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Firsekibart in the Treatment of Acute Gout Attack in a Patient with Chronic Kidney Disease and Progressive Renal Dysfunction: A Case Report

Jing Hu

Department of Nephrology, The Third Xiangya Hospital of Central South University, Changsha 410000, Hunan, China

Abstract: Currently, glucocorticoids are the only treatment option for acute gout attacks in patients with chronic kidney disease (CKD) and progressive renal dysfunction. Management becomes particularly challenging when steroid therapy is contraindicated, poorly tolerated, or ineffective. IL-1β monoclonal antibodies offer potent, targeted anti-inflammatory effects and have been approved for the treatment of gout, providing new therapeutic hope for this population. Here, we report a case of a patient with CKD and progressively worsening renal function who experienced an acute gout attack requiring dialysis. Treatment with the IL-1β monoclonal antibody Firsekibart produced rapid and remarkable anti-inflammatory and analgesic effects, effectively controlling the gout attack, improving renal function, and allowing discontinuation of dialysis.

Keywords: Gout; Inflammation; Acute kidney injury; Chronic kidney disease; Dialysis

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1. Background

Gout is an inflammatory arthritis caused by the deposition of monosodium urate crystals, with a prevalence of up to 3.2% in China [1]. The prevalence of gout is significantly higher in patients with chronic kidney disease (CKD) compared to those without CKD, and gout is recognized as a risk factor for the progression of CKD to end-stage renal disease. Studies have shown that individuals with an eGFR ≤ 60 mL/min/1.73 m^2 have a five-fold higher prevalence of gout than those without kidney disease. Among gout patients, over 70% have CKD stage 2 or higher, and approximately 24% have CKD stage 3 or higher [2]. Managing gout in CKD patients is highly challenging due to the lack of high-quality evidence and guideline-based recommendations. Considerable variability exists among rheumatologists, nephrologists, and other clinicians regarding drug selection and treatment efficacy for this population [3]. Gout attacks trigger systemic inflammatory responses, which markedly increase the risk of acute kidney injury (AKI) and rapid deterioration of renal function, further complicating treatment. Recent guidelines recommend IL-1 inhibitors, such as Firsekibart, to optimize anti-inflammatory

therapy in gout patients with CKD, potentially providing additional renal benefits [4]. Here, we report a case of a CKD patient with gout who experienced progressively worsening renal function requiring renal replacement therapy. Following targeted anti-inflammatory treatment, renal function improved, and dialysis was successfully discontinued. We summarize the clinical course and analyze the case in the context of current literature, highlighting the importance of long-acting, targeted anti-inflammatory therapy for CKD patients with gout.

2. Patient history

The patient was a 68-year-old man admitted on July 24, 2025, for progressively worsening renal function over one month. In early June 2025, he had been hospitalized for a cerebral infarction of the left basal ganglia extending to the periventricular region and received anti-coagulation and anti-platelet therapy for one week. At that time, renal abnormalities were noted, including a serum creatinine of 107 μmol/L and proteinuria (1+) on urinalysis. His renal function deteriorated rapidly, with creatinine rising to 189.9 μmol/L within one week and then to 309.9 μmol/L 20 days later, prompting readmission. During hospitalization, he developed fever, cough, and gross hematuria, accompanied by progressive anemia and worsening renal function, with creatinine peaking at 529 μmol/L. Despite anti-infective therapy, transfusions, and adjustment of anti-coagulation, hematuria improved but renal function remained impaired. His past medical history included over 10 years of poorly controlled hypertension, with a maximum blood pressure of 200/110 mmHg, gout for more than 10 years with attacks every 1–2 months typically self-managed with celecoxib or colchicine, and a history of pulmonary tuberculosis that had been treated. On admission, he appeared chronically ill and anemic, with left cardiac border enlargement, slightly reduced breath sounds, multiple white tophi on the right-hand finger joints, bilateral knee swelling with mild warmth, mild lower limb edema (**Figure 1**), and limited right-sided limb movement with decreased muscle tone and strength graded as one. Laboratory findings showed a white blood cell count (WBC) of 10.22×10^9/L, hemoglobin (Hb) of 66 g/L, platelets of 146×10^9/L, schistocytes 0.38% rechecked negative, proteinuria (2+), hematuria (2+), high-sensitivity C-reactive protein (hsCRP) 142.79 mg/L, erythrocyte sedimentation rate (ESR) 74 mm/h, ferritin 1328 ng/mL, procalcitonin (PCT) 0.405 ng/mL, urea 28.06 mmol/L, creatinine 476 μmol/L, uric acid 564 μmol/L, albumin 25.1 g/L, globulin 33.1 g/L, serum iron 4 μmol/L, and transferrin saturation 9.52%. Autoantibodies including antinuclear antibody (ANA), antineutrophil cytoplasmic antibody (ANCA), anti-glomerular basement membrane (anti-GBM) antibody, PLA2R antibody, anticardiolipin antibody, as well as IgG4, complement, Coombs test, ADAMTS13, and tumor markers, were all negative. Blood, urine, sputum, and fungal cultures were negative. TB-SPOT showed minimal reactivity, and sputum next-generation sequencing (NGS) detected *Enterococcus faecalis*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and *Candida albicans*. Renal ultrasound showed both kidneys with increased parenchymal echogenicity and normal blood flow. The left kidney measures 110 × 46 mm and the right kidney 113 × 44 mm, with multiple cysts in the left kidney. The bladder wall appears irregular. The prostate is enlarged with signs of prostatitis and multiple calcifications. Chest computed tomography (CT) revealed multiple polymorphic lesions suggestive of secondary pulmonary tuberculosis (TB) with fibrosis and calcifications, traction bronchiectasis, mild chronic inflammation, small bilateral pleural and pericardial effusions, and multiple rib fractures. Bone marrow biopsy demonstrated active trilineage hematopoiesis with normal morphology, scattered or locally increased plasma cells, 11% intracellular iron, and abundant extracellular iron.

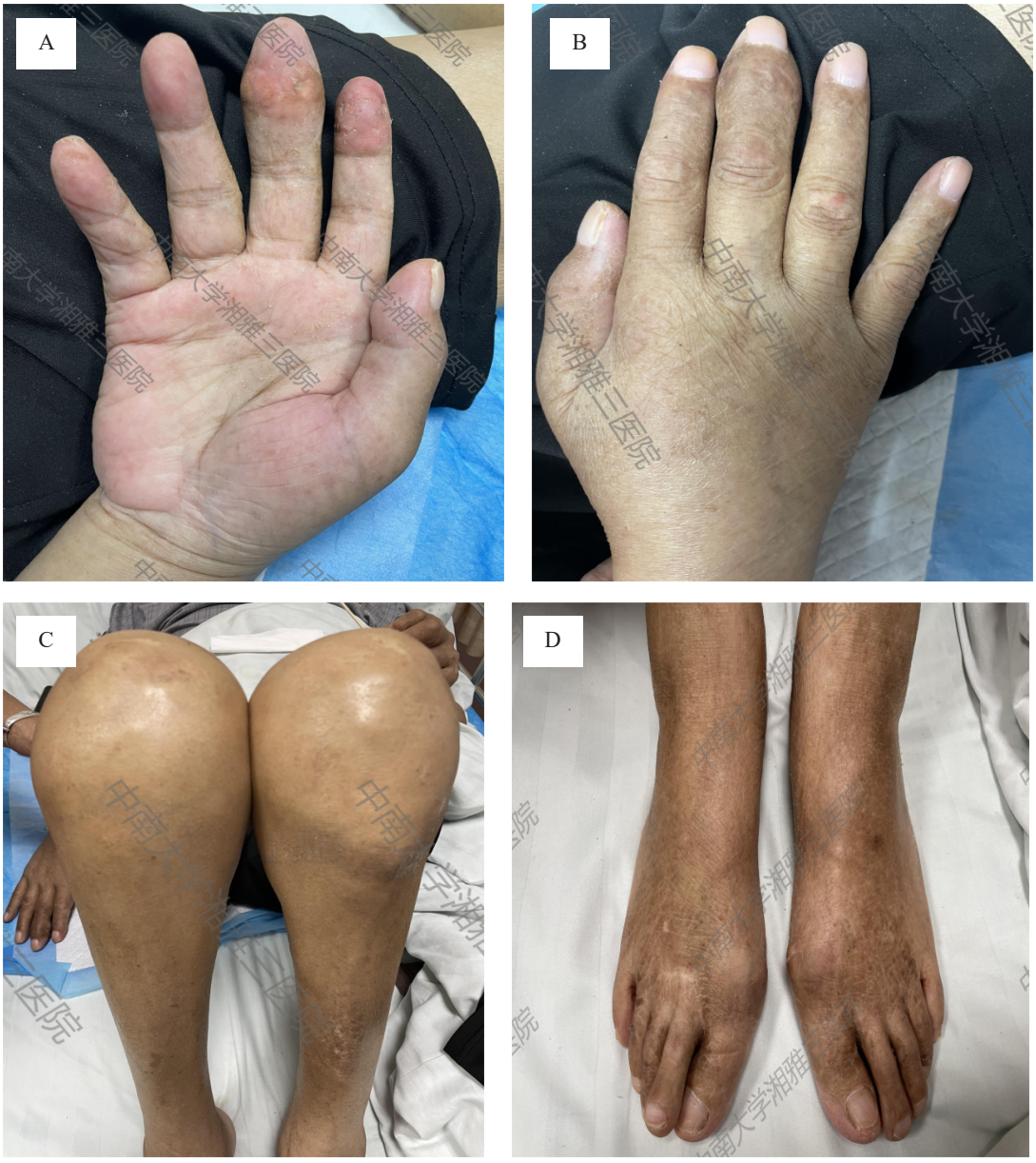

Figure 1. Multiple tophi on the right hand with mild redness before treatment (A), hand swelling (B), bilateral knee joint swelling with mildly increased skin temperature (C), and mild lower limb edema (D).

3. Treatment and outcome

The patient initially received broad-spectrum anti-infective therapy, including piperacillin-tazobactam, linezolid, caspofungin, sulfamethoxazole-trimethoprim, and meropenem, alongside acid suppression and gastric protection, rosuvastatin, concentrated red blood cell transfusions for anemia correction, and nutritional support. Despite these interventions, anemia and renal function showed minimal improvement, with hemoglobin nadir level of 55 g/L. The patient remained fatigued, experienced poor appetite, intermittent fevers, and maintained a daily urine output of 500–700 mL. On July 30, he developed redness, swelling, and pain in the right knee, with a visual analog scale (VAS) pain score of six, consistent with an acute gout attack. Intravenous methylprednisolone 40 mg daily was administered for seven days, resulting in partial relief of the gout symptoms. However,

renal function continued to decline, with decreasing urine output and a peak serum creatinine of 620 μmol/L. Hemodialysis was initiated on August 4, after which serum creatinine fluctuated between 300–400 μmol/L, and urine output remained 500–700 mL/day. Inflammatory markers, including hsCRP and ferritin, remained markedly elevated despite intensive anti-infective therapy, suggesting persistent systemic inflammation from recurrent gout attacks as a contributor to progressive renal dysfunction. Given the poor response and adverse effects from corticosteroids, methylprednisolone was discontinued. The patient was then treated with a 200 mg subcutaneous injection of Firsekibart, an IL-1β monoclonal antibody, for targeted anti-inflammatory therapy. Joint pain improved dramatically, with complete resolution of swelling and pain within three days (VAS score 0), and urine output subsequently increased to 800–900 mL/day. The patient was discharged on August 24, 2025, with serum urea of 22.42 mmol/L and creatinine of 339 μmol/L, and continued maintenance hemodialysis twice weekly after discharge.

Follow-up after discharge: Seven days after discharge, the patient's joint swelling and pain had completely resolved, and the edema in the lower limbs and knee swelling had completely alleviated (**Figure 2**). Repeat laboratory tests showed a urea level of 15 mmol/L, serum creatinine of 199 μmol/L, uric acid reduced to 346 μmol/L, and urine output increased to approximately 1,000 mL/day, allowing dialysis frequency to be reduced to once every five days. Two weeks after discharge, renal function improved further, with urea decreasing to 10.02 mmol/L and serum creatinine to 145 μmol/L, approaching baseline levels, and dialysis was subsequently discontinued.

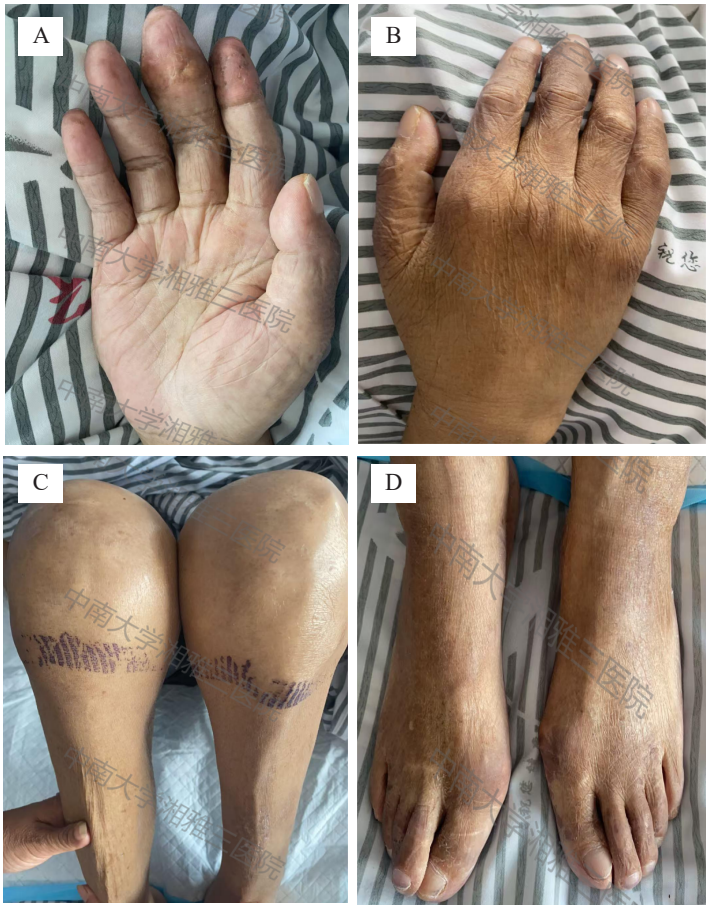

Figure 2. Multiple tophi on the right hand before treatment (A), resolution of hand swelling (B), disappearance of bilateral knee joint swelling (C), and resolution of mild lower limb edema (D) after treatment.

4. Discussion

Gout and CKD frequently coexist, posing a significant clinical management challenge [2]. Gout is not only a common comorbidity in CKD, but the chronic systemic inflammation it drives also represents a key risk factor for CKD progression to end-stage renal disease [2].

Research has shown that monosodium urate crystals activate the NLRP3 inflammasome, triggering massive release of pro-inflammatory cytokines such as IL-1β, which serves as the initiating and central mechanism in the inflammatory cascade of gout [5]. This process is not limited to the joints, as IL-1β-mediated chronic inflammation can result in multi-organ damage. In the kidneys, IL-1β promotes renal fibrosis and contributes to the development and progression of CKD via regulation of the NLRP3 signaling pathway. A multicenter retrospective study demonstrated that anakinra can be used safely and effectively to treat gout attacks in patients with stage 4–5 CKD or in those who have undergone renal transplantation [6]. Targeted inhibition of the IL-1β pathway not only effectively controls gout attacks but may also facilitate recovery of renal function. In the present case, the patient exhibited markedly elevated hsCRP, ESR, and ferritin, along with a poor response to anti-infective therapy. These findings suggest systemic inflammation driven by urate deposition and recurrent acute gout attacks accelerated renal function deterioration and reflected gout-related target organ damage. Following treatment with the IL-1β monoclonal antibody Firsekibart, the patient experienced rapid resolution of gouty arthritis symptoms, sustained improvement in inflammatory markers, and progressive recovery of renal function. Serum creatinine returned to baseline levels, and dialysis was completely discontinued. These outcomes indicate that long-acting, targeted IL-1β blockade with Firsekibart can effectively control gout attacks and promote recovery from AKI. Targeted inhibition of the IL-1β pathway may therefore represent a preferred therapeutic strategy for patients with CKD or AKI complicated by gout.

This patient had a high-risk CKD background, including long-standing uncontrolled hypertension, a well-documented history of gout, and the presence of tophi. His renal function began to deteriorate after the cerebral infarction and progressed rapidly, suggesting an acute exacerbation superimposed on underlying CKD. A comprehensive diagnostic evaluation showed insufficient evidence for alternative etiologies such as vasculitis, glomerulonephritis, thrombotic microangiopathy, active infection, or malignancy. However, persistently elevated hsCRP, ESR, and ferritin levels indicated a sustained systemic inflammatory state accompanied by recurrent acute gout attacks. It was therefore plausible that gout-related systemic inflammation, compounded by the acute stress from the cerebral infarction, was the primary driver of renal function deterioration. As a major physiological stressor, the cerebral infarction may have amplified pre-existing chronic inflammation driven by monosodium urate crystals through neuroendocrine responses and cytokine release. Excessive production of inflammatory mediators, including IL-1β, IL-6, and tumor necrosis factor-α (TNF-α), can induce renal microvascular constriction, endothelial injury, promote tubulointerstitial fibrosis, and directly inhibit tubular epithelial cell regeneration. These processes can precipitate AKI on a background of CKD, leading to rapid and severe renal function decline.

Intensive anti-inflammatory therapy was critical to improving this patient's prognosis. However, nonsteroidal anti-inflammatory drugs (NSAIDs) are contraindicated or require extreme caution in CKD because of their nephrotoxic potential. Colchicine requires substantial dose reduction in renal impairment and carries risks of myotoxicity. Although corticosteroids were administered early in this case, their therapeutic effect was limited and they posed additional risks, including worsening hypertension, fluid and sodium retention, and increased susceptibility to infection. Despite intensive anti-infective therapy, supportive care, and corticosteroid treatment,

inflammatory markers and renal function failed to improve, ultimately necessitating dialysis. Given the markedly elevated hsCRP, a key downstream effector of the IL-1β pathway, and the central role of IL-1β in gout-related inflammation, treatment with the IL-1β monoclonal antibody Firsekibart was initiated. Following treatment, the patient experienced rapid resolution of gout symptoms, increased urine output, and gradual recovery of renal function, ultimately allowing successful discontinuation of dialysis. These findings suggest that targeted blockade of the IL-1 signaling pathway can effectively suppress the inflammatory cascade driving renal deterioration and create a critical window for renal recovery.

Firsekibart is a fully humanized IL-1β monoclonal antibody that binds and neutralizes IL-1β, thereby inhibiting the inflammatory cascade at its source. This mechanism differs fundamentally from traditional therapies that target downstream inflammatory mediators. Phase III clinical trials have demonstrated that a single dose produces rapid therapeutic effects and reduces the risk of gout safety recurrence over six months by nearly 90% [7]. Moreover, Firsekibart has demonstrated a favorable safety profile in patients with gouty arthritis and CKD stage three or higher. The most recent gout anti-inflammatory treatment guidelines recommend IL-1 inhibitors such as Firsekibart for patients across CKD stages G1 through G5, providing new therapeutic options and guidance for the management of gout in patients with CKD or AKI [8].

To date, no published reports have described recovery of renal function following Firsekibart treatment in patients with gout and CKD experiencing acute renal deterioration. This case highlights that in patients with gout complicated by CKD and cardiovascular or cerebrovascular disease, multiple comorbidities interact with recurrent gout attacks to create a persistent high-inflammatory state that increases treatment complexity and the risk of organ damage. Long-acting, targeted IL-1β blockade with Firsekibart not only effectively controlled the gout attack and improved renal function but also suggests that incorporating IL-1β monoclonal antibody therapy into long-term disease management may help sustain suppression of chronic inflammation. This approach may contribute to long-term stabilization of CKD and prevention of cardiovascular and other related complications.

5. Conclusion

This case report describes the first documented use of Firsekibart, an IL-1β monoclonal antibody, in a patient with CKD and progressive renal dysfunction experiencing an acute gout attack. Following treatment, the patient achieved rapid and complete resolution of gout symptoms, sustained improvement in inflammatory markers, and progressive recovery of renal function, ultimately allowing discontinuation of dialysis. These findings suggest that targeted IL-1β blockade with Firsekibart may represent a valuable therapeutic option for managing acute gout in patients with CKD, particularly when conventional therapies are contraindicated or ineffective. The dual benefits of controlling gout-related inflammation and facilitating renal recovery warrant further investigation in larger clinical studies to establish the role of IL-1β inhibitors in this challenging patient population.

Disclosure statement

The author declares no conflict of interest.

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