# A Case of Recurrent Renal Infarction Following Transient Resolution: Evidence From Serial Computed Tomography

In Hong Choi<sup>1,2</sup>, Chang Seong Kim<sup>1,2</sup>, Eun Hui Bae<sup>1,2</sup>, Seong Kwon Ma<sup>1,2</sup>, Soo Wan Kim<sup>1,2</sup> and Hong Sang Choi<sup>1,2</sup>

<sup>1</sup>Department of Internal Medicine, Chonnam National University Medical School, Gwangju, Republic of Korea; <sup>2</sup>Department of Internal Medicine, Chonnam National University Hospital, Gwangju, Republic of Korea

Received: December 27, 2023 Revised: April 10, 2024 Accepted: April 16, 2024 Corresponding Author: Hong Sang Choi, MD, PhD Department of Internal Medicine Chonnam National University Medical School 42 Jebongro, Gwangju 61469, Republic of Korea Tel: +82-62-220-6296; Fax: +82-62-225-8578 E-mail: choihs@jnu.ac.kr Although renal infarction (RI) is not a rare disease, its outcomes have not been well-documented. Furthermore, transient resolution and recurrence of RI have not been captured through imaging. We report a case of idiopathic RI that recurred within a short period following transient resolution, as demonstrated by serial computed tomography (CT). A 53-year-old man diagnosed with RI was transferred to the emergency room. An abdominal CT scan at the local hospital revealed a segmental wedge-shaped perfusion defect in the left kidney and a focal thrombotic filling defect in the anterior segmental branch of the left renal artery. Since his left flank pain improved, another CT scan was performed again 6 hours after the initial CT scan. A repeat CT scan showed that the thrombus in the renal artery remained, but the perfusion defect had spontaneously resolved. We initiated anticoagulant therapy using unfractionated heparin. On the sixth day of hospitalization, the left flank pain recurred, prompting another CT scan. The follow-up CT scan confirmed that RI had recurred in the same area as before. We continued anticoagulant therapy and switched to warfarin. After treatment, his symptoms improved, and he was discharged. RI can recur at any time, even after it has spontaneously resolved, as evidenced by our case. Therefore, it is crucial to closely monitor patients who experience resolution of RI for any recurrence of symptoms, and repeat radiological evaluation should be performed even within a short period.

Key Words: Renal infarction, Transient resolution, Recurrence, Computed tomography

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (https://creativecommons.org/licenses/by-nc/4.0/) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

## INTRODUCTION

Renal infarction (RI) is an uncommon condition that can result from acute occlusion of blood flow in the renal arteries or their segmental branches<sup>1-6)</sup>. Many risk factors are known to cause RI, including cardiogenic factors, such as atrial fibrillation, valvular heart disease, and endocarditis, as well as renal artery injury, hypercoagulation disorders, and hematologic diseases<sup>1,2)</sup>. However, in some cases, the cause is unknown<sup>1,2)</sup>.

The outcomes of RI can include acute kidney injury, pro-

gression to chronic kidney disease or end-stage renal disease, and death<sup>1)</sup> Moreover, thromboembolic events are likely to recur in other organs, including the kidneys<sup>3,7)</sup>. However, to our knowledge, there have been no published large-scale retrospective studies investigating the outcomes of RI, so these have not been well reported<sup>1)</sup>. Furthermore, there have been no reports demonstrating the recurrence of RI after transient resolution through imaging. We report a case of idiopathic RI that recurred within a short period after transient resolution, as demonstrated by serial computed tomography (CT).

## CASE REPORT

A 53-year-old man suspected of having RI based on an abdominal CT scan was transferred to our emergency room (ER) from a local hospital. He complained of Lt flank pain about five hours ago, and the symptom persisted until he was at the local hospital, but gradually improved after arriving at our hospital. He was taking 80 mg of valsartan, 5 mg of amlodipine, and 0.2 mg of tamsulosin per day for hypertension and benign prostatic hyperplasia diagnosed 10 years ago and was not taking any other medications such as antiplatelet agents or anticoagulants. He was a current smoker for about 15 pack-years and a social alcohol drinker. His blood pressure was 150/80 mmHg, his pulse rate was 82 beats/min, and his body temperature was 36. 6°C. Laboratory studies showed a white blood cell count of  $12.9 \times 10^3 / \mu L$  and an elevated lactate dehydrogenase (LDH) level (640 IU/L), while his C-reactive protein level (0.15 mg/dL) was normal. Additionally, he had a normal creatinine level (0.95 mg/dL) and a normal eGFR (91.02 mL/ min/1.73 m<sup>2</sup>). No hematuria or pyuria was observed; however, mild proteinuria (1+) was detected in the urinalysis. An abdominal CT scan at the local hospital revealed a segmental wedge-shaped perfusion defect in the left kidney and a focal thrombotic filling defect in the anterior segmental branch of the left renal artery (Fig.1A, 2A). Since his flank pain improved after admission to our ER, another abdominal CT scan was performed 6 hours after the initial

CT scan at the local hospital. The thrombus in the anterior segmental branch of the left renal artery remained, but the segmental wedge-shaped perfusion defect in the left kidney had spontaneously resolved in the repeated CT scan (Fig. 1B, 2B). We initiated anticoagulant therapy with intravenous unfractionated heparin and adjusted the dose to target activated partial thromboplastin time of 60-90 seconds. We tested his blood for hypercoagulability and autoimmune diseases and performed electrocardiography and echocardiography to investigate the cause of the RI. The blood tests for hypercoagulability such as protein C, protein S, antithrombin and homocysteine, and tests for antiphospholipid syndrome did not reveal any abnormal findings. Electrocardiography did not detect any arrhythmias during his hospitalization, and there was no evidence of thromboembolic sources such as thrombi or vegetations on the echocardiogram. We, therefore, diagnosed him with idiopathic RI. On the sixth day of hospitalization, he again complained of left flank pain similar to his previous pain. He did not have a fever, and there was no evidence of a urinary tract infection, so another abdominal CT scan was performed to ascertain the possibility of recurrent RI. The follow-up abdominal CT scan showed that the segmental wedge-shaped perfusion defect had recurred in the same area as previously observed, and the thrombus in the anterior segmental branch remained (Fig. 1C, 2C). We continued anticoagulant therapy with unfractionated heparin for a week and transitioned to warfarin 6-7 mg/day for the goal of prothrombin time 2-3 INR, with a period of overlap between the two



**Fig. 1.** Serial transverse views of abdominal CT scans of the patient with renal infarction. (**A**) An abdominal CT scan at the local hospital revealed a perfusion defect in the left kidney (arrowhead); (**B**) The perfusion defect spontaneously resolved (arrowhead) in our emergency room; (**C**) The perfusion defect recurred (arrowhead) on the sixth day of hospitalization. A thrombus in the anterior segmental branch of the left renal artery (arrow) persisted on all serial abdominal CT scans.



Fig. 2. Serial coronal views of abdominal CT scans of the patient with renal infarction. (A) An abdominal CT scan at the local hospital showed a perfusion defect in the left kidney (arrowhead); (B) The perfusion defect spontaneously resolved (arrowhead) in our emergency room; (C) The perfusion defect recurred (arrowhead) on the sixth day of hospitalization.

anticoagulants. After treatment, his symptoms improved, and he was discharged on the eighth day of hospitalization with ongoing anticoagulant therapy. Six months after discharge, low-dose aspirin was prescribed instead of warfarin, and it was planned to be maintained lifelong to prevent recurrence of RI. One year after the event, he had not experienced any symptoms associated with RI such as flank pain, and his renal function remained within the normal range of eGFR 96 mL/min/1.73 m<sup>2</sup>.

# DISCUSSION

The incidence of RI is not vet clearly known<sup>8)</sup>. Domanovits et al. and Huang et al. reported that 0.007% (17 of 248,842) and 0.004% (20 of the 481,540), respectively, of patients who visited the ER were diagnosed with RI<sup>3,8)</sup>. Korzets et al. also found that 11 of 151,914 patients (0.007%) admitted to their hospital were diagnosed with RI<sup>9</sup>. However, the actual prevalence is thought to be higher because RI is often misdiagnosed owing to its non-specific symptoms<sup>9</sup>. Additionally, as the use of contrast-enhanced CT increases as a diagnostic tool for abdominal problems of unknown origins, the number of patients diagnosed with RI is also increasing<sup>9)</sup>. The symptoms and signs of RI include abdominal or flank pain, nausea, vomiting, fever, and hypertension <sup>1,2,4,9,10</sup>. However, because of these non-specific features, RI is often misdiagnosed as other more common diseases, such as urolithiasis, acute pyelonephritis, and back pain of musculoskeletal origin, leading to delayed diagnosis<sup>8,9</sup>. Increased serum LDH is the most sensitive laboratory finding, and inflammation markers, such as white blood cells and C-reactive protein, are sometimes elevated<sup>1-3,10</sup>. Hematuria and proteinuria are also sometimes present<sup>3,10</sup>. However, these same laboratory findings and urinalysis results can be associated with other diseases<sup>3,10</sup>. Therefore, imaging tools, such as CT, magnetic resonance imaging, renal angiography, and/or scintigraphy, are required to confirm Rl<sup>1,3</sup>. Since contrast-enhanced CT is non-invasive and can be performed within 24 hours, it is currently the gold standard for diagnosing Rl<sup>3</sup>.

Treatment options for RI include radiologic or surgical percutaneous endovascular therapy, anticoagulant therapy, and antiplatelet therapy $^{1,2)}$ . However, there are currently no prospective randomized clinical trials to determine which treatment is superior<sup>2,3)</sup>. Thus, there is still no established definitive treatment for RI<sup>2,3)</sup>. Treatment for RI should be chosen considering various factors such as the time taken to diagnose the infarction, the underlying cause of the infarction, and the severity of the infarction<sup>2,3,7,8)</sup>. If revascularization is deemed to be more beneficial, radiologic or surgical percutaneous endovascular therapy may be considered as the initial intervention<sup>2,3,7,8)</sup>. If revascularization is not deemed beneficial, antiplatelet therapy or anticoagulant therapy may be selected based on the underlying cause of the RI<sup>2,3,7,8)</sup>. In cases diagnosed as idiopathic renal infarction, like our case, anticoagulant therapy is typically initiated.

If the diagnosis of RI is delayed, appropriate treatment cannot be administered, which may result in deterioration of renal function and even death<sup>6,8)</sup>. Therefore, early diagnosis is important for improving outcomes<sup>3)</sup>. Domanovits et al. suggest that contrast-enhanced CT should be performed early for all patients exhibiting the triad of high risk for thromboembolic events: persistent back pain, elevated serum LDH, and/or hematuria within 24 hours of the onset of pain<sup>3)</sup>. However, this triad has limitations: many patients diagnosed with RI have a low risk of thromboembolism and often do not have hematuria<sup>5,8)</sup>. Huang et al. also proposed a flow chart for the diagnosis and treatment of RI<sup>8)</sup>. Due to the current lack of established guidelines for the early diagnosis of RI, further studies are needed. It is also important for physicians to be aware that RI is not a rare disease<sup>8)</sup>.

The outcomes of RI are not yet clearly known<sup>1)</sup>. Specifically, there are few studies that have reported the frequency and timing of recurrent thromboembolic events in RI<sup>1,5)</sup>. Oh et al. found that 2.8% (12 of 438) of patients diagnosed with RI experienced recurrence, and the median time to recurrence was 11.5 months (range, 1-108 months)<sup>1</sup>. García-García et al. reported that 11.9% (7 of 59) of patients diagnosed with RI had recurrent arterial thrombosis (three with RI, two with cerebrovascular disease, and three with ischemic heart disease) and 3.4% (2 of the 59 patients) had recurrent venous thromboembolism<sup>5)</sup>. While Oh et al. found that there was no significant difference in the recurrence rate based on the cause of RI, García-García et al. determined that the recurrence rate of arterial thrombosis was higher in the group with clear underlying pathogenetic mechanisms compared with the idiopathic group<sup>1,5)</sup>. Additional studies are needed to understand the frequency, timing, and risk factors associated with recurrent RI.

There is a lack of established guidelines for monitoring patients following the initiation of treatment for RI. Our case demonstrated that thromboembolic events can recur during treatment, and the recurrence interval can be remarkably short. As early diagnosis and appropriate treatment are important for the prognosis of RI<sup>3,4,6-8)</sup>, it is also crucial to detect the recurrence of thromboembolic events early. To achieve this, proper monitoring of the patient's status following treatment is required. This entails ongoing

assessment of their clinical condition, laboratory findings, and urinalysis. Physicians' alertness to the possibility of recurrent RI is also crucial for early detection. If there is suspicion of recurrence of RI during monitoring, it is necessary to actively perform radiologic examinations (especially contrast-enhanced CT) to confirm recurrence.

The first is the presence of undetected arrhythmias such as paroxysmal atrial fibrillation (Af). Af is one of the main causes of RI, and it is important to suspect the presence of paroxysmal Af even if arrhythmia is not found when RI occurs (cereus paper). Although we were monitoring the patient very closely and maintaining anticoagulation, this possibility cannot be completely ruled out. Second, the patient's thrombotic risk may have been higher than we expected. The patient was a long-term current smoker. Smoking is known to be one of the risk factors for RI. Huang et al. reported that current smoking had a significant adverse impact on thromboembolic complications after RI<sup>11)</sup>. Therefore, continuous monitoring of the expression of Af will be very important in determining the treatment policy for patients with renal infarction, and educating current smokers such as our patient to quit smoking will likely improve the patient's prognosis.

As we experienced during the management of this patient, the recurrence of RI may be observed during the administration of anticoagulation therapy despite the prior spontaneous resolution of the RI. Furthermore, RI recurrence can occur within a short period following spontaneous resolution. Radiologically documented cases of spontaneous resolution and recurrence of RI during initial anticoagulation are rare. Therefore, physicians' awareness of the potential for recurrent thromboembolic events is essential. Also, if recurrence is suspected, it is important to actively perform radiologic examinations for early diagnosis.

#### Acknowledgements

This work was supported by a National Research Foundation of Korea (NRF) grant funded by the Korean government (Ministry of Science and ICT) (NRF-2022R1C1C1007573)

#### Disclosure

The authors have no potential conflicts of interest to disclose.

### REFERENCES

- 1. Oh YK, Yang CW, Kim YL, et al.: Clinical Characteristics and Outcomes of Renal Infarction. Am J Kidney Di 2016;67(2): 243-250.
- Bourgault M, Grimbert P, Verret C, et al.: Acute renal infarction: a case series. Clin J Am Soc Nephrol 2013;8(3): 392-398.
- Domanovits H, Paulis M, Nikfardjam M, et al.: Acute renal infarction. Clinical characteristics of 17 patients. Medicine (Baltimore) 1999;78(6):386-394.
- 4. Bae EJ, Hwang K, Jang HN, et al.: A retrospective study of short- and long-term effects on renal function after acute renal infarction. Ren Fail 2014;36(9):1385-1389.
- García-García A, Demelo-Rodríguez P, Ordieres-Ortega L, et al.: Idiopathic versus Provoked Renal Infarction: Characteristics and Long-Term Follow-Up of a Cohort of Patients in a Tertiary

Hospital. Kidney Blood Press Res 2019;44(6):1432-1440.

- Antopolsky M, Simanovsky N, Stalnikowicz R, et al.: Renal infarction in the ED: 10-year experience and review of the literature. Am J Emerg Med 2012;30(7):1055-1060.
- Hazanov N, Somin M, Attali M, et al.: Acute renal embolism. Forty-four cases of renal infarction in patients with atrial fibrillation. Medicine (Baltimore) 2004;83(5):292-299.
- 8. Huang CC, Lo HC, Huang HH, et al.: ED presentations of acute renal infarction. Am J Emerg Med 2007;25(2):164-169.
- 9. Korzets Z, Plotkin E, Bernheim J, et al.: The clinical spectrum of acute renal infarction. Isr Med Assoc J 2002;4(10):781-784.
- Chu PL, Wei YF, Huang JW, et al.: Clinical characteristics of patients with segmental renal infarction. Nephrology (Carlton) 2006;11(4):336-340.
- 11. Huang CW, Lee MJ, Hsu CY et al.: Clinical outcomes associated with anti-coagulant therapy in patients with renal infarction. QJM 2018;111(12):867-873.