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Original Research Article

Acute mesenteric ischemia: a review of 50 cases

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ABSTRACT

Background: Acute mesenteric ischemia is a rare but highly lethal cause of abdominal pain. Diagnosis should be prompt to ensure early treatment and avoid progression of the disease. The aim of the study was to describe the population presenting with acute mesenteric ischemia submitted to surgery, evaluate their outcome and determine possible predictors of mortality.
Methods: Retrospective analysis of all patients who underwent surgery due to mesenteric ischemia between May 2013 and January 2015. A total of 50 patients were included. Pre-operative patient risk factors, diagnostic and surgical approach, post-operative complications and outcome were analysed. Statistical analysis was performed using SPSS v. 20. Univariable analysis was performed using T student and chi square tests.
Results: Mean age was 79 years. Most patients (n=43) had cardiovascular risk factors. Clinical and laboratory studies findings were non-specific. During surgery, 28% were found to have extensive ischemia. Exclusive exploratory laparotomy was done in 11 cases due to extensive ischemia. Thrombectomy was performed in 6 cases. Eleven patients were submitted to reoperation. In-hospital mortality was 68% (n=34). Patients who died had more frequently systemic findings and extensive ischemia. Patients with extensive vs. segmental ischemia were also compared and no statistically significant differences regarding preoperative findings were found. Mortality was significantly higher in patients with extensive ischemia.
Conclusions: Timely diagnosis and treatment are essential to improve the outcome of AMI but tools to perform this are still lacking. Mortality remains high, especially when ischemia is extensive at the time of surgery.

Keywords: Mesenteric ischemia, Nonocclusive mesenteric ischemia, Occlusive mesenteric arterial ischemia

INTRODUCTION

Acute mesenteric ischemia (AMI) is a rare cause of abdominal pain in the emergency department. It is frequently life-threatening or even lethal.[1] Although there are advances in diagnosis and treatment approach, the most important factor determining outcome is a rapid diagnosis and intervention.[2] Clinical presentation of patients with acute mesenteric ischemia is frequently dominated by abdominal pain out of proportion to the physical examination, but other forms of presentation might occur. Serum biomarkers are not as valuable for early diagnosis as it would be expected.[3] Computed tomographic angiography (CTA) has become the standard for the diagnosis of acute mesenteric ischemia. Management of these patients, when hemodynamically unstable, implies careful fluid replacement and avoidance of vasoconstrictor drugs, as much as possible. Treatment may include endovascular therapies, which aim to restore perfusion of the bowel, and surgery which, in addition to revascularization, also allows assessment of viability and resection of necrotic bowel.[2]

Our aim was to describe the population presenting with acute mesenteric ischemia submitted to surgery, evaluate

their outcome and determine possible predictors of mortality.

METHODS

We performed a retrospective analysis of all patients who underwent surgery due to mesenteric ischemia between May 2013 and January 2015 in Hospital Beatriz Ângelo, Loures, and a secondary hospital in Portugal. A total of 50 patients fulfilled these criteria No exclusion criteria was applied. We analysed pre-operative patient risk factors, clinical presentation, diagnostic and surgical approach, post-operative complications and outcome.

Statistical analysis was performed using SPSS v. 20. Univariable analysis was performed using T student and chi square tests as appropriate. Statistical significance was considered for $p<0.05$.

RESULTS

Mean age was 79 years and 80% of patients were 75 or more years old. Twenty four patients (48%) were female. Twenty seven patients had at least one risk factor for mesenteric ischemia (Table 1), 23 had cardiac risk factors and 43 had cardiovascular risk factors.

Table 1: Risk factors for mesenteric ischemia, cardiac risk factors for mesenteric ischemia and cardiovascular risk factors.

Risk factors		N (%)
Risk factors for mesenteric ischemia	Cardiac disease	21 (42)
	Aortic surgery/instrumentation	2 (4)
	Peripheral artery disease	4 (8)
	Haemodialysis	4 (8)
	Bowel strangulation	1 (2)
Cardiac risk factors for mesenteric ischemia	Ischemic heart disease	11 (22)
	Atrial fibrillation	16 (32)
	Valvular heart disease	2 (4)
Cardiovascular risk factors	Diabetes mellitus	20 (40)
	High blood pressure	34 (64)
	Dyslipidaemia	14 (14)
	Obesity	4 (8)
	Smoking	3 (6)

The most frequent clinical signs were abdominal pain and systemic symptoms (Table 2). Regarding laboratory findings, elevation of lactate, creatinine, urea and C reactive protein (CRP) were the most prevalent (Table 3).

Imaging methods were performed in almost every patient. CT/angio-CT was performed in 49 out of the 50 patients evaluated. Abdominal X-ray, colonoscopy, ultrasonography and upper endoscopy were other diagnostic tools. The main findings reported in imaging tests are summarized in Table 4.

Table 2: Symptoms and signs at presentation.

		N (%)
Symptoms	Acute abdominal pain	35 (70)
	Nausea/vomiting/refusal to eat	28 (56)
	Melena haematochezia	4 (8)
	Hematemesis	5 (10)
	Diarrhoea	7 (14)
	Constipation	9 (18)
	Respiratory distress	6 (12)
	Altered mental status	5 (10)
Signs	Diffuse abdominal pain on palpation	28 (56)
	Localized abdominal pain on palpation	10 (20)
	Peritoneal irritation signs	12 (24)
	Abdominal distention	20 (40)
	Tachycardia	3 (6)
	Hypotension	22 (44)
	Poor peripheral perfusion	3 (6)

Table 3: Preoperative laboratory tests results.

Results	Mean	N (%)
Haemoglobin (g/dL)	12.6	49 (98)
Haematocrit (%)	38.7	49 (98)
Leucocyte count (n/µL)	15 724	49 (98)
Ldh (mg/dL)	330.8	36 (72)
Lactate (mg/dL)	36.4	39 (78)
Creatinine (mg/dL)	2.3	49 (98)
Urea (mg/dl)	90	49 (98)
Amylase (ul/L)	115	30 (60)
Phosphate (mg/dL)	4.1	12 (24)
CRP (mg/dL)	19.6	49 (98)
D dimer (mg/L)	43.3	10 (20)

N indicates the number of available results. CRP: C reactive protein; LDH: Lactate dehydrogenase.

After reviewing clinical, biochemical and imaging results, a preoperative diagnosis was determined. Most patients were submitted to surgery already with the suspicion of AMI, in others only complications of the disease were recognized, such as visceral perforation (Table 5).

During surgery, 28% were found to have extensive ischemia, affecting the small intestine in 40 patients and the colon in 25. Regarding surgical procedures, most patients underwent a combination of resection and primary anastomosis, stoma construction or stapling off bowel ends. Exclusive exploratory laparotomy without any further procedure was done in 14 cases-in 3 of them due to viability of the whole bowel and in the remaining

cases due to extensive ischemia that precluded further surgical procedures (Figure 1). Thrombectomy was performed in 6 cases. In one case, correction of an incarcerated hernia was performed.

Table 4: Main preoperative imaging findings.

Findings	N (%)
Bowel wall injury	37 (74)
Parietal pneumatosis	18 (36)
Bowel wall thickening	18 (36)
Pneumoperitoneum/visceral perforation	10 (20)
Vascular findings	11 (22)
Arterial thrombosis	9 (18)
Venous and arterial thrombosis	1 (2)
Atherosclerosis plaques	1 (2)
Nonspecific findings	39 (78)
Visceral distension	29 (58)
Free peritoneal fluid	14 (28)
Mesenteric fat stranding	12 (24)
Hydroaeric level	5 (10)
Bowel strangulation	5 (10)
Internal hernia/adhesion	3 (6)
Faecaloma	1 (2)
Intestinal invagination	1 (2)
No relevant findings	1 (2)

Table 5: Preoperative diagnosis.

Preoperative diagnosis	N (%)
Mesenteric ischemia	33 (66)
Acute abdomen of unknown cause	4 (8)
Intestinal oclusion	4 (8)
Visceral perforation	5 (10)
Intestinal invagination	1 (2)
Complicated acute diverticulitis	1 (2)
Other	2 (4)

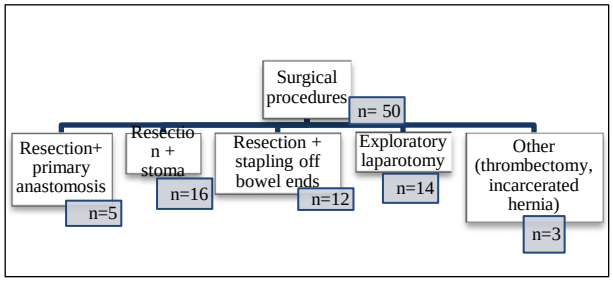

Figure 1: Surgical procedures.

Eleven patients were submitted to reoperation, 9 as a second look or planned reoperation and 2 due to complications (Figure 2). One underwent bowel anastomosis due to good evolution. Among the unplanned reoperations, one was due to anastomosis dehiscence with subsequent ileostomy and mucous fistula and the other presented progression of ischemia (no bowel had been resected in the first surgery-only an incarcerated hernia had been corrected) and resection of the ischemia bowel was performed, with simultaneous primary anastomosis.

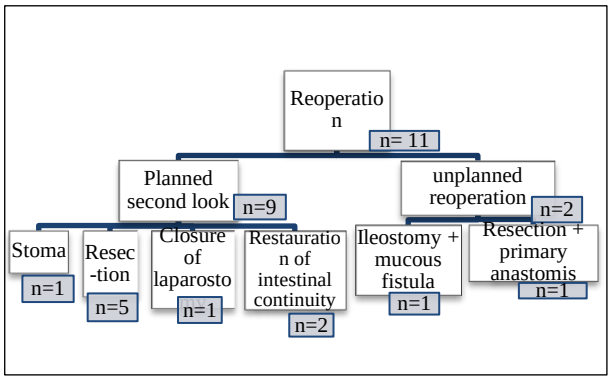

Figure 2: Surgical procedures at reoperation.

The most frequent postoperative complications were renal failure (n=9) and nosocomial infection not related with surgical site (n=9). Three patients had multiple organ failure and 2 had a new episode of mesenteric ischemia. In-hospital mortality was 68% (n=34).

We compared patients who died and those who survived regarding clinical, laboratorial, imaging and surgical findings. Patients who died had more frequently systemic findings, such as altered mental status, respiratory distress and hypotension, although it was not a statistically significant difference. The only laboratory results significantly different between patients who died and those who survived were preoperative haemoglobin and postoperative lactate levels. Patients who died had extensive ischemia more frequently (Tables 6 and 7).

Table 6: Comparison of patients alive vs. dead regarding clinical presentation and surgical treatment.

Variables	Alive (n=16)	Dead (n=34)	P value
Time between admission and surgery (days)	Mean: 0.93 85.7% <48h	Mean: 0.93 77.8% <48h	0.99
Altered mental status N (%)	1 (6.3)	4 (11.8)	1
Respiratory distress N (%)	1 (6.3)	5 (14.7)	0.65
Hematemesis N (%)	0 (0)	5 (14.7)	0.16
Hypotension N (%)	4 (25)	18 (52.9)	0.213
Reoperation N (%)	4 (25)	7 (20.6)	0.725

We also compared patients with extensive vs. segmental ischemia regarding laboratory and imaging findings and status at discharge. There were no statistically significant differences regarding preoperative findings. Mortality was significantly higher in patients with extensive ischemia (92.3% vs 58.3%, p= 0.021) (Table 8).

Table 7: Comparison of patients dead vs. alive regarding age, serum markers, surgical and imaging findings.

Parameters		Alive (n=16)	Dead (n=34)	P value
Age (mean± S.D.)		74.4 ± 13.4	81.1±6.4	0.069
LDH (mg/dL) (mean± SD) (mg/dL)		307.6 ±110.0	341.0 ±104.3	0.391
Lactate (mg/dL) (mean± SD)		27.9±20.2	39.3±22.2	0.159
Haemoglobin (g/dL) (mean± SD)		14.0±2.7	12.0±2.8	0.021
Leukocytes (n/µl) (mean±SD)		15675.0±10198.3	15748.5±12191.7	0.983
Creatinine (mg/dL) (mean±SD)		2.0±1.2	2.4±1.9	0.401
Phosphate (mg/dL) (mean±SD)		2.9±0.1	4.3±2.0	0.050
CRP (mean±SD) (mg/dL)		15.9±14.32	21.5±17.2	0.269
D-dimer (mg/dL) (mean±SD)		117.8±165	11.41±12.6	0.380
Amylase (mg/dL) (mean±SD)		101.8±75.5	124.4±97.5	0.503
CRP post-op (mg/dL) (mean±SD)		28.2±10.1	33.9±8.7	0.144
Lactate post-op (mg/dL) (mean±SD)		11.1±10.0	41.5±37.0	0.01
Surgical findings:	Extensive ischemia	1 (6.25%)	13 (38.2%)	0.021
Imaging findings	Bowel suffering	12 (75%)	25 (73.5%)	1
	Vascular alterations	2 (12.5%)	9 (26.5%)	0.466

CRP: C reactive protein; LDH: Lactate dehydrogenase, T student test/ Fisher test: p value significant <0.05.

Table 8: Comparison of patients with extensive vs. segmental ischemia regarding age, serum marker, outcome and imaging findings.

Parameters		Extensive ischemia (n=14)	Segmental ischemia (n=36)	*p* value
Age (mean± S.D.)		81.6±5.8	78.0±10.7	0.243
LDH (mg/dL) (mean±SD)		377.8±71.6	312.7±112.1	0.098
Lactate (mg/dL) (mean± SD)		37.6±23.1	36.0±22.0	0.846
Haemoglobin (g/dL) (mean±SD)		12.7±2.8	12.6±3.0	0.903
Leukocytes (mean±SD) (n/µl)		14342.9±6841.8	16277.1±12914.4	0.599
Creatinine (mg/dL) (mean±SD)		1.7±1.0	2.6±1.8	0.090
Phosphate (mg/dL) (mean±SD)		4.5±2.3	3.9±1.8	0.694
CRP (mean±SD)		19.9±20.4	19.6±14.8	0.955
D-dimer (mg/dL) (mean±SD)		11.7±13.8	90.8±145.2	0.356
Amylase (mg/dL) (mean±SD)		112.8±57.1	117.1±106.3	0.899
CRP post-op (mg/dL) (mean±SD)		24.8±12.4	31.4±9.5	0.285
Lactate post-op (mg/dL) (mean±SD)		9.7±2.1	30.6±33.3	0.08
In hospital mortality		13 (92.3%)	21 (58.3%)	0.021
Imaging findings	Bowel suffering	8 (57.1%)	29 (80.6%)	0.149
	Vascular findings	6 (42.9%)	5 (13.9%)	0.052
	Only nonspecific findings	1(7.7%)	4 (11.1%)	1

CRP: C reactive protein; LDH: Lactate dehydrogenase; T student test/ Fisher test: p value significant <0.05

DISCUSSION

Acute mesenteric ischemia (AMI) is a rare disease, accounting for less than 1 out of every 1000 hospital admissions. It is a serious condition with a high mortality rate that ranges between 30% and 90%.[2,3] AMI usually presents with nonspecific symptoms and a relatively benign physical examination, which leads to a low index of suspicion and subsequent diagnostic delays.[2,3] The main challenge in diagnosis is to differentiate acute mesenteric ischemia from other more common causes of acute abdominal pain.[3] A high mortality rate is frequently associated with late diagnosis.

AMI may be non-occlusive (NOMI) or occlusive. The most commonly aetiology is superior mesenteric artery embolism (40%-50), followed by acute mesenteric artery thrombosis (20%-30%) and nonocclusive mesenteric ischemia (25%). Mesenteric and portal venous thrombosis represent less frequent aetiologies (5-15%).[3]

Nonocclusive mesenteric ischemia is most often related to low cardiac output states that may occur in association with hypovolemia, heart failure and haemodialysis.[2] It can also occur when vasoconstriction drugs are used. Predisposing factors for mesenteric vein thrombosis include hypercoagulability, portal hypertension, portal vein thrombosis, abdominal inflammation and a history

of previous surgery or abdominal trauma.[4] It can also develop secondary to volvulus, intussusception, or strangulation of the bowel.[5]

Most of the patients in our cohort had cardiovascular risk factors. Mesenteric ischemia can be a manifestation of cardiovascular disease. Occlusive aetiology could only be documented by CT as vascular thrombosis in 10 patients.

AMI typically presents in three stages. Firstly, acute, severe abdominal pain dominates, which may be accompanied by diarrhoea and the first signs of shock. Then the silent phase follows, and rapid deterioration of the general condition occurs without evidence of symptoms. After 12hours, ileus and bacterial peritonitis with sepsis are evident, and multi-organ failure ensues. Therefore treatment should be instituted emergently in the early stages (<12 hours) in order to obtain good results.[6]

The classical clinical presentation is "pain out of proportion to examination".[2] However, depending on the exact aetiology of AMI and the timing of presentation, this presentation may be absent in 20% to 25% of cases.[7] Many patients also complain of sickness, vomiting or diarrhoea.[4] Acute upper gastrointestinal bleeding has also been reported as the sole mean of presentation, as well as there are patients who report no abdominal pain.[8] Some patients may present with tenderness on palpation when full thickness bowel injury has already occurred and peritoneal irritation is installed.[2]

Huang et al reported that all 124 patients included in their study complained of abdominal pain or distension, accompanied by vomiting in 43.5% of patients, gastrointestinal bleeding in 20.1%, and hypotension in 12.1%.[9]

In a study including 58 patients in a 10 year period in the Mayo Clinic, 95% of patients presented with acute abdominal pain with a median duration of 24 hours. Nausea was the second most frequent symptom (44%), and 3 patients were already in shock.[10]

Kougias et al reported that 94% of a total of 72 patients complained of abdominal pain as the presenting symptom, 56% of nausea, 38% of vomiting and 31% of diarrhoea.[11] In our cohort study, 70% of patients presented with abdominal pain. Nausea and vomiting were also frequent (56%). Almost 50% were already hypotensive. These results are consistent with those reported in literature.

Regarding diagnosis, there is no specific laboratory test for acute mesenteric ischemia. When evaluating a potential case of acute mesenteric ischemia it is important to assess fluid, electrolyte, and acid-base status and infection, and the results will generally demonstrate leucocytosis, hemoconcentration, elevated amylase levels, abnormal liver enzymes, and/or metabolic acidosis.[2,3] The latest indicates that bowel injury is already severe or irreversible and therefore, intervention should occur before it develops, so that intestine can be saved from full-thickness injury.[2]

Lactates, creatinine, urea and CRP were the most frequently elevated in our study which underlines the lack of specific serum markers for mesenteric ischemia. Furthermore, no marker was significantly different between the groups dead vs. alive nor extensive vs. segmental ischemia. Only haemoglobin was significantly lower in the non survivors, but this may only reflect associated comorbidities or it may even be a marker of worse prognosis. However, due its lack of specificity it is not useful for diagnosis.

Computed tomography angiography (CTA) is generally the first-line imaging modality in cases of suspicion of AMI as it is a fast and non-invasive test with high sensitivity (96%) and specificity (94%) in diagnosing this condition. It is also helpful in excluding other causes of abdominal pain.[3] Vascular CT findings include arterial stenosis, embolism, thrombosis, arterial dissection, and mesenteric vein thrombosis; nonvascular CT findings include bowel wall thickening, hypoperfusion and hypoattenuation, bowel dilatation, bowel wall haemorrhage, mesenteric fat stranding, pneumatosis intestinalis, and portal venous gas.[3]

In a study with 124 patients, the most common CT imaging findings included bowel wall thickening (n=36), intramural pneumatosis (n=15), mesenteric artery or vein thrombosis (n=14), and mesenteric or portal vein gas (n=9).[9]

In our study, CT showed signs of bowel wall injury in 74% of the cases (parietal pneumatosis 36%, bowel wall thickening 36%, pneumoperitoneum/visceral perforation 20%) and vascular findings in 22%. Nonspecific findings such as free fluid, distension and fat stranding were also frequently described (78%). CT was the imaging method of choice in our group of patients and yielded important information to prompt surgical indication.

Radiography is usually the first imaging modality ordered for patients with acute abdominal pain but has a limited role in acute mesenteric ischemia, as findings are usually nonspecific and late.[6] Ultrasound (US) is primarily helpful in diagnosing other causes of acute abdominal pain and can also demonstrate proximal mesenteric vasculature occlusion.[3]

Catheter angiography, with a sensitivity of 74%-100% and specificity of 100%, has been the gold standard to aid in diagnosis and preoperative planning in acute mesenteric ischemia allowing aetiological classification of mesenteric ischaemia (occlusive versus non-occlusive, thrombotic versus embolic), the exact localization of the obstruction and having an important role in the initial

therapy.[2-4] Endovascular therapies can then be combined to restore blood flow.[2]

Early angiography remains associated with increased survival rates but has a controversial role in the acute setting when the patient has already developed peritoneal signs, with some authors favouring immediate surgery and others advocating the important role of angiography in the preoperative surgical planning.[3] Moreover, it is impossible to perform selective mesenteric angiography for every patient with suspected AMI in many hospitals.[5]

The initial goal of treatment is to resuscitate and stabilize the patient, restore blood flow to the ischemic intestine and resect nonviable bowel. [12] An acute occlusion can be treated with a combination of endovascular strategies, with initial treatment aimed at rapidly restoring perfusion to the viscera, most often by means of mechanical thrombectomy or angioplasty, stenting and arterial bypass. Thrombolysis can also be helpful in restoring perfusion to occluded arterial branches in patients without peritonitis.[2]

The goals of open surgical therapy for acute mesenteric ischemia are to revascularize the occluded vessel, assess the viability of the bowel, and resect the necrotic bowel.[2] Exploratory laparotomy remains the gold standard for the determination of bowel viability and resection of non-salvageable bowel. [10] Overtly necrotic bowel has to be resected and, if the margins are unequivocally viable, primary anastomosis may be done, otherwise a stoma must be constructed.[4]

Second look procedures allow for the reassessment of bowel viability and further bowel resection as needed.[10] It may be performed 12 to 36 hrs later to reinspect areas of questionably viable intestine, aiming at reducing the extent of resection at primary exploration.

It is still controversial when to perform a second-look laparotomy, as some surgeons choose to schedule second-look procedure in every patient undergoing bowel resection and primary anastomosis, whereas others prefer a more selective approach, making the decision about the need for a second-look operation based on findings at the initial operation.[13] Leaving bowel of questionable viability may avoid massive enterectomy, as frequently bowel will improve with supportive measures and viable and non-viable segments can be better distinguished at the time of a second-look operation.[4] Second-look laparotomy may be excluded when there are clear margins of demarcation between well-vascularized and necrotic bowel, adequate perfusion of the remaining bowel, safe anastomosis, exteriorized viable bowel, minor spillage from perforated bowel, hemodynamic stability and improving lactate levels.[13]

In our patients, thrombectomy was performed during surgery in 6 cases. Resection of nonviable bowel was performed in 66% and exclusive exploratory laparotomy in 28%. Eleven patients were submitted to reoperation-nine of them as a planned second look. Further bowel resection was performed only in two patients (one planned second look and one unplanned), indicating that there was no progression of ischemia in most patients.

Park et al reported a 30 day mortality rate of 32%.[10] Gupta et al obtained a 30 day postoperative mortality of 56.6% in a total of 861 patients who underwent bowel resection for AMI.[14] Another multicentric study including 131 patients reported an overall mortality rate at the end of hospitalization of 74.8%. Among those patients, 60% died within the first 72 hours.[15] Rates of mortality are higher among patients with renal insufficiency, older age, metabolic acidosis, a longer duration of symptoms, and bowel resection at the time of a second-look operation.[2] Predictors of mortality that have been identified in other studies include older age (>65 years), previous cardiac pathology, elevated AST, BUN, creatinine, lactate level >2 mmol/L (18 mg/dL), metabolic acidosis, time delay to surgery.[9-11,16]

In our cohort, mortality (68%) was comparable to that reported in literature which is in the range of 59-93%.[17] Creatinine, CRP and phosphate were higher in non-survivors, but with no statistical significance. As was expected, non survivors had more frequently extensive ischemia. Low haemoglobin and elevation of lactate pre-operatively were significantly correlated with mortality. We also observed that extensive ischemia at the time of surgery was also a significant risk factor for death, which comes as expected. Regarding patients with extensive ischemia, more than half of these patients had imaging findings consistent with bowel suffering.

Major complications found in other studies include respiratory, renal, hepatic and multiple organ failure, recurrent bowel infarction, embolism to other areas, resulting in myocardial infarction and stroke. Gastrointestinal haemorrhage and infections can also occur.[10,11]

In our group of patients, renal failure and nosocomial infection were the most frequent complications. Multiple organ failure also occurred, as well as new episodes of ischemia, as described in literature.

The main limitations to this study are the reduced number of patients and the limitations associated with retrospective studies-as lack of some data, and only patients submitted to surgery were analysed.

CONCLUSION

Acute mesenteric ischemia is still difficult to diagnose in its early stages, with dreadful consequences. We attempted to understand how the patients who end up being submitted to surgery due to mesenteric ischemia present initially and what determines a poorer prognosis.

More studies with larger populations are needed to draw firmer conclusions.

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