. He had good urinary output. His creatinine started decreasing. Stool routine & culture showed no growth. Blood culture & urine culture showed no growth. Urologist on review advised conservative management.

On day 16, as motions became more voluminous & increased frequency, Gastroenterologist's review was done. Colonoscopy was advised.

Colonoscopy showed-

Rectum & distal sigmoid colon showed normal mucosa. Rest of the colon up to caecum showed diffuse ulcerations throughout suggestive of ischemic colitis. Multiple biopsies were taken. Terminal ileum up to distal 10 cm showed normal mucosa.

Opinion of Surgeon was sought. He advised medical management. Mesenteric Angiography was withheld in view of high creatinine.

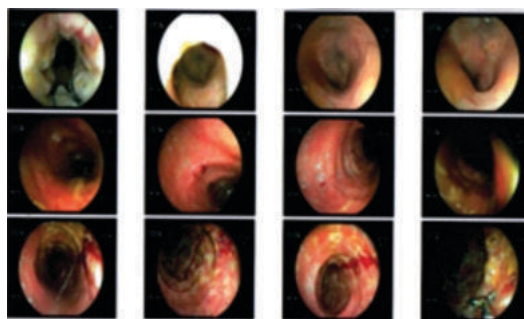


Figure No. 3 1st Colonoscopy

Patient continued to have voluminous & frequent loose motions, no Malena. Repeat Stool routine was normal and stool culture no growth. Stool for Clostridium & Bio fire came negative.

Biopsy was suggestive of Ischemic colitis.

On day 21 patient started getting fever & was not getting relief from loose motions. Gastroenterologist and surgical opinion was reviewed. CT mesenteric Angio done and was normal. Repeat stool culture & Clostridium sent. Decided conservative management.

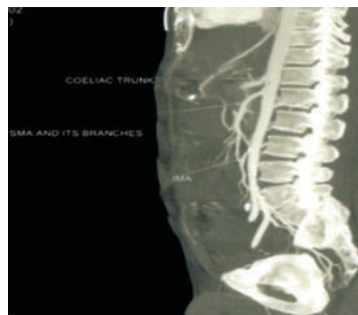


Figure No 4. Mesenteric Angio

On day 25, patient continued to have fever and voluminous and frequent loose motions, no Malena. Blood culture came positive staph epidermidis. CRBSI treated accordingly, central line removed.

Till Day 29, patient was deteriorating, getting continuous fever remaining obtunded, drowsy requiring inotropes, tachycardia, metabolic acidosis, hypernatremia was getting loose motions 15 to 17 times a day. Review gastro and surgical reference done, decided conservative management. Repeat ECHO ruled out vegetation.

Day 30 repeat Colonoscopy done showed significant improvement Rectum, sigmoid colon and descending colon appeared normal. Transverse colon and ascending colon showed presence of scattered erythema, erosions and few ulcerations. Impression: ischemic colitis. **SO CONTINUED WITH CONSERVATIVE MANAGEMENT**

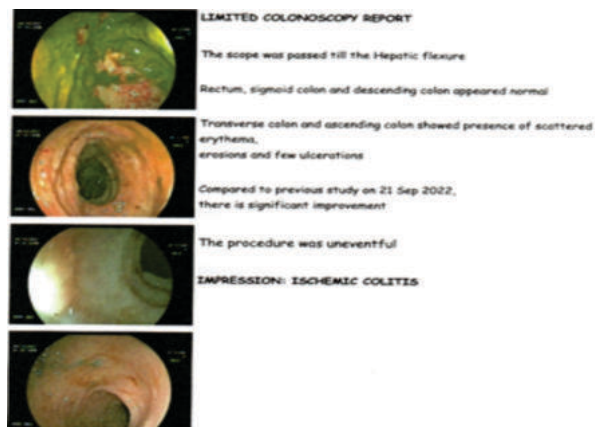


Figure No. 5 Repeat Colonoscopy

Day 36 continued to have fever, drowsiness, hypernatremia, loose motions 11 to 13 times per day. In between fungaemia is ruled out. Then had CRUTI-klebsiella.

From day 37 onward his fever started settling down. Over next 7 days he continued to have loose stools but with reduced frequency and quantity. improved, sensorium, stable hemodynamics and improvement in all blood parameters. switched to complete oral diet from parental & partial parenteral nutrition.

Day 46 patient was discharged in walking condition with complete enteral diet.

DISCUSSION:

Our patient was an elderly patient with Diabetes Mellitus for 15 years, presented in cardiogenic shock. He had Loose motions, abdominal pain, leukocytosis, metabolic acidosis, remained obtunded for long time, which was in support of advanced intestinal ischemia but may not be indicative of either reversible or frank necrosis.

His age, previous morbidity & symptoms raised a high index of suspicion of acute mesenteric ischemia (AMI). Colonoscopy confirmed the diagnosis. Mesenteric Angio was normal, so confirms diagnosis of NOMI.

Patient management and interpretations were complicated due to obstructed left kidney, dengue shock and acute renal failure due to contrast in addition.

Fluid management and nutrition was challenging due to low EF and limited use of enteral route as patient had loose motions more than 8 to 12 times / day which lasted more than 3 weeks.

Being recent PTCA and borderline platelets, managed on single antiplatelet throughout stay. Prolonged stay got complicated due to CRBSI and CRUTI.

Patient could be managed by supportive medical treatment alone successfully.

Murono K et al report a rare case of NOMI in a 72-year-old woman localized in the transverse colon. It was diagnosed by computed tomography (CT) angiography. Emergency laparotomy was performed, which revealed a segmentally necrotic transverse colon. The necrotic bowel was resected.[9]

Versyck G et al reported two cases of NOMI. In the first case, the patient developed NOMI after septic shock. She was successfully treated with surgery. The second patient experienced NOMI after two episodes of cardiac arrest. He received only supportive care.[10]

Nieh CC et al reported a case of a 76-year-old Chinese female with multiple comorbidities, a case of large bowel ischemia secondary to non-occlusive mesenteric ischemia. She was managed surgically.[11]

CONCLUSION:

Vague symptoms and physical findings, and nonspecific laboratory results can make the diagnosis of non-occlusive mesenteric ischemia (NOMI) a challenge to clinicians. Recognizing risk factors and a high index of suspicion helped us to give timely care. colonoscopy was diagnostic.

This clinical case intends to alert and remind clinicians of the possibilities of NOMI, especially in elderly with different comorbidities, particularly heart failure.

If diagnosed and treated early patient gets better in spite of high mortality and challenges in management.

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