Chirurgia (Bucur). 2025 October, Online Ahead of Print

http://dx.doi.org/10.21614/chirurgia.3127

Clinical Features and Outcomes of Patients with Acute Mesenteric Ischaemia in a Retrospective Study

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Rezumat

Context: Ischemia intestinală este o urgență abdominală caracterizată printr-o reducere drastică a fluxului sanguin în vasele mezenterice, cu posibilă apariție a necrozei intestinului subțire şi/sau a colonului. Incidența sa este destul de rară, iar diagnosticul este foarte dificil deoarece tabloul clinic nu este specific şi nu există teste de laborator patognomonice.
Metode: A fost efectuat un studiu retrospectiv pe 28 de pacienți cu infarct intestinal, incluzând analiza factorilor de risc, comorbidităților, simptomelor, testelor de laborator şi investigațiilor instrumentale, pentru a determina prezența eventualelor semne de ischemie mezenterică secundară insuficienței vasculare.
Rezultate: Douăzeci şi patru de pacienți (85%) au fost supuşi unei intervenții chirurgicale şi la toți s-a găsit necroză intestinală. Printre aceştia, s-a observat o rată a mortalității destul de ridicată (64%).
Concluzie: Studiul retrospectiv a confirmat frecvența scăzută, mortalitatea ridicată şi dificultatea de diagnosticare a ischemiei mezenterice în diferitele sale forme clinice. În prezent, nu există teste de laborator şi nici tehnici instrumentale care să poată oferi un diagnostic cert de ischemie mezenterică acută într-o fază precoce. Cu toate acestea, suspiciunea clinică puternică, un diagnostic rapid şi o abordare terapeutică agresivă ar putea îmbunătăți rezultatele clinice şi reduce mortalitatea ridicată.

Cuvinte cheie: ischemie intestinală, ischemie mezenterică acută, ischemie intestinală, ocluzie arterială mezenterică

Received: 02.02.2025
Accepted: 13.05.2025

Abstract

Background: Intestinal ischaemia is an abdominal emergency characterized by a drastic reduction in blood flow in the mesenteric vessels with the possible

onset of necrosis of the small intestine and/or colon. Its incidence is rather rare and the diagnosis is very difficult as the clinical presentation is not specific and there are no pathognomonic laboratory tests.
Methods: A retrospective study was carried out on 28 patients with intestinal infarction, including analysis of the risk factors, comorbidities, symptoms, laboratory tests and instrumental investigations, to determine the presence of eventual signs of mesenteric ischaemia secondary to vascular insufficiency.
Results: Twenty-four patients (85%) underwent surgery and intestinal necrosis was found in all. Among these, a quite high mortality rate (64%) was observed.
Conclusion: The retrospective study confirmed the low frequency, high mortality and diagnostic difficulty of mesenteric ischaemia in its various clinical forms. Currently, there are neither laboratory tests nor instrumental techniques that can give a certain diagnosis of acute mesenteric ischaemia in an early phase. However, strong clinical suspicion, a rapid diagnosis and an aggressive therapeutic approach could improve the clinical results and reduce its high mortality.

Keywords: intestinal ischaemia, acute mesenteric ischaemia, bowel ischaemia, mesenteric arterial occlusion

Introduction

Acute mesenteric ischaemia (AMI), described for the first time by Virchow in 1852, is an abdominal emergency characterized by a sudden and important reduction of arterial or venous perfusion in the intestines. It can be caused by occlusion or hypoperfusion in the area of the mesenteric vessels with the onset of transitory ischaemia or necrosis of the small intestine and/or colon. AMI has an incidence of 1 case every 1,000 hospitalizations and occurs principally in 70-80 year olds with a frequency three times greater in females than in males (1) and is characterized by a high global mortality, between 59 and 93% of cases (2).

The incidence of this pathology is expected to grow given that the increasing average age of the population. AMI includes a heterogeneous group of different pathophysiological processes that have as a common outcome intestinal necrosis with onset of severe metabolic alterations that cause a series of events that can culminate in multi-organ dysfunction (MOF) and death of the patient. AMI can be classified in: 1) embolic or thrombotic occlusive arterial type, 2) non-occlusive arterial type and 3) thrombotic venous type. Arterial intestinal ischaemia from embolic occlusion is responsible for about 50% of all the cases of acute intestinal ischaemia. The clot frequently originates at the cardiac level (atrial or ventricular) such as epiphenomenon of atrial arrythmias, myocardial ischaemia, cardiomyoathy, ventricular aneurisms, valve disorders, vegetal endocarditis and rarely caused by endovascular procedures or by an aortic aneurismectomy or correction of cardiac valve pathologies. The clot is preferentially localized in the superior mesenteric artery (SMA) due to its acute-angle origin from the aorta, frequently it is situated at the level of Reiner's critical segment, rarely at the origin of the SMA or in the collateral branches when they are small (3).

Mesenteric arterial thrombosis (about 20% of cases) is principally caused by atherosclerosis; secondary causes are fibromuscular dysplasia, Takayasu's arteritis, luetic arteriopathy and hematic hypercoagulability. Non-occlusive intestinal ischaemia (about 20% of cases) is caused by a severe hypoperfusion of the mesenteric vascular area. Possible mechanisms underlying the development of non-occlusive intestinal ischemia include shock (cardiogenic, haemorrhagic, hypovolemic or septic), severe diarrhoea, acute pancreatitis, severe burns, hepatic and/or renal pathologies and use of vasoconstrictive drugs that worsen intestinal perfusion. Mesenteric venous thrombosis (MVT) is the cause of about 10% of cases of AMI. MVT can be divided into a primitive form (20%), determined by states of congenital or acquired hypercoagulability, and in a secondary form (80%), caused by infections and sepsis, previous abdominal surgery, intestinal inflammatory diseases, hepatic cirrhosis and portal hypertension, abdominal trauma, abdominal tumours, paraneoplastic syndromes and use of oral contraceptives (4).

Although the pathogenesis of AMI seems clearly comprehensible, in about 50% of cases it is not possible to establish the triggering etiological factor. About 70% of the mesenteric blood flow is distributed to the mucosa tunic and submucosa

and the remaining 30% is directed to the muscular and sierosa layers, therefore it is the intestinal mucosa surface that is initially affected by ischaemia when there is a reduction of blood flow. A prolonged period of intestinal ischaemia of more than 6 hours causes necrosis of the whole intestinal wall. In this study we aimed at investigating the mortality and diagnostic difficulty of mesenteric ischaemia in its various clinical forms.

Materials and Methods

A retrospective study was carried out on 28 cases of mesenteric ischaemia enrolled at the Unit of Surgery of the Department of Surgery and Medical-Surgical Specialties of the University of Catania (Italy), in the period between January 2019 and December 2024. We assessed the patients' files and collected data on risk factors, comorbidities, symptoms and clinical signs, laboratory tests and instrumental investigations with the aim of determining the presence of signs of a probable diagnosis of mesenteric ischaemia secondary to vascular failure. All patients underwent blood tests and radiological examinations such as direct abdominal radiography, abdominal CT scan and colonoscopy; hemogas analytical test was carried out only on 17 patients.

Results

Of the 28 patients, 15 were males (53%), the mean age was 75 (65-92) years. The mean duration of hospital length was 12 (1-52) days. Only 2 of the 28 patients did not show comorbidities and risk factors, in the remaining 26 patients arterial hypertension was the most frequent comorbidity (13 patients, 46%). Other associated pathologies were diabetes mellitus in 9 patients (32%), ischemic cardiopathy in 7 patients (25%), chronic atrial fibrillation in 6 patients (21%), chronic renal failure and cerebral ischaemia in 5 patients (18%), heart failure in 3 patients (11%), and anaemia in 2 patients (7%); further pathologies were deep venous thrombosis, viral hepatitis HCV and correlated HBV, obstructive broncopneumopathy, ulcerative rectocolitis, dyslipidemia, chronic venous failure and vasculitis. Only 14 of the 28 patients were on medical therapy for the treatment of the comorbidities: anti-hypertensive drugs, digitalis, oral anticoagulants, mesalazine, anti-arrythmic agents. Of the 28 patients only 11 did not report previous surgery at work-up, all the others had undergone different types of surgery, among which were thromboendoarterectomy of the internal carotid artery, intestinal resection for a previous heart attack, and angioplasty of the internal iliac artery. The initial symptomatology of the patients who went to the Emergency Room were various; the most frequent symptom was diffused or localized abdominal pain in a specific abdominal quadrant, present in 25 patients (89%). Associated symptoms were intestinal obstruction in 9 patients (32%), diarrhoea in 7 patients (25%), vomiting in 6 patients (21%), sensorial numbness in 6 patients (21%), hematochezia in 5 patients (18%), fever in 2 patients (7%), hematemesis in 1 patient (3.5%), and septic shock syndrome in 1 patient (3.5%). Hemogas analytical exam showed in all cases (100%) the presence of metabolic acidosis (pH between 7.18 and 7.34) with an increase of lactic acid. Neutrophilic leukocytosis (12.500- 43.500 GB/mm^3) was found in 27 patients (96%), an increase in the values of fibrinogen (453-1187 mg/dl) in 21 patients (75%), an increase of LDH (480-7952 U/L) in 20 patients (71%), an increase of amylase (105-1335 U/L) in 11 patients (40%), an increase of CPK (408-3948 U/L) in 7 patients (25%), and an increase of D-dimer (707-5728) in 6 patients (21%); serum phosphate was dosed only in 7 patients and showed elevated values (5.6-7.8 mg/dl) in 5 patients (71%). Plain abdominal X-ray was carried out in 19 patients (68%) and was negative in 2 patients (11%), the remaining cases did not show specific signs such as the presence of distended jejunoileal loops with air-fluids levels in 6 patients (32%), air-fluids levels in the ileal loops in 9 patients (47%) or in the colon in 1 patient (5%), and meteoric distension of the intestinal loops in 1 patient (5%). Abdominal CT scan with contrast medium was carried out in only 13 patients (46%) and showed in 2 cases the poor opacity of the superior mesenteric artery associated with thickening of the ileal loops, in 1 case thrombosis of the portal vein and superior mesenteric vein was associated with wall thickening of the ileal loops and free abdominal fluid, in 1 other case there was thrombosis of the portal vein and splenic vein, in the remaining 9 cases the signs were not specific such as distended ileal loops and thickened walls with air-fluids levels, thickening of the walls of the colon, free peritoneal fluid and calcification and/or thrombi of the abdominal aorta and common iliac arteries (*Table 1*). Colonoscopy with biopsy was carried out only in 1 patient, who arrived at the Emergency Room with pain in left iliac fossa and who had hematochezia the night

Table 1. Summary table of patients' characteristics

Demographic information	**Total number n= 28**
- male	15 (53%)
- mean age (years; range)	75 (65-92)
Comorbid medical conditions	
- hypertension	13 (46%)
- diabetes mellitus	9 (32%)
- ischemic cardiopathy	7 (25%)
- chronic atrial fibrillation	6 (21%)
- chronic renal failure	5 (18%)
- cerebral ischaemia	5 (18%)
- heart failure	3 (11%)
- anaemia	2 (7%)
Symptoms	
- localized abdominal pain	25 (89%)
- intestinal obstruction	9 (32%)
- diarrhoea	7 (25%)
- vomiting	6 (21%)
- sensorial numbness	6 (21%)
- hematochezia	5 (18%)
- fever	2 (7%)
- hematemesis	1 (3.5%)
- septic shock syndrome	1 (3.5%)
Laboratory findings	
- neutrophilic leukocytosis (12.500- 43.500 GB/mm^3)	27 (96%)
- increased fibrinogen (453-1187 mg/dl)	21 (75%)
- increased LDH (480-7952 U/L)	20 (71%)
- increased amylase (105-1335 U/L)	11 (40%)
- increased CPK (408-3948 U/L)	7 (25%)
- increased D-dimer (707-5728)	6 (21%)
- increased serum phosphate (5.6-7.8 mg/dl) (dosed in 7 patients)	5 (71%)
Imaging results	
Plain abdomen X-ray (carried out in 19 patients)	
- distended jejunoileal loops with air-fluids levels	6 (32%)
- air-fluids levels in the ileal loops	9 (47%)
- air-fluids levels in the colon	1(5%)
- meteoric distension of the intestinal loops	1(5%)
- negative	2 (11%)
Abdominal CT scan with contrast medium (carried out in 13 patients)	
- poor opacity of the SMA and thickening of the ileal loops	2 (16%)
- thrombosis of the portal vein and superior mesenteric vein, thickening of the ileal loops and free abdominal fluid	1(8%)
- thrombosis of the portal vein and splenic vein	1(8%)
- not specific	9 (70%)
Mean hospital lenght (days; range)	12 (1-52)
Outcomes	
Death	
- due to cardio-respiratory failure	17 (60%)
- due comorbidities present before surgery	1 (4%)
Discharged	10 (36%)
Histological examination (only in 19 patients)	
- intestinal transmural infarction of the ileum	12 (63%)
- massive transmural infarction of the distal ileum and the colon	2 (10.5%)
- necrosis of the ileal mucosa	3 (16%)
- necrosis of the colic mucosa	2 (10.5%)
- necrosis of the margins of surgical resection	7 (37%)

before, with a previous anterior resection of the rectum due to carcinoma of the upper rectum; endoscopy showed at 15 cm from the anal margin, the presence of oedematous mucosa that was congested, friable and raised in plaques not flattened with gas insufflation; the histological examination confirmed the diagnosis of ischemic colitis.

Of the 28 patients, 24 underwent surgery: 1 patient affected by ischemic colitis was treated medically, 2 patients refused surgery and 1 patient died of cardio-respiratory failure in the operating room before surgery. Five explorative laparotomies were carried out finding massive intestinal infarction from the second jejunal loop to the right colon, 12 partial resections of the ileum were done (with manual side to side ileo-ileal anastomosis in 6 cases, mechanical side to side in 3 cases, manual end to end in 1 case, mechanical end to end in 1 case and mechanical end to side ileo-cecal in 1 case); 3 massive intestinal resections of the ileum and of the right colon (with mechanical side to side jejuno-colic anastomosis in 2 cases and mechanical side to end in 1 other case), 1 resection of the sigma (with colostomy), 1 right hemicolectomy (with mechanical side to side ileo-colic anastomosis), 1 resection of distal ileum and transverse colon (in a patient operated on 4 days before with a right hemicolectomy) and 1 double intervention in a patient with first a descending colon resection with colostomy of the transverse colon and then a right hemicolectomy with removal of the sigma, of the intraperitoneal rectum and ileostomy due to the onset of diffused colic ischaemia (*Table 2*). During each surgical intervention intestinal infarction was found. The intestinal necrosis was localized at the level of the ileum (segmentary necrosis) in 12 patients, in 7 patients it extended from the second jejunal loop to the right colon, in 1 case to the sigma, in another case it extended to all the colon and in 3 cases it affected the distal ileum and the right and transverse colon (in the patient who previously underwent right hemicolectomy). All the surgical procedures were carried out in laparotomy, no patient received surgical revascularization of the mesenteric vessels. The average duration of surgery was 210 (180-270) minutes. Post-operatively 11 patients (40%) needed to be transferred to an intensive care unit (ICU) with the following conditions: cardio-respiratory insufficiency (7 patients), septic state (3 patients) or need for monitoring of vital signs (1 patient); in 4 patients total parenteral nutrition was necessary post-operatively. Other post-operative complications were anemization in 9 patients who required blood transfusion, the onset of acute pulmonary oedema in 2 patients,

Table 2. Sites of intestinal necrosis and corresponding surgical treatment

Site of intestinal necrosis	Surgery type (n=24)
Infarction from the second jejunal loop to the right colon	5 Explorative laparotomy
Ileal necrosis	12 Partial resection of the ileum
Ileal and right colon necrosis	3 Massive intestinal resection of the ileum and right colon
Necrosis of the distal ileum and cecum	1 Right hemicolectomy
Necrosis of the distal ileum and right colon	1 Resection of distal ileum and right colon
Necrosis of the sigma	1 Resection of the sigma
Necrosis of the transverse and descending colon with successive inclusion of right colon, sigma and rectum	1 Resection of the descendent colon + colostomy on the transverse colon with successive right hemicolectomy + exeresis of the sigmoid colon, rectum + ileostomy

atrial fibrillation in 1 patient, and septic shock in 2 patients. There were surgical complications in 4 patients (16%): 1 case of dehiscence of the colic jejunal anastomosis after two weeks in a patient operated on for massive intestinal infarction, 1 case of peristomal abscess after one month in a patient who had undergone resection of the terminal ileal loops with ileostomy, 1 case of evisceration (tenth post-operative day) and 1 case of necrosis of cecum, sigma and intraperitoneal rectum in a patient who had undergone, two weeks earlier, a resection of the distal transverse and descending colon. Of the 28 patients, 17 (60%) died post-operatively due to cardio-respiratory failure, 1 (4%) died in the operating room due to the same clinical condition present before surgery and 10 (36%) were discharged. The histological examination of a surgical biopsy was possible in only 19 of the 28 patients, excluding the 5 patients who underwent explorative laparotomy with successive abdominal wall suturing. Pathological anatomy confirmed the presence of intestinal transmural infarction of the ileum in 12 patients (63%), massive transmural infarction of the distal ileum and the colon in 2 cases (10.5%), necrosis of the ileal mucosa in 3 cases (16%) and necrosis of the colic mucosa in 2 cases (10.5%); presence of intestinal necrosis of the margins of surgical resection was observed in 7 cases.

Discussion

A correct approach to AMI should include, other than clinical suspicion, a rapid pre-operatory diagnostic evaluation, an aggressive surgical treatment based on the revascularization of the mesenteric vessels or the resection of the necrotic intestine with eventual further control of intestinal vitality ("second look") in cases of doubt and intensive post-operative care, with the aim of preventing the onset of MOF. Suspicion should be high in the presence of a compatible clinical history and unclear clinical signs. Two studies have found that only in one third of patients, a correct diagnosis of AMI was made before surgical exploration or death (5). AMI should be suspected in patients over 60 years of age with a history of atrial fibrillation, recent infarction of the myocardium, congested cardiac deficit, arterial embolism, or postprandial abdominal pain with weight loss, characterized by the referred abdominal pain that is disproportional to the objective abdominal examination. Clinical diagnosis of acute intestinal ischaemia is difficult as most patients do not present specific symptoms. The most common clinical symptom is abdominal pain that typically is "out of proportion" to the objective abdominal examination. The pain is of the pure visceral type, of notable intensity, with sudden onset, constant in time, and is localized in the mesogastric region with irradiation to all the abdominal quadrants and sometimes to the lumbar region. If abdominal pain is present for more than 2 hours and intestinal occlusion or perforation have been excluded, acute intestinal ischaemia should be suspected. Other possible symptoms are nausea, food or biliary vomiting and abdominal distension preceded by episodes of diarrhoea or intestinal haemorrhage. A third of patients present in a confused state. The presence of acute abdomen and hypotension may suggest an advanced clinical picture. In cases of MVT the clinical picture is initially less clear and insidious and is characterized by fever, anorexia, general malaise, distension and vague abdominal pain, moderate peritoneal fluid. The outcome of the different causes of mesenteric ischaemia is the onset of intestinal infarction that is associated with signs of peritonitis, hemodynamic instability and sepsis with multi-organ failure (MOF). Acute intestinal ischaemia has to be differentiated from other

pathologies that cause abdominal pain such as acute pancreatitis, acute cholecystitis, perforation of viscera, acute diverticulitis, rupture of aneurisms of the abdominal aorta, intestinal occlusion and renal colic. There are no laboratory tests that can detect the initial phase of acute intestinal ischaemia; however, some markers can indicate advanced intestinal necrosis or sepsis. Most patients present an evident neutrophil leukocytosis (up to 30-50,000 GB/mm^3) not proportionate to clinical symptoms and hemoconcentration. In 50% of cases there is a metabolic acidosis that is similar to that found in every other intra-abdominal catastrophe, indicative of late diagnosis. Hyperamylasemia is present in 50% of cases and can give rise to an incorrect diagnosis of acute pancreatitis. Elevated serum levels of the enzymes LDH, CPK, alkaline phosphatase and gamma-GT have been found, but none singularly or in combination have been found to be sufficiently sensitive or specific in early diagnosis of acute intestinal ischaemia. Often there is an increase of lactic acid and hyperphosphatemia following the release into the blood stream of inorganic phosphate by enterocytes. Various studies have been conducted on new possible serological markers of intestinal ischaemia such as D-dimer, α-glutathion, S-transferasis (αGST), D-lactate, fatty acid binding protein (FABP), however none of these serological markers has been found to be sufficiently sensitive and specific for an early diagnosis (6-9). The instrumental examinations used in the diagnosis of acute intestinal ischaemia include non-invasive procedures such as plain abdominal X-ray, eco-colour-doppler, CT scan of the abdomen with contrast medium and MR angiography, and invasive procedures such as angiography of the mesenteric vessels. Plain abdominal X-ray is not really useful in diagnosis but can exclude other causes of acute abdomen such as perforation of the viscera or intestinal occlusion. This method is negative in 25% of patients at onset of mesenteric ischaemia; in the initial phases there can be paralytic ileum with gaseous distension of the loops and/or presence of air-fluid levels, in the late phases there can be the presence of ischemic loops with wall thickening (wall oedema: thumbprinting), absence of peristalsis or pneumatosis. In cases of intestinal perforation this method identifies the presence of free air in the peritoneal cavity. The radiological signs obtainable with abdominal radiography are not specific, late and associated with high mortality when present (10). The eco-colour-doppler of the splanchnic vessels is a non-invasive and repeatable method. Even if limited by intestinal meteorism, it can identify the presence of embolisms or thrombi in the proximal tract of the celiac trunk or SMA, the presence of free endoperitoneal fluid, wall thickening of the intestinal loops and the absence of peristalsis. This method has a high specificity (92-100%) in identifying occlusions or critical stenosis of the splanchnic vessels and a sensitivity of 70-89% (11) in as much as it cannot exclude a distal embolism of the principal splanchnic vessels or of their branches giving false negatives with potentially fatal consequences (12). Abdominal CT scan with contrast medium can show the triggering cause of intestinal ischaemia and its possible complications, excluding other causes of abdominal pain. It can also show stenosis or vascular occlusions of a thromboembolic nature or non-specific signs of AMI in the late phase such as thickening (5-15 mm) of the intestinal walls (MVT or mesenteric arterial occlusion after reperfusion), thinning of the wall in cases of transmural infarction due to arterial occlusion, presence of dilated intestinal loops full of gas and/or liquid, free intraperitoneal fluid and oedema of mesenteric adipose tissue, intestinal pneumatosis and presence of gas in the venous portal-mesenteric system (present in almost 100% of the cases of acute intestinal ischaemia from venous mesenteric thrombosis). A highly specific sign, but not sensitive for AMI visible with the use of contrast medium, is the absence or reduced uptake of the contrast medium by the intestinal wall (13). Another radiological sign indicative of intestinal ischaemia at the CT scan is the "halo" appearance of the intestinal loops caused by hyperaemia and hyperperfusion associated with surrounding wall oedema; this sign can be found in cases of arterial occlusion after reperfusion and non-occlusive or venous-occlusive intestinal ischaemia (*Fig. 1*). CT angiography with three dimensional reconstruction of the image can show the vascular anatomy comparable to arteriography thus increasing the accuracy of this method in diagnosing intestinal ischaemia with a sensitivity of 96% and a specificity of 94%. Nowadays, CT angiography has overtaken angiography as the diagnostic test of choice thanks to its ease of use and capacity to reconstruct the vascular anatomy and evaluate secondary signs of intestinal ischaemia (*Fig. 2*). MR angiography is a non-invasive test that is becoming more used to evaluate patients with suspected intestinal ischaemia; its three dimensional reconstruction can evaluate anatomy, the

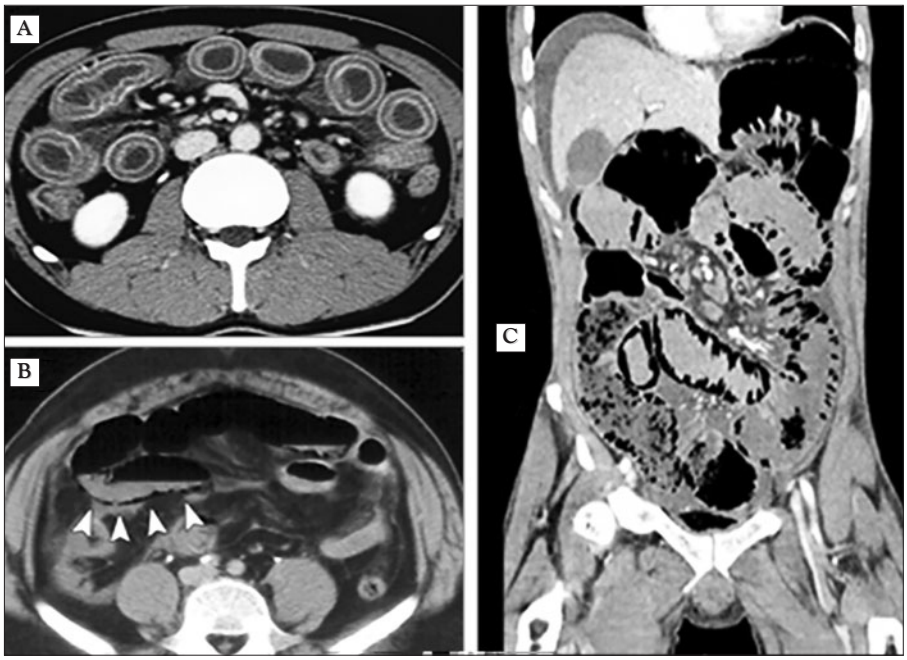

Figure 1. **(A)** Abdominal CT scan with contrast, axial plan: diffuse thickening of the small bowel walls with double-track pattern; **(B)** Abdominal CT scan without contrast, axial plan: air-fluid levels with small bowel loop pneumatosis (white arrows); **(C)** Abdominal CT scan with contrast, coronal plan: diffuse intestinal pneumatosis of the small intestine with left sub-diaphragmatic air crescent.

patency of vessels and the presence of blood flow. This method avoids the risk of an allergic reaction and nephrotoxicity associated with the use of contrast medium and is not operator-dependent. The weak point of this method is the relatively poor spatial resolution, limited to 1 mm^3, which does not allow a correct visualization of the distal embolism;

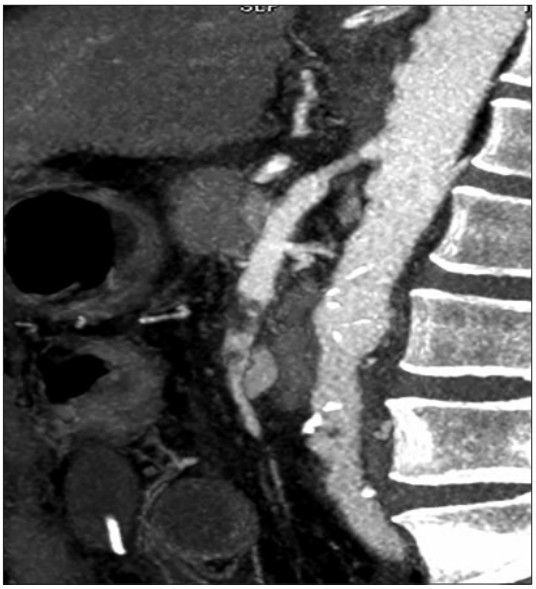

Figure 2. Abdominal CT scan with contrast, sagittal plan: stenosis at the origin of the superior mesenteric artery and thrombotic filling defect in the middle-third of the artery

also, the greatest difficulty with respect to the CT scan is that of identifying secondary signs of acute intestinal ischaemia such as intestinal wall thickening (14). The use of MR angiography is principally limited to the study of the celiac trypod and superior mesenteric artery. Angiography is an invasive procedure that today plays a prime role in diagnosis and treatment of AMI. Without clinical indications for an emergency laparotomy, it is, for some surgeons, the examination of choice for suspected AMI. It is potentially a nephrotoxic technique that needs some time to carry out, thus not practical in conditions of shock or peritonitis. This investigation, through the correct visualization of the anatomy of the splanchnic vessels, can distinguish the arterial from the venous cause of ischaemia and determine if the cause is occlusive or non-occlusive. In selected cases angiography can indicate the correct endovascular procedure such as loco-regional infusion of vasodilators or thrombolytics or carrying out a dilatation of the stenotic vessel with eventual positioning of a stent. Laparoscopy has a limited role in the evaluation of intestinal vitality. The colour of the intestinal serosa tunica can be difficult to evaluate and can appear altered due to bad functioning or an incorrect calibration of the colours of the video camera. Moreover, segmentary intestinal ischaemia can be misinterpreted due to the difficulty of exploring the entire intestine and of visualizing all the surfaces. With the aim of

improving the sensitivity of this method, some authors have successfully used fluorescein with laparoscopic ultraviolet light (15), however the diagnostic role of laparoscopy has not been widely accepted due to the impossibility of visualizing some areas of the necrotic intestine. The therapy for AMI is generally surgery if the cause is thromboembolic while it is conservative, in the absence of peritonitis or intestinal necrosis, in its non-occlusive form and in MVT. The actions necessary to stabilize the patient from the hemodynamic and metabolic points of view are primary and include the positioning of a central venous catheter and a urine catheter, endovenous infusion of liquids, broad-spectrum antibiotics, suspension of vasoconstrictor drugs and correction of metabolic acidosis and hydro-electrolytic imbalances. Surgery should rapidly confirm the diagnosis, evaluate the site and extension of intestinal ischaemia/necrosis, exclude extrinsic causes of intestinal ischaemia such as adhesions or volvolus, carry out revascularization when possible and remove the necrotic intestine (*Fig. 3*). Intestinal revascularization should be carried out only when there is a real possibility of restoring blood flow and improving the ischemic picture. In cases of acute arterial ischaemia of embolic nature an arteriotomy is carried out with thromboembolectomy through a Fogarty catheter. In cases of intestinal ischaemia due to acute arterial thrombosis various surgical approaches are possible: the venous (or prosthetic) bypass, reimplantation or thrombo-endoarterectomy. In emergency, the procedure most recommended is bypass with a bridge using a venous graft (great saphenous vein or inverted superficial femoral vein) or prosthesis in Dacron or in polytetrafluoroethylene (PTFE). The thrombo-endoarterectomy is an alternative surgical approach to a bypass in cases of unavailability of an autologous vein or when there are contraindications to the use of prosthesis; moreover, it is considered potentially risky in as much as flaps can be created in the lumen after removal of the thrombotic plaque. After surgery for revascularization it is necessary to wait for 30-45 min to evaluate the state of intestine. In patients with acute mesenteric thromboembolism at high surgical risk and with early stage intestinal ischaemia, locoregional thrombolysis or angioplasty with eventual positioning of a stent can be used. The use of anticoagulants in patients with acute thromboembolic mesenteric ischaemia is controversial in as much as early administration of heparin reduces thromboembolic progression and thus intestinal damage but it is correlated with an increased risk of haemorrhage in the ischemic intestinal segment. In cases of acute non-occlusive intestinal ischaemia the initial therapy consists of infusion in the superior mesenteric artery of vasodilators such as glucagon, nitroglycerine, sodium nitroprussiate, prostaglandin E,

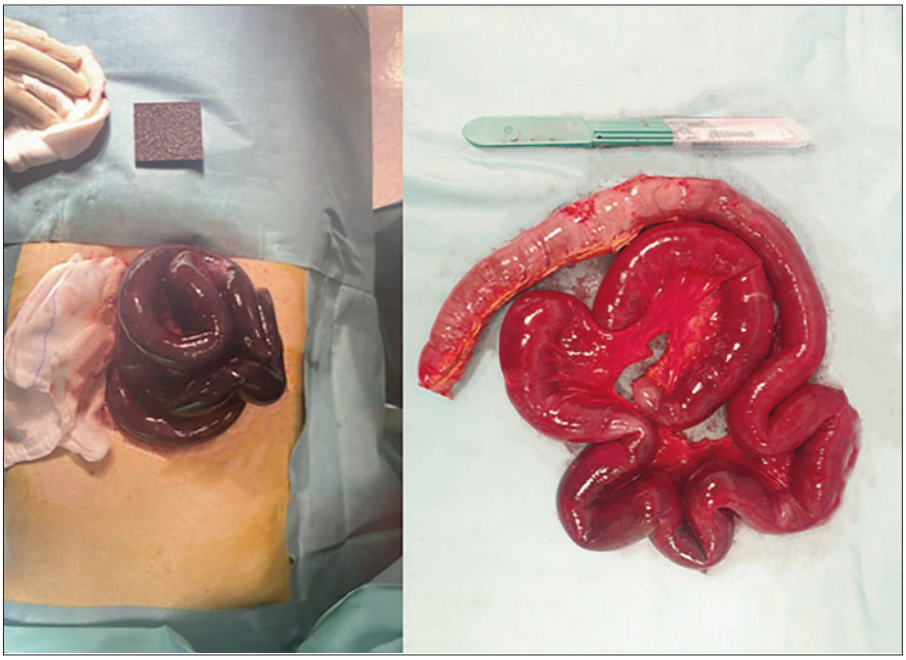

Figure 3. Massive small bowel infarction

isoproterenol and above all papaverin with the regression of the vasospasm and reduction of mortality from 70-90% to 0-55% (10). In cases of MVT the therapy is based on either the administration of heparin for some days followed by chronic administration of oral anticoagulants (dycumarol) or on the locoregional thrombolysis with infusion of lytic agents (streptokinase, urokinase). The surgical thrombectomy of the mesenteric veins is not very successful and is rarely indicated. The correct determination of intestinal vitality is fundamental as if left in the abdomen the necrotic intestine could become perforated with successive peritonitis and sepsis; on the other hand if an excessive quantity of the intestine is removed, "short intestine" syndrome can develop. The evaluation of intestinal vitality is rather difficult and is based on clinical parameters such as the colour of the tunica serosa, the presence of peristalsis and arterial mesenteric pulse, but this evaluation is not always possible. Therefore, the evaluation of intestinal vitality with only the clinical criteria previously mentioned is not sure and is characterized by a sensitivity of 82% and a specificity of 91% (1). There are other methods to determine intestinal vitality such as the use of vital colourants (fluorescein) or the use of doppler for the study of blood flow in the mesenteric vessels; the combination of the macroscopic picture, fluorescein test and the intra-operatory use of a doppler probe gives the best results. However, none of these methods is decisive in evaluating final vitality of the ischemic intestine that can progress after surgery: a second laparotomy or laparoscopy to "just check" is the gold standard in evaluating the intestinal vitality. If vitality is doubtful then a laparotomic (or laparoscopic) "second-look" within 24-48 h is suggested, even if some surgeons prefer a laparoscopic "second-look" within 72 hours to evaluate eventual intestinal anastomotic dehiscence, given that most of them occur from the third to the fifth post-operative day (16). Even if a second operation is accepted by many surgeons to evaluate intestinal vitality, there is still no universal consensus on correct indications for timing, type of procedure (laparotomic or laparoscopic) and if this method should be programmed or on demand (17). Some surgeons choose an aggressive strategy with a planned second intervention in all patients who undergo intestinal resection with primary entero-enteric anastomosis (18), while other surgeons suggest a more selective approach due to the risk of infection, malnutrition, hemodynamic instability and onset of MOF. A review of the data in the literature of the last 20 years has shown that just over 50% of operated patients have "second look" surgery, and that about 40% of these benefited from the surgery with further resection of the necrotic intestine. It has been demonstrated that during first surgery, despite the clinical evaluation and the use of doppler and fluorescein, a correct evaluation of the extension of the necrotic intestine was not made of the non-occlusive mesenteric ischaemia and of MVT. The second surgical operation is thus very important in the final evaluation of intestinal necrosis. The principal limit in defining the exact role of the surgical "second-look" is the lack of prospective studies that compare the results of this procedure and controls. As regards the different methods of the surgical "second-look", programmed or on demand, different choices have been made by surgeons (19). The initial post-operative course of the patient could distract the clinician from the real vitality of the remaining part of the intestine; therefore the decision to recheck intestinal vitality should be made at the moment of the first surgery and not influenced by the post-operative course of the patient. Mortality and the morbidity can be reduced by a laparoscopic "second look" thanks to the shorter duration of the intervention and less trauma with respect to traditional surgery. At the second surgery intestinal vitality is generally clear even if sometimes a laparoscopic (third-look) is necessary before carrying out definitive intestinal anastomosis. In cases of massive intestinal infarction, the decision to proceed to a massive intestinal resection has to be subordinate to the general evaluation of the case and depends closely on the probability that the patient has of survival. The post-operative medical treatment includes continuing antibiotic therapy, administrating liquids with balanced hydro-electrolytes, and anticoagulants. The return to normal food is secondary to the restoration of intestinal motility and parenteral nutrition can be used in cases of extensive intestinal resection. The prognosis of AMI is influenced by the aetiology, by the speed of diagnosis and by early therapeutic treatment. A quantitative analysis of the data from 45 observational studies of 3,692 patients in 2003, with subdivision of the patients into various categories based on the various aetiologies of AMI and on the type of treatment (surgical or non-surgical), showed that the prognosis of venous mesenteric thrombosis is better than acute arterial mesenteric ischaemia and that with the latter, embolic occlusion presents a better prognosis with respect to thrombotic

occlusion and non-occlusive ischaemia. This study also showed that the percentage of mortality in patients not undergoing surgery is high, varying from 87 to 99.4% (with minor and major incidence to patients with venous and arterial thrombosis respectively) and that the percentage of post-operative mortality from venous thrombosis (32.1%) and arterial embolism (54.1%) were lower than those from arterial thrombosis (77.4%) and non-occlusive ischaemia (72.7%) (2). Other studies have shown that an age lower than 60 and intestinal resection are associated with better prognosis, while advanced age (>65 years), hepatic and renal failure, metabolic acidosis, tissue hypoxia, intestinal pneumatosis and sepsis are associated with a worse prognosis (20-22). To date there are no guidelines for the diagnosis and the treatment based of the results of randomized controlled studies (23). The percentage of survivors has not improved over the past 70 years due to the difficulty of diagnosis before the onset of intestinal necrosis.

Conclusions

Our retrospective study confirmed the elevated mortality and the diagnostic difficulty of mesenteric ischaemia in its various clinical forms. There are currently neither laboratory tests nor instrumental diagnostic tests that can diagnose early phase AMI with any certainty. Given the various and not specific symptoms it is necessary that the clinician has a high index of suspicion to come to a rapid and correct diagnosis. The prognosis of this pathological condition depends on the underlying cause, the extension of the ischemic/necrotic process, the time between symptoms and the diagnosis; indeed, outcomes are positively influenced by the rapidity of the diagnosis and an appropriate and fast medical-surgical treatment. This study showed that in all patients diagnosis was late with the consequent onset of partial intestinal necrosis or total intestinal necrosis at surgery. With the aim of reducing the incidence of mesenteric ischaemia, it is necessary that in patients at risk there is a correct preventive medical therapy (i.e. the use of oral anticoagulants in patients with atrial fibrillation) and a diagnostic screening by means of eco-colour-Doppler to identify eventual critical stenosis of splenic vessels.

Conflict of Interests

The authors declare no conflict if interests.

Funding

The authors received no financial support for the research, authorship and/or publication of this article.

References

1. Bala M, Catena F, Kashuk J, De Simone B, Gomes CA, Weber D, et al. Acute mesenteric ischemia: updated guidelines of the World Society of Emergency Surgery. World J Emerg Surg. 2022;17(1):54.
2. Schoots IG, Koffeman GI, Legemate DA, Levi M, van Gulik TM. Systematic review of survival after AMI according to disease aetiology. Br J Surg. 2004;91:17-27.
3. Reintam Blaser A, Coopersmith CM, Acosta S. Managing acute mesenteric ischaemia. Intensive Care Med. 2024;50(4):593-596.
4. Shrestha AK, Shrestha A, Ghimire B, Ghimire PR, Kunwar S, Luitel P, Adhikari N. Mesenteric Ischemia and Its Need for Timely Recognition and Management. Case Rep Surg. 2022:7370634.
5. Mamode N, Pickford THE, Leiberman P. Failure to improve outcome in AMI: seven year review. Eur J Surg. 1999;165:203-208.
6. Block T, Nilsson TK, Bjorck M, Acosta S. Diagnostic accuracy of plasma biomarkers for intestinal ischaemia. Scand J Clin Lab Invest. 2008;68(3):242-248.
7. Chiu YH, Huang MK, et al. D-Dimer in patients with suspected AMI. Am J Em Med. 2009;27:975-979.
8. Montagnana M, Danese E, Lippi G. Biochemical markers of acute intestinal ischemia: possibilities and limitations. Ann Transl Med. 2018;6(17):341.
9. Tilsed JV, Casamassima A, Kurihara H, Mariani D, Martinez I, Pereira J, et al. ESTES guidelines: acute mesenteric ischaemia. Eur J Trauma Emerg Surg. 2016;42(2):253-70. 10) AGA Technical review on intestinal ischaemia. Gastroenterology. 2000;118:954-968.
11. Bowersox JC, Zwolak RM, Walsh DB, Schneider JR, el al. Duplex ultrasonography in the diagnosis of celiac and mesenteric artery occlusive disease. J Vasc Surg. 1991;14:780-786.
12. Bjorck M, Acosta S, Lindberg F, Troeng T, Bergqvist D. Revascularization of the superior mesenteric artery after acute thromboembolic occlusion. Br J Surg 2002;89:923-927.
13. Florim S, Almeida A, Rocha D, Portugal P. Acute mesenteric ischaemia: a pictorial review. Insights Imaging. 2018;9(5):673-682.
14. Meaney JF, Prince MR, Nostrant TT, et al. Gadolinium-Enhanced MR angiography of visceral arteries in patients with suspected chronic mesenteric ischaemia. J Magn Reson Imaging. 1997;7:171-176.
15. Paral J, Ferko A, Plodr M, et al. Laparoscopic diagnostic of acute bowel ischaemia using ultraviolet light and fluorescein dye: an experimental study. Surg Laparosc Endosc Percutan Tech. 2007;17:291-295.
16. Yanar H, Taviloglu K, Ertekin C, et al. Planned second-look laparoscopy in the laparoscopy in the management of AMI. World J Gastroenterol 2007;13(24): 3350-3353.
17. MacSweeney ST, Postlethwaite JC. Second-look laparoscopy in the management of AMI. Br J Surg. 1994;81:90.
18. Meng X, Liu L, Jang H, et al. Indications and procedures for second-look surgery in AMI. Surgery Today. 2010;40:700-705.
19. Lendzion RJ, Frahm-Jensen G, Keck J. Acute Mesenteric Ischemia. Clin Colon Rectal Surg. 2022;35(3):227-236.
20. Bala M, Kashuk J, Moore EE et al. Acute mesenteric ischemia: guidelines of the World Society of Emergency Surgery. World J Emerg Surg. 2017;12:38
21. Chang RW, Chang JB, Longo WE. Update in management of mesenteric ischemia. World J Gastroenterol. 2006;12(20):3243-7.
22. Garzelli L, Ben Abdallah I, Nuzzo A, Zappa M, Corcos O, Dioguardi Burgio M, et al. Insights into acute mesenteric ischaemia: an up-to-date, evidence-based review from a mesenteric stroke centre unit. Br J Radiol. 2023;96(1151):20230232.
23. Terlouw LG, Moelker A, Abrahamsen J, Acosta S, Bakker OJ, Baumgartner I, et al. European guidelines on chronic mesenteric ischaemia - joint United European Gastroenterology, European Association for Gastroenterology, Endoscopy and Nutrition, European Society of Gastrointestinal and Abdominal Radiology, Netherlands Association of Hepatogastroenterologists, Hellenic Society of Gastroenterology, Cardiovascular and Interventional Radiological Society of Europe, and Dutch Mesenteric Ischemia Study group clinical guidelines on the diagnosis and treatment of patients with chronic mesenteric ischaemia. United European Gastroenterol J. 2020;8(4):371-395.