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RESEARCH ARTICLE

SEVERE ACUTE PANCREATITIS REVEALING EXTENSIVE AORTIC DISSECTION: A CASE REPORT AND LITERATURE REVIEW

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Abstract

Severe acute pancreatitis is a frequent reason for hospitalization in the intensive care unit. Biliary and alcoholic etiologies are the most frequent. Vascular etiology, particularly ischemic etiology, is rare. Its often-serious consequences raise the question of how to proceed: should surgical treatment be provided, or should medical care be delayed? This study reports the case of an 87-year-old patient, known to be hypertensive, admitted to the emergency department of the Oued Eddahab military hospital with excruciating epigastric abdominal pain. Investigations revealed severe acute pancreatitis of ischemic origin following thoracoabdominal aortic dissection extending to the superior mesenteric artery. A review of the literature revealed only 15 similar cases of acute pancreatitis of ischemic origin associated with aortic dissection. This underlines the rarity of this situation, and the absence of standardized recommendations for the management of this exceptional situation, which presents the clinician with the dilemma of whether to opt for surgical or medical management. This case demonstrated the exceptional role of medical imaging in the context of severe acute pancreatitis, in terms of diagnosis, prognosis and etiology. It also highlighted the importance of an individualized approach based on multidisciplinary consultation, given the absence of recommendations and the paucity of literature.

Introduction:-

Acute pancreatitis is defined as inflammation of the pancreatic gland. It can be triggered by reflux of biliopancreatic fluid into the main pancreatic duct or by increased pressure (gallstone, abnormality of the biliopancreatic junction) or by direct toxic action (alcoholic pancreatitis).

The incidence is estimated at 70 cases per 100,000 inhabitants, representing the third leading cause of gastrointestinal pathology in the United States and 4% of abdominal pain syndromes.

Gallstone formation and alcohol remain by far the primary etiopathogenic factors of the disease.

The ischemic etiology of acute pancreatitis is very rare; its pathophysiology is mainly associated with ischemia-reperfusion injuries. It can be encountered in various clinical situations, particularly during states of shock, postoperatively following major vascular surgeries, in post-hypotensive anesthesia, or in the context of extensive aortic dissection.

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We report the case of a patient hospitalized for severe acute pancreatitis, and the etiological investigation revealed an ischemic cause due to an extensive thoraco-abdominal aortic dissection extending to the superior mesenteric artery, causing obstruction. The objective of reporting this case is to study and compare, with the literature, a case of acute ischemic pancreatitis due to untreated aortic dissection and to emphasize this rare ischemic etiology of severe acute pancreatitis and the challenges in its management, particularly in the decision-making regarding the operability of the patient.

Case Report:

Patient information:

The patient is an 87-year-old individual who has chronic hypertensionfor 30 years with a combination of a calcium channel blocker and an angiotensin II receptor antagonist (amlodipine/valsartan). He has no history of chronic alcohol consumption or smoking.

He presented to the emergency department with a transfixing epigastric painthat had been evolving for two days, worsened by food intake, accompanied by vomiting, fever, and a general deterioration of his overall condition.

Clinical findings:

On admission, the clinical examination revealed hypertension at 180/90 mmHg, a heart rate of 79 bpm, a respiratory rate of 18 cpm. He had a fever of 38°C. His pulse oxygen saturation was 97% in ambient air. His capillary blood glucose level was 2.8 g/L.

The abdominal examination revealed a distended abdomen with no scar or collateral vein circulation, along with epigastric tenderness, without guarding or palpable mass, and the presence of diffuse tympany. Hernial orifices were free. The rest of the clinical examination was unremarkable.

Diagnostic evaluation and therapeutic intervention:

Face to the intense epigastric pain, an electrocardiogram was performed, ruling out acute coronary syndrome. However, it did reveal left ventricular hypertrophy without electrical changes in the ST segment.

Faced with the transfixing epigastric pain contrasting with a limited clinical examination, acute pancreatitis was strongly suspected. The lipase level, exceeding three times the normal range, with a value of 1130 IU/L, confirmed the diagnosis.

To evaluate the severity of acute pancreatitis, a laboratory test was requested. It revealed an elevated C-reactive protein at 279 mg/L, leukocytosis at 17,600/mm³, and a renal deficiency suggestive of functional impairment with a creatinine clearance of 47 ml/min. The creatinine level was 15 mg/L (132.6 micromoles/L). Plasma urea was 1.2 g/L.

Moreover, the assessment revealed moderate hepatic cytolysis without cholestasis: AST = 123 IU/L; ALT = 105 IU/L; normal alkaline phosphatase (ALP); normal total bilirubin levels. Additionally, there were electrolyte imbalances: hypokalemia at 2.6 mmol/L without electrical signs on the electrocardiogram; hyponatremia at 131 mmol/L with a corrected sodium level of 134 mmol/L, and normal calcium levels at 2.4 mmol/L.

The rest of the laboratory results revealed a hypochromic microcytic anemia with a hemoglobin level of 8.8 g/dL.

The diagnosis of severe acute pancreatitis was established based on the elevation of CRP exceeding 150 mg/L.

The patient was transferred to the intensive care unit for initial management, which included:

1. Preparation: Insertion of large-caliber intravenous line, nasogastric tube, and bladder catheter.
2. Stopping oral feeding
3. Multimodal analgesia, including titrated initiation of morphine
4. Vascular filling with isotonic saline 0,9 %
5. Correction of electrolyte imbalances
6. Insulin therapy to correct hyperglycemia.

As part of the etiological investigation, a hepatobiliary ultrasound was requested. The results came back normal: the gallbladder was of normal size with a thin wall and no stones. The common bile duct was not dilated. The lipid profile did not reveal hypertriglyceridemia.

48 hours after admission, there was a notable clinical improvement in renal function, with an increase in creatinine clearance to 75 ml/min. An abdominopelvic CT scan with contrast injection was performed, revealing a

Balthazar grade E pancreatitis associated with an incidentally discovered dissection of the superior mesenteric artery.

Complementary thoracic angioscan showed extensive thoracoabdominal aortic dissection extending to the superior mesenteric artery, obstructing it.

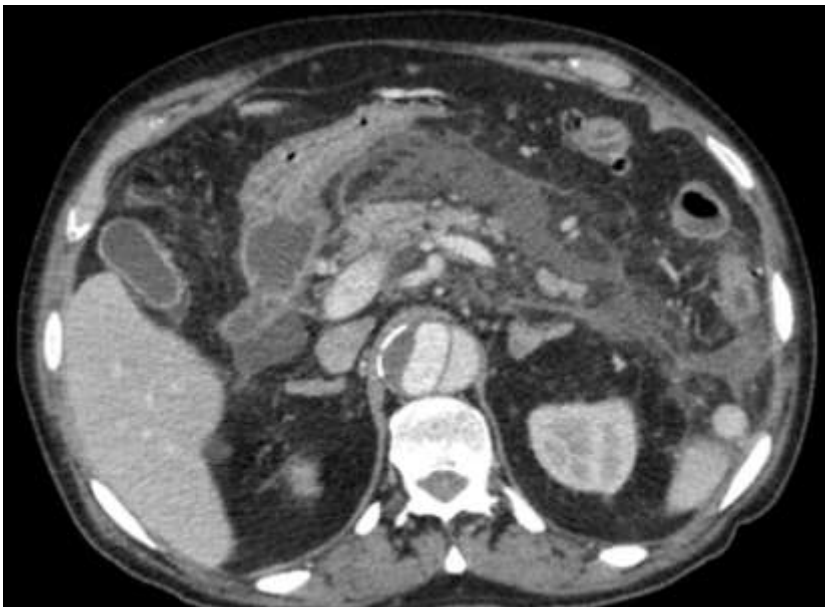

Figure 1: Horizontal CT section showing BALTHAZAR stage E acute pancreatitis.

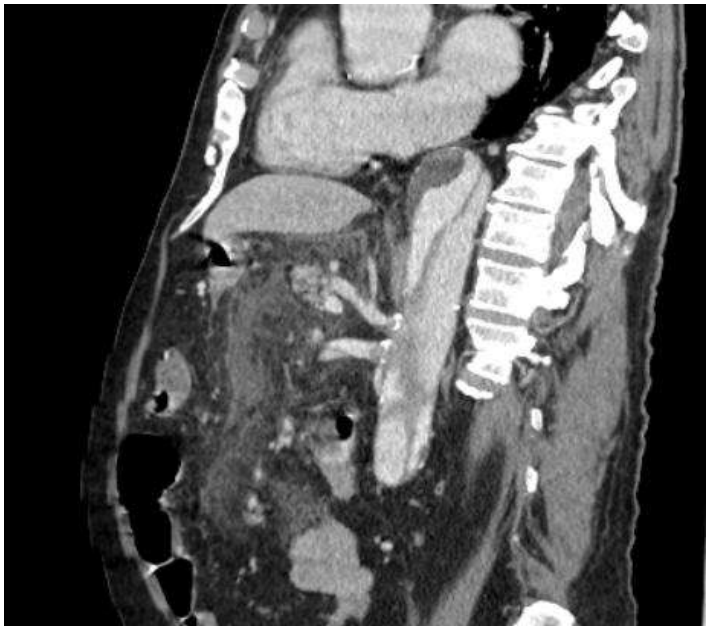

Figure 2: RMP scan showing aortic dissection reaching the superior mesenteric artery.

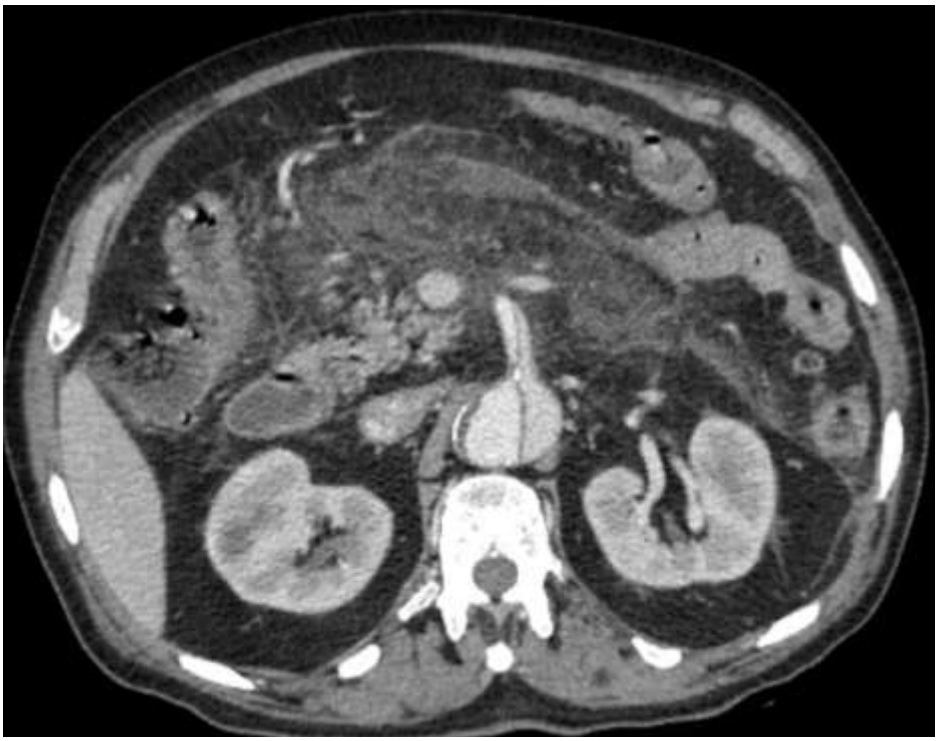

Figure 3: Horizontal CT section showing extension of aortic dissection to the superior mesenteric artery.

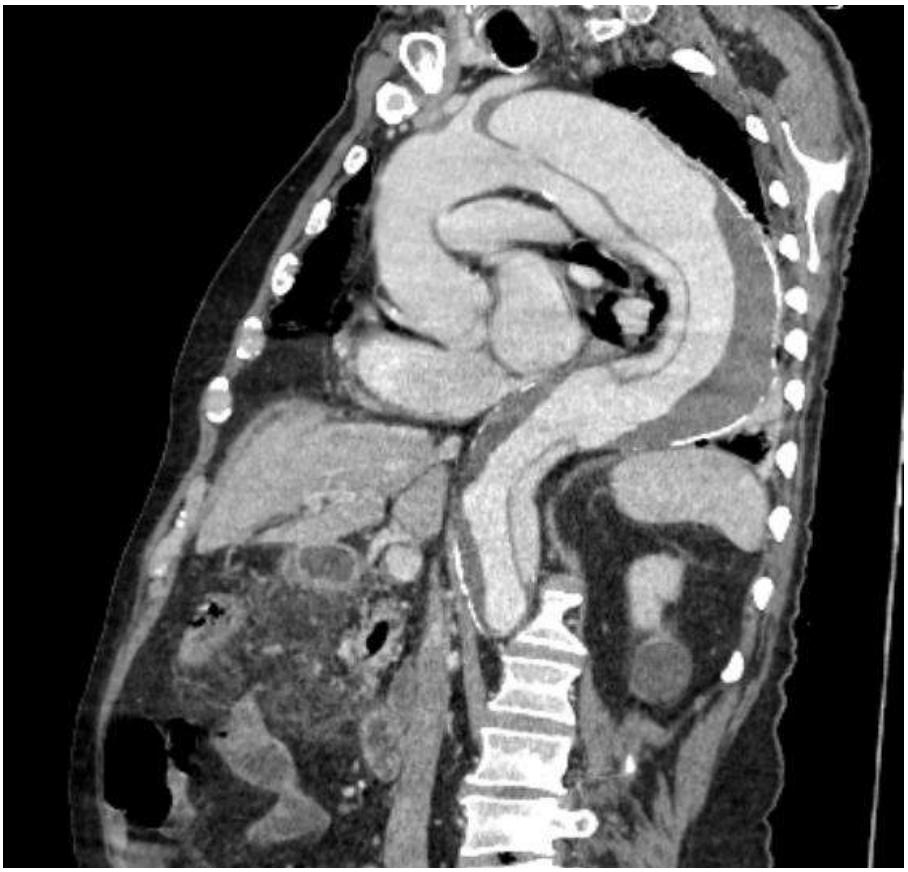

Figure 4:RMP scan showing STAMFORD stage B dissection.

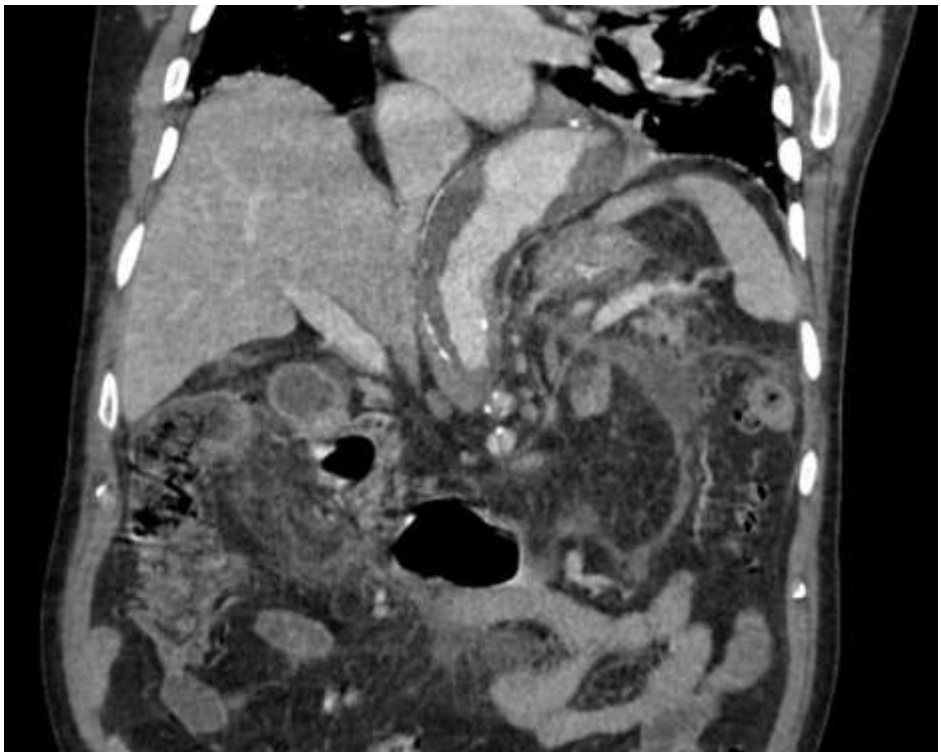

Figure 5: RMP scan showing reduced mesenteric artery lumen, Over 80% occlusion.

In view of these findings, an ischemic cause of severe acute pancreatitis was identified. In a joint meeting involving anesthesiologists, intensivists, vascular surgeons, and cardiologists, the decision was made to pursue medical treatment with blood pressure normalization and close monitoring. Regular CT scan surveillance was recommended for ongoing assessment.

On the 5th day of hospitalization, given the clinical improvement with the disappearance of pain and a gradual decrease in lipase levels, oral feeding was reintroduced very slowly, in addition to his usual antihypertensive treatment.

On day 5 of hospitalization, and in view of the clinical improvement with disappearance of pain and biological decrease in lipasemia, oral feeding was reintroduced very gradually in addition to her usual antihypertensive treatment.

On day 10, the patient was discharged with regular follow-up in cardio-vascular surgery consultations. His CRP level was at 54 mg/L.

On day17, one week after his discharge, the patient was readmitted through the emergency department due to fever and a decline in general condition. The patient was hemodynamically unstable with arterial hypotension at 80/40 mmHg, tachycardia at 112 beats/min, fever at 39°C, tachypnea at 25 cycles/min, and oxygen saturation at 97% on room air.

The abdomen was distended with hypertympanism and epigastric tenderness.

The rest of the clinical examination was unremarkable. Initial management consisted of:

1. A large-calibre venous line and bladder catheterization.
2. Careful filling with 0.9% isotonic saline.
3. Introduction of vasoactive drugs: noradrenaline with a target mean arterial pressure greater than or equal to 80mmHg, given the history of hypertension.

A laboratory assessment revealed a pronounced inflammatory syndrome with a CRP of 379 mg/L and leukocytosis of 22,500/mm³. Renal function was also impaired, with an increase in plasma creatinine to 14 mg/L and a creatinine clearance of 51.01 ml/min without electrolyte disturbances. The serum procalcitonin was elevated at 14 ng/L.

Given the suspicion of superinfection of the pancreatic necrosis flows, an abdomino-pelvic CT scan was performed, showing the appearance of air bubbles within the necrosis flows.

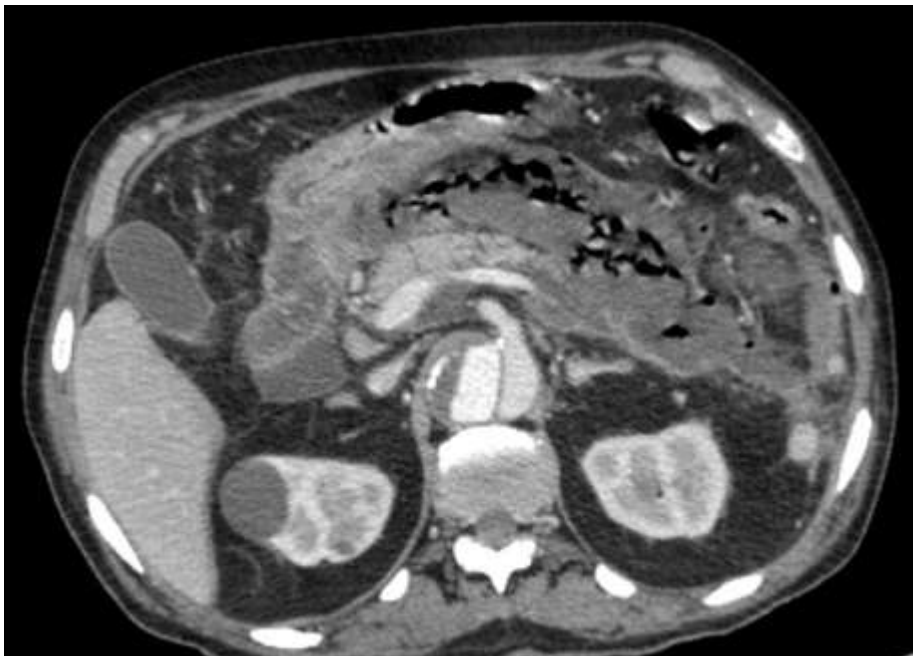

Figure 6:Horizontal scan section showing the appearance of air bubbles within pancreatic necrosis flows.

Given the state of shock, the biological inflammatory syndrome and the scannographic sign of superinfection of necrotic flows, the diagnosis of septic shock, whose starting point was superinfection of pancreatic necrotic flows, was retained. The patient was readmitted to the intensive care unit for management.

The patient was put on:

1. Vascular filling and vasoactive amines
2. Probabilistic bi-antibiotherapy: imipenem 500mg / 6 hours
3. Metronidazole 500mg/ 08h.
4. Gastroprotection: omeprazole 40mg/ 24h
5. Thromboprophylaxis

The evolution was marked by clinical and biological improvement with the withdrawal of vasoactive drugs. The patient was transferred to the gastrology department on day +10 for additional management.

Revisited one year later, the patient reported no episodes of abdominal pain since discharge. A thoracic-abdominal-pelvic angioscan was ordered, which showed stabilization of the vascular lesions with a scannographically normal pancreas with no visible cystic images.

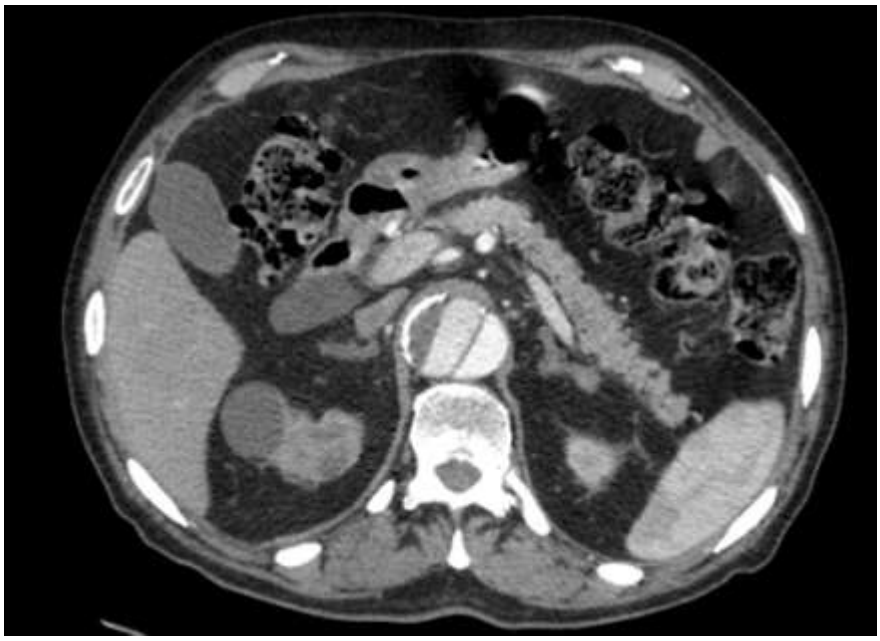

Figure 7: Horizontal section of the one-year scan, showing pancreatic parenchyma cleansing without the appearance of false cysts.

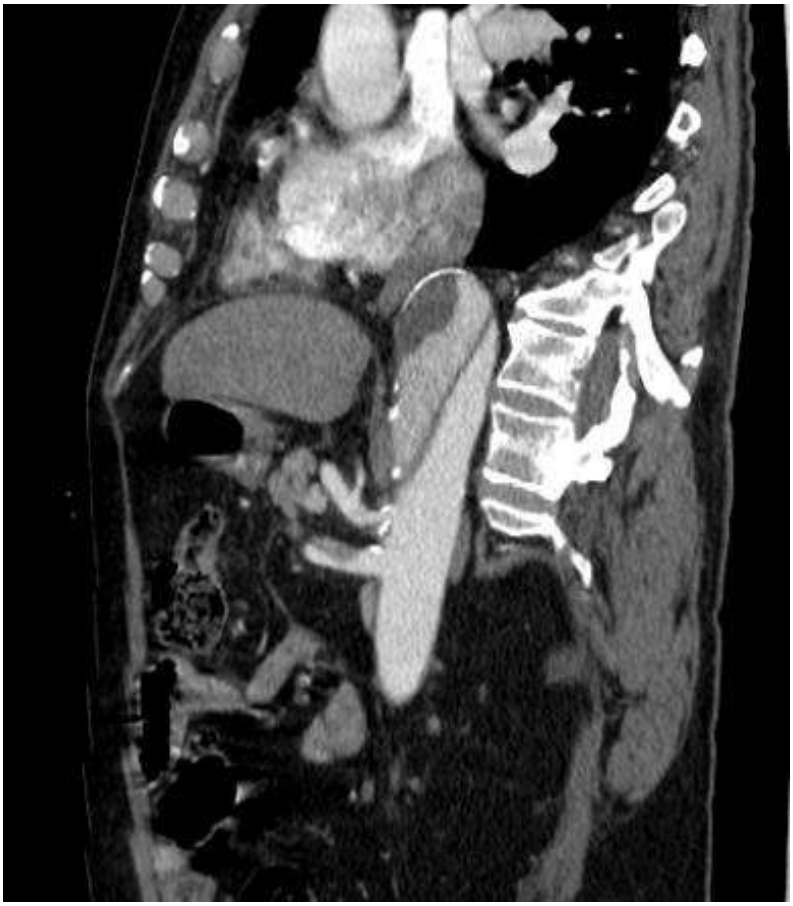

Figure 8:RMP scan, from 1-year follow-up, showing stabilization of vascular lesions.

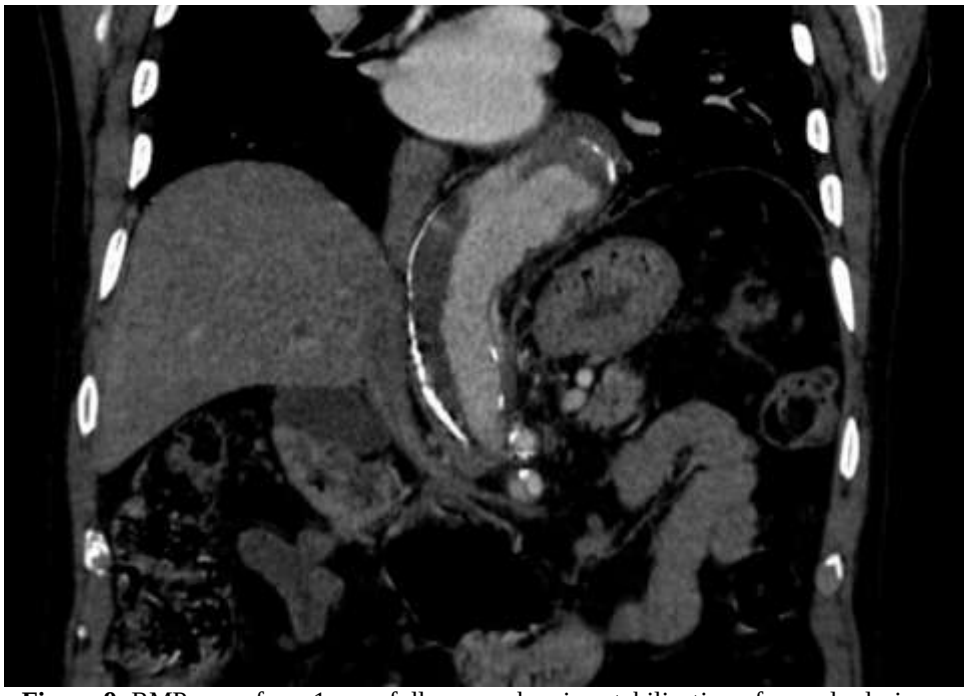

Figure 9: RMP scan, from 1-year follow-up, showing stabilization of vascular lesions.

Discussion:

This case of acute ischemic pancreatitis associated with aortic dissection diagnosed at the Military Hospital Oued Eddahab in Agadir appears to be the first case in Morocco. Indeed, no national cases have been found in the literature.

On an international scale, 15 cases of acute ischemic pancreatitis associated with aortic dissection have been reported. There are 9 isolated sporadic cases and a series of 6 cases by Wang et al [3].

Now, we will discuss our case in light of this data.

1. Epidemiological aspect:

The average age was 53 years, with a range from 42 to 68 years, while the age of our patient was 87 years.
As for medical history, uncontrolled hypertension was the only common pathological history among all patients, including ours.

Indeed, uncontrolled hypertension is a risk factor for aortic dissection (AD). The latter can be complicated by acute pancreatitis.

One-third of the reported cases were women, while two-thirds were men, including our case, indicating a clear male predominance, consistent with the literature. [4]

2. Clinical presentation:

The clinical presentation was generally marked by intense pain, either epigastric in 11 cases (73%), dorsal in one case (7%), or thoracic in one case (7%), with a combination of epigastric and thoracic pain in two cases (13%). The onset was sudden, accompanied by nausea and/or vomiting.

Our case had also consulted for acute, transfixing epigastric pain associated with food vomiting.

3. Initial diagnosis:

In the series studied, seven patients were initially diagnosed with acute pancreatitis. Diagnosis was based on clinical examination and lipasemia or amylasemia assays in the earliest series.

In eight cases, the diagnosis was initially established as aortic dissection and later evolved into acute pancreatitis due to the persistence or reappearance of pain despite initial management. This diagnostic chronology for the same symptom can be attributed to the experience and instinct of the practitioner, as well as the patient's medical history.

In our patient's case, the initial diagnosis was acute pancreatitis, and etiological investigation revealed that the underlying cause was ischemia due to aortic dissection.

It is important to emphasize that the precise diagnosis and recognition of the relationship between aortic dissection and acute pancreatitis can be complex. A thorough clinical evaluation and appropriate etiological investigation are essential to identify the underlying cause and guide proper management.

4. Type of dissection:

In 9 out of the 15 cases found in the literature, a Stanford type B aortic dissection (Debakey type III) was diagnosed, representing 60% of the documented cases. The remaining 6 cases (40%) had a Stanford type A aortic dissection (Debakey type I).

Our patient had a Stanford type B aortic dissection, aligning with the predominance of type B dissections.

5. Associated complications:

Aortic dissection can extend to the peripheral feeding arteries of organs, leading to hypoperfusion and complications such as acute pancreatitis. Among the studied series, five patients presented complications of aortic dissection associated with pancreatitis: three patients developed renal failure, and two other patients had digestive complications such as ischemic gastropathy and visceral dysfunction. In one of them, there was also lower limb peripheral arterial disease (PAD).

These complications can make management challenging and have a negative impact on prognosis. In the case of our patient, the acute renal failure he presented at admission was corrected through appropriate volume restoration.

6. Therapeutic intervention:

Among the documented cases, seven out of 15 patients underwent surgical treatment, either through endovascular repair or open surgery.

Two patients refused the recommended surgical treatment. Unfortunately, the outcome was fatal for them.

Regarding medical treatment modalities, six patients underwent a medical approach, including volume restoration, somatostatin injection, beta-blocker administration, and blood pressure control. Our patient benefited from this approach.

It is worth noting that currently, there is no consensus on the management of aortic dissection complicated by acute pancreatitis. Therapeutic approaches may vary depending on the specificity of each case and multidisciplinary consultation among healthcare professionals.

7. Following up and outcomes:

Whether the therapeutic approach was medical or surgical, following a local multidisciplinary professional consensus, the prognosis was favorable in 13 documented cases and in the case of our study. The outcome was unfavorable for the two patients who refused the surgical approach that was recommended to them.

The calculated average mortality rate among the documented cases of patients with acute pancreatitis secondary to aortic dissection is 13%, which is paradoxical compared to the mortality rate associated with severe acute pancreatitis of other origins, which is 30%.[5]

In the case of our patient, the current follow-up extends beyond one year. Monitoring is conducted regularly, and no complications have been observed. The CT scan appearance of the dissection has remained stable, and the pancreatic lesions have shown gradual improvement.

These results suggest that, despite the complexity of the situation and the absence of codified recommendations, the therapeutic approach based on the collaboration of the medical-surgical team provides, at least in the 15 cases documented in the literature, a favorable outcome for patients with acute ischemic pancreatitis secondary to aortic dissection.

It is important to emphasize that additional research is needed to better understand the risk factors, pathological mechanisms, and optimal therapeutic approaches for this rare condition. This will further enhance the prognosis and management of affected patients.

Conclusion:

Severe acute pancreatitis is frequently observed in intensive care units. The main causes are gallstone disease and alcoholism, while vascular etiology, particularly of ischemic origin, is rare. Due to the serious consequences it entails, it is challenging to determine the optimal management approach: should one opt for immediate surgical intervention or choose a medical approach with careful monitoring?

The literature review has identified only 15 similar cases that have been documented since 1991 of acute pancreatitis of ischemic origin associated with aortic dissection. This highlights the rarity of this situation and the absence of standardized recommendations for managing this exceptional scenario, which poses a dilemma for the clinician regarding the decision between surgical or medical treatment. In 54% of the cases described in the literature, non-surgical management was favored, with a favorable prognosis in 75% of these cases.

This case highlights the exceptional importance of medical imaging in the context of severe acute pancreatitis, both for diagnosis, prognosis, and etiological investigation. It also underscores the importance of an individualized approach based on multidisciplinary collaboration, given the lack of recommendations and the rarity of cases in the medical literature.

Table 1: Comparison of literature data with our case.

Author	Year	Age/Sexe	Clinic	Initial Diagnosis	DA	Therapeutic intervention
Pombo et al [6]	1991	42/M	Epigastric pain	PA	B	Surgical reconstruction
Goff et al [7]	1992	54/M	Epigastric pain	PA	B	Conservator
Baydina et al [8]	2008	52/M	Epigastric pain	PA	A	Conservator and surgical proposal
Umeda et al [9]	2011	49/M	Thoracic + Epigastric pain	DA	A	Conservator
Hamamoto [10]	2012	47/M	Back pain	DA	B	Conservator
Hideaki et al [11]	2012	60/M	Asymptomatic	PA	B	Conservator
Pham et Nable [12]	2013	69/F	Epigastric pain	PA	A	Conservator (Refusal of surgery)
Zheng et al [13]	2014	47/M	Epigastric pain	DA	B	Surgical reconstruction
JinJie [14]	2018	52/M	Epigastric pain	DA	B	Surgical reconstruction
Wang et al [3]	2018	63/F 48/M 48/M 46/F 68/F 54/M	Epigastric pain Epigastric pain Epigastric pain Epigastric pain Epigastric pain Epigastric pain	DA PA DA PA DA DA	A B A A B B	Surgical reconstructionSurgicalreconstructionSurgicalreconstructionSurgi (Refusal of surgery)
Our case	2022	87/M	Epigastric pain	PA	B	Conservator

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