



Acute Mesenteric Ischemia, Atypical Presentation as Acute Appendicitis in Two Young Patients: Case Report

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ABSTRACT

Introduction: Mesenteric ischemia occurs when visceral tissues receive an inadequate supply of blood supply. This can result from arterial thrombus, arterial embolus, venous thrombosis limiting arterial flow, and extrinsic compression of the blood vessels. **Clinical Cases:** **Case 1:** A 27-year-old female with a clinical picture of burning pain of 10 out of 10 intensity in the lower abdomen that radiates to the navel and right iliac fossa, accompanied by headache, diarrhea and vomiting for six days prior to admission, fever of 38 °C. He has a torpid evolution in his post-surgery with progression to multiple organ failure 3 days after surgery, beginning with hemodynamic failure with a requirement for vasoactive amines, in addition to bleeding from a nasogastric and transvaginal tube, severe thrombocytopenia. Intubation was decided due to acute respiratory failure Heart rate of 160 x',

increased respiratory pattern RF of 45x'. She died three days later, due to multiple organ failure. **Case 2:** A 32-year-old female with Polycystic Ovary Syndrome diagnosed at 13 years of age under treatment for the first 3 years with discontinued hormonal contraceptives. Due to risk and age, admission to the intensive care unit was decided, where 3 days later the oral route was started, with adequate tolerance with liquids, for which it was decided to go to the floor in charge of our service. The patient is stable. **Conclusions:** Acute mesenteric ischemia is a rare pathology, but due to its high morbidity and mortality it requires extensive knowledge about its risk factors, symptoms, diagnosis and treatment, since timely recognition leads to a satisfactory evolution. In this type of young patients with no recognized risk factors, diffuse abdominal pain and imaging and imaging studies with non-specific findings make it necessary to rule out the most frequent abdominal pathologies and suspect them.

Keywords: Mesenteric ischemia, young female, Survival, Death.

INTRODUCTION

Mesenteric ischemia occurs when visceral tissues receive an inadequate supply of blood supply. This can result from arterial thrombus, arterial embolus, venous thrombosis limiting arterial flow, and extrinsic compression of the blood vessels.

It predominantly affects male patients over 60 years of age, which occurs in 1 in 1000 hospital admissions with a mortality of up to 5%, represents a vascular emergency which is associated with a mortality greater than 60%. A delay in the diagnosis greater than 12 hours is above 90% and greater if it takes more than 24 hours.

Unfortunately, physical examination and laboratory findings are neither sensitive nor specific for the diagnosis of acute mesenteric ischemia. It is often the combination of clinical, laboratory and radiological findings with the performance of angiotomography that helps to establish the diagnosis and its possible cause.

Among the etiologies, mesenteric arterial embolism (50%), arterial mesenteric thrombosis (15-25%), or mesenteric venous thrombosis (5-15%) are discussed. Of the cases, two thirds of them correspond to women ¹.

Two clinical cases are presented which begin as a presentation of acute reactive appendicitis, secondary to mesenteric thrombosis, one patient with thrombosis of the superior mesenteric artery secondary to clinical findings and one patient associated with segmental mesenteric thrombosis.

CLINICAL CASE. 1

A 27-year-old female with a clinical picture of burning pain of 10 out of 10 intensity in the lower abdomen that radiates to the navel and right iliac fossa, accompanied by headache, diarrhea and vomiting for six days prior to admission, fever of 38 °C.

Background

Not relevant to the case. On examination soft and depressible abdomen with pain on superficial and deep palpation, positive MacBurny, positive Rovsing, positive obturator sign and positive Blumberg.

Cabinet leukocytosis 19.15, urea 21.5 Cr 0.86

X-ray of the abdomen standing and decubitus (figures 1, 2) where air-fluid levels are observed, pelvic ultrasound with a 3-cm right adnexal cyst, free fluid and it is not possible to visualize the cecal appendix, an interconsultation with a gynecologist is requested, who comments that he has no pathology of his service; At the time of reassessment, there were data of peritoneal irritation located in the right iliac fossa, for which it was decided to proceed to exploratory laparotomy on suspicion of acute appendicitis.

Trans-surgical findings: erythematous cecal appendix enlarged in size of 15x2 cm with abundant free fluid, approximately 1000 cc of sallow fluid; upon recovery he presented rectal bleeding.

In her post-surgery, the patient continued with mild pain in the right iliac fossa, on the second day, she presented fever 38°C and data of localized peritoneal irritation and leukocytes 20.31, acute abdomen was suspected due to residual abscess, she proceeded to exploratory laparotomy with a plan of Abscess drainage where abundant free sallow fluid is located, approximately 1000 cc, ischemia of approximately 115 cm of terminal ileum, cecum, ascending colon, transverse colon, and splenic angle with data of ischemia and necrosis. Hartmann pouch closure and ileostomy are performed.

He has a torpid evolution in his post-surgery with progression to multiple organ failure 3 days after surgery, beginning with hemodynamic failure with a requirement for vasoactive amines, CR 1.86 Urea 89.3 Alb 1.9 0.19. With urinary output in 24 h of 0.19 mL, in addition to bleeding from a nasogastric and transvaginal tube, severe thrombocytopenia of 35,000 PT 45.4 Sec, TPT 55.1 Sec, INR 4.1, D-dimer 37,185, severe metabolic acidosis pH 7.20 and HCO₃ 8.2 mmol/L, BE -19.8. Hypocalcemia 6.9 mg/dL, Hyperkalemia 5.2 mmol/L, Hyperphosphatemia 6.7 mg/dL, Hyperlactatemia 14.7 mmol/L. Intubation was decided due to acute respiratory failure Heart rate of 160 x', increased respiratory pattern RF of 45x'. Pale, anuric. She died 3 days later, due to multiple organ failure.

Multiple pieces were sent to pathology where they report: [Fig. 3]

Cecal appendix with data of incipient acute appendicitis, small recanalized wall thrombi. Sample of terminal ileum, 50 cm segment, with violaceous-brown serosa, with reddish areas with a hemorrhagic appearance and areas of poor fixation. Upon opening, light-brown, velvety mucosa is identified, with scant bile, on multiple cuts it is soft with a thickness 0.2 cm wall, representatives are included in two capsules. Product of resection of the ileum, viable resection edges, vascular congestion and frequent hemorrhage of the serous vessels, hyperplasia of the germinal follicles of the submucosa, negative for malignancy.

Sampling of the cecum, ascending and transverse colon, and proximal descending colon, 65

cm long and variable in diameter, the largest at 6 cm from the cecum, less than 1.9 cm at the descending level, with congestive violaceous-brown serosa with areas of hemorrhagic appearance with predominance of the right side of the colon, diffuse bleeding of 90% of the mucosal surface is observed upon opening, a transmural hemorrhage is identified on multiple cuts that thickens the wall of the cecum up to 2 cm thick, the rest of the wall varies from 15-18 cm, it has an adipose appearance, lobulated with vascular congestion, recent hemorrhage with ischemic necrosis of the colonic wall, in the ascending, transverse and descending portion, blood vessels with thrombi in the process of organization, with fibrinoid necrosis of the wall, congested serosa with recent and old hemorrhage, omentum tissue with recent hemorrhage and congestion, negative for malignancy.



Figure 1. X-ray of the abdomen standing upright.



Figure 2. Supine abdominal X-ray



Figure 3. Piece sent to pathology after the surgical event.

CLINICAL CASE 2.

A 32-year-old female with Polycystic Ovary Syndrome diagnosed at 13 years of age under treatment for the first 3 years with discontinued hormonal contraceptives.

Her condition began 11 days prior to her admission with a picture of sudden onset pain, located in the right iliac fossa, nausea and vomiting, for which she went to a private facility where a laparoscopic appendectomy was performed the following day, discharged the same day due to improvement. , however, four days later, he complained of sudden onset pain, located in the flank and right iliac fossa, oppressive, irradiating to the back, exacerbated by food intake, without extenuating symptoms, accompanied by nausea and vomiting on two occasions, denies fever , with a decrease in the caliber of the feces, which is why he went to the emergency room for evaluation.

On physical examination, vital signs were normal, on physical examination, the abdomen was globose at the expense of adipose tissue, tender on superficial palpation throughout the abdomen, absent peristalsis, tympani on percussion, with a well-addressed surgical wound without signs of infection or hematoma. , pen rose with serohematic expenditure of approx. 30cc

It is decided for a matter of hemodynamic stability and only a decrease in saturation, to request a cabinet, complete laboratories and a simple and contrasted CT scan. Due to availability, only a simple CT scan of the abdomen is performed

Admission laboratories: normal. CT on admission: periileal inflammatory changes secondary to probable post-surgical changes. Free fluid at the level of the pelvic cavity. [Figs. 1 and 2]

We continue under clinical surveillance and on the third day of hospitalization he presented increased pain and frank data of acute abdomen, for which it was decided to go to the operating room for Exploratory Laparotomy where the following was found: Segmental intestinal infarction approximately 30 centimeters to 60 centimeters from the ileocecal valve, free peritoneal fluid 700 ml swallow, not purulent. Normal appendiceal stump.

The following diagnoses are passed to the floor: Segmental intestinal infarction distal ileum 60 cm from the ileocecal valve + surgical convalescence from laparoscopic appendectomy.

After 3 days of evolution of Laparotomy, he presented symptoms of intestinal occlusion, intolerance to the oral route, tachycardia, laparotomy was performed where anastomosis stenosis was found due to Lembert's stitches, significant loop distension, closure was performed with adequate evolution.

Due to risk and age, admission to the intensive care unit was decided, where 3 days later the oral route was started, with adequate tolerance with liquids, for which it was decided to go to the floor in charge of our service. The patient is stable. When going to his appointment, a reported piece of 29 cm in length by 3 cm in diameter was found, with a brown, gray, smooth and soft serosa, with a yellow lobulated mesentery, with hemorrhagic areas, black, scattered in the mesentery, when cut from the 4 cm wall is mucosa with preserved folds, in the central area there is a change in color with a necrotic appearance and a soft friable consistency, Acute ischemic enteritis.



Figure 1. Computed tomography on admission of the patient.



Figure 2. Ileal changes evidenced in computed tomography.

DISCUSSION

The superior mesenteric artery is the main blood supply to the small intestine with some collateral from the celiac vascular system, with the superior and inferior pancreaticoduodenals, coming from the inferior mesenteric artery. The venous return goes through the portal vein. The splanchnic circulation receives 15-30% of the cardiac output, depending on the state if you are eating, but the oxygen extraction is low, compared to the oxygen supply that the portal carries to the liver. This oxygen supply can be reduced by more than 50%, after which the intestine becomes ischemic.

The pathophysiology involved, about 50% of Acute Mesenteric Ischemia, is generated by embolism of the mesenteric artery. The embolus can originate in the right atrium, associated with arrhythmias such as atrial fibrillation, left ventricular dysfunction with poor ejection fraction, or infective endocarditis. Sometimes an embolus can be generated in the aorta by atherosclerosis. Most emboli occur 3-10 cm distal to the origin of the superior mesenteric artery. They classically supply the proximal jejunum and colon. 20% are associated with previous embolism to another organ such as the spleen and/or kidney.

Acute arterial mesenteric thrombosis accounts for approximately 25% of cases and is generally associated with chronic atherosclerotic disease leading to a state of stenosis. Most patients have a history of chronic mesenteric ischemia (postprandial pain, weight loss, and/or fear of eating). It generally originates from visceral arteries, with an underlying atherosclerotic plaque, which later becomes thrombosis, generally accompanied by celiac occlusion. It can occur in pictures of vasculitis, mesenteric dissection, and mycotic aneurysm. If only the ileocolic is affected, there is only necrosis of the proximal portion of the colon ².

It must always be remembered that acute mesenteric ischemia is not common and that diagnostic delay is common; which is generally accompanied by comorbidities and high morbidity and mortality. An accurate and rapid diagnosis requires careful attention in clinical history and physical examination, such as angiotomography. The most frequent clinical data is acute abdominal pain syndrome in 70%.

The presentation usually begins with abdominal pain, which is classically out of proportion to the physical examination. It is generally moderate to severe, diffuse constant. Other symptoms that may occur are nausea/vomiting, diarrhea and transrectal bleeding. A study of acute mesenteric ischemia was performed, 95% had abdominal pain, 44% nausea, 35% vomiting, 35% diarrhea and 16% transrectal bleeding ³.

Only approximately one third of patients will present the classic triad of embolic disease, which is characterized by abdominal pain, bloody stools, and fever, associated with acute mesenteric ischemia. Among the risk factors most frequently associated with this pathology are: atherosclerosis, advanced age, heart disease, arterial hypertension, atrial fibrillation, smoking, obesity, use of digitalis ⁴.

In some situations there is no significant risk factor and only appendicitis itself with stigmata of complications (which are: mesenteric phlebitis, mesenteric perivascular inflammation) are a risk factor for mesenteric thrombosis; this is due to the appendiceal inflammatory process that causes thrombosis of intramural vessels, and as a consequence of a state of procoagulability. It was found in a report of a case of appendicitis with mesenteric venous thrombosis which was managed conservatively, in its follow-up for another month a CT scan with contrast was performed, observing the periappendicular circulation completely thrombosed and with collateral circulation.

Among the most used biomarkers for the diagnosis of this pathology are Lactate, Albumin modified by ischemia and alpha glutathione S transferase. L-lactate is generally increased by anaerobic metabolism; D-lactate is mainly generated by intestinal bacterial metabolism. L-LDH is generated endogenously in humans, but L-Lactate is considered to be increasing and is proportional to tissue hypoperfusion and cellular hypoxia. It increases up to 10 times more in patients with occlusion of the intestinal arteries. It was observed that it rises 4 hours after the start of the episode of mesenteric ischemia with a maximum of 6 hours, but it rises in other non-intestinal pathologies such as gastric perforation, pancreatitis, perforated appendicitis, ketoacidosis and malignancy. Albumin modified by ischemia, is associated with the fact that serum albumin binds to metal ions such as cobalt, copper and nickel, in episodes of acute ischemia such as Pulmonary Thromboembolism, Cerebrovascular Accident, Rhabdomyolysis and systemic sclerosis, the binding capacity of albumin, decreases as a result of hypoxia, acidosis, oxidative damage, sodium and calcium. Alpha glutathione S transferase is another stress marker that is associated with cellular detoxification. It is present in intestinal and liver cells, it can be observed in intestinal or hepatic ischemia, and the latter are measured by ELISA (Enzyme Linked Immunosorbent Assay).

Within the biomarkers of inflammation and/or infection, leukocytosis is found within normal values of 4,000-10,000; values greater than 20,000 are generally found in these patients, except in immunosuppressed or treated with steroids.

CRP (C-reactive protein), IL-6 (interleukin-6) are other biomarkers that are elevated in multiple abdominal pathologies, such as pancreatitis, complicated diverticulitis and intestinal ischemia, a cohort value of approximately 27.7 pg/mL of IL-6 is characteristic with high sensitivity and specificity. Procalcitonin (PCT) is a prohormone, secreted from liver parenchyma and other cells (including leukocytes), which can be elevated during severe infections. A disruption of the intestinal wall, after ischemia, can lead to bacterial translocation of the microbiota and/or generation of toxic products.

In thrombosis biomarkers, D-dimer is one of the best known, it is a degradation product of stabilized fibrin, it is a marker of coagulation activation and fibrinolysis, an increase in plasma suggests thrombus formation and dissolution. It is a good marker for the diagnosis of suggestive symptoms and occlusive thromboembolic disease of the superior mesenteric artery, but its value is less satisfactory in ischemic diseases with a sensitivity of 60% and a specificity of 82%.

Markers of damage to the wall are found, one of them is citrulline, which is identified and isolated from watermelon juice. It is a non-protein amino acid synthesized from glutamine in small intestine enterocytes and is a de novo formation of arginine. After being generated from the enterocytes, it passes into the portal circulation, and after it enters the systemic circulation, where it is metabolized by the kidneys and converted into arginine and released into the plasma. The value of citrulline depends on intestinal synthesis and renal metabolism, it is dependent on the mass of enterocytes. Pathologies that reduce the mass of enterocytes (short intestine, villous atrophy, Crohn's disease, acute mucosal enteropathy, among others) are associated with decreased citrulline. The half-life is 3 hours; the damage generates a decrease of this marker ⁵⁻⁷.

Among the diagnostic modalities are conventional radiography, which has non-specific findings and more than 25%, can be found normal. There is no dilation of intestinal loops, air-fluid levels, the typical is an ileus. There are nonspecific signs and intestinal infarction may appear. However, there may be the presence and absence of perforation, intestinal pneumatosis (submucosal gas), intestinal wall in thumb impression sign, portal gas is a late sign ⁸.

Abdominal ultrasound and color Doppler can provide indirect data of intra-abdominal pathology, such as thickening of the intestinal wall and ascitic fluid. Color Doppler helps to recognize mesenteric flow, but not that of the small distal vessels. The application of intravenous contrast increases the visualization of the vessel and facilitates the evaluation of intravascular thrombi. It must be taken into account that it is operator dependent and its sensitivity is variable. Take into account the gas that sometimes limits the ultrasound window. Six of the 14 cases studied were performed as the first modality and ascitic fluid was found and thickening of the intestinal wall was identified in only 2 and the absence of portal flow in another two ⁹.

Computed Axial Tomography has a high specificity and sensitivity of approximately 90%, it helps to distinguish abdominal pain from other pathologies. It has to be done with IV contrast medium. It is performed with 100-150 mL of non-iodinated contrast, preferably with an infusion of 2.5-4 mL per second, with a 30-second scan in the arterial phase and 60-70 seconds in the venous phase. With cuts of approx. 5 mm for adequate reconstruction, there are new CT scanners with 1-2 mm cuts. Among the findings are filling defects in the middle colic and/or in

distal branches, as well as decreased diameter of the superior mesenteric artery and/or superior mesenteric vein. The filling contrast in the intestinal wall is absent or diminished. Ileus can be observed, in addition to transmural infarction with thinning of the wall, intramural gas or free air can be seen ¹⁰.

That is why the use of CT with IV contrast is the initial modality of choice when clinically suspected to diagnose mesenteric thrombosis, being diagnosed in more than 90% of cases, giving guidelines for timely and rapid management, reducing its time of evolution (which has an average of 5-14 days of evolution until its diagnosis) and an early exploratory laparotomy where the objective is the resection of the necrotic intestinal segment and preserving the greatest amount of viable intestine.

Contrast angiography and 3D reconstruction are excellent non-invasive techniques for patients with suspected mesenteric ischemia from any cause. It has a good resolution and rapid acquisition, in places where it is available, it allows seeing the viability of all the visceral peripheral vessels and the inferior mesenteric artery, it helps to identify atheromatous or calcified plaques, the only risk is the contrast, radiation and the risk of kidney failure.

Gadolinium-enhanced magnetic resonance angiography has shown high specificity and sensitivity with visualization of stenosis and obstruction of the Superior Mesenteric Artery and/or celiac trunk ¹¹.

Within the initial treatment, it lies in aggressive hemodynamic support, which corrects hypovolemia and avoids vasoconstrictors (especially alpha-agonists), in order to maintain perfusion in the splanchnic territory. Metabolic acidosis should improve and broad-spectrum antibiotic therapy should be initiated to prevent and treat bacterial translocation. Anticoagulation is essential to reduce the risk of embolization in patients with atrial fibrillation, regardless of possible bleeding during the subsequent procedure. Second, as long as the patient's clinical situation allows it, revascularization should be considered, taking into account the etiology, duration of the symptoms, patient comorbidities, and technical resources. Those with data suggestive of peritonitis or radiological data of advanced intestinal ischemia will be treated directly with laparotomy. To evaluate the extension of the affected intestine (color, distension, peristalsis), pulse at the level of the SMA, mesenteric arcades and possible occupation of the SMV; some teams use intraoperative Doppler and even fluorescein injection to examine intestinal perfusion, although its utility is controversial. In cases in which there is no massive necrosis of the entire small intestine, but if clearly non-viable areas are found, a rapid and conservative resection is performed with damage control and within the same surgical time, revascularization is performed in occlusive cases. In unstable, elderly and comorbid patients, with a short or very distal segment, an isolated excision can be performed without correction of the arterial or venous lesion ¹².

Zhang Z, et al, comment in their comparative study between endovascular therapy and open surgery, where 30 patients were analyzed, whose diagnosis was confirmed based on angiography or angiotomography. The endovascular therapies used were aspiration, thrombolysis, anticoagulation, balloon dilation, stent implantation, and surgical management, which included laparotomy, excision of necrotic bowel, arterial embolectomy, intestinal fistula, and delayed abdominal closure. Laparotomy was preferred in patients with clinical

deterioration or pain data suggestive of intestinal necrosis. Endoscopic therapy was performed on 18 patients, 12 men and 6 women with a mean age of 60 +/- 10.9, of which 2 of them had 75-90% occlusion and the other more than 90% or until complete occlusion. 8 cases were seen before 12 hours and 10 cases in less than 12 hours. All underwent catheter thrombolysis, 9 (50%) received balloon dilation, 2 (11.1%) and 1 (5.6%) required stenting. The technique was completed in 8 (44.4%) cases and partial in 10 (55.6%). After endovascular management, some presented deterioration, for which surgical management was performed in 6 patients who underwent laparotomy with an average resection of 88 +/- 44 (20-150) cm. It shows us the type of management; timely treatment and early recognition play a crucial role in the management of this pathology^{13, 14}.

CONCLUSIONS

Acute mesenteric ischemia is a rare pathology, but due to its high morbidity and mortality it requires extensive knowledge about its risk factors, symptoms, diagnosis and treatment, since timely recognition leads to a satisfactory evolution. In this type of young patients with no recognized risk factors, diffuse abdominal pain and imaging and imaging studies with non-specific findings make it necessary to rule out the most frequent abdominal pathologies and suspect them. The lack of resources in first or second level units makes it a challenge to approach, but early suspicion and recognition would allow referral to a third level or to an optimal unit where adequate treatment can be carried out, since the endovascular therapy reduces morbidity, decreased hospital stay, short-term mortality, in addition to decreased intestinal resection.

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