



Atrial Fibrillation Complicated by Mesenteric and Renal Infarction: Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Background: Very rarely, atrial fibrillation can be complicated by acute renal or intestinal infarction. The diagnosis of this condition can therefore be challenging, and treatment guidelines have not yet been established. In this article, we present a case report of a patient with acute mesenteric embolic ischemia and renal infarction as a complication of mitral stenosis-related atrial fibrillation.

Case Report: We report the case of a patient with mesenteric and renal infarction of thromboembolic origin due to rheumatic valvulopathy in atrial fibrillation who presented to the emergency department with acute abdominal pain. The angioscanner showed bilateral renal ischaemia, more extensive on the right, and occlusion of the superior mesenteric artery with signs of visceral distress. An effective dose of anticoagulation was started and an emergency laparotomy was performed, revealing a completely necrotic and distended bowel. The clinical outcome was unfavourable and the patient did not survive.

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Conclusion: This case study highlights the importance of diagnosing renal and mesenteric infarction in the presence of acute abdominal pain in patients with embolic heart disease or other risk factors for thrombosis.

Keywords: Atrial fibrillation; mesenteric; renal infarction.

ABBREVIATIONS

MS : Mitral Stenosis
AMI : Acute Mesenteric Ischemia

1. INTRODUCTION

Renal ischemia and acute mesenteric ischemia (AMI) are two rare life-threatening emergencies responsible for abdominal and back pain, which are often under-diagnosed. The prevalence of renal infarction is estimated at between 4 and 7 cases per 100,000 inhabitants, and its incidence is estimated at less than 2% [1]. Mesenteric ischemia accounts for 1% of hospitalizations for acute abdomen. Atrial fibrillation is responsible for 25-65% of cases of acute mesenteric ischemia and renal infarction [2-6].

This case study underlines the importance of early diagnosis, to prevent ischemia progressing to infarction, and to define the origin and severity of the condition, on which treatment will depend.

2. CASE PRESENTATION

This was a 72-year-old hypertensive patient on calcium channel blocker 20mg and Indapamide 1.5mg. Since 2004, she had been known to have

mitral stenosis (MS) dilated by percutaneous mitral commissurotomy in atrial fibrillation and on anti-vitamin K anticoagulant therapy. Pathological history includes phlebitis of the lower limb in 2004, polypectomy in 2013, wheezing bronchitis on inhaled corticosteroids and sciatica for 2 months on symptomatic treatment. Since the day before admission, she had reported severe acute abdominal pain of abrupt onset, associated with nausea and vomiting, in a context of afebrile. On admission, the patient reported no cardiovascular symptoms. Clinical examination revealed a conscious GSC 15/15 patient with a hypertensive peak of 192/83mmHg, tachycardic at 112 bpm, 89% saturated on room air. Cardiovascular examination reveals rapid, irregular heartbeats without murmurs or signs of heart failure. Abdominal examination revealed diffuse tenderness. The electrical tracing performed on admission showed atrial fibrillation with a ventricular rate of 94bpm and secondary repolarization disorders such as inferolateral ST-segment shift. Thoracoabdominal angio-CT showed bilateral renal ischemia more extensive on the right without arterial stenosis, mesenteric artery occlusion superior to 3cm from its origin, moderate peritoneal effusion and intestinal distension. Biological findings included elevated

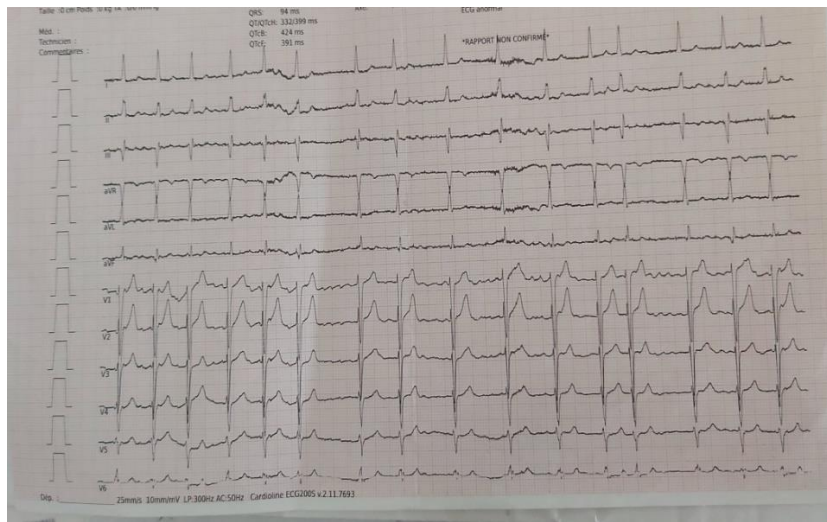


Fig. 1. The 12-lead electrocardiography findings. On admission, the patient had atrial fibrillation with rapid ventricular rate 110b/m

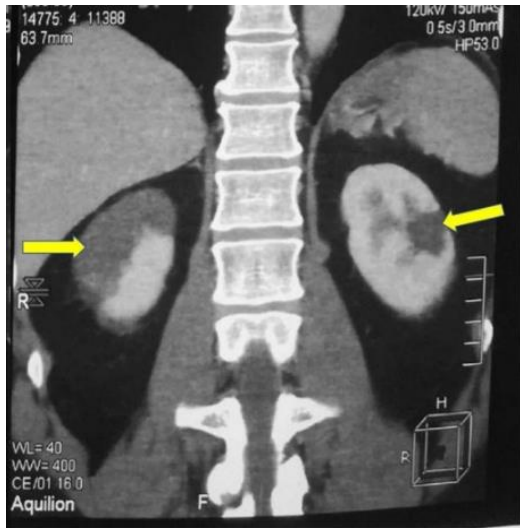


Fig. 2. CT image showing bilateral renal parenchyma infarction (ischemia of 2/3 of the right renal parenchyma)



Fig. 3. Laparotomy performed as an emergency procedure, revealing completely necrotic and distended intestinal loops

CRP 282.1mg/l, procalcitonin 37ng/ml, positive troponin 0.027pg/ml in stationary kinetics, elevated d-dimer 1200pg/ml, creatinine 10.1mg/l, urea 0.56g/l and GFR 57.3ml/min/1.73m², positive ECBU Escherichia coli. AST/ALT 108/68, LDH 942IU/L, CBC without abnormalities. Trans-thoracic echocardiography (TTE) revealed a tight mitral stenosis (MS) with a surface area of 1.1cm², a mean gradient of 8.8mmHg with minimal leakage, a loose aortic stenosis, grade II aortic insufficiency and dilated atria. Left ventricular systolic function was normal, with LVEF at 55-60%. Curative-dose anticoagulation was started, along with antibiotic therapy, proton pump inhibitors and Nicardipine in a self-pulsating syringe. The patient presented with profuse sweating, a hypertensive peak of

200mmHg systolic, 80% desaturation, severe abdominal distension, impaired renal function (creatinine 23mg/l), hypokalemia 2.8mmol/l and anuria. An emergency laparotomy was performed, revealing completely necrotic and distended bowel. The patient died a few hours later.

3. DISCUSSION

The clinical expression of acute renal ischemia is not very specific, which makes diagnosis difficult for the clinician, not only because of the non-specific presentation of this pathology, but also because of the existence of numerous differential diagnoses linked either to the clinical presentation (renal colic, pyelonephritis), or to the terrain (mesenteric ischemia) [7]. Microscopic hematuria is present in 60-90% of cases. Renal function is impaired in almost half of patients, to varying degrees. The mean time to diagnosis of renal infarction is generally long, with a median of around 15 hours, and may take several days [8].

The clinical picture of mesenteric ischemia depends on the etiology; classically, arterial origin is associated with intense abdominal pain contrasting with an initially poor clinical examination (abdominal distension, transit disorders, hyperperistalsis, more or less bloody diarrhea). At the stage of constituted infarction, general signs appear, marked by collapse which may be associated with fever, abdominal contracture and adynamic ileus. [9] The risk factors to look for are atheromatous terrain and emboligenic heart disease. Venous origin is responsible for a less noisy sometimes over several weeks [10]. Our patient presented late to the emergency department at H20 with abdominal pain complicated by irreversible intestinal necrosis, impaired renal function and death.

Abdominal angioscanner is the examination of choice for the diagnosis of acute renal ischemia, with a sensitivity of over 95%. It can be used to rule out differential diagnoses (tumor, abscess), and typically reveals a hypodense area with a sharp, homogeneous, triangular (cortical-based) border that is unenhanced after injection of contrast medium [11] Renal Doppler ultrasonography has a low sensitivity for the diagnosis of renal ischemia (10%) and should not be considered as a first-line procedure [12]. Our patient underwent an emergency abdominal CT scan showing bilateral renal involvement, more

marked on the right, with the same scannographic features found in the literature [13] Magnetic resonance imaging (MRI) of the kidney is a test that can reveal areas of infarcted renal parenchyma at an early stage, and should be considered in centers where it is easily accessible in an emergency [14].

In the case of acute mesenteric ischemia, CT is the diagnostic tool of choice, enabling us to see the thrombosis and its extent, as well as the condition of the intestine and other intra-abdominal organs [15]. Arteriography can show vasospasm, lack of visualization of the venous system and absence of flow in the necrotic part of the bowel, and can be used to inject a thrombolytic agent; it is not routinely performed [16]. Ultrasound is not the reference examination; its quality is mediocre due to air interpositions accompanying dilatation of the small intestines. [17].

Any suspicion of AMI on ultrasound should be followed by a CT scan to assess the severity of ischemia [18]. In our patient, abdominal CT showed total occlusion of the superior mesenteric artery, with signs of visceral distress. Cardio-embolic origin is found in around 90% of cases of renal infarction and 60 to 75% of acute mesenteric ischemia [19], with a poor prognosis and a very high mortality rate of 60 to 90% [20]. The most frequent cause is embolism due to cardiac arrhythmia caused by atrial fibrillation, followed by left-sided valvular disease, myocardial infarction, left atrial myxoma and iatrogenic causes due to arterial manoeuvres. It occurs in healthy arteries and leads to massive, sudden ischemia in the absence of a bypass network [21]. Thrombotic causes present in 20-30% of cases include atherosclerosis, aortic and mesenteric dissection, hypercoagulability and hyperviscosity syndromes [22].

All causes of vasoconstriction, particularly drug-induced, can lead to the same lesions. Thrombotic ischemia is generally not very severe, and has a much better prognosis than arterial ischemia, since it is usually reversible with anticoagulant treatment. The thromboembolic origin of renal infarction and superior mesenteric thrombosis was retained in our patient because of the very high thromboembolic risk (history of atrial fibrillation, mitral stenosis and phlebitis of the lower limb), which was confirmed on imaging. Patients with emboligenic heart disease should therefore be anticoagulated to avoid these complications [23].

Management of patients admitted with acute mesenteric ischemia is aimed at preventing progression to irreversible intestinal necrosis, treating the cause and symptomatically managing organ failure [24]. It includes a medical protocol combining digestive rest, curative anticoagulation, anti-platelet aggregation, proton pump inhibitor and probabilistic antibiotic therapy, arterial revascularization to save viable bowel and resection of necrotic digestive segments. The revascularization strategy depends on the mechanism of arterial occlusion, the morphological appearance of the lesions and the indication for a laparotomy for digestive exploration [25]. Endovascular revascularization is preferred whenever possible. Open surgical revascularization is indicated when endovascular revascularization fails or is impossible, and when a laparotomy is required for digestive exploration. Effective-dose heparin anticoagulation is the standard treatment for renal infarction, and should be initiated as a matter of urgency. It may or may not be supplemented by local or systemic thrombolysis, which is associated with bleeding complications [25].

Our patient underwent curative anticoagulation with heparin, and emergency exploratory laparotomy revealed completely necrotic and distended bowel. Elevation of LDH, a marker of tissue necrosis, is frequently described in the literature, but is not specific [26]. Our patient presented with an elevated LDH level.

In terms of long-distance prognosis, a retrospective study of 47 cases of renal infarction found varying degrees of renal function deterioration in 90% of patients within three years of renal infarction. Our patient presented with acute impairment of renal function with anuria; her creatinine increased from 10mg/l (GFR 57.3ml/min) to 23mg/l (GFR 22ml/min). The mortality rate for patients hospitalized for acute mesenteric ischemia and renal infarction remains very high, at around 60% [27]. Unfortunately, the clinical course was unfavorable in our patient, who died.

4. CONCLUSION

The clinical presentation of renal infarction is non-specific, making diagnosis difficult. Abdominal pain is the main symptom of mesenteric ischemia. Abdominal angioscan is the key examination for diagnosis and severity of necrosis. Therapeutic management, including resuscitation and possible digestive resections,

should be carried out as early as possible to limit the extent of necrosis.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of manuscripts.

CONSENT

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standards or university standards written ethical approval has been collected and preserved by the author(s).

DECLARATIONS

I, the Corresponding Author, declare that this manuscript is original, has not been published before and is not currently being considered for publication elsewhere. I can confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. I further confirm that the order of authors listed in the manuscript has been approved by all of us. I understand that the Corresponding Author is the sole contact for the Editorial process and is responsible for communicating with the other authors about progress, submissions of revisions and final approval of proofs. We have no conflict of interest to declare.

Signed by the Corresponding Author on behalf of the all other authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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