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Case Report

# Acute interstitial nephritis manifesting as a persistent nephrogram ☆

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ABSTRACT

We report a case of a 61-year-old woman who presented to the emergency department with high inflammatory parameters and acute renal failure. Marked enlarged kidneys with persistent nephrogram were found on contrast-enhanced computed tomography . Renal biopsy showed acute interstitial nephritis. Acute interstitial nephritis must be included in the differential diagnosis in patients presenting with a persistent nephrogram.



## Introduction

Acute interstitial or tubulointerstitial nephritis (AIN) is implicated in up to 15%-25% of patients hospitalized for acute renal failure and is therefore one of the most frequent causes of acute renal failure [1–3]. Renal injury in AIN results from immune-mediated tubulointerstitial injury and is drug-induced in more than 75% of cases. In about 10%, it is infection-induced or related to autoimmune disorders. Rarely, it is idiopathic, accounting for less than 5% of cases [1,4,6]. Men and women are affected equally. AIN can be seen in any age group, but the wider use of medication in the elderly combined with reduced renal clearance places them at greater risk of adverse events including AIN [7].

Patients with AIN typically present with nonspecific symptoms of acute renal failure (malaise, anorexia, nausea, …). Since laboratory and standard imaging (ultrasound) findings are non-specific, renal biopsy is the only definitive method of establishing the diagnosis [1,2,4,6]. Here, a case is reported of a persistent nephrogram on contrast-enhanced computed tomography (CT) corresponding to biopsy proven acute interstitial nephritis.

## Case report

A 61-year-old woman was referred to the emergency department for high infectious parameters and mild diarrhea with-

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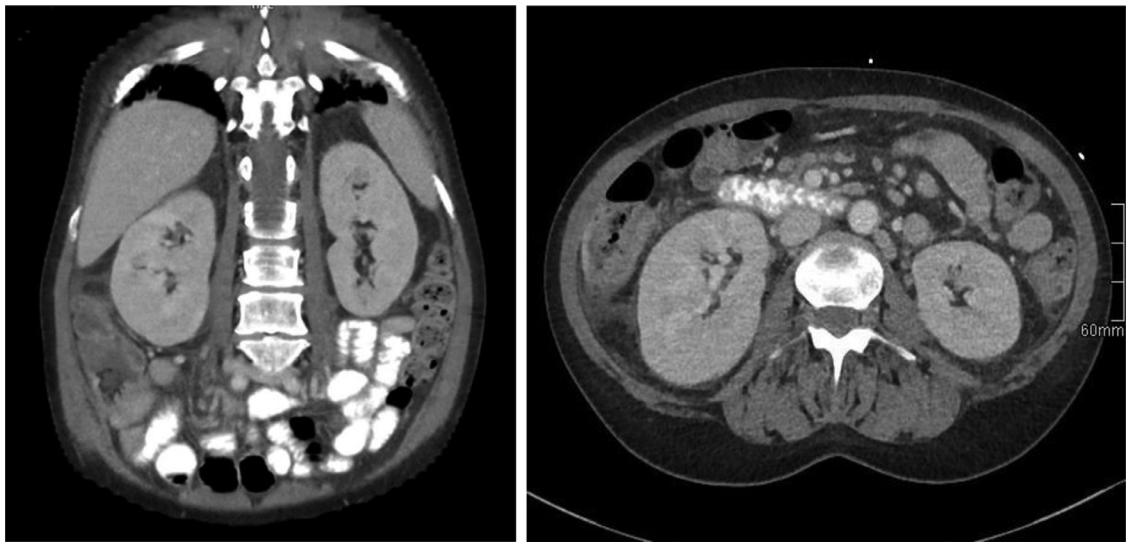


**Fig. 1 – Paracoronal (left) and axial (right) abdominal contrast-enhanced images in the nephrographic phase show diffusely enlarged kidneys. There is bilateral fat infiltration and fluid in the pararenal loges. The contour of the renal capsule is intact and regular without wall thickening.**

out an evident focus. She had been feeling unwell and weak for the past 3 days with loss of appetite. The patient has a history of insulin dependent diabetes mellitus type 2, positron emission tomography/CT confirmed polymyalgia rheumatica and bipolar disorder treated with prednisolone and lithium respectively. She also had a history of Hashimoto thyroiditis 9 years ago. On admission, the patient was hemodynamically stable and afebrile.

She experienced no headache, thoracic or abdominal pain. She denied any fever, rash, difficulty urination, or discoloration of urine. She mentioned a recent transient discrete upper respiratory infection without residual complaints.

Physical exam revealed no cardiovascular, thoracoabdominal, neurologic, musculoskeletal, or dermatologic abnormalities.

Initial laboratory work-up showed significant inflammation with elevated white blood cell count of 12,000/mm (reference range: 5000-10,000/mm) and C-reactive protein of 400 mg/dL (reference range: <4 mg/dL). An increased creatinine of 4.16 mg/dL (reference range: 0.5-1.1 mg/dL) and decreased glomerular filtration rate 35 mL/min/1.73 $m^2$ (reference range: 90-120 mL/min/1.73 $m^2$) were consistent with acute renal failure. Urinalysis showed no hematuria, pyuria, or proteinuria.

The referring physician ruled that the glomerular filtration rate was still sufficient to perform a contrast-enhanced CT (CECT) of the lungs and abdomen to exclude pneumonia, pyelonephritis, or diverticulitis provided that hydration was carried out afterward.

The chest CT scan was normal and showed no inflammatory focus. However, the nephrographic phase of the contrast-enhanced abdominal CT revealed marked enlargement of both kidneys (Fig. 1). The nephrogram is normal. Bilateral perirenal fat stranding and fluid in the pararenal compartments were observed. There was a little amount of ascites in the left and right paracolic gutter and multiple normal and slightly enlarged mesenteric and retroperitoneal lymph nodes. Renal veins were patent and without congestion. There was neither hydronephrosis nor kidney stones. A few type I Bosniak cortical cysts were present bilaterally.

A delayed phase scan performed 1 h after intravenous contrast administration showed contrast media retention in both kidneys, also known as a persistent nephrogram (Fig. 2) [8–10]. Renal contours were unsharply delineated due to limited motion artefacts. There was no striated nephrogram as seen in pyelonephritis. The findings are in favor of acute inflammation of the kidneys.

The patient was hospitalized at the nephrology department. Medical treatment with methylprednisolone (Medrol, Pfizer, Brussels, Belgium) 32 mg/d and amoxicillin/clavulanate potassium (Augmentin®, Pfizer, Brussels, Belgium) 1 g 3×/d intravenously was started. Prompt ultrasound-guided percutaneous renal needle biopsy was performed to establish a definitive diagnosis.

Anatomopathological results showed interstitial infiltrates characteristic for interstitial nephritis. There was little tubular damage with some mild chronic changes being present. Ultrastructural evaluation of the glomeruli was normal. There were no arguments for lithium toxicity.

Serology showed elevated immunoglobulin G antibody titers, suggestive for a recent mycoplasma infection, although clinically silent. All other screens for typical and atypical infections were negative.

With a history of Hashimoto thyroiditis and polymyalgia rheumatica, both autoimmune disorders, targeted immunologic tests were performed to exclude an autoimmune AIN. All test results were normal.

## Discussion

We highlight the unexpected CT-graphic appearance of acute interstitial nephritis presenting with a persistent nephrogram.

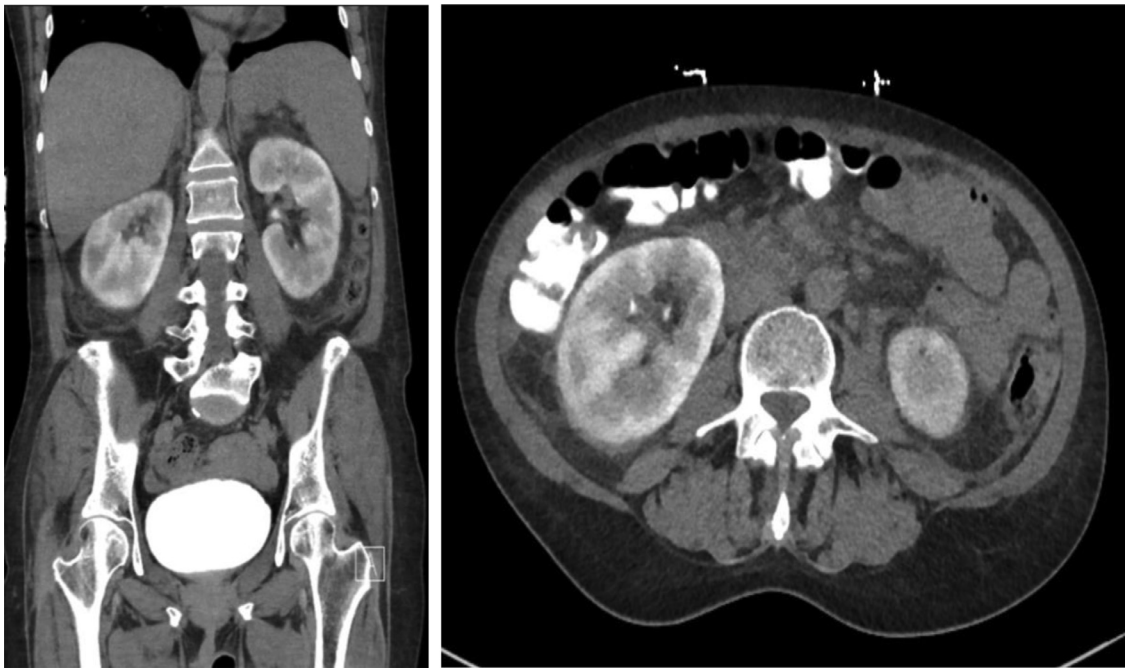

**Fig. 2 – Coronal (left) and axial (right) abdominal contrast-enhanced images in the delayed phase show homogeneous contrast media retention in both kidneys, known as a persistent nephrogram. Renal contours are unsharply delineated due to limited motion artefacts. The bladder is completely filled with contrast.**

A nephrogram is defined as the radiographic image of opacified renal parenchyma after water-soluble iodinated contrast material administration [11]. The nephrogram varies with the scanning phase. In the corticomedullary phase, typically 25-80 seconds after initiation of contrast injection, the cortex is clearly distinguishable from the medulla. This is called the cortical nephrogram. In the nephrographic phase, usually 85-120 seconds after contrast injection, contrast has entered the loop of Henle and the collecting ducts. In this tubular or generalized nephrogram, differentiation between the cortex and medulla is lost. In late or excretory nephrogram, usually 3 minutes after contrast injection, renal parenchyma attenuation is much less intense and contrast usually has entered the pyelum [11,12].

Eight major abnormal nephrograms have been described each with their own differential diagnostics (ie, absent, delayed, persistent, hyperdense, rim, reverse rim, striated and spotted nephrogram) [11,13].

With a persistent nephrogram, both kidneys remain in the corticomedullary or nephrographic phase for more than 3 minutes [13].

In the literature, acute interstitial nephritis has been previously described as presenting with a striated nephrogram [14–16] but no previous case reports or articles could be found were AIN presents with persistent nephrogram.

A persistent nephrogram can be seen in a few conditions: bilateral obstructive uropathy, renal artery or renal vein stenosis, systemic hypotension, and acute tubular necrosis [9,15,17].

The absence of nephro- or urolithiasis and hydronephrosis excluded bilateral obstructive uropathy.

A vascular cause such as bilateral renal artery or vein stenosis could also be excluded as there is normal contrast enhancement of both the arteries and veins without significant stenosis.

As blood pressure is normal, systemic hypotension was also highly unlikely.

Acute tubular necrosis is characterized by renal tubular cell damage and cell loss usually caused by ischemic or nephrotoxic insults. CECT typically demonstrates a striated or persistent nephrogram [15].

Clinical and laboratory findings excluded pyelonephritis. On postcontrast CT, there was no striated nephrogram typical for pyelonephritis [16].

Renal biopsy remains the gold standard for diagnosis of all forms of nephritis, including interstitial nephritis [1,2,4,6].

On renal biopsy, the infiltration of the interstitium with inflammatory cells (in this case, lymphocytes, macrophages, and plasma cells), associated edema and sparing of glomeruli and blood vessels was pathognomonic for AIN [1,5,6].

The exact cause of interstitial nephritis in this case has not been definitively established but by exclusion, mycoplasma infection will be the most likely cause. No new medication was started that could have triggered the AIN. Also, the lack of a significant number of eosinophils on histopathology is not in favor of drug-induced TIN [6]. Autoimmune investigation was also completely normal which makes an autoimmune cause unlikely.

The patient responded well on corticosteroid therapy and renal function quickly restored.

The precise mechanism of the radiologic image of prolonged renal cortical retention of contrast media remains unclear. Histopathologically, it could be explained by altered glomerular hemodynamics due to interstitial inflammation [18]. We also hypothesize that there must be compression

of the arterioles, glomeruli, and tubules secondary to the interstitial infiltrates in combination with a rigid renal capsule that doesn't allow a lot of tissue expansion. Because AIN usually presents with acute renal failure, a CECT is often contraindicated. This could explain why this was not previously reported.

## Conclusion

Acute interstitial nephritis must be included in the differential diagnosis in patients presenting with a persistent nephrogram on CECT. When diffuse enlarged kidneys are observed on CT, performing a late excretory phase can reveal a persistent nephrogram and narrow the differential diagnostics.

## Authors' contributions

Lien Van Liedekerke was the main contributor in constructing and writing of the manuscript, performed literature search, and prepared the figures and figure captions.

Roel Beckers contributed to drafting of the manuscript and critical revision.

Laurens De Cocker and Kenneth Carels performed critical revision.

## Patient consent

The author did obtain written informed consent from the patient for submission of this manuscript for publication.

## REFERENCES


[1] Kodner CM, Kudrimoti A. Diagnosis and management of acute interstitial nephritis. Am Fam Physician 2003;67(12):2527–34.

[2] Naik RH, Annamaraju P. Interstitial nephritis. StatPearls [Internet]. Treasure Island, FL: StatPearls Publishing; 2022.

[3] Raghavan R, Eknoyan G. Acute interstitial nephritis—a reappraisal and update. Clin Nephrol 2014;82(3):149–62. doi:10.5414/cn108386.

[4] Bhandari J, Thada PK, Arif H. Tubulointerstitial nephritis. StatPearls [Internet]. Treasure Island, FL: StatPearls Publishing; 2022.

[5] Praga M, González E. Acute interstitial nephritis. Kidney Int 2010;77(11):956–61. doi:10.1038/ki.2010.89.

[6] Joyce E, Glasner P, Ranganathan S, Swiatecka-Urban A. Tubulointerstitial nephritis: diagnosis, treatment, and monitoring. Pediatr Nephrol 2017;32(4):577–87. doi:10.1007/s00467-016-3394-5.

[7] Clarkson MR, Giblin L, O'Connell FP, O'Kelly P, Walshe JJ, Conlon P, et al. Acute interstitial nephritis: clinical features and response to corticosteroid therapy. Nephrol Dial Transplant 2004;19(11):2778–83. doi:10.1093/ndt/gfh485.

[8] Persistent nephrogram. Available at: https://radiopaedia.org/articles/persistent-nephrogram. Baba Y. Accessed 15 Oct 2021.

[9] Gagne SM, Newbury A, Nowitzki KM, Chen BY, Lo HS. Name that nephrogram: asymmetric renal enhancement in the acute care setting. Curr Probl Diagn Radiol 2019;48(6):616–25. doi:10.1067/j.cpradiol.2018.10.004.

[10] Machida S, Imai N, Shibagaki Y. Persistent nephrogram. Intern Med 2016;55(24):3687–8. doi:10.2169/internalmedicine.55.7448.

[11] Saunders HS, Dyer RB, Shifrin RY, Scharling ES, Bechtold RE, Zagoria RJ. The CT nephrogram: implications for evaluation of urinary tract disease. Radiographics 1995;15(5):1069–85 discussion 1086-8. doi:10.1148/radiographics.15.5.7501851.

[12] Yuh BI, Cohan RH. Different phases of renal enhancement: role in detecting and characterizing renal masses during helical CT. AJR Am J Roentgenol 1999;173(3):747–55. doi:10.2214/ajr.173.3.10470916.

[13] Wolin EA, Hartman DS, Olson JR. Nephrographic and pyelographic analysis of CT urography: differential diagnosis. AJR Am J Roentgenol 2013;200(6):1197–203. doi:10.2214/AJR.12.9692.

[14] Moinuddin I, Bracamonte E, Thajudeen B, Sussman A, Madhrira M, Costello J. Allergic interstitial nephritis manifesting as a striated nephrogram. Case Rep Med 2015;2015:250530. doi:10.1155/2015/250530.

[15] Sato T, Kawasaki Y, Ito A, Izumi H, Kawamorita N, Yamashita S, et al. Infliximab-induced tubulointerstitial nephritis with image findings of striated nephrogram in Crohn's disease. Tohoku J Exp Med 2018;245(3):149–52. doi:10.1620/tjem.245.149.

[16] Striated nephrogram. Available at: https://radiopaedia.org/articles/striated-nephrogram. Bell DJ. Accessed 23 Sep 2022.

[17] Acute tubular necrosis. Available at: https://radiopaedia.org/articles/acute-tubular-necrosis. Weerakkody Y. Accessed 19 Nov 2021.

[18] Sagiv I, Koslowsky B, Korem M, Hiller N, Heyman SN. Scattered striated persistent nephrogram in sepsis. Nephrol Dial Transplant 2011;26(6):2053–5. doi:10.1093/ndt/gfr113.