Acute Renal Infarction Secondary to Membranous Glomerulopathy

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Abstract

Background: Acute renal infarction often presents with abdominal pain, nausea, vomiting, and fever. With other more common illnesses presenting with the same symptoms, it is often misdiagnosed leading to delayed treatment. We present a case of a young female diagnosed to have Membranous Glomerulopathy who presented with sudden onset flank pain in whom was initially treated as urinary tract infection.

Case: A 19-year-old female diagnosed with membranous glomerulopathy presented at the Emergency Room (ER) with severe, right sided, flank pain of acute onset, associated with nausea and vomiting. No fever, dysuria, hematuria, or history of trauma. Her vital signs were within normal range. Abdominal examination revealed a distended but soft non-tender abdomen with positive shifting dullness and fluid wave test. Right sided costovertebral angle tenderness was elicited. Initial diagnostics showed leukocytosis with neutrophilic predominance, serum creatinine of 0.77mg/dL, and proteinuria of >600mg/dL. Abdominal ultrasound showed non-specific findings, thus contrast-enhanced computed tomography scan (CT-Scan) of the abdomen was done which revealed areas of non-enhancement in the upper to middle portions of the right kidney which may relate to areas of ischemia and/or infarction, likely due to thrombosis involving the more distal portion of the right renal artery and massive ascites. Result was confirmed by computed tomography angiography (CTA) of the kidneys showing right renal artery thrombosis. Evaluations for other causes of renal artery thrombosis aside from patient's concurrent membranous glomerulopathy were done and were negative. Anti-coagulation therapy was initiated using low molecular weight heparin (LMWH) and was thereafter maintained on warfarin.

Conclusion: A high index of clinical suspicion is needed to diagnose acute renal infarction because of its non-specific symptoms which can mimic other conditions. Early diagnosis and prompt initiation of anti-coagulation therapy is important to avoid irreversible kidney damage. Acute renal infarction should be considered as a cause of acute onset flank pain in patients with risk factors and normal initial screening test.

Keywords: acute renal infarction, membranous glomerulopathy, flank pain, case report

Introduction

Acute renal infarction occurs when blood flow stops to a part of the kidney causing damage to the renal parenchyma. It has an estimated incidence of 0.007% out of the 250,000 patients and clinically diagnosed only in 0.014% of patients in the ER.[1,2] It commonly presents with abdominal pain, nausea, vomiting, and fever. Since abdominal pain is a common complaint at the ER and is associated with other more common illnesses, it is often misdiagnosed leading to delayed treatment. We present a case of a young female diagnosed to have Membranous Glomerulopathy who presented with sudden onset flank pain in whom a diagnosis of acute renal infarction secondary to renal artery thrombosis was confirmed after contrast enhanced CT-Scan of the abdomen and CTA of the kidneys, respectively.

Case

A 19-year-old woman was admitted at St Luke's Medical Center Quezon City due to sudden onset, right sided flank pain accompanied by nausea and vomiting episodes. She had no history of fever, dysuria, hematuria, constipation, loose bowel movement, or trauma. She was recently diagnosed to have membranous glomerulopathy stage II and is maintained on steroid and diuretic therapy. No history of surgery or allergies. Her last menstrual period was a week prior to her admission. There was no family history of kidney disease, connective tissue disease or vascular diseases. There was neither any history of oral contraceptive use.

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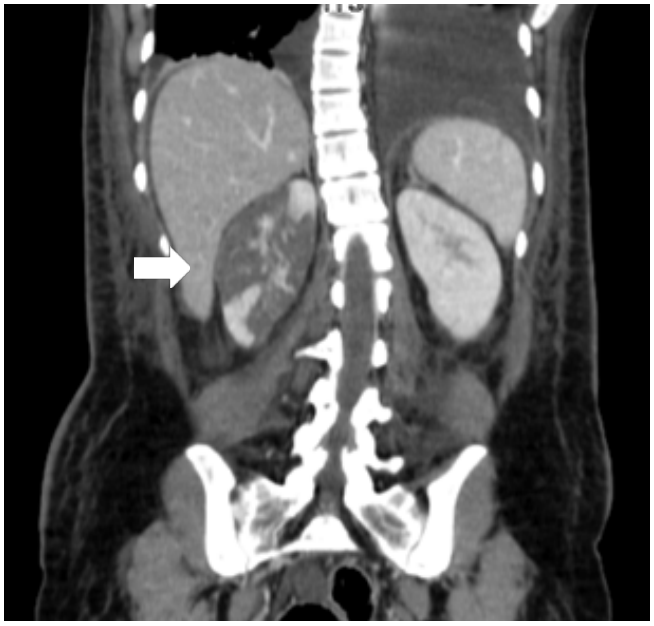

Figure 1: Contrast-enhanced whole abdomen CT-Scan showing filling defects on the right kidney (white arrow)

In the emergency room, she was awake, not in distress, with a blood pressure of 110/70 mmHg, heart rate of 82 beats/minute with regular rhythm, respiratory rate of 18 breaths/min, and afebrile. Physical examination findings were unremarkable except for the abdominal examination which revealed a distended abdomen with normoactive bowel sounds, and soft to touch. Shifting dullness and fluid wave test were positive. There were no scars, no tenderness nor organomegaly. Right sided costovertebral angle tenderness was elicited on percussion. Grade 1 pitting bipedal edema was also noted.

Differential diagnosis during that time included infections like urinary tract infection or acute gastroenteritis, and surgical abdomen like acute appendicitis and acute cholecystitis. Initial diagnostic investigations showed leukocytosis with neutrophilic predominance in complete blood count, proteinuria with 2 RBC/hpf in urinalysis, and normal serum creatinine (0.77 mg/dL), which were non-specific. Thus, whole abdomen ultrasound was requested and showed prominent gallbladder wall; perihepatic, perisplenic and pelvic ascites; normal pancreas; and an anteverted uterus. However, the right kidney appeared to be isoechoic when compared to the liver parenchyma, which was not present in the kidney ultrasound done 10 days prior, raising the possibility of non-specific renal parenchymal disease.

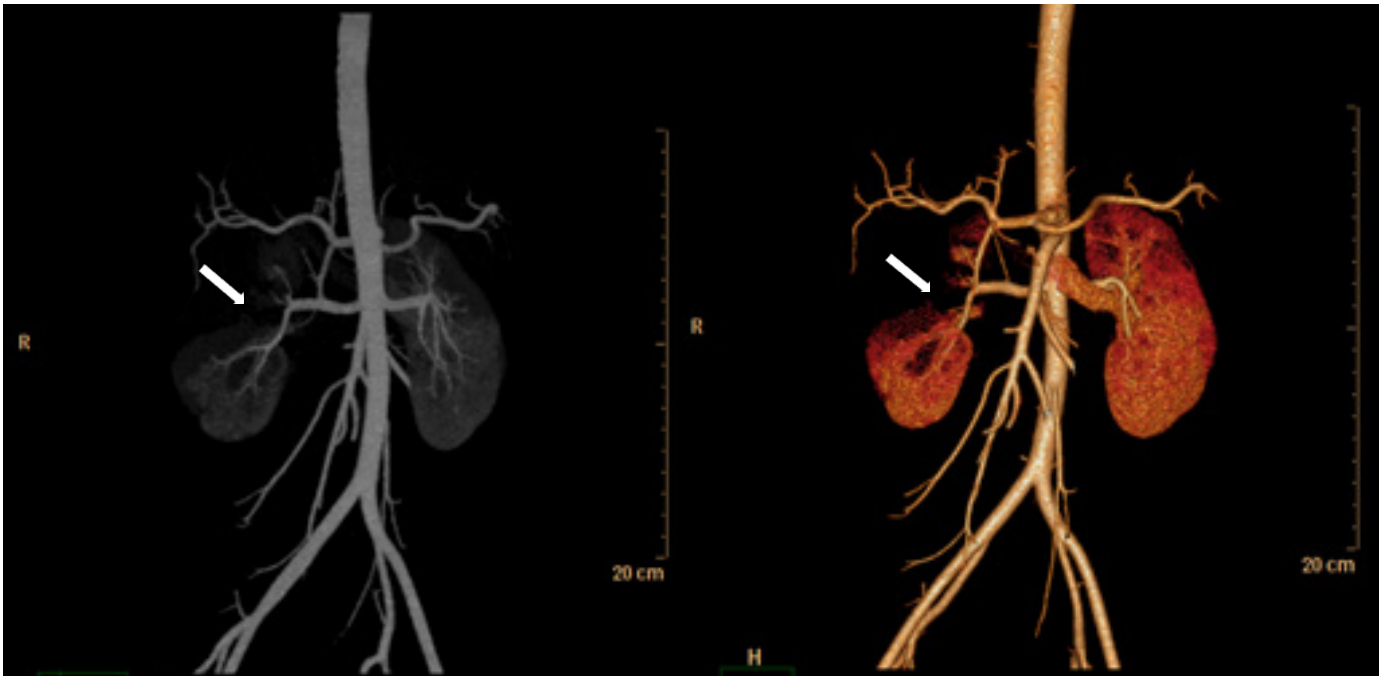

Figure 2: CT-angiography of the kidneys showing tubuar filling defects involving the right superior renal arteries (white arrow)

Due to persistence of right-sided flank pain despite aggressive pain control and non-specific findings in the whole abdomen ultrasound, further investigation for the cause of flank pain was done. Contrast enhanced CT scan of the whole abdomen showed cortico-medullary areas of non-e
nhancement from a portion of the upper pole down to the middle third of the right kidney. There was adequate enhancement of the right renal artery from its take-off at the aorta up to about 3.3 cm distally wherein a tubular filling defect was noted into one of its branches, likely containing thrombosis and massive ascites (Figure 1). This was confirmed with a tubular filling defect relating to thrombosis involving the right superior, anterior superior and anterior inferior segmental renal arteries on computed tomography angiography of the kidneys (Figure 2).

She was immediately started on anti-coagulation therapy using low molecular weight heparin. With the diagnosis of renal artery thrombosis, further evaluation for the probable cause of hypercoagulability was requested. Results showed elevated lactate dehydrogenase (2,508 U/L), normal bleeding parameters, decreased homocysteine levels (3), decreased protein S (45%), elevated protein C (176%), decreased anti-thrombin III (51%), negative LAC, and hypoalbuminemia, which were inconclusive for specific hypercoagulable disorders.

She was managed as a case of acute renal infarction secondary to right renal artery thrombosis due to membranous glomerulopathy stage II since other possible causes of hypercoaguability were inconclusive. Patient underwent pulse steroid therapy with methylprednisolone 1gm / day for three days. During the course of her admission, she developed vaginal bleeding and hematuria with presence of fine and course casts on urinalysis. Anti-coagulation therapy was temporarily stopped and was resumed at a lower dose after the bleeding subsided. Warfarin was overlapped with low molecular weight heparin and after achieving an INR range of 2-3, LMWH was discontinued. She was eventually discharged with warfarin and monitored closely on an out-patient basis.

Discussion

The patient was brought to the ER with severe right flank pain with no fever or hematuria and inconclusive abdominal examination findings. Abdominal pain is the most common reason for consult at the ER with non-specific abdominal pain as the main operative diagnosis.[3,4] This is despite the presence technological advances in laboratory and imaging.[4]

Acute renal infarction due to thromboembolic occlusion of the renal artery was first reported by Traube in 1856 in Germany.[5] Renal infarction is rare. It has an estimated incidence of 0.007% out of the 250,000 patients and is clinically diagnosed only in 0.014% of patients in the ER.[1,2] In 1940, a study showed that the incidence of renal infarction was only 1.4% out of 14,411 autopsies.[10] The frequency of renal infarction is probably higher since the clinical diagnosis is frequently missed or delayed because the symptoms mimic other common conditions like nephrolithiasis and pyelonephritis.

Our patient is 19 years old, contrary to the mean age of 53 years when acute renal infarct usually occurs.[5,7,9] But she presented the typical symptoms of acute renal infarct like acute onset flank pain with nausea, vomiting, and fever. Her abdominal findings were vague and non-specific. A patient with acute renal infarct would reveal costovertebral angle tenderness.[6] Laboratories that could have support the diagnosis include hematuria, leukocytosis, and elevated lactate dehydrogenase (LDH).[1] Our patient had leukocytosis and elevated LDH.

The gold standard for diagnosing renal infarction is revealing a non-opacified kidney of normal or increased size with non-dilated pelvicalyceal system, cortical rim sign, and a wedge-shaped area of non-enhancement in patients with branch occlusion that can be seen in contrast-enhanced CT scan.[2] Our patient had these findings at her right kidney concluding the diagnosis of acute renal infarct.

With no apparent risk factor for renal infarction, nephrotic syndrome, which is present in our patient, is a possible cause of renal infarction in our patient. In a case series of renal infarction, nephrotic syndrome is present in only one out of 94 patients diagnosed with nephrotic syndrome.[5] Patients with nephrotic syndrome have a higher frequency of arterial thromboses compared to the general population with an annual incidence of 5.5%.[11-14] Among the causes of nephrotic syndrome, membranous glomerulopathy appears to have the highest risk of thrombosis, which is present in our patient.[11,15-18]

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Treatment options include anticoagulation, thrombolysis and embolectomy. Anticoagulation typically consists of

giving low molecular weight Heparin followed by Warfarin and low dose aspirin. Selective intra-arterial thrombolysis is considered for unilateral renal infarction.[2] The prognosis of acute renal infarction is determined by the etiology and the size of the infarct and the success of the intervention is limited by the duration of the ischemia.[2,5]

Conclusion

A high index of clinical suspicion is needed to diagnose acute renal infarction because of its non-specific symptoms, which can mimic other conditions such as nephrolithiasis and pyelonephritis. Early diagnosis and prompt initiation of anti-coagulation therapy is important to avoid irreversible kidney damage. Acute renal infarction should be considered as a cause of acute onset flank pain in patients with risk factors and normal initial screening tests. This should be an eye opener to physicians to think of other less common differentials for flank pain.

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