The Impact of Ibuprofen on Acute Kidney Injury in Brucellosis: A Case Report

Klejda ÇOLLAKU
Regional Hospital of Peshkopi, Albania
Corresponding Author klejda.collaku@yahoo.com

Neada HOXHA
Regional Hospital of Peshkopi, Albania

Armanda SARACI
Hospital of Kruja, Albania

Olta ZENELI
Regional Hospital of Peshkopi, Albania

Abstract

Purpose: To report a case of acute kidney injury (AKI) in a patient with brucellosis, highlighting the role of nephrotoxic medications, particularly ibuprofen, in its development. Brucellosis is a systemic zoonotic infection with diverse clinical manifestations, while renal involvement remains an uncommon but recognized complication [1–3,6].

Methodology: A 65-year-old male with poorly controlled type 2 diabetes mellitus presented with fever, anemia, hepatosplenomegaly, and bilateral pleural and pericardial effusions, consistent with severe systemic brucellosis [1,3]. Serological testing confirmed the diagnosis (Wright agglutination test 1:320, positive). The patient was treated with rifampicin and doxycycline, in accordance with recommended

therapy for brucellosis [2,5], along with supportive medications including ibuprofen, spironolactone, and furosemide. Clinical course, laboratory findings, and therapeutic adjustments were carefully reviewed.

Findings: After two weeks of therapy, the patient developed AKI (urea 159 mg/dL, creatinine 3.2 mg/dL, potassium 5.5 mmol/L) with preserved urine output. Nephrotoxic agents, including NSAIDs and spironolactone, were discontinued, while anti-brucellosis therapy was maintained. Supportive management, including hydration and electrolyte correction, resulted in complete renal recovery within two weeks (urea 54 mg/dL, creatinine 1.22 mg/dL, potassium 4.5 mmol/L). No recurrence of effusions or hepatosplenomegaly was observed. These findings are consistent with previously reported cases of reversible renal involvement in brucellosis [4,6].

Conclusion: Acute kidney injury in brucellosis is rare but possible [3,6]. Its development may be influenced by a combination of infectious factors, drug-induced nephrotoxicity, and patient-related risk factors such as diabetes and hypertension, which can increase renal vulnerability [4,6].

Early recognition of AKI is essential for favorable outcomes. Careful monitoring of renal function and cautious use of potentially nephrotoxic agents, including NSAIDs or aminoglycosides, can help prevent permanent kidney damage.

Originality: This case highlights the rarely reported contribution of ibuprofen to AKI in the setting of brucellosis, underscoring the need for vigilance when prescribing potentially nephrotoxic medications in infected patients.

***Keywords**: Brucellosis; Acute kidney injury; Ibuprofen; NSAIDs; Drug-induced nephrotoxicity; Case report*

Introduction

Brucellosis is a common zoonotic infection with systemic involvement, particularly affecting the reticuloendothelial, musculoskeletal, and hepatobiliary systems [1–3]. Renal complications are relatively uncommon, reported in approximately 2–20% of cases, and most frequently manifest as membranoproliferative glomerulonephritis, tubulointerstitial nephritis, or IgA nephropathy [3,6]. Rapidly progressive glomerulonephritis (RPGN) associated with brucellosis is exceedingly rare and has been described only in isolated case reports [6].

The pathogenesis of renal involvement in brucellosis is believed to be primarily immune-mediated, with immune complex deposition playing a central role [3,6]. In addition, acute kidney injury (AKI) in patients with brucellosis may be exacerbated by contributing factors such as dehydration, systemic inflammation, hemodynamic instability, and exposure to nephrotoxic medications, including non-steroidal anti-inflammatory drugs (NSAIDs) [4,6].

This case highlights the multifactorial etiology of AKI in a patient with brucellosis, with particular emphasis on the contributory role of ibuprofen in precipitating renal injury.

Case Presentation

Patient

A 65-year-old male with a history of poorly controlled type 2 diabetes mellitus, arterial hypertension, anemia, gastritis, peptic esophagitis, sigmoid diverticulitis, non-proliferative diabetic retinopathy, and severe COPD (GOLD stage IV) presented with fever, fatigue, anorexia, unintentional weight loss of 12 kg in one month, and night sweats. Imaging revealed bilateral pleural effusion and minimal pericardial effusion.

Diagnosis

Brucellosis confirmed by serology (*Brucella abortus* / Wright agglutination test 1:320, positive).

Hospitalizations

- First admission (30 June – 16 July 2025): Brucellosis diagnosis confirmed.
- Second admission (16 July – 1 August 2025): Transferred to Infectious Diseases Department.
- Third admission (8 – 15 August 2025): Worsening renal function/AKI.

Initial therapy

- Rifampicin 600 mg/day, Doxycycline 200 mg/day
- Gentamicin 240 mg/day during initial hospitalization
- Supportive medications: Ibuprofen 600 mg/day, Spironolactone 25 mg/day, Furosemide 40 mg/day
- Antidiabetic therapy: Oral agents (Siofor 850 mg, Maninil 5 mg), later switched to insulin therapy

TABELE 1. Medications Administered During Hospitalizations and Nephrotoxicity Risk

Drug	Dose	Duration	Indication	Nephrotoxicity Risk
Rifampicin	600 mg/day	16.07 – ongoing	Standard anti-brucellosis therapy	Low
Doxycycline	200 mg/day	16.07 – ongoing	Standard anti-brucellosis therapy	Low
Gentamicin	240 mg/day	16.07 – 01.08	Short course Anti-brucellosis adjunct	High
Spironolactone	25 mg/day	17.07 – 08.08	Chronic antihypertensive	Moderate
Furosemide	40 mg/day	16.07 – 08.08	Chronic antihypertensive	Moderate
Ibuprofen	400 mg ×3/day	25.07 – 08.08	Added for inflammatory control	High
Colchicine	1 mg/day	25.07 – 08.08	Added for pericardial involvement	Low

High risk / nephrotoxic: Medications with a strong potential to cause kidney injury.
Moderate risk / potential hemodynamic impact: Medications that may affect renal function indirectly through hemodynamic changes or mild nephrotoxicity.
Low risk / considered safe: Medications generally considered safe for renal function under monitored conditions.

Results

FIGURE 1. Serum Creatinine and Urea Trend (July – October 2025)

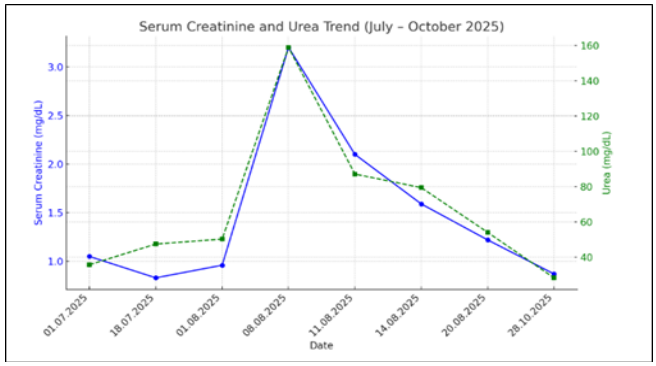


TABLE 2. Laboratory changes

Date	Creatinine (mg/dL)	Urea (mg/dL)
01.07.2025	1.05	35.6
18.07.2025	0.83	47.4
01.08.2025	0.96	50.2
08.08.2025	3.20	159.0
11.08.2025	2.10	87.1
14.08.2025	1.59	79.4
20.08.2025	1.22	54.0
28.10.2025	0.87	28.2

Evolution of Renal Function and Therapeutic Adjustments

Acute Kidney Injury (AKI)

After two weeks of therapy, the patient developed AKI with preserved urine output. Laboratory values at onset: urea 159 mg/dL, creatinine 3.2 mg/dL, potassium 5.5 mmol/L.

Therapeutic Adjustments

- NSAIDs (ibuprofen), spironolactone, and colchicine were discontinued due to nephrotoxic and hemodynamic effects.
- Anti-brucellosis therapy (rifampicin + doxycycline) was continued; gentamicin was administered only during the initial hospitalization.
- Supportive care included hydration, electrolyte correction, monitoring of urine output, renal function and hemodynamics, and nutritional support.

Renal Function Recovery

Gradual improvement was observed following discontinuation of nephrotoxic drugs. Within two weeks, renal function normalized (urea 54 mg/dL, creatinine 1.22 mg/dL, potassium 4.5 mmol/L). No recurrence of pleural or pericardial effusions; hepatosplenomegaly resolved.

Interpretation

Findings confirm multifactorial AKI due to infection, nephrotoxic medications, and possible hemodynamic factors. Early recognition and intervention led to complete renal recovery.

Discussion

AKI in this patient was multifactorial, resulting from:

1. Inflammatory effects of brucellosis [1–3,6]
2. Nephrotoxic effects of ibuprofen (NSAIDs) [4,6]
3. Potential hemodynamic factors (diuretics, spironolactone) [4]

Early recognition and discontinuation of nephrotoxic agents, along with supportive therapy led to complete renal recovery [4,6].

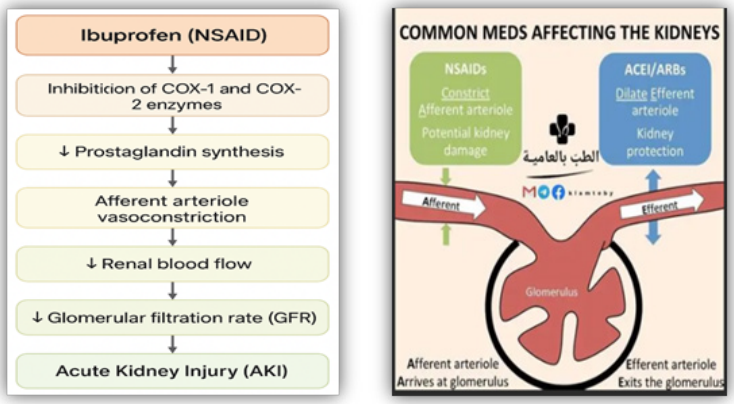
This case highlights the importance of monitoring renal function in brucellosis patients receiving potentially nephrotoxic drugs [3,6].

Mechanism of Ibuprofen-Induced AKI

Ibuprofen, a nonsteroidal anti-inflammatory drug (NSAID), inhibits COX-1 and COX-2 enzymes, leading to decreased prostaglandin synthesis [4].

- Role of prostaglandins: Normally, prostaglandins mediate vasodilation of the afferent arteriole, maintaining adequate renal blood flow and glomerular filtration rate (GFR) [4].
- Effect of NSAID use: Reduced prostaglandin levels cause afferent arteriole vasoconstriction, resulting in decreased renal perfusion and reduced GFR, which can precipitate acute kidney injury (AKI) [4,6].

FIGURE 2. Mechanism of Ibuprofen-Induced Acute Kidney Injury



Risk factors for NSAID-induced AKI include:

- Volume depletion / dehydration [4,6]
- Congestive heart failure [4]
- Liver cirrhosis [4]
- Pre-existing kidney disease [4,6]

In this patient, *the combination of ibuprofen therapy, brucellosis-related inflammatory effects, and underlying comorbidities likely contributed to the multifactorial AKI observed. [3,4,6].* Early discontinuation of ibuprofen and supportive care facilitated complete renal recovery [4,6].

Conclusion

Acute kidney injury in brucellosis is rare but possible. Its development may be influenced by a combination of infectious factors, drug-induced nephrotoxicity, and patient-related risk factors such as diabetes and hypertension, which can increase renal vulnerability. Early recognition of AKI is essential for favorable outcomes. Careful monitoring of renal function and cautious use of potentially nephrotoxic agents, including NSAIDs or aminoglycosides, can help prevent permanent kidney damage. This highlights the importance of assessing renal risk factors during brucellosis treatment.

Research Limitations

This is a single-case report; therefore, findings are not generalizable. However, it provides insight into the contributory role of nephrotoxic drugs in brucellosis-associated AKI.

Practical Implications

Early recognition of renal dysfunction and prompt discontinuation of nephrotoxic agents are essential to prevent permanent kidney damage, particularly in patients with systemic infections [4,6].

Social Implications

Increased awareness of drug-induced renal injury may improve patient safety and reduce healthcare burden.

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