CLINICAL MEDICINE

# Unani Medicine Management of Acute Kidney Injury with Anuria, Generalized Oedema, Severe Abdominal Pain and High-Grade Fever: A Case Report

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Published November 2026

**Abstract**

A case report highlighting an acute kidney function disorder associated with oliguria and anuria, generalized oedema, and markedly elevated serum creatinine, requiring urgent treatment and close clinical monitoring; considered an AKI syndrome. This case report describes a 60-year-old man who initially developed fever with chills and rigors, with abdominal pain. Initial laboratory evaluation showed markedly elevated serum AST/SGOT (940 U/L) and ALT/SGPT (508 U/L). Subsequently, he developed generalized oedema, markedly elevated serum creatinine of approximately 10.07–11.74 mg/dL and serum urea of approximately 112.8–118.27 mg/dL. Hemodialysis was advised, but the patient and his family refused. The patient received Unani medicines: sharbat Bazuri 20 mL, Arq Mako 30 mL, and Arq Kasni 30 mL. The patient was secured from hemodialysis because serum creatinine, blood urea, and other biochemical tests returned to normal within days.

**Keywords:** acute kidney injury; anuria; case report; Su-i-Mizaj Hār Kulya; Unani medicine

## 1 Background

AKI is not a disease, but rather a clinical syndrome with multiple aetiologies. An abrupt decline in glomerular filtration rate (GFR) impairs excretion of nitrogenous waste products and disrupts fluid volume and electrolyte homeostasis. According to the standardized Kidney Disease: Improving Global Outcomes (KDIGO) criteria, progression to Stage 3 AKI is defined by a threefold or greater rise in serum creatinine from baseline, an absolute serum creatinine level exceeding 4.0 mg/dL, or anuria lasting more than 12 hours [1]. Severe Acute Kidney Injury signs may require renal replacement therapy [2].

AKI occurs in about 10–15% of hospitalized patients. Kidney dysfunction or damage can occur over a longer period or follow AKI in a continuum with acute and chronic kidney disease [2].

Acute kidney injury, or *Su-i-Mizaj Hār* with *Tafarrūq al-Ittiṣāl,* is characterized by a sudden deterioration in renal function with reduced urine output (Ihtibās al-Bawl), Al-Taftīr (presence of interval in fever), headache, insomnia, and oedema [3].

Ibn Sina stated that acute nephritis and nephrotic injury present differently, depending on which kidney cells are injured; they may affect other organs and cause delirium, or lead to scarring of the kidney within a short time [4].

The Unani system of medicine uses compound formulations to manage acute inflammatory conditions of the viscera. Sharbat Bazuri, Arq Mako, and Arq Kasni are reported to be effective in acute inflammatory conditions of the kidney, urinary tract, liver, and other internal organs [5]. They help soothe the heat of inflammation and facilitate the power of exertion (Quwwat Dafiya) [6]. As *Istīfrāgh* (evacuation of waste) is essential, the kidneys and urinary tract are the gateway of evacuation through diuresis [7].

## 2 Case presentation

### 2.1 Patient information

A 60-year-old male had previously been diagnosed with malaria and had received treatment. Approximately one month later, he relapsed with fever, chills, and rigors, accompanied by severe abdominal pain and headache.

The patient was subsequently admitted through the emergency department with these complaints, along with complete cessation of urine output. His condition was complicated by lower body oedema, especially in both lower limbs, ankles, and feet. The oedema was described as pitting. Later facial swelling also developed. The patient has experienced breathlessness but remains stable.

### 2.2 Initial laboratory investigations

**Table 1.** Laboratory tests done on 5 August and 9 August 2026.

| Investigation (5 August 2026) | Findings | Reference range |
|---|---|---|
| Haemoglobin | 15.8 g/dL | 11.5%–14.5% |
| Red cell distribution width | 14.1% | 11.5%–14.5% |
| Total leukocyte count | 6,910 cells/mm$^3$ | 4,000–11,000/mm$^3$ |
| Platelet count | 135,000/µL | 150,000–450,000/µL |
| HbA1c | 5.00% | <5.7% |
| Mean blood glucose | 96.80 mg/dL | <100 mg/dL |
| Malaria parasite smear | Not seen | - |
| AST/SGOT | 940 U/L | 5–40 U/L |
| ALT/SGPT | 508 U/L | 7–56 U/L |
| Alkaline phosphatase | 125 U/L | 44–147 U/L |
| Investigation (9 August 2026) | Findings | Reference range |
| Serum creatinine | 10.07 mg/dL | 0.70–1.30 mg/dL |
| Serum urea | 114.27 mg/dL | 15–45 mg/dL |
| Blood urea nitrogen | 53.40 mg/dL | 7–20 mg/dL |
| Serum sodium | 138.0 mmol/L | 135–145 mmol/L |
| Serum potassium | 4.22 mmol/L | 3.5–5.1 mmol/L |
| Serum chloride | 102.0 mEq/L | 96–106 mEq/L |

### 2.3 Diagnosis of acute kidney injury

AST and ALT were markedly elevated; the patient subsequently developed severe renal dysfunction. Severe azotaemia was demonstrated. The report findings (Table 1) showed serum creatinine of 10.07 mg/dL and serum urea of 114.27 mg/dL, both markedly high.

The patient remained anuric for approximately two days. Catheterization initially yielded no appreciable urine output. With persistent anuria, markedly elevated serum creatinine and urea, haemodialysis was advised. The patient and his family were apprehensive about dialysis and further complications and refused the procedure. The patient's relatives subsequently presented his medical records to the Colocation Centre of RRIUM at J.J. Hospital for further advice.

### 2.4 Unani intervention

Following clinical assessment, the patient was managed with the Unani medicines listed in Table 2 under supervision [5, 8–10].

**Table 2.** Unani treatment regimen.

| No. | Formulation | Botanical/component source | Dosage | Frequency | Mode of administration |
|---|---|---|---|---|---|
| 1 | Sharbat Bazūrī | Compound pharmacopoeial syrup | 20 mL | Three times daily | Oral |
| 2 | ʻArq Mākō | Distillate of Solanum nigrum L. | 30 mL | Three times daily | Oral |
| 3 | ʻArq Kasnī | Distillate of Cichorium intybus L. | 30 mL | Three times daily | Oral |

**Table 3.** Monitoring of urine output after the Unani intervention.

| Investigation | Result | Date | Reference range |
|---|---|---|---|
| 24-hour urine volume | 3,000 mL | 11 August 2026 | 800–2,000 mL/day |
| 24-hour urinary protein | 990 mg | 11 August 2026 | <150 mg/24 hours |
| Spot urine protein | 33 mg/dL | 11 August 2026 | Negative |
| Estimated GFR | 37.73 mL/min/1.73 $m^2$ | 17 August 2026 | >90 mL/min/1.73 $m^2$ |
| Blood urea nitrogen | 14.8 mg/dL | 17 August 2026 | 7–20 mg/dL |

**Table 4.** Selected renal biochemical investigations during follow-up.

| Parameter | 11 August 2026 | 12 August 2026 | 17 August 2026 | 31 August 2026 |
|---|---|---|---|---|
| Serum creatinine | 11.69 mg/dL | 11.74 mg/dL | 1.99 mg/dL | 1.10 mg/dL |
| Serum urea | 112.80 mg/dL | 118.27 mg/dL | 31.75 mg/dL | 16.26 mg/dL |
| Serum uric acid | 11.60 mg/dL | 12.80 mg/dL | 9.00 mg/dL | 4.75 mg/dL |
| Serum calcium | 7.60 mg/dL | 7.80 mg/dL | 8.10 mg/dL | 8.80 mg/dL |
| Serum phosphorus | 5.70 mg/dL | 6.50 mg/dL | 3.80 mg/dL | 2.25 mg/dL |
| Serum sodium | 138 mmol/L | 144 mmol/L | 141 mmol/L | 139 mmol/L |
| Serum chloride | 106 mmol/L | 103 mmol/L | 99 mmol/L | 107 mmol/L |

**Table 5.** Immunological and endocrine investigations.

| Investigation | Result | Date | Reference range |
|---|---|---|---|
| Anti-dsDNA antibody | 19.316 IU/mL; reported as negative | 12 August 2026 | Negative |
| Complement C3/C4 | 108.8/24.9 mg/dL | 14 August 2026 | 90–180/10–40 mg/dL |
| Intact parathyroid hormone | 235.50 pg/mL | 12 August 2026 | 15–65 pg/mL |

### 2.5 Clinical outcomes

The patient was clinically monitored, including assessment of general condition, urine output, oedema, blood pressure, and breathlessness. During follow-up, repeat renal function tests, liver function tests, complete blood count, urine investigations, serum electrolyte measurements, and immunological and parathyroid hormone tests were performed.

Urine output resumed the morning after starting the Unani regimen. The initial recorded urine output was approximately 200 mL in the morning and 500 mL in the evening. Over the next 24 hours, total urine output was approximately 3,000 mL (Table 3).

The patient's generalized oedema, including facial, severe ankle, and pedal, gradually subsided. His general condition improved. The patient continued to pass urine under supervision.

### 2.6 Follow-up laboratory findings

The follow-up renal biochemistry is given in Table 4, Figure 1 and Figure 2. Serum creatinine remained markedly elevated during the early phase of treatment, with values of 11.69 mg/dL on 11 August and 11.74 mg/dL on 12 August 2026. It subsequently declined to 1.99 mg/dL by 17 August 2026 and to 1.10 mg/dL by 31 August 2026. Serum urea declined from 112.8–118.27 mg/dL during the acute phase to 31.75 mg/dL on 17 August and 16.26 mg/dL by 31 August 2026. Serum uric acid decreased from 11.6–12.8 mg/dL to 9.0 mg/dL, then to 4.75 mg/dL.

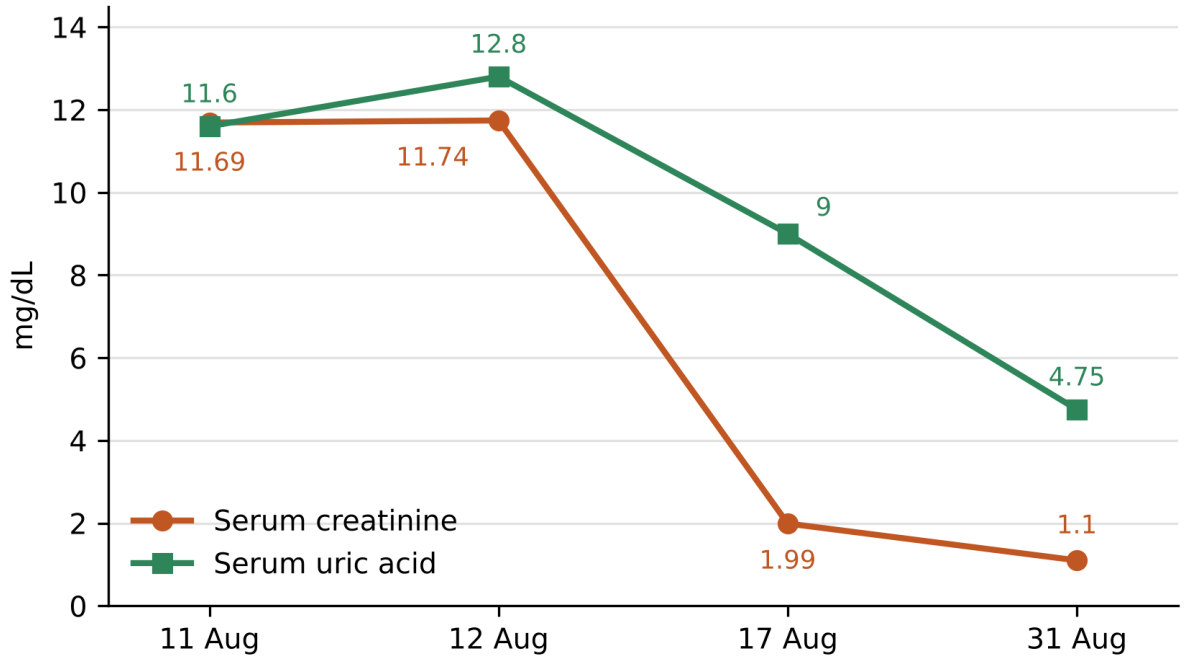



**Figure 1.** Serum creatinine and serum uric acid during follow-up (11–31 August 2026).

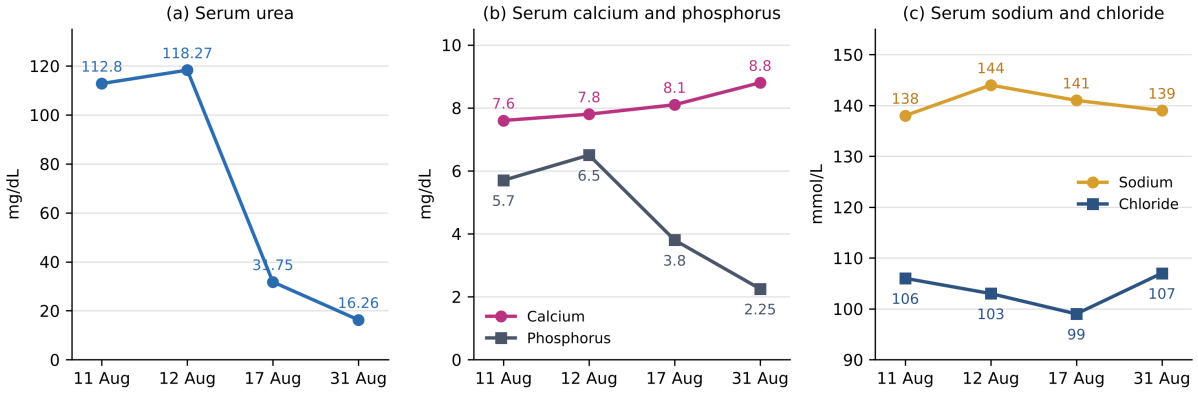



**Figure 2.** Serum urea (a), calcium and phosphorus (b), and sodium and chloride (c) during follow-up (11–31 August 2026).

The estimated glomerular filtration rate was reported as 37.73 mL/min/1.73 $m^2$ on 17 August 2026. Blood urea nitrogen was 14.8 mg/dL, with a BUN-to-creatinine ratio of 7.46 and a urea-to-creatinine ratio of 15.95. Other investigations included negative anti-double-stranded DNA antibody testing, normal C3 and C4, and an elevated intact parathyroid hormone level of 235.50 pg/mL (Table 5).

## 3 Discussion

This case describes a patient who developed severe acute renal dysfunction characterized by acute anuria, generalized edema, marked elevation of serum creatinine 10.07 mg/dL, urea 114.27 mg/dL and blood urea nitrogen, and was diagnosed with acute kidney injury. According to classical Unani philosophy, past history is important; the patient had a history of malaria and subsequent treatment highlighted the possibility of AKI. Potential considerations include infection-associated kidney injury and drug-associated organ injury; accordingly, the line of treatment included Sharbat Bazūri, a moderate-type diuretic [5], Arq Mako, and Arq Kasni, given orally for 21 days. All these formulations had already undergone physicochemical standardization;[10] Arq Mako and Arq Kasni were also standardized for quality control [8]. Urine output and renal biochemicals returned to normal; clinical features fully resolved. The elevated intact parathyroid hormone level may reflect mineral metabolism disturbances during acute or chronic renal dysfunction [1]. [2] The reported 24-hour urinary protein excretion was 990 mg, and spot urine protein was 33 mg/dL, indicating increased protein excretion. Constant monitoring and laboratory testing were performed from 11 August to 17 August, and C3 and C4 immunological tests were within the normal range. Intact parathyroid hormone was 235.50 pg/mL, above the reference range, and eGFR was 37.73 mL/min/1.73 $m^2$; the change started on 14 August. The tests done on 31 August was almost within the normal range, showing a significant response and recovery, suggesting that Sharbat Bazūri (a moderate-type diuretic), Arq Mako, and Arq Kasni, which are anti-inflammatory and help evacuate urine along with waste products, restored normal kidney function and played a significant role.

## 4 Conclusion

This is a single case study; although it responded magnificently, it cannot demonstrate efficacy or effectiveness. The findings suggest that hypothesis-generating data should not be interpreted as evidence that the Unani regimen can replace dialysis or standard emergency management of acute kidney injury. This study describes the resolution of a highly challenging condition and a critical hemodialysis case, a major achievement that prompted the researcher to recruit a large sample of

such emergency cases to generalize the use of Unani Medicine to conserve kidney function and manage the syndrome.

## Declarations

**Ethical considerations:**The authors followed ethical guidelines for recording and analyzing the intervention case.

**Consent for publication:** Before starting the intervention, written consent was obtained, and for the case report publication, consent was also obtained from the patient's relatives.

**Conflict of interest:**The authors have no conflicts of interest regarding the data and literature.

**Funding:**The authors acknowledge RRIUM and CCRUM for financial and infrastructure support.

**Authors' contributions:** Humaira Bano diagnosed the condition, conceptualized the study and prepared the manuscript. Ziaur Rahman added literature. Masroor Ali Qureshi served as the study observer and coordinator.

**Data availability:** As per the policy of the journal.

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