World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(RESEARCH ARTICLE)

Check for updates

Acute mesenteric ischemia in the intensive care unit: A seven-year retrospective study

Mehdi HALOUI *, Aissam EL RHARI, Soumaya TOUZANI, Nawfal HOUARI, Abderrahim EL BOUAZZAOUI, BrahIM BOUKATTA and Nabil KANJAA

Department of anesthesia and intensive care, Plyvalent ICU unit A4, Hassan II university hospital, Fez, Morocco.

World Journal of Advanced Research and Reviews, 2026, 30(02), 1132-1139

Publication history: Received on 03 April 2026; revised on 12 May 2026; accepted on 14 May 2026

Article DOI: https://doi.org/10.30574/wjarr.2026.30.2.1274

Abstract

Background: Acute mesenteric ischemia (AMI) is a rare vascular and digestive emergency associated with high mortality. Its clinical presentation is often nonspecific, which contributes to delayed diagnosis and progression to intestinal necrosis.

Methods: We conducted a retrospective, longitudinal, descriptive and analytical study in the polyvalent intensive care unit A4 of Hassan II University Hospital in Fez over a seven- year period, from January 2013 to December 2019. All adult patients hospitalized for AMI were included when the diagnosis was established preoperatively on clinical, biological and radiological grounds, or intraoperatively. Incomplete records and patients managed only in the resuscitation room were excluded. Statistical analysis was performed using SPSS 20. Quantitative variables were expressed as means and qualitative variables as percentages. Univariate analysis was performed, with statistical significance set at $p<0.05$.

Results: Sixteen patients were included, representing approximately 0.3% of ICU admissions during the study period. The mean age was 64.8 ± 12.68 years and 62.5% of patients were women. Atrial fibrillation was the most frequent comorbidity (43.75%). The mean delay before consultation was four days. All patients presented with abdominal pain, 62.5% with vomiting and 31.25% with an occlusive syndrome. CT/CT angiography established the preoperative diagnosis in 10 patients. Fifteen patients underwent surgery; intestinal necrosis was found in all operated patients. Invasive mechanical ventilation was required in 81% of patients, vasoactive drugs in 62%, and therapeutic anticoagulation in all patients. Overall mortality was 62.5%. Factors significantly associated with mortality were central venous catheterization, invasive mechanical ventilation and septic shock.

Conclusion: AMI in the ICU remains associated with high mortality. Prognosis depends on early clinical suspicion, rapid CT angiography and multidisciplinary management involving intensive care, digestive surgery and vascular surgery.

Keywords: Acute Mesenteric Ischemia; Intensive Care; CT Angiography; Intestinal Necrosis; Mortality; Septic Shock

1. Introduction

Acute mesenteric ischemia (AMI) is a severe clinical entity that is often difficult to recognize because its initial manifestations are nonspecific. It corresponds to intestinal injury secondary to sudden vascular insufficiency, causing a mismatch between blood supply and the metabolic requirements of the gastrointestinal tract [1,2]. Although rare,with an incidence classically estimated at approximately one case per 1000 hospital admissions [3], it represents an absolute medical and surgical emergency.

* Corresponding author: Mehdi HALOUI

Mortality remains high and is frequently reported at around 70% [4,5]. The mechanisms include arterial embolism, arterial thrombosis, mesenteric venous thrombosis and non- occlusive mesenteric ischemia in the setting of low splanchnic flow. AMI may also reveal or complicate pre-existing chronic mesenteric ischemia [6].

The main challenge is to establish the diagnosis before irreversible lesions develop. At the potentially reversible ischemic stage, restoration of splanchnic blood flow may preserve the bowel. Conversely, progression to transmural necrosis requires intestinal resection and is associated with a major risk of multiple organ failure and death [7].

Advances in CT angiography, vascular surgery, digestive surgery, interventional radiology and intensive care have changed the management of this multidisciplinary disease. However, prognosis remains poor, especially when diagnosis is delayed or when patients are admitted to the ICU with shock or organ failure [8]. The objective of this study was to analyze the epidemiological, clinical, biological, radiological, therapeutic and outcome characteristics of patients hospitalized in intensive care for AMI, and to identify factors associated with mortality in our series.

2. Materials and Methods

This was a retrospective, longitudinal, descriptive and analytical study conducted in the polyvalent intensive care unit A4 of Hassan II University Hospital in Fez. The study period extended from January 2013 to December 2019, i.e., seven years.

All adult patients admitted to the ICU during the study period with a diagnosis of acute mesenteric ischemia were included. The diagnosis could be established preoperatively based on suggestive clinical, biological and radiological findings, or intraoperatively during exploratory laparotomy. Incomplete files were excluded when clinical observations, biological or radiological assessments, or operative reports were missing. Patients managed only in the resuscitation room were not included.

Data were collected from the ICU admission registry, Hosix.Net electronic medical records, imaging reports and operative reports. Variables included demographic data, medical history, clinical signs, biological and radiological findings, management in the resuscitation room, operating theatre and ICU, as well as outcomes, complications and mortality.

Data were entered in Microsoft Excel 2016 and analyzed using SPSS 20. Quantitative variables were expressed as means and qualitative variables as percentages. Univariate analysis compared means and percentages using Student's t-test and the chi-square test, respectively. A p value <0.05 was considered statistically significant. The study was retrospective and based on hospital medical records. No intervention was performed for research purposes. Data were analyzed anonymously.

3. Results

During the study period, 16 patients were hospitalized in intensive care for AMI among 5204 admissions for all causes, corresponding to an approximate prevalence of 0.3%. The mean age was 64.8 ± 12.68 years, with extremes of 40 and 80 years. Patients aged 60 years or older represented 75% of the cohort. The population included 10 women (62.5%) and 6 men (37.5%), with a male/female sex ratio of 0.6.

Comorbidities were dominated by atrial fibrillation, found in 7 patients (43.75%), known in 3 patients and newly diagnosed during hospitalization in 4. Diabetes was present in 5 patients (31.25%), hypertension in 4 (25%) and ischemic heart disease in 3 (18.75%). Other less frequent histories included chronic smoking, ischemic stroke, Leriche syndrome, history of malignancy, recent cardiac catheterization, recent abdominal surgery, toxic hyperthyroidism with cardiothyreosis and old post-caesarean incisional hernia.

Table 1 Different epidemiological variants of the study

Variable	Result
Number of patients	16
Prevalence among ICU admissions	0.3%
Mean age	64.8 ± 12.68 years
Age ≥60 years	75%
Women	62.5%
Men	37.5%
Sex ratio M/F	0.6
Atrial fibrillation	43.75%
Diabetes	31.25%
Hypertension	25%

The time from symptom onset to consultation ranged from 0 to 9 days, with a mean of 4 days. Abdominal pain was present in all patients. It was sudden and intense, diffuse in 14 cases and localized in 2 cases. Vomiting was observed in 10 patients (62.5%); it was alimentary in 8 cases, bilious in 1 case and fecaloid in 1 case. Digestive bleeding in the form of rectal bleeding was noted in one patient. Transit disorders were present in 7 patients (43.75%), including absence of stool without absence of gas in 2 cases and absence of both stool and gas in 5 cases. No case of liquid diarrhea was noted.

At general examination, fever was found in 6 patients, signs of dehydration in 4, tachycardia in 8, hemodynamic collapse in 4 and impaired consciousness in 2. Abdominal examination found diffuse tenderness in 10 patients, distension in 4, guarding in 2, contracture in 1 and preserved meteorism in 1 patient. Digital rectal examination, performed in 6 patients, was normal in 3 cases and found an empty rectal ampulla in 3 cases.

All patients underwent an initial biological assessment. Neutrophilic leukocytosis was documented in 13 patients (81.25%) and CRP was elevated in all patients. Urea was elevated in 6 patients and creatinine in 8. Electrolyte disturbances were common, with hyponatremia in 5 patients, hypokalemia in 3 and hyperkalemia in 4. Troponin was positive in 7 of the 11 tested patients. Lactate was measured in only 4 patients and was elevated in 2.

Chest radiography was performed in 4 patients and plain abdominal radiography in 2. Abdominal ultrasound, performed in 3 patients, did not provide significant diagnostic information. Abdominal CT or CT angiography was performed in 13 patients and correctly established the diagnosis in 10. The main CT signs were peritoneal effusion, intestinal distension, mesenteric fat infiltration, bowel wall thickening, abnormal parietal enhancement and mesenteric vascular occlusion.

The diagnosis of AMI was established preoperatively in 10 patients. Six diagnoses were made in the emergency department on CT criteria, with a delay between admission and precise diagnosis ranging from 1 to 48 hours, for a mean of 15 hours. In the remaining cases, the diagnosis was made intraoperatively during exploration for subocclusive syndrome, occlusive syndrome, suspected postoperative peritonitis or strangulated incisional hernia.

All patients received initial resuscitation including standard monitoring, oxygen therapy, two large-bore peripheral venous lines, biological assessment and vascular filling. A central venous line was placed in 5 patients in the resuscitation room. Preoperative invasive mechanical ventilation was required in 1 patient. All patients received analgesic treatment and 6 received preoperative antibiotic therapy.

Fifteen patients underwent surgery. Surgical exploration found intestinal necrosis in all operated patients. Small bowel involvement was total in 2 patients and segmental in 13. Associated caecal involvement was observed in 4 patients and associated colonic involvement in 2. Intestinal resection was performed in 14 patients, with a mean resection length of 1.50 m. A Bouilly-Volkmann double stoma was performed in 13 patients. Therapeutic abstention was decided in 1 patient because of the extent of lesions. Vascular surgeons were consulted in 3 cases; one mesenteric-mesenteric bypass and one Fogarty catheter embolectomy were performed in two separate cases.

In the ICU, all patients received clinical and biological monitoring, oxygen therapy and respiratory physiotherapy. Invasive mechanical ventilation was required in 13 patients (81%), with a mean duration of 5.4 days. Tracheostomy was performed in 2 patients and noninvasive ventilation in 5. Catecholamines were required in 10 patients (62%), mainly for septic shock.

Management also included optimization of volume status, prevention of nephrotoxicity, initial fasting, parenteral nutrition, empirical postoperative antibiotic therapy, therapeutic anticoagulation in all patients and etiological treatment according to context. Enteral feeding was authorized in 5 patients after a mean duration of 5 days.

Mean ICU stay was 11 days, with extremes of 1 and 32 days. The main complications were septic shock in 7 patients, functional acute kidney injury in 8, acute respiratory distress syndrome in 3, cardiogenic shock in 1, hemorrhagic shock in 1 and recurrent AMI in 5.

Outcome was favorable in 6 patients. Ten deaths were recorded, corresponding to an overall mortality of 62.5%. The main cause of death was multiple organ failure.

Univariate analysis showed that three factors were significantly associated with mortality: central venous catheterization in the ICU, invasive mechanical ventilation and septic shock. Among the 11 patients with a central venous catheter, 9 died, compared with 1 death among the 5 patients without a central venous catheter (p=0.036). Ten of the 13 mechanically ventilated patients died, whereas no death was observed among non- ventilated patients (p=0.036). All patients who developed septic shock died (p=0.01).

4. Discussion

Our study confirms that acute mesenteric ischemia admitted to the ICU is rarely an early and isolated form. It is instead a vascular, digestive and systemic emergency, often diagnosed when intestinal lesions are already irreversible. In our series, all operated patients had intestinal necrosis, explaining the burden of surgical management, the frequency of organ failure and the overall mortality of 62.5%. This illustrates the central issue in AMI: the useful therapeutic window is short, and prognosis depends less on one isolated treatment than on the rapid sequence of clinical suspicion, CT angiography, resuscitation, surgical decision-making and possible revascularization [9,10].

Epidemiologically, the mean age of our patients was 64.8 years, with a high proportion of patients aged 60 years or more. This finding is consistent with the literature, where AMI preferentially affects older patients with cardiovascular comorbidities [11,12]. Published international series report comparable mean ages, often above 60 years [13-20]. This predominance is explained by the increasing frequency of atherosclerotic lesions, rhythm disorders, heart failure and diffuse vascular disease with age [21]. In our cohort, age was higher among deceased patients than survivors, but without a statistically significant difference. This absence of significance should be interpreted cautiously because of the small sample size.

Our series was characterized by female predominance, with a male/female sex ratio of 0.6. Published data are variable, with some series reporting male predominance and others female predominance [13,22-24,14-20]. Sex alone therefore does not appear to be a major prognostic determinant. Individual risk should instead be interpreted according to vascular background, diagnostic delay, initial hemodynamic status and extent of intestinal lesions.

Atrial fibrillation was the most frequent comorbidity, found in 43.75% of patients. This is consistent with the major role of arterial embolism in the pathophysiology of AMI, particularly in the superior mesenteric artery [25-27,10]. Detection of atrial fibrillation during hospitalization in some patients highlights the value of systematic electrocardiography and early rhythm monitoring in any older patient with unexplained acute abdominal pain. Diabetes, hypertension and ischemic heart disease were also frequent, reflecting an atheromatous background that favors mesenteric arterial thrombosis. In a compatible clinical context, these factors should lower the threshold for CT angiography.

The mean consultation delay of four days was probably one of the major determinants of the poor prognosis observed. AMI is classically described as intense abdominal pain, sometimes disproportionate to initially limited physical findings [28-30]. This misleading presentation exposes patients to diagnostic errors and delayed treatment. In our study, abdominal pain was constant and vomiting frequent, but many patients already had signs of advanced disease, including occlusive syndrome, distension, guarding, contracture, shock or impaired consciousness. These findings suggest that most patients were no longer at a reversible ischemic stage but at the stage of established intestinal infarction.

The clinical analysis of our cohort reinforces that the absence of an initial peritoneal sign should never be reassuring when the context is suggestive. The diagnosis should be considered in any acute, sudden, intense or unusual abdominal pain, especially in an older patient with atrial fibrillation, heart disease, atherosclerotic disease or a low-flow state. The literature emphasizes that survival is clearly better when diagnosis is established within the first 24 hours and decreases when management is delayed [9]. In our series, the frequency of intestinal necrosis among operated patients was therefore probably the direct consequence of both prehospital and in-hospital delay.

The biological findings observed in our study were dominated by neutrophilic leukocytosis and consistently elevated CRP. These abnormalities reflect inflammation, tissue injury and sometimes sepsis, but they are not specific to AMI. Similarly, hydroelectrolyte disturbances, functional renal failure, hepatic cytolysis or elevation of muscle enzymes may accompany severe disease without confirming the diagnosis. Their main value is therefore to assess severity, guide resuscitation and identify associated organ failures.

Lactate deserves special attention. In our series it was measured in only four patients and was elevated in two. This should not be interpreted as low disease severity, since all operated patients had intestinal necrosis. It mainly reinforces that normal lactate cannot exclude AMI, especially early in the course of disease or when hepatic clearance is preserved [10,31,32]. Lactate should therefore be used as a severity and resuscitation marker, not as an exclusion test. Waiting for hyperlactatemia before requesting CT angiography may cause harmful delay.

CT angiography played a central diagnostic role. It established the preoperative diagnosis in 10 of the 13 patients explored by CT. The most frequent signs were peritoneal effusion, intestinal distension, mesenteric fat infiltration, wall thickening, abnormal enhancement and vascular occlusion. The combination of vascular and bowel-wall signs is essential because CT angiography does not merely confirm ischemia; it helps determine the mechanism, assess the extent of intestinal involvement, detect signs of necrosis and guide therapy [33,34].

Our results support recommendations placing contrast-enhanced CT angiography at the forefront whenever AMI is suspected [35,10]. Ideally, the examination should include non- contrast, arterial and venous phases with multiplanar reconstructions. In our series, three patients underwent surgery without prior CT, probably because of an obvious surgical abdomen or life-threatening emergency. This remains acceptable when peritonitis or instability requires immediate laparotomy, but it should not lead to underuse of imaging in patients who can still be explored.

Therapeutic management in our series was dominated by digestive surgery. Fifteen patients underwent surgery and fourteen had intestinal resection. This high frequency of resection reflects delayed diagnosis at the necrotic stage. The surgical objective was to remove nonviable segments, control peritoneal contamination and limit progression toward multiple organ failure. Stoma creation in most cases was consistent with sepsis, hemodynamic instability and the risk of anastomotic leakage.

Surgical revascularization was performed in only two patients and no patient underwent endovascular treatment. This absence is explained by limited emergency access to angiography but also by the advanced lesion stage at diagnosis. Recent data suggest that endovascular techniques may reduce mortality and avoid some laparotomies in selected patients, especially in the absence of signs of intestinal necrosis [36,37]. However, once necrosis is established, surgical exploration remains indispensable. The challenge is therefore not only to have an endovascular platform available, but above all to identify patients earlier, before transmural infarction develops.

Intensive care was central to outcome. All patients required hemodynamic, respiratory, nutritional, anti-infective and anticoagulant management. The initial goal is to restore adequate tissue perfusion, correct hypovolemia related to third spacing, maintain oxygenation and prevent worsening splanchnic ischemia [10,38]. Catecholamines were required in 62% of patients. Although vasopressors may theoretically worsen splanchnic vasoconstriction, they remain indispensable in persistent shock after volume optimization because systemic perfusion pressure also conditions mesenteric perfusion.

Postoperative antibiotic therapy was administered to all patients, which appears justified by the frequency of intestinal necrosis, peritoneal contamination and risk of bacterial translocation. Therapeutic anticoagulation was also prescribed in all patients. This approach is consistent with thrombotic or embolic mechanisms and the need to prevent lesion extension or recurrence [10,38,39]. In our series, AMI recurrence occurred in 31.25% of patients and required repeat surgery, highlighting the difficulty of etiological control and the need for close postoperative monitoring.

Overall mortality of 62.5% is comparable to several published series [22,24,14,18]. It is explained by diagnostic delay, extent of intestinal lesions, frequency of shock, need for mechanical ventilation and organ failure. Deaths were mainly

related to multiple organ failure. Pathophysiologically, this evolution results from intestinal necrosis, mucosal barrier disruption, bacterial translocation, systemic inflammatory response, septic shock and ischemia-reperfusion injury [40-43]. Mortality is therefore not only the consequence of a local digestive lesion, but of an established systemic disease.

In univariate analysis, septic shock, invasive mechanical ventilation and central venous catheterization were significantly associated with mortality. Septic shock was the strongest marker, with 100% mortality among affected patients. Mechanical ventilation also reflected severity, whether neurological, respiratory or hemodynamic. Central venous catheterization should be interpreted as an indirect marker of instability and catecholamine requirement rather than an independent causal factor.

These findings are broadly consistent with prognostic factors reported in the literature. Leone et al. identified advanced age, high SOFA score at diagnosis and lactate above 2.7 mmol/L as poor prognostic factors [18]. Studer et al. emphasized the value of duration of digestive necrosis and hyperlactatemia [19]. Alhan et al. reported diabetes, symptom duration before surgery, shock, metabolic acidosis and relaparotomy as factors associated with death [44]. Gupta et al. proposed a prognostic score based on several preoperative factors [45]. In our series, the small sample size did not allow confirmation of all these associations, but the observed trend was similar.

Several practical lessons can be drawn from this study. First, AMI should be considered early and not as a diagnosis of exclusion. Second, CT angiography should be requested without delay when AMI is suspected, even when biological signs are not contributive. Third, management should be multidisciplinary from the outset, involving emergency physicians, intensivists, radiologists, digestive surgeons and vascular surgeons. Fourth, postoperative monitoring should actively search for respiratory, hemodynamic, renal, septic complications and recurrent ischemia.

This study has limitations. Its retrospective design exposes it to missing data and heterogeneity in practice over time. The small sample size limits statistical power and precludes reliable multivariate analysis. Important variables, including repeated lactate measurements, severity scores, histopathological assessment and precise etiological characterization, were not available for all patients. The absence of endovascular management also limits comparison with current strategies in centers with emergency interventional radiology. Despite these limitations, this series reflects the reality of a rare, severe and often late-diagnosed condition in the ICU setting.

Overall, our work shows that improving AMI prognosis primarily depends on reducing diagnostic delay. Prevention of intestinal necrosis should be the central objective. A strategy based on early clinical suspicion, urgent CT angiography, aggressive but reasoned resuscitation, anticoagulation, appropriate antibiotic therapy, rapid vascular discussion and resection of nonviable segments is the approach most consistent with the literature and with the results of our series [35,10,46].

5. Conclusion

Acute mesenteric ischemia remains a rare but formidable condition in the ICU. It mainly affects older patients, often with cardiovascular comorbidities. In our series, delayed consultation, frequent intestinal necrosis and organ failure explain the high mortality of 62.5%.

The central message is that prognosis depends primarily on early diagnosis and treatment. Any intense acute abdominal pain, particularly in an older patient or in the presence of atrial fibrillation or vascular risk factors, should raise suspicion of AMI and lead rapidly to CT angiography. Aggressive and multidisciplinary management involving intensive care, digestive surgery, vascular surgery and, when available, interventional radiology represents the best strategy to limit intestinal necrosis and reduce mortality.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

References

[1] Dewitte A, Biais M, Coquin J, Fleureau C, Cassinotto C, Ouattara A, Janvier G. Diagnosis and management of acute mesenteric ischemia. Ann Fr Anesth Reanim. 2011;30(5):410-20.

[2] Giabicani M, Corcos O, Paugam-Burtz C. Acute mesenteric ischemia: what management in intensive care? Le Praticien en anesthésie réanimation. 2020;24:141-150.

[3] Corcos O. Acute mesenteric ischemia. Colon Rectum. 2010;4:4-13.

[4] Cho JS, Carr JA, Jacobsen G, Shepard AD, Nypaver TJ, Reddy DJ. Long-term outcome after mesenteric artery reconstruction: a 37-year experience. J Vasc Surg. 2002;35:453-60.

[5] Park WM, Gloviczki P, Cherry Jr KJ, Hallett Jr JW, Bower TC, Panneton JM, et

[6] al. Contemporary management of acute mesenteric ischemia: factors associated with survival. J Vasc Surg. 2002;35:445-52.

[7] Kougias P, Lau D, El Sayed HF, Zhou W, Huynh TT, Lin PH. Determinants of mortality and treatment outcome following surgical interventions for acute mesenteric ischemia. J Vasc Surg. 2007;46:467-74.

[8] Parliteanu C, Gavillet M, Gié O, Bize P, Maillard MH. Diagnosis and treatment of mesenteric ischemia. Revue Med Suisse. 2016;12:1419-23.

[9] Gnanapandithan K, Feuerstadt P. Mesenteric ischemia. Curr Gastroenterol Rep. 2020;22(4):17.

[10] Berland T, Oldenburg WA. Acute mesenteric ischemia. Curr Gastroenterol Rep. 2008;10(3):341-6.

[11] Tilsed JVT, Casamassima A, Kurihara H, et al. ESTES guidelines: acute mesenteric ischaemia. Eur J Trauma Emerg Surg. 2016;42:253-270.

[12] Andersson R, Parsson H, Isaksson B, Norgren L. Acute intestinal ischemia: a 14 year retrospective investigation. Acta Chir Scand. 1984;150(3):217-21.

[13] Klar E, Rahmanian PB, Bucker A, Hauenstein K, Jauch KW, Luther B. Acute mesenteric ischemia: a vascular emergency. Dtsch Arztebl Int. 2012;109(14):249- 56.

[14] Crawford RS, Harris DG, Klyushnenkova EN, Tesoriero RB, Rabin J, Chen H, Diaz JJ. A statewide analysis of the incidence and outcomes of acute mesenteric ischemia in Maryland from 2009 to 2013. Front Surg. 2016;3:22.

[15] Akyildiz HY, et al. The length of necrosis and renal insufficiency predict the outcome of acute mesenteric ischemia. Asian Journal of Surgery. 2014.

[16] Yildirim D, Hut A, Tatar C, Donmez T, Akinci M, Toptas M. Prognostic factors in patients with acute mesenteric ischemia. Turk J Surg. 2017;33(2):104-109.

[17] Huang HH, et al. Clinical factors and outcomes in patients with acute mesenteric ischemia in the emergency department. J Chin Med Assoc. 2005;68(7).

[18] Zhao Y, Yin H, Yao C, Deng J, Wang M, Li Z, Chang G. Management of acute mesenteric ischemia: a critical review and treatment algorithm. Vasc Endovascular Surg. 2016;50(3):183-92.

[19] Leone M, Bechis C, Baumstarck K, Ouattara A, Collange O, et al. Outcome of acute mesenteric ischemia in the intensive care unit: a retrospective, multicenter study of 780 cases. Intensive Care Med. 2015;41(4):667-676.

[20] Studer P, et al. The value of serial serum lactate measurements in predicting the extent of ischemic bowel and outcome of patients suffering acute mesenteric ischemia. J Gastrointest Surg. 2015;19:751-755.

[21] Reissfelder et al. Ischemic colitis: who will survive? Surgery. 2011;149(4):585-92.

[22] Thomas JH, Blake K, Pierce GE, Hermreck AS, Seigel E. The clinical course of asymptomatic mesenteric arterial stenosis. J Vasc Surg. 1998;27:840-4.

[23] Kassaiaa FZ. Mesenteric infarction: about 19 cases. Medical thesis no. 91. Faculty of Medicine and Pharmacy of Marrakech; 2010.

[24] Bocoum A. Acute mesenteric ischemia: epidemiological, diagnostic and therapeutic aspects at CHU Gabriel Toure. Medical thesis; 2021.

[25] Aouni F, et al. Acute mesenteric ischemia: study of predictive factors of mortality. La Tunisie Médicale. 2012;90(7):533-536.

[26] Leon A, Lepousé C, Floch T. Mesenteric ischemia: clinical presentation and resuscitation. Le Praticien en Anesthésie Réanimation. 2005;9(1).

[27] Björck M, Koelemay M, Acosta S, et al. Management of diseases of mesenteric arteries and veins: ESVS clinical practice guidelines. Eur J Vasc Endovasc Surg. 2017;53(4):460-510.

[28] Acosta S, Björck M. Acute thrombo-embolic occlusion of the superior mesenteric artery. Eur J Vasc Endovasc Surg. 2003;26(2):179-83.

[29] Bartholin F, Delabrousse E, Destrumelle N, Capellier G. Management of suspected acute mesenteric ischemia. Réanimation. 2001;10:654-65.

[30] Nuzzo A, Corcos O. Mesenteric ischemia in the era of intestinal vascular emergency structures. Journal Européen des Urgences et de Réanimation. 2017;29(4):313-326.

[31] Corcos O, Bouhnik Y. Acute mesenteric ischemia. Post'U. 2013:177-188.

[32] Kärkkäinen JM, Lehtimäki TT, Saari P, et al. Endovascular therapy as a primary revascularization modality in acute mesenteric ischemia. Cardiovasc Intervent Radiol. 2015;38(5):1119-1129.

[33] Jonas J, Schwarz S, Dehkordy A. Behavior of the lactate level in occlusion and reperfusion of the superior mesenteric artery. Langenbecks Arch Chir. 1996;381:1- 6.

[34] Wiesner W, Khurana B, Ji H, Ros PR. CT of acute bowel ischemia. Radiology.

[35] 2003;226:635-50.

[36] Menke J. Diagnostic accuracy of multidetector CT in acute mesenteric ischemia: systematic review and meta-analysis. Radiology. 2010;256:93-101.

[37] Bala et al. Acute mesenteric ischemia: guidelines of the World Society of Emergency Surgery. World J Emerg Surg. 2017;12:38.

[38] Beaulieu RJ, Arnaoutakis KD, Abularrage CJ, et al. Comparison of open and endovascular treatment of acute mesenteric ischemia. J Vasc Surg. 2014;59:159- 164.

[39] Acosta S, Sonesson B, Resch T. Endovascular therapeutic approaches for acute superior mesenteric artery occlusion. Cardiovasc Intervent Radiol. 2009;32:896- 905.

[40] Clair DG, Beach JM. Mesenteric ischemia. N Engl J Med. 2016;374(10):959-968.

[41] Acosta S. Mesenteric ischemia. Curr Opin Crit Care. 2015;21(2):171-178.

[42] Goris RJ, Beokhorst PA, Nuytinck KS. Multiple organ failure: generalized autodestructive inflammation. Arch Surg. 1985;120:1109-1115.

[43] de Smet AMGA, et al. Decontamination of the digestive tract and oropharynx in ICU patients. N Engl J Med. 2009;360(1):20-31.

[44] Aranow JS, Fink MP. Determinants of intestinal barrier failure in critical illness. Br J Anaesth. 1996;77:71-81.

[45] Biffl WL, Moore EE. Splanchnic ischemia/reperfusion and multiple organ failure. Br J Anaesth. 1996;77:59-70.

[46] Alhan E, et al. A study on 107 patients with acute mesenteric ischemia over 30 years.

[47] Int J Surg. 2012;10:510-513.

[48] Gupta PK, Natarajan B, Gupta H, Fang X, Fitzgibbons RJ Jr. Morbidity and mortality after bowel resection for acute mesenteric ischemia. Surgery. 2011;150(4):779-787.

[49] Corcos O, Castier Y, Sibert A, et al. Effects of a multimodal management strategy for acute mesenteric ischemia on survival and intestinal failure. Clin Gastroenterol Hepatol. 2012.