ACUTE MESENTERIC ISCHAEMIA IN SURGICAL PERSPECTIVE- A SINGLE INSTITUTE EXPERIENCE

Shilpa Mariappa Casaba¹, Nagesh N. S², Prasanna Basavarajappa³, Venugopal H. G⁴, Vinay B. №

¹Assistant Professor, Department of Surgical Gastroenterology and Liver Transplantation, Bangalore Medical College and Research Institute, Bangalore.

²Professor and HOD, Department of Surgical Gastroenterology and Liver Transplantation, Bangalore Medical College and Research Institute, Bangalore.

³Senior Resident, Department of Surgical Gastroenterology and Liver Transplantation, Bangalore Medical College and Research Institute, Bangalore.

⁴Associate Professor, Department of Surgical Gastroenterology and Liver Transplantation, Bangalore Medical College and Research Institute, Bangalore.

⁵Professor, Department of Surgical Gastroenterology and Liver Transplantation, Bangalore Medical College and Research Institute, Bangalore.

ABSTRACT

BACKGROUND

Acute mesenteric ischaemia is a life-threatening abdominal emergency. It may be the result of an arterial or venous occlusion, a vasospasm secondary to low-flow states in ICU patients. The overall incidence is 1 in 1000 hospital admissions. The objective of this descriptive study was to determine the clinical presentations and outcome after surgery of patients with acute mesenteric ischaemia. It was conducted in Department of Surgical Gastroenterology and Liver Transplantation, BMCRI, Bangalore.

MATERIALS AND METHODS

Descriptive study of all patients having operative diagnosis of acute mesenteric ischaemia from June 2009 to December 2016 were included.

RESULTS

Total of 31 patients were studied. Their mean age was 60.12 years, 17 were males and 14 were females. Mean BMI was 32.4. Abdominal pain was present in 26 patients, vomiting in 21 and anorexia in 13 patients. Abdominal tenderness was present in 21 patients, abdominal distension and rebound tenderness in 18 patients. Eighteen patients had hypertension, 10 had coronary artery disease and 20 had diabetes mellitus. Eight patients were in haemodynamic instability and 4 patients in renal failure. Except one patient, all underwent CECT abdomen and pelvis. Four patients were managed conservatively with close monitoring after diagnostic laparoscopy. Resection of bowel was done in 27 patients. Two cases had re-exploration. Nine patients deteriorated haemodynamically as sepsis was irreversible and died subsequently. Common immediate postoperative complications are wound infection, dehiscence, pneumonia, septicaemia and MODS. On followup at 1 month, 9 patients were seen with improved nutritional status without any complications. The common complications noted at 1st month are persistent diarrhoea, deep wound infection, stromal complications, respiratory infection, melena, etc.

CONCLUSION

Acute Mesenteric Ischaemia (AMI) is a common condition at a GI referral centre. It is commonly distributed among elderly men and women with pre-existing risk factors. The prognosis depends upon time at presentation, general condition at presentation, associated risk factors, extent of bowel gangrene and extent of bowel resected. In high-risk patients with pain abdomen of unexplained cause, we should have a low threshold for diagnosing acute mesenteric ischaemia as our study showed PPV clinical diagnosis of AMI is 57%. Timely diagnosis will prevent the progression of bowel gangrene and early surgical intervention is warranted for better clinical outcome.

KEYWORDS

Acute Mesenteric Ischaemia (AMI), Bowel Gangrene, Stoma, Heparin.

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Corresponding Author:
Dr. Prasanna Basavarajappa,
No. 250, 1st Main, 6th Phase,

Mahaganapathinagar, Rajajinagar, Bangalore-560010. E-mail: prasisibaka@gmail.com

E-mail: prasisibaka@gmail.com DOI: 10.18410/jebmh/2017/339



BACKGROUND

Acute Mesenteric Ischaemia (AMI) is defined as a clinical entity secondary to sudden arterial or venous occlusion or drop in circulating pressure resulting in insufficient blood flow in the splanchnic circulation.¹

AMI accounts for about 1:1000 acute hospital admissions in Europe and the USA. 2 In Japan, it is $1:10,000.^3$ The incidence appears to be increasing due to an ageing general population with increasing prevalence of comorbidities. This pre-existing disease worsens the prognosis of intestinal gangrene. 4

Sudden occlusion of the superior mesenteric artery by an embolus orthrombus in patients with pre-existing wall alterations. 5Predisposing factors for AMAT or AMAE are cardiac arrhythmia particularly a trial fibrillation, coronary heart disease, clinical status following myocardial infarction and Peripheral Arterial Occlusive Disease (PAOD). Clinical features are sudden-onset abdominal pain, vomiting, melena, etc.

Arterial nonocclusive ischaemia caused by reduction in cardiac output with reactive mesenteric vessel spasm. Predisposing factors are heart surgery with extra corporeal circulation, particularly with complicated disease course, long-term haemodialysis, digitalis medication, etc. Clinical features are increasing abdominal pain, abdominal distension, increase in inflammatory parameters and signs of sepsis.

Venous thrombosis of the mesenteric portal axis occurs secondary to paraneoplasia, pancreatitis, pancreatic carcinoma, congenital thrombophilia (e.g.AT III deficiency, protein C deficiency, protein S deficiency), HCC (hepatocellular carcinoma) with macrovascular invasion.

In spite of the major progresses in the diagnostic tools and treatment in the past years due to the low rate of clinical suspicion, the late presentation at the hospital, the advanced age of the patients and the present comorbidities, the mortality rate is still estimated to be 40-70%. Fin this article, we presented 31 cases of AMI that have been diagnosed with a spectrum of risk factors for AMI. Twenty seven managed with emergency laparotomy and 4 cases managed conservatively.

AIMS AND OBJECTIVES

The aim of this descriptive study is to find the clinicodemographic domain and short-term outcome after surgical intervention in AMI (acute mesenteric ischaemia).

MATERIALS AND METHODS

We studied patients at our institute (Department of Surgical Gastroenterology and Liver Transplantation, BMCRI, Bangalore), having operative diagnosis of acute mesenteric ischaemia from June 2009 to December 2016. The patients were studied for mode of presentation, intraoperative findings, postoperative course and followed up for 1 month.

Inclusion Criteria

We included all patients who had intraoperative diagnosis (both on exploratory laparotomy and diagnostic laparoscopy) of acute mesenteric ischaemia.

Exclusion Criteria

- 1. Patients clinically suspected AMI, but with different diagnosis on exploration.
- 2. Patients with AMI with surgical intervention elsewhere.

RESULTS

Total of 31 patients were studied. Their mean age was 60.12 years, 17 were males and 14 were females. Mean BMI was 32.4.Abdominal pain was present in 26 patients, vomiting in 21 and anorexia in 13 patients. Abdominal tenderness was present in 21 patients, abdominal distension and rebound tenderness in 18 patients. Eighteen patients had hypertension, 10 had coronary artery disease and 20 had diabetes mellitus. Eight patients were in haemodynamic instability and 4 patients in renal failure. Except one patient, all underwent CECT abdomen and pelvis (Table 1).



Image 1. X-Ray Abdomen -Dilated Small and Large Bowel Loops

Case	Age/ Sex	Comorbidities	Haemodynamic instability	RF	CECT±CT Angiogram	Intraoper ative Findings	Length of Bowel Resected	Stoma Mucus Fistula
1.	58/F	DM Obesity	No	No	SMV Thrombosis Ischaemic changes in the terminal ileum Minimal Ascites	Ischaemic changes in the distal ileum ~50 cm Minimal Ascites	60 cm distal ileum(10 cm from ICV left as it was normal)	Ileostomy+ Mucus fistula
2.	61/M Sm., Alc.	CAD DM	No	No	Minimally-distended ileal bowel loops Vessels normal	Dusky discoloration of mid ileal loop ~ 5 cm	-	-
3.	57/F	DM	No	No	SMV+PV thrombosis Ischaemic changes in the jejunal bowel loops Minimal ascites	Gangrenous changes in the distal jejunal bowel loops~10 cm Minimal ascites	~20 cm distal jejunum	Primary anastomosis
4.	50/M Sm	CAD (H/O AMI) HTN	Yes	No	Ischaemic changes in the terminal ileum and ascending colon	Extensive gangrene of bowel from mid jejunum upto descending colon	Resection from mid jejunum upto descending colon ~300cm	Jejunostomy+ mucus fistula
5.	71/F	DM HTN	No	No	SMV+PV Thrombosis Ischaemic changes in the jejunal bowel loops Minimal ascites	Gangrenous mid-jejunal bowel loop Minimal ascites	60 cm mid- jejunum	Jejunostomy+ mucus fistula
6.	56/M Sm. Al.	CAD ST.CABG (H/O AMI) HTN	No	No	SMV+PV partial thrombosis Distended large and small bowel	Ascending colon	Right hemicolectomy~ 70 cm	Ileostomy+ colonic mucus fistula
7.	49/M	HTN Obesity, DM	No	No	Ischaemic changes in jejunum and ileum	Extensive gangrene of bowel from proximal jejunum ~15 cm from DJF upto distal ileum Haemorrhagic peritoneal fluid +	250 cm	Jejunostomy+ ileal mucus fistula
8.	52/F Alc.	HTN	No	No	Distended small bowel Minimal ascites Vessels normal	Patchy dusky discoloration of distal jejunal loop	-	-
9.	51/M Alc.	DM HTN	No	No	Atherosclerosis in Aorta SMA partial thrombosis Bowel normal	Gangrenous changes in mid jejunum	40 cm of mid- jejunum	Primary anastomosis

10.	60/F	CAD HTN PAOD	Yes	No	Middle SMA embolus Ischaemic changes Distal jejunum and Proximal ileal loops Clot in the left ventricle of heart	Gangrenous changes in distal jejunum and proximal ileum	70 cm of distal jejunum and proximal ileum	Jejunostomy +ileal mucus fistula
11.	53/M Sm.	DM	Yes	Yes	SMV+PV+IMV thrombosis Distended small and large bowel loop Moderate ascites	Extensive gangrene of bowel from mid ileum up to transverse colon Purulent collection+	100 cm of distal ileum and colon	colonic mucuic
12.	81/F	DM CAD (H/O AMI) HTN OBESITY	Yes	Yes	Not done direct surgery	Extensive gangrene of small bowel from proximal jejunum up to distal haemorrhagic peritoneal fluid ileum	~340 cm of small bowel	Jejunostomy+ leal Mucus fistula
13.	50/M Sm Alc.	-	No	No	Vessels normal Thickening of mid jejunal segment	Dusky discoloration of mid jejunal loop	-	-
14.	61/M	HTN Obesity DM	No		SMV complete thrombosisdistal jejunum and proximal ileal loops Moderate ascites	Gangrenous changes in distal jejunum and proximal ileum	60 cm of distal jejunum and proximal ileum	Jejunostomy+i leal Mucus fistula
15.	59/F	DM Obesity	No	No	SMV thrombosis Ischaemic changes in the terminal ileum	Gangrenous changes in the distal jejunal bowel loops~20 cm Minimal ascites	~30 cm distal jejunum	Primary anastomosis
16.	66/M Sm. Alc.	CAD DM	No	No	Minimally-distended jejunal bowel loops Vessels normal	Extensive gangrene of bowel from mid jejunum upto descending colon	Resection from mid jejunum upto descending colon ~320cm	Jejunostomy+ mucus fistula
17.	58/F	DM	No	No	SMV+PV thrombosis Ischaemic changes in the jejunal bowel loops Minimal ascites	Ischaemic changes in the distal ileum ~40 cm Minimal ascites	50 cm distal ileum(10 cm from ICV left as it was normal)	Ileostomy+ mucus fistula
18.	53/M Sm	CAD (H/O AMI) HTN	Yes	No	Ischaemic changes in the terminal ileum and ascending colon	Dusky discoloration of mid ileal loop ~ 10 cm	-	-

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19.	74/F	DM HTN	No	No	SMV+PV thrombosis Ischaemic changes in the proximal ileal bowel loops Minimal ascites	Gangrenous distal-jejunal bowel loop Minimal ascites	60 cm distal- jejunum	Jejunostomy+ mucus fistula
20.	51/M Sm. Al.	CAD ST.CABG (H/O AMI) HTN	No	No	SMV+PV partial thrombosis Distended large and small bowel	Dusky distal ileum and ascending colon	Right hemicolectomy~ 60 cm	Ileostomy+ colonic mucus fistula
21.	48/M	HTN Obesity DM	No	No	Ischaemic changes in jejunum and ileum Minimal ascites	Extensive gangrene of bowel from proximal jejunum ~20 cm from DJF upto distal ileum Haemorrhagic peritoneal fluid +	230 cm	Jejunostomy+i lealmucus fistula
22.	54/F Alc.	HTN	No	No	Distended small bowel Minimal ascites Vessels normal	Patchy dusky discoloration of distal jejunal loop	-	-
23.	56/M Alc.	DM	No	No	SMA subtotal thrombosis Bowel normal	Gangrenous changes in mid jejunum	55 cm of mid- jejunum	Primary anastomosis
24.	62/F	CAD HTN	Yes	No	SMA subtotal thrombosis Bowel distended	Gangrenous changes in distal jejunum and proximal ileum	80 cm ofdistal jejunum and proximal ileum	Jejunostomy+i lealmucus fistula
25.	55/M Sm.	DM	Yes	YES	SMV+PV+IMV thrombosis Distended small and large bowel loop Moderate ascites	Extensive gangrene of bowel from mid ileum up to transverse colon Purulent collection+	90 cm of distal ileum and colon	Ileostomy+ colonic mucus fistula
26.	83/F	DM Obesity	Yes	YES	SMV complete thrombosis Distended small bowel loops Moderate ascites	Dusky discoloration of mid jejunal loop	-	-
27.	52/M Sm Alc.	-	No	No	vessels normal Thickening of mid jejunal segment	Extensive gangrene of small bowel from proximal jejunum up to distal haemorrhagic peritoneal fluid ileum	~310 cm of small bowel	Jejunostomy+i ilealmucus fistula
28.	63/M	HTN Obesity DM	No	No	SMV complete thrombosis distal jejunum and proximal	Gangrenous changes in distal jejunum	50 cm of distal jejunum and	Jejunostomy+i lealmucus fistula

					ileal loops Moderate ascites	and proximal ileum	proximal ileum	
29.	70/F	DM HTN	No	Yes	SMV+PV thrombosis Ischaemic changes in the jejunal bowel loops Minimal ascites	Extensive gangrene of bowel from mid jejunum upto descending colon	Resection from mid jejunum upto descending colon ~280cm	Jejunostomy+ mucus fistula
30.	59/M	HTN CAD	No	No	SMV thrombosis Ischaemic changes in the terminal ileum	Dusky discoloration of proximal jejunal loop	-	-
31.	68/F	DM OBESITY	No	No	Pneumatosis intestinalis in distal ileal and ascending colon Ascites +	Dusky distal ileum and ascending colon	Right hemicolectomy~ 70 cm	Ileostomy+ colonic mucus fistula
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Table 1:Clinico-Epidemiological and Operative Findings

- EL- Exploratory Laparotomy
- DL- Diagnostic Laparoscopy
- FJ- Feeding Jejunostomy

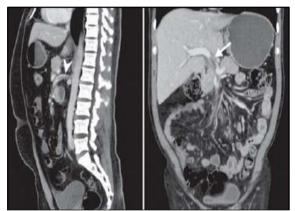


Image 2. CECT-SMA Thrombosis (Left)
PV-SMV Thrombosis (Right)



Image 3. CECT- Dilated and Thickened Small Bowel



Image 4. CECT-Long Segment SMA Thrombosis

Once in triage, the patient with suspected mesenteric ischaemia was quickly assessed for haemodynamic stability, acidotic status, renal function, etc. Depending on severity of the condition, patients will be admitted to ICU, HDU or ward. Some patients had come with CECT and other investigations done outside, which is suggestive of AMI. If not done after optimisation of condition, CECT

abdomen with or without CT angiogram abdomen was obtained. Once diagnosed, patient will be put on IV heparin therapy (titrated to keep APTT 60-80 secs.). The type of intervention surgery/diagnostic laparoscopy/conservative was depend on the patient's clinical condition and progress and CECT findings. Table 2 depicts operative synthesis of our case series.

Case No.	Postoperative Course/Complication	Re-Exploration	PODs	Mortality	Followup at 1 Month
1.	Pneumonia Stomal complications Deep wound infection	-	9	-	Stomal complications
2.	Improved	-	6	-	Gained weight
3.	Improved Deep wound infection	-	7	-	Deep Wound infection Short bowel syndrome
4.	Septicaemia MODS	-	2	Yes	-
5.	Improved Deep wound infection	-	9	-	Intractable diarrhoea
6.	Pneumonia Wound infection Melaena	-	8	-	Deep Wound infection
7.	Blackish discoloration of stoma Septicaemia	Yes Resection of proximal jejunum	4	Yes	-
8.	Improved	-	5	-	Uneventful
9.	Pneumonia DVT	-	12	-	Improved
10.	Pneumonia Improved	-	6	-	Improved
11.	Septicaemia persistent MODS Melena	-	1	Yes	-
12.	Bleeding from jejunostomy site Septicaemia	Yes Resection of proximal jejunum	2	Yes Died immediately after re- exploration	-
13.	Improved	-	6	-	Improved nutrition
14.	Improved deep wound infection	-	11	-	Persistent diarrhoea Deep wound infection Melaena
15.	Pneumonia Stomal complications Deep wound infection	-	9	-	Stomal complications
16.	Improved	-	6	-	Gained weight
17.	Improved deep wound infection	-	7	-	Deep wound infection Short bowel syndrome
18.	Septicaemia MODS	-	2	Yes	-
19.	Improveddeep wound infection	-	9	-	Intractable diarrhoea

20.	Pneumonia Wound infection Melaena	-	8		Deep wound infection			
21.	Blackish discoloration of stoma Septicaemia	Yes Resection of proximal jejunum	4	YES	-			
22.	Improved	-	5	-	Uneventful			
23.	Pneumonia DVT	-	12	-	Improved			
24.	Pneumonia Improved	-	6	-	Improved			
25.	Septicaemia persistent MODS Melaena	-	1	Yes	-			
26.	Improved	-	8		Improved nutrition			
27.	Septicaemia	-	3	Yes	-			
28.	Improved deep wound infection	-	11	-	Persistent diarrhoea Deep wound infection Melena			
29.	Wound dehiscence Septicaemia	-	2	Yes	-			
30.	Improved	-	7	-	Improved nutrition			
31.	Improved deep wound infection	-	12	-	Persistent diarrhoea Deep wound infection			
	Table 2. Postoperative Course and Follow Up							

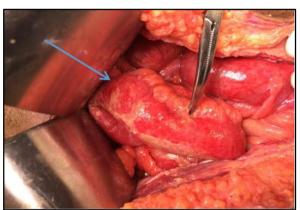


Image 5. Thickened and Dusky Mid Jejunum (Arrow)



Image 6. Gangrene of Proximal Jejunal Segment



Image 7. Extensive Gangrene of Small and Large Bowel



Image 8. Post Resection of Distal Jejunum

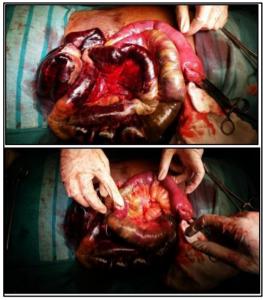


Image 9 and 10. Extensive Gangrene of Small and Large Bowel



Image 11. Gangrenous Ascending Colon



Image 12. Completion of S-S Jejuno jejunostomy

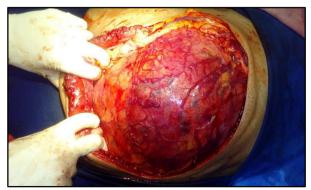


Image 13. On Exploration of Case 11-Purulent Collection



Image 14. Dusky Distal Jejunum and Proximal Ileum



Image 15. Jejunostomy and Ileal Mucus Fistula

All patients were managed with IV heparin therapy, TPN, fluid and electrolyte supplementation according to ESTES guidelines. Patients were put on oral anticoagulation (T.warfarin) to maintain INR of 2-3 for minimum 6 months or lifelong (if procoagulation abnormality identified). Four patients who underwent diagnostic laparoscopy were closely observed for any clinical signs of progressive bowel ischaemia with IV heparin therapy. All 4 patients improved clinically without any clinical signs of progressive bowel ischaemia. They were allowed orally after 2 days and tolerated well. Discharged subsequently. Two cases had reexploration. Case 7 had blackish discoloration of stoma on POD2. Re-explored to resect proximal jejunum. Patient died on POD 4 with persistent septicaemia and MODS. Case 12 had bleeding from jejunostomy with hypotension. Reexplored immediately to take-down stoma and resect bleeding proximal segment of jejunum. But, patient died in immediate postoperative period. Four patients deteriorated haemodynamically as sepsis was irreversible and died subsequently. Three patients had pneumonia and wound infection, but recovered in 6 days. As there was a clot in the left ventricle of case 10, patient was referred to Jayadeva Cardiac Centre. On followup at 1 month, 9 patients were seen with improved nutritional status without any complications. The common complications noted at 1st month are persistent diarrhoea, deep wound infection, stromal complications, respiratory infection, melena, etc. One patient had lost followup.



Image 16. Re-Exploration of Case-7



Image 17. Re-Exploration of Case-12

DISCUSSION

The aetiology of acute mesenteric ischaemia can be embolism or acute thrombosis. The SMA, mesenteric venous thrombosis or splanchnic vasoconstriction leading to regional ischaemia that is called Non-occlusive Mesenteric Ischaemia (NOMI). Although, acute mesenteric vessel thrombosis is most frequently caused by the migration of thrombus to the systemic circulation due to any underlying cardiac problems or any procoagulant state. To any case series, acute mesenteric vein thrombosis was most common cause (16-18/31).

Clinical evaluation need to be focused on the inability to tolerate RT feeds, GI bleed, metabolicacidosis despite adequate resuscitation and septic indicators. CECT with angiography remain a better imaging option for visualisation of the vessels and a therapeutic intervention. Its routine use is questioned in emergency situations due to its complications, and therefore, it is employed selectively. Suggested indications for selective mesenteric angiography as a- (i) Absence of defecation for more than three day safter surgery despite maximal supportive treatment, (ii) Severe abdominal distension, (iii) Clinical and radiologic signs of paralytic ileus or(iv) Raised serum lactate (Klotz et al) when used in the absence of peritonitis signs, angiography has been shown to improve the survival rate.¹⁰ A normal CT finding in a patient with high suspicion of mesenteric ischaemia should prompt an angiography without delay.11 In our series, 11/31 patients had CT angiography as an adjunct diagnostic procedure.

Early intervention of AMI (within 6-8 hours) has resulted in a good postoperative outcome. Diagnostic laparoscopy has a high sensitivity (94%) for the correct diagnosis of intra-abdominal complications after major cardiacsurgery. Probably, since our institute is a super speciality referral center, most of the cases (24/31) we receive were cases with late presentation and with CECT diagnosis or suspicion of AMI (acute mesenteric ischaemia). The de novo cases (7/31) with pain abdomen, which turn out to be AMI are only few. In our case series, we had 4 such patients (PPV-57%). Another 3 patients, we suspected AMI, but turnout to be different diagnosis on CECT/diagnostic laparoscopy. In our series, 4/31 patients had underwent diagnostic laparoscopy were closely observed for any clinical signs of progressive bowel

ischaemia with IV heparin therapy. All 4 patients improved clinically without any clinical signs of progressive bowel ischaemia. They were allowed orally after 2 days and tolerated well. Discharged subsequently. Another mode of investigation been described in literature is diagnostic peritoneal lavage. Lee and colleague in their retrospective study have concluded diagnostic peritoneal lavage is associated with reduced operative intervention, yet improved survival when compared with patients evaluated with either CT or no diagnostic modality. These data support that for critically ill patients suspected of harbouringintra-abdominal pathology such as acute mesenteric ischaemia, diagnostic peritoneal lavage should be a mainstay in the preoperative diagnostic evaluation. We had no such patients.

Emergency surgical intervention remains the gold standard treatment for central occlusion of the SMA, failure of endovascular treatment or peritonitis. Cooperation between visceral and vascular surgeons is paramount. The principle of arterial reperfusion before intestinal resection is considered. From a vascular point of view, good experience of embolectomy as well as vascular reconstruction techniques for visceral arteries is required. 13 Eradicating abdominal infection on the other hand involves identifying and resecting gangrenous segments of the intestine. If relative ischaemia of nonresected portions of the intestine is not noticed and an anastomosis is created, this results in anastomotic failure and a high mortality rate.14 In case of doubt, the ends of the intestine are kept out as stoma. It also allows endoscopic follow-up. If any preserved portions of the intestine have uncertain reperfusion, second-look surgery must be planned within 12 hours of initial surgery. We had 2 such patients who underwent re-exploration. In addition, repeat exploration is indicated if the patient's condition fails to stabilise. Short bowel syndrome is expected if there is inability to following minimal length of bowel, 100 cm for terminal jejunostomy (colon removed), 65 cm for jejunocolic anastomosis (colon retained) and 35 cm for jejunoileal anastomosis with retention of the ileocecal region.15

In postoperative period, intensive care must be continued until all acute phase parameters return to normal values and all organ functions are stable. In particular, the end products of infection, ischaemia and bacterial

translocation can lead to a septic clinical picture in the longer term. In the initial postoperative course, one should be vigilant for the effects of ischaemia/reperfusion on abdominal, cardiac, pulmonary and renal function. In our series, all patients were managed with IV heparin therapy, TPN, fluid and electrolyte supplementation according to ESTES Guidelines. Four cases who underwent diagnostic laparoscopy were closely observed for any clinical signs of progressive bowel ischaemia with IV heparin therapy. All 4 patients improved clinically and discharged subsequently. Two cases had re-exploration. One patient had blackish discoloration of stoma on POD2.Re-explored to resect proximal jejunum. Patient died on POD 4 with persistent septicaemia and MODS. One patient had bleeding from ieiunostomy with hypotension. Re-explored immediately to take-down stoma and resect bleeding proximal segment of jejunum. But, patient died in immediate postoperative period. Two patients deteriorated haemodynamically as sepsis was irreversible and died on POD 2 and 1, respectively. One patient had a clot in the left ventricle. Patient was referred to a higher cardiac centre.

CONCLUSION

Acute Mesenteric Ischaemia (AMI) is a common condition at a GI referral centre. It is commonly distributed among elderly men and women with pre-existing risk factors. The prognosis depends upon time at presentation, general condition at presentation, associated risk factors, extent of bowel gangrene and extent of bowel resected. Older patients with delayed presentation and abdominal signs of peritonitis or organ failure generally have a worse prognosis. In high-risk patients with pain abdomen of unexplained cause, we should have a low threshold for diagnosing acute mesenteric ischaemia as our study showed PPV clinical diagnosis of AMI is 57%. Timely diagnosis will prevent the progression of bowel gangrene and early surgical intervention is warranted for better clinical outcome.

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