

## Intestinal Infarction Through Arterial Vascular Obstruction – Case Series from 1<sup>st</sup> and 3<sup>rd</sup> Surgery Clinics Cluj-Napoca

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### Rezumat

#### *Infarctul intestinal prin obstrucție vasculară arterială la adult - serie de cazuri din Clinicile de Chirurgie I și Chirurgie III Cluj-Napoca*

Articolul prezintă o serie de cazuri de infarct intestinal prin obstrucția arterei mezenterice superioare - două cazuri de embolie mezenterică, două cazuri de tromboză mezenterică și un caz de volvulus.

**Cuvinte cheie:** infarct intestinal, embolie mezenterică, tromboză mezenterică, volvulus

### Abstract

This article presents a case series of intestinal infarction through obstruction of superior mesenteric artery - two cases of acute mesenteric artery embolism, two cases of acute mesenteric artery thrombosis and a case of volvulus.

**Key-words:** intestinal infarction, acute mesenteric artery embolism, acute mesenteric artery thrombosis, volvulus

### Introduction

Acute mesenteric ischemia (AMI) consists of the rapid, partial or complete, interruption of the blood flow in the irrigation area of the superior or inferior mesenteric artery, that results in intestinal infarction - the hemorrhagic necrosis of the intestines (1).

The rate of AMI (diagnosed at either autopsy or operation) in Malmö, Sweden (1970-1982) has been estimated to 12.9:100,000 per year. The incidence was exponentially increasing with age, and no difference has been observed between sexes (2). In Finland, the incidence of AMI (in-hospital) between 2009 and 2013 was 7.3:100,000 per year (4.5 for the obstructive forms) (3). Despite the major progresses in the diagnostic tools and treatment in the past years, due to the low rate of clinical suspicion, the late presentation at the hospital, the advanced age of the patients and the present comorbidities, the mortality rate is still estimated to be 40-70% (4). Delayed diagnosis rapidly results in declining survival rates, with 50% survival when diagnosed within 24 hours of symptom onset, and 30% survival when diagnosed beyond this time frame (5).

In approximately 50% of the cases, the cause of AMI is acute mesenteric artery embolism (AMAE), in 25% non-occlusive mesenteric ischemia (NOMI), in 10% acute mesenteric artery thrombosis (AMAT), in another 10% mesenteric venous thrombosis and in 5% focal segmental ischemia (6-8).

The causes of arterial obstruction of the mesentery can be further divided into internal (embolia or thrombosis) or external mechanisms (incarcerated transmesenteric intestinal hernia (9), neighboring tumour (10) or volvulus (11)).

In this article we present 5 cases of AMI that have been classified according to the physiopathologic mechanisms of their production. These cases aim to highlight the difficulty of

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the diagnosis and treatment of the condition and to show the poor prognostic that these patients have.

### Case series presentation

#### Case 1 and 2. Intestinal infarction through mesenteric artery embolism

Case 1. A 75 year-old man presented to the emergency room (ER) on 05.29.2015 with an abdominal pain of medium intensity in the epigastric region with a relatively sudden onset 72h prior to admission, vomiting, and four watery stools. The patient has a history of essential hypertension treated with triple therapy, hypertensive and ischemic heart disease, moderate degenerative mitral insufficiency, tricuspid insuffi-

ciency, mild pulmonary hypertension, mild degenerative aortic stenosis, major right bundle branch block, permanent atrial fibrillation with oral anticoagulant treatment, congestive heart failure class II according to the New York Heart Association (NYHA) and diabetes mellitus type 2 under treatment with oral antidiabetic drugs.

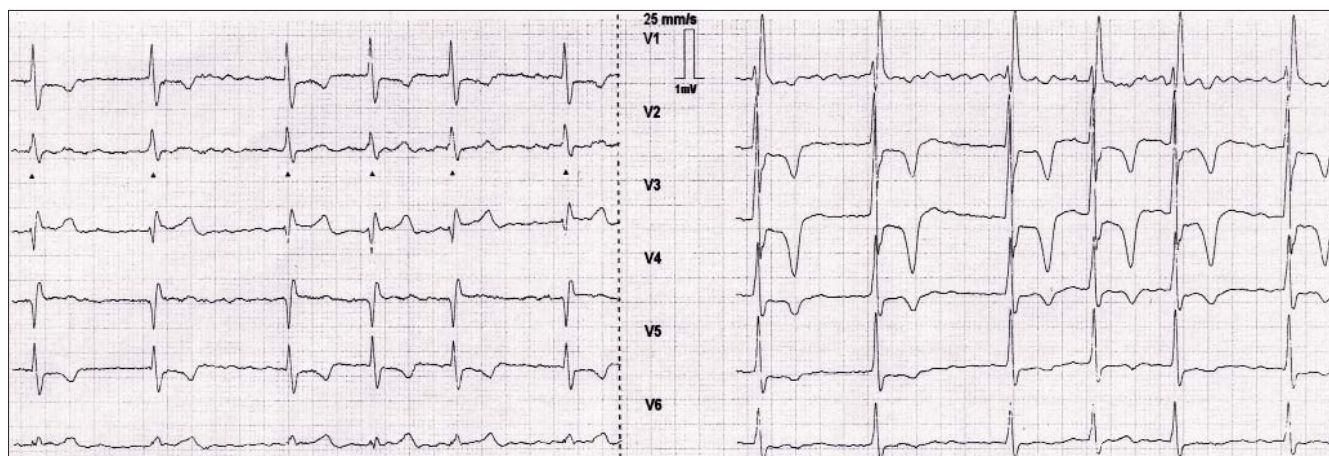
On the clinical examination the patient was hard to cooperate with, had a normal orientation in time and space, but was restless and agitated. He presented cold teguments, polypnea, and an abdomen increased in volume, with signs of cyanosis on its sides, without hydroaeric sounds.

Laboratory results showed a severe metabolic acidosis, leukocytosis, increased levels of urea and uric acid and efficiently anticoagulated (Table 1). The EKG showed atrial fibrillation with a medium ventricular frequency, a complete

Table 1. Comparative synthesis of the cases

	Case 1 S.G., 75 year-old, male	Case 2 T.A., 70 year-old, male	Case 3 P. M., 80 year-old, female	Case 4 P. N., 62 year-old, male	Case 5 F. I., 80 year-old, male
Clinical picture	Abdominal pain of medium intensity in the epigastric region with a relatively sudden onset 72h prior to admission, vomiting, and four watery stools, abdomen increased in volume, without hydroaeric sounds	Severe abdominal pain, which was accompanied by distension	Diffuse abdominal pain that started 6h prior admission, vomiting, and three watery stools	Lack passing stool over the past 5 days, hematemesis, lipothymia, without hydroaeric sounds	Epigastric pain of sudden onset 12h prior admission
Risk factors	Ischemic heart disease, mitral insufficiency, tricuspid insufficiency, aortic stenosis, permanent atrial fibrillation	Ischemic heart disease, mitral insufficiency, aortic insufficiency, tricuspid insufficiency, recurrent atrial flutter ventricular extrasystoles	Essential hypertension, ischemic heart disease	Chronic glomerulonephritis hemodialysis, secondary arterial hypertension	
Laboratory	WBC=24200x10 <sup>9</sup> /L Ne=81,8% pH=7,12 INR=2,05 Creatinine= 2,08 mg/dL Urea=65 mg/dL	WBC=10640 x10 <sup>9</sup> /L Ne=85,2% LDH=521 U/L K <sup>+</sup> =5,1 mmol/L INR=1,22 ASAT=35U/L ALAT=45U/L	WBC = 16730 x10 <sup>9</sup> /L pH=7,18 Lact=12,8, mmol/L Na=162mmol/L INR = 1,63	pH= 7,15, Lact=10,87 mmol/L, K=4,82 mmol/L, Creatinine=6,78mg/dL, Urea=88 mg/dL	WBC=12100 x10 <sup>9</sup> /L pH=7,35, pCO <sub>2</sub> =28,6 mmHg, K <sup>+</sup> =4,9mmol/L,
Abdominal radiography				Hydroaeric levels all over the abdominal cavity	
Computed tomography angiography in SMA		Dilated intestinal loops with the thickening and poor substance uptake of the parietal walls, hydroaeric levels, endoluminal thrombus	Atherosclerosis of the abdominal aorta and its main branches, thrombi in SMA, pneumatosis and hydroaeric levels		Dilated intestinal loops with poor substance uptake of the parietal walls, twisted of SMA, ascites
Surgery	During the exploration entero-mesenteric infarction have been observed, from the Treitz angle until 30cm distal from ileocecal valve		During the exploration have been observed entero-mesenteric infarction, approximately 20cm distal from Treitz angle until ileocecal valve		During the exploration the volvulation of the last 60cm of the ileon has been observed, with cyanotic intestinal wall and absence of peristaltism. Segmental enteral resection latero-lateral anastomosis
Evolution	hemodynamic and respiratory instability, death after 7 hours from his presentation	death after 50 hours from diagnosis	lactic acidosis and hiperpotasemia, death after 27 hours from his presentation	hemodynamic and respiratory instability, death after 8 hours from his presentation	favorable evolution, discharge after 7 days
Diagnosis	AMAE. Intestinal infarction	AMAE. Intestinal infarction	AMAT. Intestinal infarction	AMAT. Intestinal infarction	Volvulus. Intestinal infarction

WBC - white blood cells; Ne - neutrophils; INR - International Normalised Ratio; LDH - lactat dehydrogenase; ASAT - aspartat-aminotransferase; ALAT - alanine aminotransferase; Lact - lactic acid



**Figure 1.** EKG: atrial fibrillation, complete right bundle branch block

right bundle branch block with appropriate T wave discordance (Fig. 1).

The diagnosis of acute surgical abdomen has been stated, and the decision of an emergency intervention has been made. During preoperative preparations, a big quantity of gastric fluid has been evacuated that initially had a bilious appearance, which later was found to have a dark brown color. An exploratory laparoscopy has been performed. On a closer examination, the intestines appeared to have an abnormal colour. Conversion was executed. During the exploration of the abdominal cavity entero-enteral and entero-parietal adhesions have been observed together with the almost complete entero-mesenteric infarction, from the Treitz angle until 30cm distal from Bauhin's ileocecal valve. The peristalsis of the jejunum and ileum was absent. No pulse was palpable over the superior mesenteric artery (SMA). The last 30 cm of the ileum has also presented signs of incipient ischemia. The case has been considered to have no potential surgical therapy.

After the intervention, the patient presented severe hemodynamic and respiratory instability that required the administration of noradrenalin in resuscitatory doses. He has been intubated, mechanically ventilated and major pain control has been established. 7 hours after his presentation at the ER the patient succumbed.

Case 2. A 70 year-old male patient presented on 3.30.2015 to the ER with a colliquative abdominal. The pain was severe, located in the epigastric and right hypochondriac region, non-radiating. The clinical complaints started insidiously approximately 48h before presentation, and suddenly became severe after a meal 3h prior admission, accompanied by an episode of vomiting with food content and three watery stools. The patient had a known history of severe essential hypertension, partially treated with quadruple therapy, hypertensive and ischemic heart disease, moderate degenerative mitral insufficiency, aortic insufficiency, tricuspid insufficiency, moderate pulmonary hypertension, major right bundle branch block, recurrent atrial flutter with various transmission under oral anti-coagulant therapy, nonsystematic ventricular extrasystoles and

NYHA grade III left ventricular failure.

On the clinical examination, the patient appeared pale, sweaty, with a positive Murphy sign.

Laboratory findings included leukocytosis with a mild neutrophilia, lightly elevated hepatic enzymes and INR outside of the therapeutic range (Table 1). An abdominal echography has been performed which showed an enlarged gall bladder (long axis 100cm, transvers axis 36mm) with multiple hyperechogenic regions and a double-contoured wall.

A diagnosis of acute lithiasic cholecystitis has been established. After the evaluation of the patient's cardiovascular and pulmonary comorbidities, the surgical intervention has been postponed for the following day. Conservative treatment with antibiotics (Cefoperazonum + Sulbactam 2 x 1g/1g/day IV), pain medication (Metronidazolium 4 x 0.5g/day IV), correction of the hydroelectrical disturbances, and anticoagulant therapy (Nadroparinum 3800 UI/0.4 ml) has been instituted.

On 03.31.2015, at 6am, the patient presented the clinical signs of a right carotid artery stroke. A brain CT has been performed, which was suggestive of a medial cerebral artery thrombosis with an ischemic stroke in evolution. The patient has been transferred to the neurology department, with the recommendation to continue the antibiotic therapy and his abdomen to be reevaluated ecographically. At 8 pm the patient complained of pain in the right hypochondriac region. The clinical examination showed palpatory sensibility and a soft abdomen that was lightly increased in volume with signs of meteorism. Pain medication together with antispasmodic and anti-inflammatory agents have been administered.

On 4.1.2015 the patient was evaluated by an internal medicine specialist and a new abdominal echography was performed, concluding that abdominal pathology had a stationary evolution.

On 4.2.2015 the patient complained of severe abdominal pain, which was accompanied by distension. After a surgical consult, a thoraco-abdominal Computed tomography angiography (CTA) has been ordered. This showed dilated intestinal loops ( $\varnothing_{max}$  4,5 cm) with the thickening (max 6mm) and poor



**Figure 2.** Abdomen-pelvis CTA, axial section: SMA without substance uptake, dilated intestinal loops

substance uptake of the parietal walls, hydroaeric levels, and a cecum and colon with poor parietal substance uptakes. Approximately 5 cm distal to the emergence of the SMA an endoluminal thrombus has been identified, which extended over the majority of the distal branches (Fig. 2-3). The images also showed a lithiasic hydrops of the gallbladder, without signs of acute cholecystitis.

Laboratory findings included an elevated level of LDH and hyperpotassemia (Table 1).

The diagnostic of an AMI has been established. After the pre-anesthetic evaluation the possibility of an anesthesia has been excluded. In these conditions, the patients was considered to be outside of the sphere of the surgical therapeutic possibilities. Medical treatment has been instituted to correct hydro-electrolytic disturbances, with additional anticoagulant and pain therapy.

On 4.4.2015 at 5pm the patient succumbed.

#### *Case 3 and 4. Intestinal infarction through mesenteric artery thrombosis*

Case 3. An 82 year-old woman, with a previous history of essential hypertension treated with triple therapy, ischemic heart disease, inferior myocardial infarction and a large degenerative aortic stenosis, has presented to the ER on 6.12.2015 with a diffuse abdominal pain that started 6h prior admission, vomiting, and three watery stools.

The clinical examination revealed: a polypneic patient with increased arterial blood pressure (230/70 mmHg), ventricular frequency 103/min, general palpatory sensibility of the abdomen, discrete hydroaeric sounds..

Laboratory findings included leukocytosis and an elevated LDH (Table 1).

An abdominal CTA has been performed, which showed atherosclerosis of the abdominal aorta and its main branches, several thrombi of the SMA from its origin until 7 cm distal to it, pneumatosis of the intestines and hidroaeric levels. (Fig. 4-7)

A diagnosis of AMI has been established, and a surgical



**Figure 3.** Abdomen-pelvis CTA, coronal reconstruction: SMA, jejunal and ileal arteries without substance uptake



**Figure 4.** Abdomen-pelvis CT, axial section: stenosis through calcified atheromatous plaque to the origin of SMA



**Figure 5.** Abdomen-pelvis CT, axial section: stenosis through calcified atheromatous plaque in SMA



**Figure 6.** Abdomen-pelvis CTA, axial section: SMA without substance uptake

intervention has been performed. During the preoperative phase, 300 ml of clear gastric fluid has been evacuated. The intraoperative exploration of the abdominal cavity revealed intestines of purple colour, approximately 20cm distal from Treitz angle until Bauhin's ileocecal valve and impalpable pulse of the SMA. The case has been considered to be beyond the current surgical treatment options.

After the intervention, the patient was hemodynamically and respiratory stable. Presents lactic acidosis and hyperpotassemia. Major pain therapy has been instituted. 27h after the admission, the patient suffered of cardio-respiratory insufficiency with bradycardia, followed by asystole.

Case 4. A 62-year old woman presented to the ER on 4.2.2015 at 8 am because of lack passing stool over the past 5 days, three episodes of digestive hemorrhages manifested through hematemesis, and lipothymia over the morning. The patient has a known history of chronic glomerulonephritis that is chronically treated with hemodialysis, secondary arterial hypertension, right carotid artery stroke with sequelae, and mitral, aortic and tricuspid insufficiency.

10 days prior presentation to the ER, the patient has been admitted for four days to a gastroenterology department accusing pain located in the epigastrium and right hypochondrium, meteorism, and weight loss of 5kg over the previous 3 months. The pain first appeared 1 month earlier and had a regular pattern occurring 1h after meal. A gastroscopy has been performed which revealed reflux esofagitis and antral erosive gastritis; the colonoscopy showed erosive colitis in the descendent colon, suspicious of ischemic colitis.

The clinical examination revealed: pale and cold teguments, excessive sweating, hemodynamical instability with a blood pressure of 60/40 mmHg and a silent abdomen.

Laboratory findings include decompensated metabolic acidosis, elevating levels of lactic acid, hyperpotassemia, and high values of creatinine and urea.

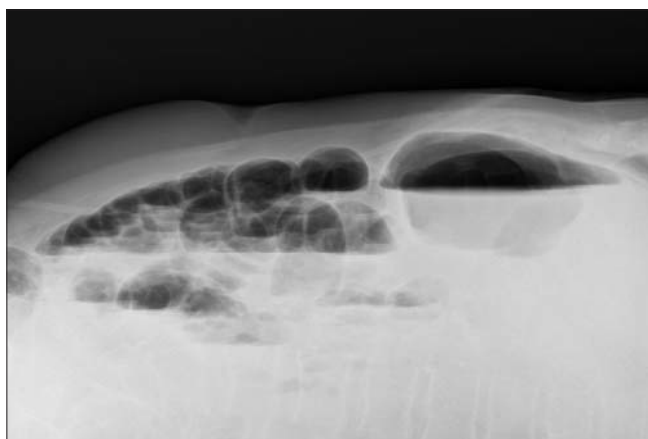
An abdominal radiography has been performed, which showed hidroaeric levels all over the abdominal cavity (Fig. 8).

Gastroscopy revealed gastric fluid with coffee ground appearance.

An attempt to restore the patient's volemic and electro-



**Figure 7.** Abdomen-pelvis CTA, coronal reconstruction: jejunal and ileal arteries without substance uptake



**Figure 8.** Abdominal radiography: hidroaeric levels all over the abdominal cavity

lytic equilibrium has been made, but it was unsuccessful. 8h after presentation the patientsuccumbs.

Necropsy showed significant atherosclerosis of the SMA, covered by a friable, purple-black coloured material that was adherent to the vascular wall (Fig. 9), elements that are highly suggestive for thrombosis. The esophagus, stomach and small intestines contained inside a purple-black material. The serous membranes of the small intestines and colon presented purple-black areas (Fig. 10) and the mucous membranes showed signs of hemorrhage and erosion.

Microscopically, we observed extensive necrosis of intestinal



**Figure 9.** Necropsy: atherosclerotic plaques at SMA

mucosa, the presence of a transmural neutrophilic infiltration.

#### *Case 5. Intestinal infarction through volvulus*

An 80 year-old man, without known history of significant diseases, presented to the ER on 6.1.2015 with epigastric pain of sudden onset 12h prior admission.

The clinical examination revealed an abdomen that was sensible during palpation both superficially and profoundly and general muscular contraction.

Laboratory results included compensated metabolic acidosis, hyperpotasemia and leukocytosis (Table 1).

The abdominal CTA was suggestive for an AMI caused by the obstruction of the SMA due to a volvulus and ascites in medium quantity (Fig. 13-14).

The diagnosis of an acute surgical abdomen has been stated, and the decision of an emergency intervention has been made. During the exploration of the abdominal cavity the volvulation of the last 60cm of the ileon has been observed, with cyanotic intestinal wall and absence of peristaltism. Segmental enteral resection has been performed with latero-lateral anastomosis. The post-operative evolution was favorable, the patient receiving treatment to maintain his hydroelectrolitic and acido-basic balance, antibiotics, anticoagulant and pain relievers.

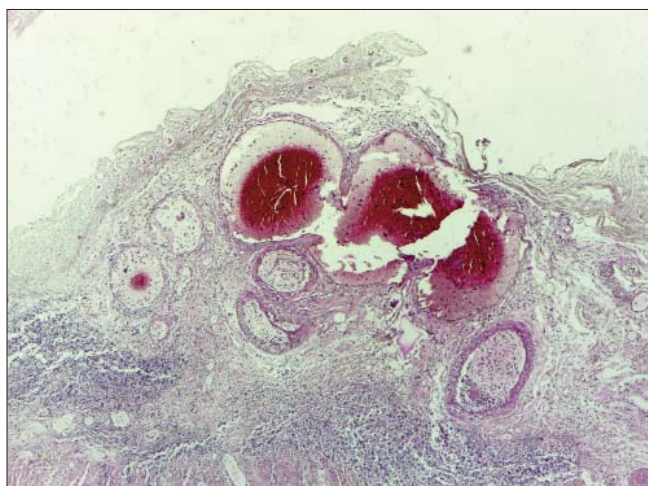
The following table presents a comparative synthesis of the cases.

## **Discussions**

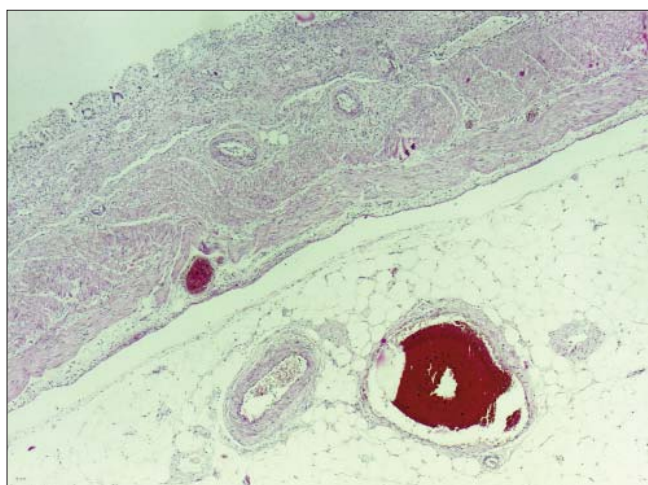
Acute mesenteric arterial embolism is most frequently caused by the migration of an intracardiac thrombus to the systemic circulation, formed under the conditions of atrial tachycardia, valvulopathy, ischemic heart disease or myocardial infarction, cardiomyopathy, ventricular aneurysm (12-16), endocarditis (17), intracardiac tumors (18-20). Other cases that have been



**Figure 10.** Necropsy: intestinal infarction



**Figure 11.** Jejunal mucosa necrosis, thrombosis



**Figure 12.** Colonic mucosa necrosis, thrombosis

reported include AMI due to aortic dissection (21), thrombosis of the ascending aorta (22), thrombangiitis obliterans (23)



**Figure 13.** Abdomen-pelvis CTA, axial section: dilated intestinal loops, poor substance uptake of the parietal walls, reduced size of SMA (arrow), ascites

and osteosarcoma (24).

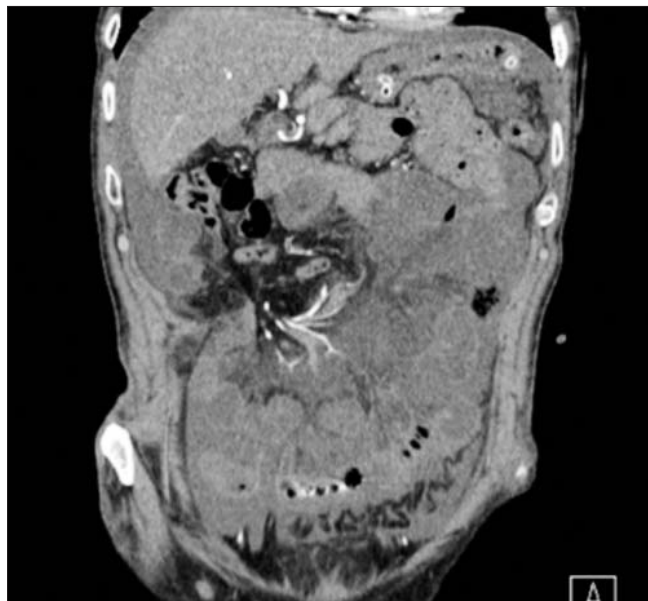
All the cases described in this article presented numerous risk factors for AMAE. Atrial fibrillation is the most common source of arterial emboli (25): during atrial fibrillation stasis of blood in the left atrium predisposes to clot formation within the left atrial appendage; these thrombi can range in size from a few millimeters to 4 cm and are found in 5-14% of patients with atrial fibrillation (26).

It's worth noting that in Case 1, the patient's INR was in the therapeutic range. Late presentation and the comorbidities played an important role in the cessation of this case.

In Case 2, the value of INR was outside of the suggested interval. Anticoagulant therapy has been quickly instituted. Nevertheless, the first embolic event occurs 24h after presentation, followed by a second one 48h later. This sequence/simultaneity of stroke and AMI is plausible in the presence of atrial fibrillation. AMAE can be synchronous with the embolism of the celiac trunk (27-28), or the vascular territory of the extremities (29). Approximately one-third of individuals with embolic AMI have had a history of a prior arterial embolization (30).

Most emboli to the mesentery lodge in the SMA. Its wide angle of origin and parallel course to the aorta makes it the most likely destination for emboli (26). Most large thrombi will lodge in the first three to eight centimeters of the vessel, often leaving the proximal jejunum well perfused (31).

A high index of suspicion in the setting of a compatible history and physical examination serves as a cornerstone of effective treatment. Many of the signs and symptoms of AMI are easily mistaken for other more common intraabdominal pathologies— pancreatitis, cholecystitis, appendicitis, diverticulitis, and bowel obstruction. The clinical presentation of Case 1 is typical to that of an AMAE. Generally, the pain has a sudden onset, and after transmural involvement of the bowel it becomes atrocious, diffuse irradiating towards the flanks, and possibly towards the inferior limbs, thorax, subcostal region associated with peritoneal irritation. The pain is frequently accompanied by vomiting and alterations of the bowel movement. The clinical



**Figure 14.** Abdomen-pelvis CTA, coronal reconstruction: twisted appearance of SMA

examination can reveal an antalgic position. Bowel sounds are generally absent. Superficial palpation typically shows no guarding, with a few signs of distention. During profound palpation rarely a tender, sausage-like mass can be felt, which, if percussed, produces a dull sound. The rest of the abdomen is resonant, except when liquid appears in the peritoneum. The installation of guarding marks the perforation of the bowels with consequent appearance of peritoneal effusion (1, 5, 32-35).

Laboratory explorations were non-specific in both cases and match the ones described in the literature: leukocytosis with neutrophilia, hemoconcentration, increased levels of urea and creatinine, high anion gap associated or not with hyperpotassemia (36, 37), metabolic acidosis (or alkalosis, if intense vomiting is present) (38). Among the many serum parameters that have been investigated there is no sufficiently sensitive or specific marker to guarantee diagnosis of AMI. The diagnostic significance of serum lactate is generally overestimated. Although AMI mortality is associated with high lactate serum values, a normal serum lactate value does not rule AMI out (39). Increased D-dimer levels are equally nonspecific (40).

CT angiography has replaced angiography as a gold standard in the diagnosis of AMI (sensitivity 0,96, specificity 0,94) (41, 42). Regular CT examination can show a thickened intestinal wall, intramural hematoma, dilated intestines filled with liquid, air in the portal vein and other localized infarctions of the abdominal viscera (43-45). Emboli typically lodge in the proximal SMA, distal to the origin of the middle colic artery. SMA embolism appears as a centrally located, hypodense intraluminal filling defect. In contrast, SMA thrombosis typically develops eccentrically at or near the ostium, on a background of atherosclerosis (46). Late presentation at the hospital with hemodynamic instability and high levels of creatinine were the impediments to perform a CT angiography in Case 1 and 4.

Conservative treatment, including bowel rest, nasogastric

drainage, intravenous fluid therapy, parenteral nutritional support, and anticoagulation therapy, was undertaken in patients with no clinical evidence of bowel gangrene with success (47).

Installation of peritonitis or intestinal gangrene exclude the possibility of using percutaneous endovascular procedures as treatment. Treatment is guided by the principle of arterial reperfusion before intestinal resection is considered (48). When restoration of blood flow is not possible through embolectomy alone, a bypass from the aorta or iliac vessels may be undertaken. Frequently, a "second-look" surgery is necessary to confirm viability of the remaining intestine 24 to 36 hours later. Systemic thrombolysis is rarely undertaken in these situations as it may delay the restoration of blood flow to the ischemic area (31).

The diagnostic laparoscopy was useful for the critical patients, for whom there was a suspicion of mesenteric intestinal infarction but who presented an atypical clinical picture.

If resection of larger portions of the intestine is necessary, the following minimum remaining intestinal lengths must be respected: 100 cm for terminal jejunostomy (colon removed), 65 cm for jejunocolic anastomosis (colon retained) or 35 cm for jejunoleal anastomosis with retention of the ileocecal region; failure to respect these minimum values leads to short bowel syndrome (49). In AMI due to SMA embolism, survival is approximately 50% when the diagnosis occurs within 24 hours after the onset of symptoms, but it drops sharply to <30% when the diagnosis is delayed (15).

Acute mesenteric artery thrombosis appears most frequently as a consequence of severe atherosclerotic disease of the vessels (50), but it can also complicate thrombangiitis obliterans (51, 52), postoperative (53) or in relations to certain pharmaceuticals (54). The clinical presentation is very similar to the one found in AMAE, although symptoms tend to appear in a progressive fashion. The intermediate rate of progression seen in patients with SMA thrombosis is due to collaterals development. Their subacute presentation may start weeks before the final acute insult that prompts them to seek medical attention. Laboratory findings are non-specific.

In Case 4, CT angiography was contraindicated because of the hemodynamic instability and renal insufficiency of the patient. An abdominal Rx has been performed, although it has very limited value: it can show initially areas of opacity, caused by the absence of gas in the affected intestine. Later on, an image that resembles the one found in paralytic ileus can be seen - pneumatosis and hydroaeric areas, as observed in our case (1).

AMAT is often preceded by a chronic mesenteric ischemia syndrome, as observed in Case 4. The patient started to present symptoms suggestive of an intestinal angina 1 month before the admission, with paroxistic pain after meals and weight loss. Because of the pain, often a 'fear' of eating develops, which leads to malnutrition. Poor absorption through the hypoxic intestinal wall also contributes to the weight loss of the affected people. With time, the frequency and intensity of the anginous episodes tend to increase, preparing the territory for an AMI.

If the diagnosis of AMAT has been made very early, endovascular procedures can still be useful to save the ischemic

organs. After the infarction, thrombectomy/thrombarterectomy /bypass with segmental enterectomy are the available treatment options.

Mesenteric ischemia may appear more frequently among patients on haemodialysis than in general population. Usually, in patients on haemodialysis develop more frequently a non-occlusive type of mesenteric ischemia and lesions in the cecum and right colon. This type of ischemia is precipitated by a severe reduction in mesenteric perfusion with secondary arterial spasm from such causes as severe hypotension during hemodialysis and myocardial infarction (55). In case 4, mesenteric ischemia is due to atherosclerotic thrombosis of the proximal portion of the SMA affecting the small bowel as shown by the necroptic exam.

Intestinal volvulus is a rare pathology among adults (56) (1% of the hospital admissions due to bowel obstruction) (57). In the elderly patients, a secondary type of volvulus with an underlying cause is frequently encountered. The possible etiology includes malrotation, congenital bands, tumours, mesenteric lymph nodes, adhesions after previous surgery, intussusception, colostomy, and internal hernias. If no underlying cause is found, as in case 5, then the volvulus is considered to be a primary. Primary intestinal volvulus linked to certain risk factors such as long mobile mesentery, short mesenteric base, long small bowel, and consumption of a large amount of fibre-rich food after prolonged fasting, with overloading of an empty intestine (59).

Besides the need to diagnose acute mesenteric ischemia early, before infarction, knowledge of the clinical risk factors that predict prognosis enables emergency physicians to use more aggressive resuscitation and treatment strategies to improve outcomes (59).

## Conclusions

The majority of the patients presented in this article deceased. Late presentation and cardiac comorbidities are the major factors that contributed to their death.

This case series presentation draws attention to the poor prognosis of the disease despite the progress of the angiographic and of the surgical techniques.

As AMI predominantly affects the older population physicians have to be aware of the possibility of this pathology when facing older patient with abdominal pain. An interdisciplinary collaboration is important, since patients are not initially evaluated by surgeons. Moreover, there is no evidence that advances in radiologic imaging have resulted in earlier recognition and a different management of our patients. Clinical decision making in patients with AMI may be difficult, but since there is a higher probability of a less-extensive intestinal infarction in these patients, curative surgical intent could be worthwhile.

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