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Case Report

Pharmacology Section

Hyperuricaemia Associated with High-dose Furosemide in a Patient with Anterior Wall Myocardial Infarction and Multifactorial Acute Kidney Injury: A Case Report

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ABSTRACT

Hyperuricaemia, defined as a serum uric acid level >6 mg/dL in women and >7 mg/dL in men, is a well-recognised metabolic complication of loop diuretic therapy. Furosemide can cause hyperuricaemia through reduced renal uric acid excretion. We report the case of a 57-year-old man with type 2 diabetes mellitus who presented with acute decompensated heart failure secondary to an anterior wall myocardial infarction. He received an initial intravenous bolus of furosemide (20 mg), followed by a continuous infusion at 20 mg/hour. By Day 4, serum uric acid rose to 12.2 mg/dL, and renal function deteriorated with an estimated Glomerular Filtration Rate (eGFR) decline to 33 mL/min/1.73 m², consistent with severe hyperuricaemia occurring during high dose furosemide therapy and concurrent acute kidney injury. While asymptomatic hyperuricaemia is often managed with lifestyle modifications, xanthine oxidase inhibitors remain first-line therapy for high-risk patients. This case emphasises the importance of recognising and carefully monitoring hyperuricaemia and renal dysfunction during prolonged, high-dose furosemide therapy, especially in patients with severe cardiovascular conditions.

Keywords: Acute kidney injury, Furosemide, Heart Failure, Hyperuricaemia, Loop diuretics, Myocardial infarction

CASE REPORT

A 57-year-old man with type 2 diabetes mellitus (HbA1c 8.4%) presented with two days of chest discomfort and left shoulder pain. He was tachycardic (heart rate 100 beats/min) with Blood Pressure (BP) 110/70 mmHg and bilateral pulmonary crepitations. Echocardiography showed Left Ventricular (LV) dysfunction (EF 30-40%) with Left Anterior Descending artery (LAD)-territory wall motion abnormalities, and chest X-ray revealed bilateral interstitial opacities. He was diagnosed with an anterior wall myocardial infarction with acute pulmonary oedema. Admission labs showed normal renal function (creatinine 0.8 mg/dL; estimated Glomerular Filtration Rate (eGFR) >60 mL/min/1.73 m²; blood urea 28 mg/dL). Baseline serum uric acid was not measured; the first recorded value was 12.2 mg/dL on Day 4, coinciding with renal deterioration. Treatment included aspirin 75 mg, ticagrelor 90 mg, rosuvastatin 20 mg, and intravenous furosemide (20 mg bolus followed by continuous infusion at 20 mg/hour, totaling 500 mg/day). Continuous infusion was preferred over intermittent bolusing to maintain sustained tubular drug concentrations and allow precise titration in the setting of haemodynamic instability.

Clinical and radiological features suggested aspiration pneumonia: bilateral crepitations, leukocytosis (WBC 12.65 × 10^3/µL), elevated procalcitonin (2.4 ng/mL), and bilateral infiltrates in the setting of reduced consciousness during acute MI. Empiric therapy with piperacillin-tazobactam 2.25 g three times daily (gram-negative and anaerobic coverage) and linezolid 600 mg twice daily (gram-positive coverage given poorly controlled diabetes, ICU-level care, and haemodynamic instability) was initiated. Endotracheal aspirate confirmed Klebsiella pneumoniae, and antibiotics were rationalised per sensitivity results.

Furosemide-Induced Hyperuricaemia and Metabolic Complications

On Day 4, when the patient was tested for uric acid levels, laboratory evaluation revealed marked hyperuricaemia (serum uric acid 12.2 mg/dL) with evolving acute kidney injury, reflected by rising creatinine from 0.8 mg/dL at baseline to 2.1 mg/dL (KDIGO Stage 2 Acute Kidney Injury (AKI), representing a 2.6-fold increase) and a decline in eGFR to ~33 mL/min/1.73 m². Although asymptomatic for gout, urate-lowering therapy with allopurinol 100 mg twice daily was initiated on Day 4 due to severe hyperuricaemia, ongoing high-dose loop diuretic exposure, concurrent AKI, and high cardiovascular risk. Close biochemical monitoring continued alongside essential diuretic therapy. Reduced glomerular filtration and furosemide-mediated impairment of uric acid excretion were considered contributory. Additional metabolic complications included severe hypokalemia (nadir 2.6 mmol/L on Day 14) requiring oral potassium chloride supplementation and intravenous potassium correction, transient hyperkalemia (6.5 mmol/L on Day 5), consistent with evolving rather than peak AKI (creatinine 2.4 mg/dL on Day 5, with peak creatinine 2.7 mg/dL on Day 9), all managed conservatively with close biochemical surveillance [Table/Fig-1].

Hospital Day	Creatinine (mg/dL)	eGFR (mL/min/1.73 m²)	Uric Acid (mg/dL)	Blood Urea (mg/dL)	Potassium (mmol/L)	Sodium (mmol/L)
1	0.8	>60	Not done	28	3.8	140
4	2.1	33	12.2	99	4.1	132
5	2.4	28	-	131	6.5	130
6	2.3	29	-	152	3.1	-
7	2.3	29	-	162	3.0	127
8	2.4	24	-	167	3.2	129
9	2.7	24	-	164	3.5	131
14	2.2	31	4.9	88	2.5	135

[Table/Fig-1]: Temporal Trends in Renal Function, Serum Uric Acid, and Electrolytes.

Volume status was assessed clinically (bilateral crepitations, peripheral oedema, jugular venous pressure) and biochemically via serial pro-BNP measurements (7,300 pg/mL on Day 3,

rising to 10,600 pg/mL by Day 14), confirming persistent volume overload necessitating continued diuretic therapy. Despite severe hyperuricaemia and AKI, furosemide could not be discontinued due to persistent life-threatening pulmonary oedema, exemplifying the therapeutic dilemma when the causative drug remains medically necessary. Conservative AKI management was implemented while maintaining diuresis. Given the acute pulmonary oedema and cardiogenic shock, aggressive fluid resuscitation was avoided; instead, management focused on decongestion via i.v. furosemide, guided by clinical volume assessment. Electrolytes, including potassium chloride and calcium gluconate, were replaced as needed. Urine output initially remained adequate with clinical improvement in pulmonary congestion [Table/Fig-2].

Hospital Day	Sodium (mmol/L)	Potassium (mmol/L)	Chloride (mmol/L)	Bicarbonate (mmol/L)
Day 1	140	3.8	107	21
Day 2	142	3.7	106	25
Day 3	139	3.2	100	29
Day 4	132	4.1	95	29
Day 5	130	6.5	89	30
Day 6	-	3.1	-	-
Day 7	127	3.0	82	32
Day 8	129	3.2	-	-
Day 9	131	3.5	-	-
Day 10	133	4.0	90	30
Day 11	-	-	-	-
Day 12	-	-	-	-
Day 13	-	-	-	-
Day 14	135	2.5	97	27
Day 15	-	4.2	-	-
Day 16	146	3.6	109	26
Day 17	146	3.5	109	29
Day 18	148	3.3	110	29
Day 19	142	3.6	107	26
Day 20	141	3.5	108	24
Day 21 (Morning)	-	2.5	-	-
Day 21 (Evening)	-	2.6	-	-
Day 22 (Morning)	141	2.8	119	16
Day 22 (Evening)	-	5.0	-	-
Day 23	132	4.1	100	21
Day 24	137	4.0	-	-
Day 26	138	3.8	105	23

[Table/Fig-2]: Serial Electrolyte Parameters During Hospitalisation

Following Percutaneous Transluminal Coronary Angioplasty (PTCA) on Day 6, creatinine remained elevated (2.3 mg/dL on Day 6) after an initial rise from baseline (0.8 mg/dL) to 2.1 mg/dL on Day 4 and 2.4 mg/dL on Day 5. Creatinine subsequently peaked at 2.7 mg/dL on Day 9, temporally associated with contrast exposure during PTCA, before improving to 2.2 mg/dL by Day 14. Uric acid normalised to 4.9 mg/dL by Day 14, correlating with improved volume status and later renal recovery. AKI was likely multifactorial, driven by cardiorenal syndrome, renal hypoperfusion, high-dose diuretic therapy, sepsis, nephrotoxic antibiotics, and contrast exposure. Severe hypokalemia likely further impaired renal concentrating ability. Following stabilisation of renal function and resolution of metabolic derangements, the patient was transitioned to oral torsemide 10 mg daily and discharged on Day 26, twelve days after uric acid normalisation.

DISCUSSION

We report a critically ill 57-year-old man with anterior wall MI and acute pulmonary oedema who developed severe furosemide-induced hyperuricaemia (uric acid 12.2 mg/dL) with concurrent multifactorial AKI, in whom the causative agent could not be withdrawn due to life-threatening pulmonary congestion. This case highlights the importance of prioritising life-saving therapies while managing metabolic complications through vigilant monitoring and individualised treatment. A uric acid level >6.8 mg/dL (404 µmol/L) indicates hyperuricaemia, a precursor to gout [1]. The patient's RFT showed a uric acid level of 12.2 mg/dL on Day 4, confirming severe hyperuricaemia well above the usual range reported with diuretic therapy [1-4]. Serum uric acid was not measured on admission because the immediate clinical priority was stabilisation of acute myocardial infarction with life-threatening pulmonary oedema; additionally, hyperuricaemia was not initially suspected given the patient's normal baseline renal function (creatinine 0.8 mg/dL; eGFR >60 mL/min/1.73 m^2). Drug-induced hyperuricaemia, caused by increased reabsorption or reduced excretion of uric acid, can lead to gout [2]. Diuretics, along with aspirin, chemotherapy, anti-tubercular drugs, testosterone, alcohol, and purine-rich diets, contribute to hyperuricaemia [5]. Diuretics are a major cause, with loop, thiazide, and thiazide-like diuretics increasing gout risk [3,4].

Although the exact mechanism is not fully elucidated, diuretic-induced hyperuricaemia is mediated by both haemodynamic and tubular factors. Loop diuretics such as furosemide cause extracellular volume contraction, leading to enhanced proximal tubular sodium and urate reabsorption and reduced uric acid excretion [6]. Furosemide competes with urate for organic anion transporters in the proximal tubule and reduces fractional uric acid clearance, thereby directly impairing renal urate handling, as demonstrated by Dalbeth N et al., [7]. Although short-term diuretic use increases uric acid excretion, prolonged therapy reduces excretion due to increased proximal tubular reabsorption and competition for organic acid transport [3]. All diuretics cause urate retention, with minor differences among classes [8]. Spironolactone causes less urate retention than thiazides [9], while loop diuretics are more strongly associated with acute gout [10]. The combination of loop diuretic use and an eGFR <60 mL/min/1.73 m^2 is a major risk factor for hyperuricaemia [11]. In this case, the decline in eGFR from >60 mL/min/1.73 m^2 on admission to a nadir of 24 mL/min/1.73 m^2 likely amplified furosemide-induced hyperuricaemia. AKI was likely multifactorial; furosemide-induced volume contraction and reduced renal perfusion were primary contributors, with additional factors including cardiogenic shock, contrast exposure during PTCA, nephrotoxic antibiotics (piperacillin-tazobactam, linezolid), and severe hypokalemia. Contrast-induced nephropathy cannot be excluded given the temporal association between PTCA (Day 6) and peak creatinine (2.7 mg/dL, Day 9).

Diuretics are routinely used to manage volume overload in chronic heart failure. Since the patient was admitted with chronic heart failure and acute pulmonary oedema, intravenous furosemide 20 mg was started on Day 1 and continued as an infusion at 20 mg/hour until Day 14. Uric acid levels increased and reached 12.2 mg/dL on Day 4. Importantly, furosemide could not be discontinued despite severe hyperuricaemia and evolving acute kidney injury because of persistent, life-threatening pulmonary oedema, highlighting a real-world therapeutic dilemma rarely described in the literature. In a retrospective study of 535 patients hospitalised with acute heart failure, Zhou HB et al., demonstrated that in-hospital uric acid increase was significantly associated with higher daily loop diuretic doses (p=0.016) and greater creatinine rise (p<0.001), and was independently associated with increased mortality (adjusted HR 1.53, 95% CI 1.02–2.30) [12]. Salem B et al., noted that nearly two-thirds of patients with drug-induced hyperuricaemia remain asymptomatic. Consequently, urate-lowering therapy is rarely required in asymptomatic cases; however, diuretic use may precipitate gout in patients with

a personal or familial predisposition, and decisions regarding urate-lowering therapy should be individualised [2]. In the present case, marked hyperuricaemia occurred in a critically ill, high-risk patient who remained asymptomatic for gout, yet required continued high-dose loop diuretic therapy, underscoring the need for vigilant biochemical monitoring rather than symptom-driven testing alone.

Xanthine oxidase inhibitors are recommended as first-line therapy for drug-induced hyperuricaemia, with most guidelines preferring allopurinol, while ACR guidelines allow either allopurinol or febuxostat. Combination therapy with XOIs and uricosurics is advised when monotherapy fails, as uricosurics remain second-line agents [11]. Acute intravenous furosemide may induce hyperuricaemia, which can be mitigated by adequate intravenous saline to prevent volume depletion [13]. Allopurinol 100 mg twice daily was chosen over febuxostat due to cardiovascular safety concerns [14,15]. A relatively conservative allopurinol dose was selected with close biochemical monitoring given concurrent renal dysfunction. Contemporary evidence supports dosing to achieve target urate levels rather than restricting dose based on eGFR alone. Serum uric acid normalised to 4.9 mg/dL by Day 14, and therapy was continued at discharge [16]. Additionally, in patients with recurrent diuretic-induced hyperuricaemia, SGLT2 inhibitors may serve as valuable adjuncts given their well-documented uricosuric effects and capacity to reduce diuretic requirements in heart failure [6].

CONCLUSION(S)

Furosemide-induced hyperuricaemia is a clinically significant metabolic complication, particularly in patients with renal impairment, heart failure, or high cardiovascular risk. Risk stratification at diuretic initiation, identifying pre-existing renal impairment or baseline hyperuricaemia, guides monitoring frequency and intervention thresholds. Most asymptomatic cases can be managed conservatively through diuretic optimisation, adequate hydration, and lifestyle modification, while urate-lowering therapy is reserved for high-risk patients when the causative agent cannot be withdrawn.

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