# Atrial fibrillation complicated by mesenteric and renal infarction : Case report


## Abstract

**Background:**Renal and mesenteric infarction are rare pathologies whose presentation is misleading for the clinician. An injected abdominal CT scan is essential for early diagnosis. Prompt initial management is crucial to subsequent prognosis.

**Case report:**We report here the case of a patient presenting with mesenteric and renal infarction of thromboembolic origin on rheumatic valvulopathy in atrial fibrillation who presented to the emergency department with acute abdominal pain. The angioscanner revealed bilateral renal ischemia, more extensive on the right, and occlusion of the superior mesenteric artery, with signs of visceral distress. Effective-dose anticoagulation was started, and emergency laparotomy was performed, revealing completely necrotic and distended intestinal tracts. The clinical outcome was unfavorable, the patient does not survive.

**Conclusion:** This case study highlights the importance of evoking the diagnosis of renal and mesenteric infarction in the presence of acute abdominal pain in patients with embolism-inducing heart disease or other risk factors for thrombosis.



**Key words:**Atrial fibrillation, mesenteric, renal infarction




## Background

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% [1-4]. 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 [5,6].

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.

## Case presentation:

This is a 72-year-old hypertensive patient on calcium channel blocker 20mg and Indapamide 1.5mg. Since 2004, she has been known to have mitral stenosis (MR) 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 apyrexia. 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 bpmin, 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

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 (MR) with a surface area of 1.1cm², a mean gradient of 8.8mmHg with minimal leakage, a loose aortic stenosis (Rao), grade II aortic insufficiency (IAo) 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.

## Discussion

Acute renal ischemia is a rare and under-diagnosed condition. Its clinical expression is not very specific, and the numerous differential diagnoses make it difficult for the clinician to diagnose. Thrombosis of the superior mesenteric vein is rare but serious, accounting for 10-15% of all cases of mesenteric ischemia [7-10]. According to the literature, the diagnosis of renal infarction is made difficult for the clinician not only by the non-specific presentation of this pathology, but also by the existence of numerous differential diagnoses linked either to the clinical presentation (renal colic, pyelonephritis), or to the terrain (mesenteric ischemia). Microscopic hematuria is present in 60 to 90% of cases. Renal function impairment is observed in almost half of patients, to varying degrees. The average time taken to diagnose renal infarction is generally long, with a median of around 15 hours, and may take several

days.The clinical picture of mesenteric ischemia depends on the etiology; classically, arterial origin is associated with intense abdominal pain of abrupt onset and continuous evolution, 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. The risk factors to look for are atheromatous terrain and emboligenic heart disease. Venous origin is responsible for a less noisy and more gradual onset, sometimes over several weeks. In low-flow AMI, the symptomatology is most often multi-visceral failure with hemodynamic instability [11].Our patient presented late to the emergency department at H20, resulting in irreversible intestinal necrosis, impaired renal function and death.

Abdominal angioscanner is the examination of choice for making the diagnosis of acute renal ischemia, eliminating differential diagnoses (tumor, abscess) and searching for other synchronous embolic disorders. Ideally, this should comprise four distinct phases (injection-free, arterial, venous and late) to clarify the nature of renal damage. In renal ischemia, it typically reveals a hypodense area with a sharp, homogeneous, triangular (cortical-based) border, unenhanced after contrast injection [12-14].The patient presented with bilateral involvement, with the same scannographic features found in the literature. Its sensitivity in the diagnosis of renal infarction is greater than 95% [1]. Renal angiography, although the gold standard, has the disadvantage of being invasive and difficult to access in an emergency. Renal Doppler ultrasonography has a low sensitivity for the diagnosis of renal ischemia (10%) and should not be considered as a first-line procedure.Renal magnetic resonance imaging (MRI) is a test that can provide early evidence of an area of infarcted renal parenchyma without the disadvantages of nephrotoxic contrast injection and radiation, as with angioscan [1-7]. It could be an examination to consider in centers where it is widely and easily

accessible in emergencies, when the diagnosis of renal ischemia is evoked. Renal MRI can also be used to monitor sequelae of ischemic lesions.As far as IMA is concerned, CT is the diagnostic tool of choice, allowing us to see the thrombosis and its extent, as well as the state of the intestine and other intra-abdominal organs. Arteriography can show vasospasm, lack of visualization of the venous system and absence of flow in the necrotic part of the bowel. This arteriogram could be used to inject a thrombolytic agent [15-18]. Ultrasound is not the reference examination for the diagnosis of IMA, as it is often of poor quality due to air interpositions accompanying dilatation of the small intestines, but it may be the first examination carried out in the case of an acute abdomen.Investigation of the wall of the digestive tract with a high-frequency probe should be systematic when exploring acute abdomens, as should Doppler ultrasound of the wall with slow flow parameters (below 10 cm/s) and of the mesenteric vessels. The superior mesenteric artery is accessible to Doppler ultrasound over its first eight centimetres in over 80% of cases [18]. Any suspicion of IMA on ultrasound should be followed by a CT scan to assess the severity of ischemia [19]. In our patient, the diagnosis was limited to TAP CT angiography, which revealed 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 in 60 to 75% of acute mesenteric ischemia (AMI), with a poor prognosis and a very high mortality rate of 60 to 90% [20,21]. The most frequent cause of AMI 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 maneuvers; occurs in a healthy artery, resulting in massive, sudden ischemia which, due to the absence of a bypass network, progresses to transparietal infarction. The embolus migrates into the superior mesenteric artery (SMA), and the ischemic territory is more extensive the more proximal the obstacle [3,5,6].Conversely, thrombosis, the cause of 20 to 30% of IMAs, occurs in an atheromatous setting, the patient is older and located

preferentially at the ostium of the AMS, and is most often progressive, completing an atheromatous stenosis with collaterality already in place. Thrombotic causes include atherosclerosis, aortic and mesenteric dissection, hypercoagulability and hyperviscosity syndromes [22]. Low-flow ischemia of non-occlusive origin is the cause of 20-30% of IMA cases.The drop in systemic pressure in the event of hemorrhagic, cardiogenic or septic shock leads to reflex vasoconstriction in the digestive tract to preserve perfusion of the cerebral, cardiac and renal tracts; the result is a reduction in flow to the digestive tract, and ischemic lesions which may predominate in areas of weakness (ileocaecal angle, left colic angle, recto-sigmoid junction) or extend over long intestinal segments, creating a "shock bowel" picture [23].All causes of vasoconstriction, particularly drug-induced, can lead to the same lesions. Thrombosis of the mesenteric vein is responsible for 5 to 15% of IMAs [24]. Ischemia is generally not very severe, and has a much better prognosis than ischemia of arterial origin, as it is usually reversible with anticoagulant treatment. However, if it is unrecognized and untreated, the increase in venous pressure due to engorgement can lead to secondary arterial ischemia, which can progress to infarction, necrosis and death [25].In 80% of cases, there are constitutional (antithrombin III deficiency, protein C, protein S, Leyden factor mutation) or acquired (myeloproliferative syndrome, anti-phospholipid antibody syndrome) coagulation abnormalities which, combined with local factors (local inflammation, digestive cancer, portal hypertension, pregnancy, abdominal surgery), favor the occurrence of venous thrombosis. The thromboembolic origin of the 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.Management of patients admitted for IMA must meet several objectives: avoid or limit progression to irreversible intestinal necrosis, treat the cause of mesenteric ischemia, and manage organ failure symptomatically. Management must be multimodal and

multidisciplinary, and includes a medical protocol combining digestive rest, curative anticoagulation, anti-platelet aggregation, proton pump inhibitor and probabilistic antibiotic therapy, arterial revascularization to save viable intestine, and resection of necrotic digestive segments.The extent of resection of necrotic digestive segments must be discussed collegially, taking into account the patient's condition and the risk of dependence on parenteral nutrition. The revascularization strategy depends on the mechanism of arterial occlusion, the morphological appearance of the lesions and the indication for a laparotomy to explore the digestive tract. 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 [26-31]. 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, but no study has demonstrated the superiority of either treatment.Systemic thrombolysis is associated with bleeding complications [32]. Elevation of LDH, a marker of tissue necrosis, is frequently described in the literature, but is not specific, but could be an additional argument, particularly in a patient at risk [33]. 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 [34]. 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). Mortality in patients hospitalized for IMA remains very high. Few data are available for intensive care units.Only one retrospective study found an ICU mortality rate of around 60%. In the long term, patients require management of cardiovascular risk factors, as well as a nutritional and surgical rehabilitation strategy to reduce dependence on long-term parenteral nutrition (up to 30% of cases, according to studies) [35].

## 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.

**List of abbreviations**: Not applicable

## Images

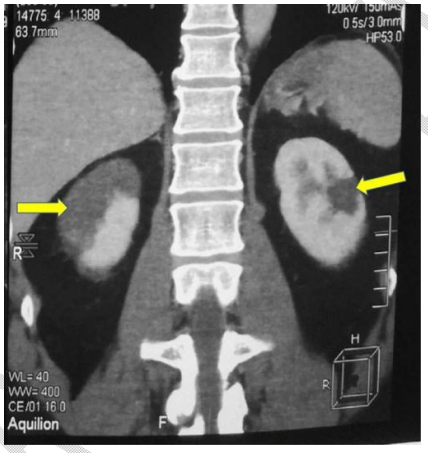


Fig1: CT image showing bilateral renal parenchyma infarction (ischemia of 2/3 of the right renal parenchyma)

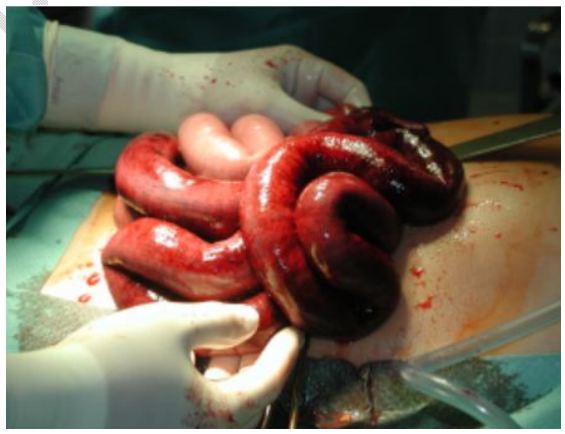

Fig2: Laparotomy performed as an emergency procedure, revealing completely necrotic and distended intestinal loops.

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