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Case report

Renal artery thrombosis. A case report and literature review

Erika Poggiali[1], *Maria Giovanna Ferrari*[2], *Camilla Botti*[3], *Emanuele Michieletti*[3], *Andrea Vercelli*[1]

[1]Emergency Department, Guglielmo da Saliceto Hospital, Piacenza, Italy; [2]Internal Medicine, Fondazione IRCCS Policlinico San Matteo, Pavia, Italy; [3]Radiology Unit, Department of Radiological Functions, Guglielmo da Saliceto Hospital, Piacenza, Italy.

Abstract. Renal artery thrombosis is a severe and often misdiagnosed entity, that represents a true medical emergency in which renal infarction can occur. The diagnosis is often a challenge for the emergency physicians since it can mimic other more common diseases, including renal colic. We report the case of an 82-year-old man who presented to our emergency department for abdominal pain, nausea, and vomiting resulting from right renal artery thrombosis and infarction caused by misdiagnosed atrial fibrillation. Starting from our experience, we recommend to always keep renal thromboembolism in the differential diagnosis in a patient with sudden onset of flank/abdominal pain, high levels of lactate dehydrogenase and/or haematuria since early diagnosis and proper treatment are the keys to rapid recovery. (www.actabiomedica.it)

Key words: renal artery thrombosis, atrial fibrillation, renal infarction, flank pain, abdominal pain

Introduction

Abdominal pain is a common cause of access to emergency department (1) that still represents a challenge for the emergency physicians. The most common causes of abdominal pain are benign, but in a smaller percentage of patients it might represent a time-dependent emergency (2), that must be identified as soon as possible to avoid a poor prognosis (3). In the less common causes of acute abdominal pain the following conditions should not be forgotten, i.e. visceral perforation, vascular complications, such as abdominal aortic aneurysm rupture, spontaneous dissection of the celiac trunk (4), mesenteric ischemia or thrombosis of others abdominal districts, including renal artery thrombosis, and pelvic disorders. Patient's medical history, physical examination, proper laboratory tests, and appropriate radiological imaging are mandatory to correctly investigate abdominal pain, discriminating between urgent and non-urgent causes and reducing the risk of missing diagnosis.

Case presentation

An 82-year-old male patient with a history of blood hypertension treated with ACE-inhibitor, presented to our emergency department with nausea, vomiting and persistent right flank pain for 3 days. He denied fever and trauma. Physical examination revealed a temperature of 36 °C, blood pressure 140/80 mmHg, pulse 110/min irregular for atrial fibrillation, respiratory rate 16/min and a pulse oximetry 98%. He complained tenderness on abdominal palpation over the right flank with normal bowel sounds, but no rebound tenderness or guarding. There was no abnormality in vesicular murmurs. He denied heart palpitations, shortness of breath or chest pain, even if electrocardiogram showed atrial fibrillation with a heart rate of 120 beats a minute. The patient had no previous history of hypercoagulability.

Point-of-care ultrasound (PoCUS) excluded bladder globe, hydronephrosis, free abdominal fluid, and bowel obstruction. Laboratory data showed increased

white blood count (23.650/mm^3, normal range 4.000-10.000) with neutrophilia (19.730/mm^3) and elevated C-reactive protein (15 mg/dL, normal range < 0.5), high values of lactate dehydrogenase (LDH) (2.027 mg/dL, normal range < 248) and transaminases (AST 137 U/L and ALT 164 U/L, normal range 10-37), and acute renal failure with serum creatinine of 1.8 mg/dL (normal range 0.60-1.20) and blood urea nitrogen of 79 mg/dL (normal range 10-50) in absence of dysionias and normal blood pH. Urine dipstick resulted positive for blood (4+) and protein (1+). The patient was hydrated with intravenous sodium chloride 0.9% 500 mL and glucose 5% 500 mL, and treated with metoprolol 5 mg rapid intravenous push, then repeated for a total of 3 doses (15 mg total dose) to reduce heart rate.

Because of persisting abdominal pain, high levels of LDH and a new diagnosis of rapid atrial fibrillation, a computed tomography angiography (CTA) of the abdomen was performed and revealed extensive renal thrombosis of the right renal artery causing renal infarction with sparing only the lower third of the right kidney, and acute thrombosis of a distal branch of the superior mesenteric artery (Figure 1). There was no evidence of renal calculi or abdominal perforation.

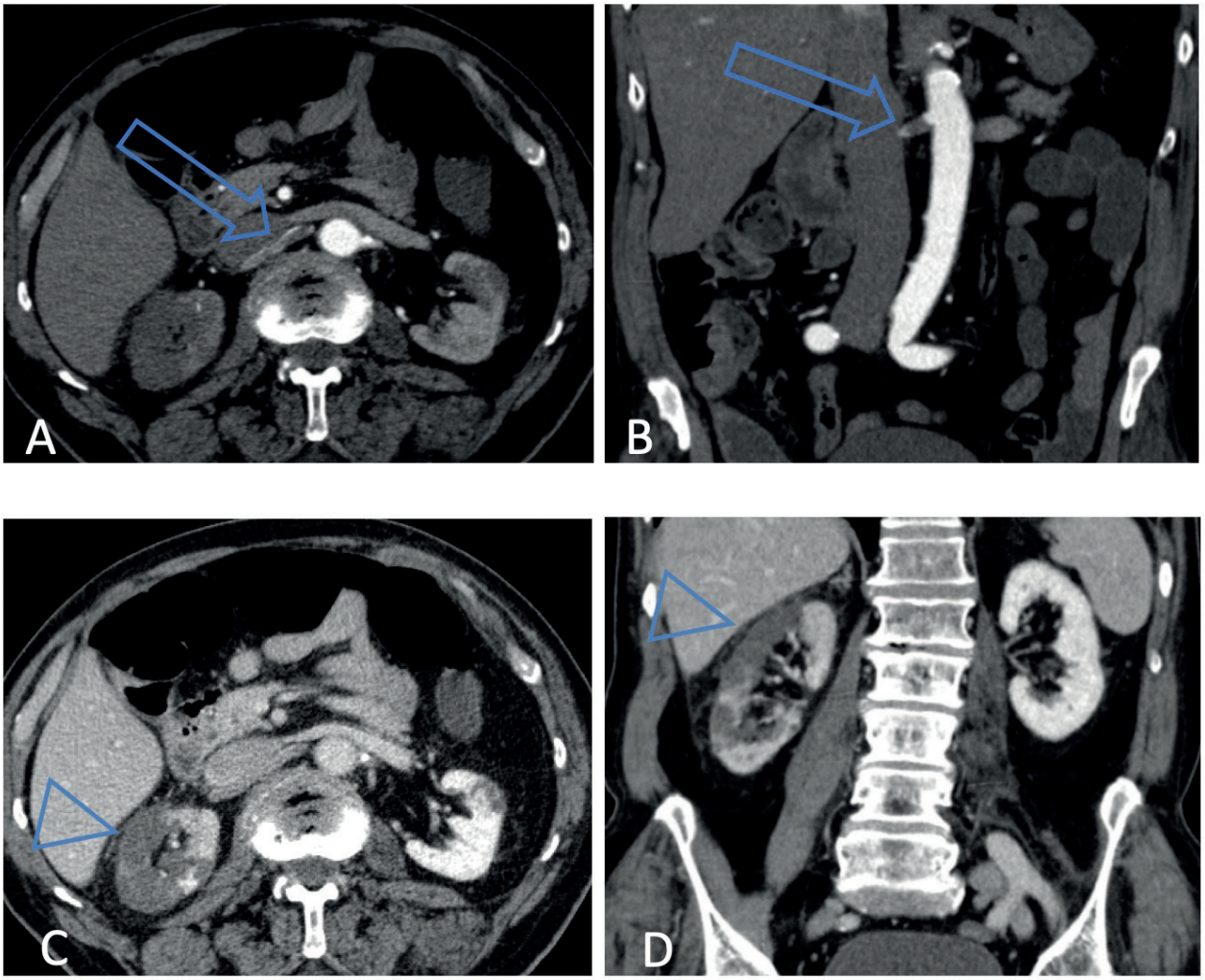

Figure 1. CTA of the abdomen at admission. Arterial phase showing thrombosis of the proximal branch of the right renal artery in axial (A) and coronal (B) multiplanar reconstruction (arrows). Venous phase axial (C) and coronal (D) multiplanar reconstruction showing multiples ischemic foci in the right kidney (arrowheads).

Considering the long delay in diagnosis, it was decided to perform conservative treatment with unfractionated heparin for 10 days to maintain an activated partial thromboplastin time of 60–80, while warfarin was started later to maintain an INR of 2–3. His clinical condition progressively improved, with right flank pain regression and amelioration of the kidney function. On day 7, CTA of the abdomen was repeated, showing maintenance of the right renal occlusion but an improved vascularization of the right renal parenchyma with compensatory left kidney function (Figure 2).

Echocardiography revealed no structural abnormalities, thrombi, vegetations, or intraluminal masses. The ejection fraction was normal (50%). A thrombophilia screen including lupus anticoagulant, anti-β2-glycoprotein-1 and anti-cardiolipin antibodies was done and resulted negative. Tumour markers (CEA, CA 19.9, alfa-fetoprotein) were all negative. During the hospitalization the patient developed an intestinal sub-occlusion managed conservatively with decompression with a nasogastric tube, intravenous hydration, and oral administration of a laxative with complete resolution after 48 hours. On day 27, he was discharged home in good clinical condition with resolution of symptoms and restoration of perfusion with anticoagulation (creatinine of 1.25 mg/dL at dis-

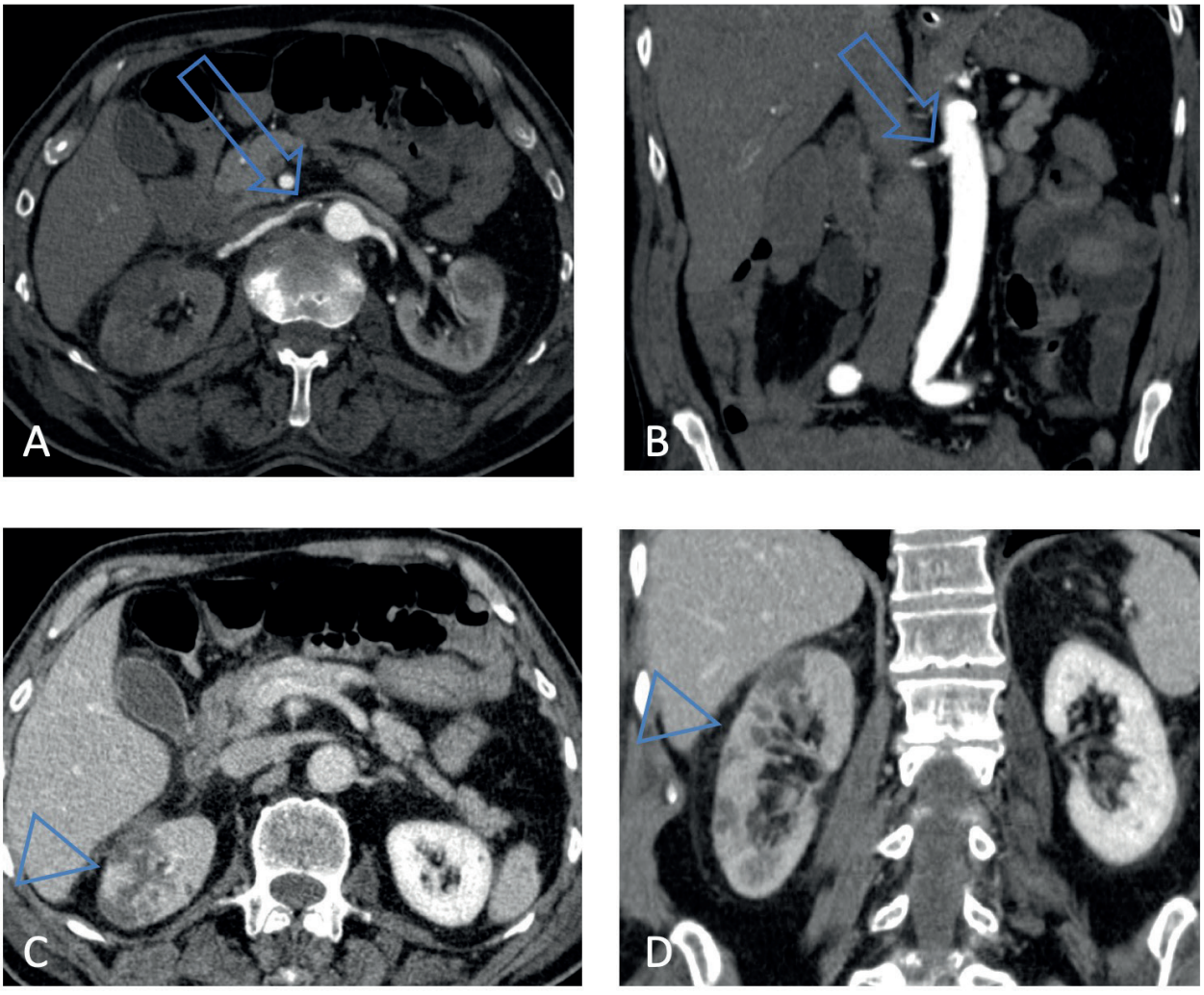

Figure 2. CTA of the abdomen performed after 7 days. Arterial phase showing the maintenance of the right renal artery thrombosis in axial (A) and coronal (B) multiplanar reconstruction (arrows). Venous phase axial (C) and coronal (D) multiplanar reconstruction showing the improved vascularization of the right renal parenchyma (arrowheads).

charge) and a final diagnosis of renal artery thrombosis and infarction due to atrial fibrillation.

Discussion

Renal artery thrombosis is a clinical condition that is often delayed or misdiagnosed due to its non-specific clinical presentation. It can occur at any age but is more frequent in patients aged group between 30 and 50 years, with no gender nor kidney predominance (5). Renal bilateral involvement is uncommon and is observed in about 10% of cases (5-7). The two main causes of renal infarction are thromboemboli, that originate in the heart or large arteries such as the aorta (8). Major risk factors include atrial fibrillation (7), atherosclerosis, valvular or ischemic heart disease, renal arteries stent, conditions of hypercoagulability, as well as the presence of a cancer, the regular use of oral contraceptives, systemic inflammation, pregnancy, obesity, smoking and diabetes (6, 9-11). Less common causes are infective endocarditis, vasculitis, nephrotic syndrome, renovascular hypertension, pyelonephritis (12), surgical arterial injury with complication, trauma, polycythaemia vera, sickle cell disease, Ehlers Danlos syndrome and cocaine use (5, 6). More recently, some cases of renal artery thrombosis secondary to SARS CoV2 infection have been reported in literature (13, 14). In our patient, misdiagnosed and untreated atrial fibrillation is the cause of renal artery thrombosis and infarction.

Clinical features

Signs and symptoms are non-specific and can mimic most of the common causes of abdominal pain, including renal colic, causing a significant delay in the diagnosis of more than 2 days in most of the cases (15). Acute and constant abdominal pain, generally refractory to analgesic therapy, and/or acute hypertension associated with high laboratory levels of white blood cells, serum creatinine and LDH, and microscopic haematuria are clues that should raise the suspicion of renal artery thrombosis or infarction. Fever, nausea, and vomiting are also common (16).

Diagnosis

A fast and proper diagnosis is mandatory to preserve the renal function. Renal infarction is the major complication that may result from an untreated renal artery thrombosis. A renal artery thrombosis and infarction should be suspected in all the patients complaining of persistent and unexplained unilateral flank or abdominal pain, associated with microscopic haematuria, leucocytosis and elevated LDH (16). LDH is a very sensitive marker for renal infarction, and it is usually five times higher than normal values (17), even if it could be elevated also in other conditions, including acute myocardial infarction, mesenteric embolism, and haemolysis (6, 18). Physicians should always investigate previous thromboembolic events, cardiac disease, atrial fibrillation, trauma, and aorta interventions, that are the most commonly associated risk factors (7).

PoCUS can be useful to exclude obstructive uropathy and nephrolithiasis. If hydronephrosis is not present and this diagnosis is suspected, CTA is mandatory to confirm the absence of renal perfusion (19). Diagnosis of renal artery embolism is established by the demonstration of an occlusion or filling defect in the renal artery on angiography. Domanovits and coll. suggested that in all patients presenting with the following triad: high risk of a thromboembolic event, persisting flank/abdominal/lower back pain, elevated serum levels of LDH and/or haematuria within 24 hours after pain onset, contrast-enhanced CT should be performed as soon as possible to prove or rule out acute renal infarction (9).

Management

There is no consensus on first-line treatment and there are no prospective studies to compare thrombolysis, anticoagulation, and embolectomy. Anticoagulation should be started immediately, firstly with intravenous heparin and then replaced with long-term oral warfarin, to prevent further embolic events, especially in patients with atrial fibrillation (5). The target INR may vary according to the cause of renal infarction. The usual goal is 2.0–3.0. A higher goal of 2.5–3.5 is

reasonable if events recur on adequate INR or in high-risk patients, such as those with rheumatic heart disease or a prosthetic valve (18).

Perfusion can be restored through thrombolysis or surgical revascularization (intra-arterial fibrinolytic therapy and mechanical thrombectomy with or without angioplasty) (18), although surgery has a high mortality rate and does not lead to better outcomes (15). Endovascular treatment is usually reserved for patients without a prolonged ischaemic time, cortical atrophy, and contraindication for thrombolysis. A very rare but important cause of acute renal failure is represented by bilateral renal artery occlusion, that can be treated with selective intrarenal artery infusion of thrombolytics with an optimal port-to-treatment time of 90-180 minutes (11).

After a diagnosis of renal artery thrombosis or renal infarction, chronic anticoagulation is always mandatory in patients with atrial fibrillation, a history of coagulopathy, and the presence of mechanical heart valves to prevent future thrombotic events.

Prognosis

The prognosis is strongly connected to the extent of thrombosis, time to diagnosis and the severity of renal failure. Most of the patients recover to normal renal function with no permanent hypertension (8, 17). Hypertension is the most common sequelae, that is caused by increased levels of renin, and should be treated with ACE-inhibitors or angiotensin receptor blockers. Only a small percentage of patients need dialysis (5) or nephrectomy (20).

Conclusions

Renal artery thrombosis is not a rare disease, but it is most often an under-diagnosed and under-reported diagnosis due to its non-specific presentation that may mimic renal colic and other more common causes of abdominal pain, as reported in the current literature. Clinicians should always consider this diagnosis in the presence of persisting flank/abdominal/lower back pain, elevated serum LDH levels and/or haematuria, especially in patients with a history of thromboembolic events or at high risk of thromboembolism, as early diagnosis and correct treatment can restore renal function, preventing severe and permanent kidney damage.

Contributions: EP, GMF and CB collected details of the case and drafted the manuscript. EP, GMF and CB cared for the patient. AV and EM critically revised the manuscript. All authors approved the final version and stated the integrity of the whole work.

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Ethics approval and consent to participate: As this was a descriptive case report and data was collected without patient identifiers, ethics approval was not required under our hospital's Institutional Review Board guidelines.

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Correspondence:
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Erika Poggiali, M.D.
Emergency Department, Guglielmo da Saliceto Hospital, Via Taverna 49, Piacenza, Italy
e-mail: poggiali.erika@gmail.com