Anuric Acute Kidney Injury in Chronic Myeloid Leukemia: A Rare Complication Case

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ABSTRACT

This report describes a rare case of anuric acute kidney injury related to suspected urate nephropathy in a 23-year-old male with chronic phase of Chronic Myeloid Leukemia (CML). The patient presented with anuria and limb edema, with a history of imatinib-treated CML. Investigations revealed probable urate crystals causing bilateral hydronephrosis and hydroureters. Management included fluid restriction to maintain euvolemic status, hypouricemic agents, urinary alkalinization, urgent hemodialysis for acute kidney injury, and blood product transfusions to address haematological imbalances. The continued use of imatinib and aforementioned treatments resulted in the restoration of renal function depicted through normalization of serum urea, creatinine and uric acid levels. This case highlights the importance of meticulous assessment and management of anuric acute kidney injury in CML patients to ensure a positive outcome.

Keywords*: anuria; acute kidney injury; chronic myeloid leukemia.*

INTRODUCTION

Chronic myeloid leukaemia (CML) is a myeloproliferative neoplasm marked by the presence of the Philadelphia chromosome, a reciprocal translocation between chromosomes 9 and 22, and positive findings of BCR-ABL1. This translocation causes a BCR-ABL1 gene fusion that expresses the p210 protein, a type of mutated tyrosine-kinase that triggers continuous cell proliferation, genomic instability, and DNA repair failure, which ultimately leads to CML.[1] About 15% of all adult leukemia cases are CML. Proliferating granulocytes in various stages of maturation is the hallmark of CML.[2] According to the American Institute for Cancer Research in 2017, the global incidence of CML ranged from 0.7 up to 1.5 per 100,000 persons.[3] The median age of CML onset was 55-65 years, which was almost half of the cases. However, CML is usually diagnosed at significantly younger age in Asia, either around 36-50 years old or even younger.[2,4,5] In relation to its pathophysiology involving tyrosine-kinase mutation, tyrosine kinase inhibitor (TKI), such as imatinib and nilotinib, was administered as the therapeutic option of CML, although sometime with suboptimal response due to pharmacokinetical variability of TKI.[6,7]

In general, cancer patients are prone to develop acute kidney injury (AKI). The incidence of AKI in patients with haematological cancer, such as myeloma and leukemia, was found to

be relatively higher compared to other types of cancers.[8] providing the basis of haematological cancer being one of the risk factors of AKI.[9] Albeit previous studies revealed that CML-related glomerular diseases were rare in comparison with other haematological cancers.[10] some pre-renal, intra-renal, and post-renal factors can still cause AKI onset, particularly among patients receiving therapies that affect renal function.[10,11] Different possible causes of anuric AKI in CML patients mandated a complex and meticulous approach. In this case report, we report a case of a CML patient with AKI presented with abrupt onset anuria.

CASE ILLUSTRATION

A 23-year-old male was referred to a tertiary hospital with a major complaint of not being able to pass urine for more than 48 hours before admission. For the last 48 hours, he could only pass less than 20 cc of darkened urine. Prior to this, the patient claimed that he was able to urinate with a frequency of four times a day and the excreted urine was not cloudy, not concentrated, not bloody, and without any urolith. Upon examination, his hands and feet were swollen. He felt pain around his waist and lower abdomen since the previous week, and the pain had gotten worse for the last four days. The patient also felt nausea with occasional vomitus and distended abdomen in the past two weeks. The patient was able to defecate regularly. He also complained of a dry cough since the previous week, but the condition improved after consuming codeine obtained from the Oncology Polyclinic. Fever, tinnitus, tingling of extremities, chest pain, and blurred vision were not identified. Before being referred, the patient had been treated with intravenous injections of pantoprazole, ondansetron, and 500 mL of physiologic saline infusion.

The patient has been diagnosed with CML since 2016 and has been receiving routine therapy with outpatient visits to the Oncology Polyclinic. At the beginning, he received 1500 mg of hydroxyurea daily. After BCR-ABL p210 fusion was qualitatively detected at the end of March 2016, the patient was started on 400 mg of imatinib mesylate daily until now. A history of discontinuation of imatinib mesylate for three months was reported in 2022. While the spleen had shrunk upon imatinib mesylate introduction, it had enlarged again since drug cessation. The patient also received 100 mg of allopurinol daily, but it was not regularly consumed. 10 mg of codeine b.i.d. was only administered when coughing.

During physical examination, the patient was alert but general weakness was observed. Blood pressure was measured at 140/90 mmHg. The patient was anemic and dyspneic, with an oxygen saturation of 98% upon administration of 4 liters per minute nasal cannula oxygen supplementation. Lymphadenopathy was found in the left neck measuring 2 x 2 x 1.5 cm, fixed, but neither hard, painful, warm, nor erythematous. Hepatomegaly was palpable at two centimetres below the right costal arch and splenomegaly was palpable (Schuffner-6). Ascites and peripheral edema were present. Other physical examinations were within normal limits.

Haematological examination and peripheral blood smear revealed normocytic-normochromic anemia with anisopoikilocytosis (haemoglobin 9.0 g/dL), leukocytosis with 3% myeloblasts and immature granulocytes (leukocytes 73.89 x 10^3/μL), and thrombocytopenia (platelets 146 x 10^3/μL); Peripheral blood smear found normocytic-normochromic erythrocytes with anisopoikilocytosis (microcytes, ovalocytes), polychromasia cells, and no normoblasts, consistent with anisopoikilocytosis and normochromic-normocytic anemia. Meanwhile, the impression of leukocytes was that the number increased, all stages of the myeloid series cells were found with a proportion of myeloblasts at 3%, promyelocytes at 7%, myelocytes at 11%, metamyelocytes at 13%, stab neutrophils at 18%, and segment neutrophils at 33%. There were also 4% monocytes, 1% eosinophils, and 10% mature lymphocytes. These findings were consistent with the chronic phase of CML (blasts proportion at 3%). The platelet seemed normal without the presence of a giant platelet or a clump.

Blood urea nitrogen and serum creatinine were found to be elevated (37.7 mg/dL and 21 mg/dL, respectively. Blood gas analysis revealed a compensated metabolic acidosis (pH

7.34, pCO2 29 mmHg, HCO3- 15.6 mmol/L, bicarbonate deficit 270 mEq). Electrolyte examination showed hypokalemia (3.1 mmol/L) and hyperuricemia (23.2 mg/dL). Other laboratory examinations were found to be within normal limits.

Chest x-ray examination revealed no heart or lung abnormalities. An abdominal USG found hepatosplenomegaly, cholelithiasis, hydronephrosis, and ascites. Abdominal CT stonography without contrast showed bilateral moderate hydronephrosis, multiple lymphadenopathies in the right cardiophrenic, paraaortic, aorta, paracava, bilateral parailiaca, and mesentery, accompanied by hepatosplenomegaly, ascites, and left side pleural effusion.

The patient was initially assessed as having AKI, suspected uropathy obstructive with bilateral hydronephrosis and hydroureters, chronic phase of CML (blast 3%), hyperuricaemia, hypertension grade I, and hypokalemia. High-calorie adequate-protein low-purine diet with a targeted calorie intake of 2100 kcal/day was given. The patient was also given 100 mEq of sodium bicarbonate in 400 mL of physiological saline daily while being advised to drink 1000-1500 cc of water a day, for future urine evaluation and euvolemic maintenance.

Based on the advice of the attending oncologist, 400 mg of imatinib mesylate q.d was continued. 100 mg of allopurinol q.d. was also administered to reduce the uric acid level. However, due to inadequate response, 80 mg of febuxostat q.d. was administered on the fifth day of admission replacing allopurinol. To alleviate the patient's current complaints, the patient was given intravenous injections of metoclopramide at 5 mg b.i.d, paracetamol at 500 mg t.i.d., and morphine sulfate at 10 mg every 48 hours with adjusted doses. 5 mg of lisinopril q.d. and 5 mg of amlodipine q.d. for hypertension, potassium chloride tablet at 600 mg t.i.d for hypokalemia, and sodium bicarbonate tablet at 500 mg t.i.d. and 500 mg of calcium carbonate b.i.d. for metabolic acidosis and urine alkalinization were also given. On the eighth day of admission, intravenous moxifloxacin at 400 mg by drip was given due to signs of infection by urinalysis and the increase of procalcitonin (from 1.12 ng/ml to 5.04 ng/ml).

CDL access was established for urgent hemodialysis which was conducted three times; on the second day, seventh day, and eleventh day of admission. During hemodialysis and admission, we provided a total of ten units of thrombocyte concentrate, apheresis platelets, and six units of packed red cells transfusion.

During hospitalization, urine production increased; with the highest production being on the thirteenth day of treatment. Fluid balance began to become deficit from the ninth day of treatment. Blood urea nitrogen level initially increased before being normalized, while both serum creatinine and uric acid levels improved gradually.

Although initially, there was a decrease in hemoglobin level from 9 g/dl to 5.1 g/dl on the fourteenth day of admission, hemoglobin level exceeded its initial level by the nineteenth day of admission (9.1 g/dl). Leukocyte level fell significantly by the nineteenth day of admission from 73.89 x 10^3/mcl to 16.41 x 10^3/mcl. However, despite platelet product transfusion, the platelet level fell considerably by the nineteenth day of admission from 146 x 10^3/mcl to 19 x 10^3/mcl. Blood urea nitrogen, serum creatinine, and uric acid levels were improved by

Table 1. Fluid monitoring during inpatient admission

Date		24/7	25/7	26/7	27/7	28/7	29/7	31/7	1/8	2/8	3/8
Input	Peroral	1000	1200	1500	1500	1400	1500	800	1200	1200	2400
	IV	500	500	500	500	500	500	500	500	250	250
	Medication	N/A	N/A	N/A	N/A	N/A	N/A	250	250	250	250
	Total	1500	1700	2000	2000	1900	2000	1550	1750	1750	2900
Output	Urine	100	200	500	700	600	200	1200	1300	1300	3200
	IWL	500	500	500	500	500	500	500	500	500	500
	Total	600	700	1000	1200	1100	700	1700	1800	1800	3700
Balance		+900	+1000	+1000	+800	+800	+1300	-150	-150	-150	-800

Table 2. Monitoring of blood urea nitrogen, serum creatinine, uric acid, urine pH, and urine specific gravity during admission

Date	21/7	23/7	25/7	28/7	31/7	3/8	8/8
BUN	43.5	47.5	64.0	46.4	56.0	73.0	28.0
SC	21.5	18.8	21.8	11.5	10.1	5.7	1.3
UA	N/A	23.2	27.0	11.5	5.0	5.1	2.7
Urine pH	5.5	N/A	5.7	N/A	5.5	N/A	6.5
Urine specific gravity	N/A	N/A	1.019	N/A	1.012	N/A	1.009

the nineteenth day of admission (28 mg/dl, 1.3 mg/dl, and 2.7 mg/dl respectively).

The patient was discharged on the nineteenth day of admission. The patient could urinate normally as much as 3,500 mL with improved peripheral edema and ascites. Three days after discharge, there was no significant complaint and CML therapy was continued, with regular consumption of allopurinol to prevent future incidents.

DISCUSSION

This patient presented with acute anuria with a suspicion of AKI, supported by several findings of kidney-related abnormalities on supporting examinations, such as increased BUN, serum creatinine, hyperuricemia, hypokalemia, compensated metabolic acidosis, and hypertension. Imaging showed bilateral hydronephrosis and hydroureters. Hence, the most important aspect to consider is the possible underlying etiologies of renal impairment.

In assessing the cause of AKI among cancer patients, the possibilities of pre-renal, intra-renal, and post-renal causes have to be considered. Pre-renal AKI is particularly common in haematological cancer, usually concerning volume depletion caused by poor fluid intake, early satiety, anorexia, emesis or diarrhea, insensible effective volume loss due to comorbidity, or medications affecting renal afferent-efferent tone. CML-associated glomerular disease as an intrarenal cause of AKI is relatively rare.[10] However, some cases also reported CML patients developing nephrotic syndrome with membranous nephropathy, membranoproliferative glomerulonephritis, or minimal change disease.[10,12,13] A renal biopsy which is the gold standard for diagnosing glomerular disease was not conducted for this patient, since such was only available in a few tertiary-level hospitals in Indonesia.[14] Thus, the probability of glomerular disease can neither be confirmed nor excluded. In addition, the nausea, vomiting, and finding of ascites by USG might indicate the pre-renal nature of AKI, but such findings may also be related to the long-term use of imatinib mesylate, one of first-line drugs of choice for CML.[15,16]

As the main therapy for CML,[7] TKI, especially imatinib, has been repeatedly reported to be associated with kidney injury in CML patients.[17] Long-term continuous use of TKI may adversely affect renal function.[18,19] Kidney impairment in CML patients is specifically associated with the consumption of imatinib and bosutinib.[4] A study by Yilmaz et al[20] also found a trend of decreasing glomerular filtration rate in CML patients taking imatinib for up to four years despite the absence of a history of chronic kidney disease (CKD). Although the specific pathogenesis of TKI consumption is not yet perfectly understood, TKI might cause damage to endothelial cells and podocytes in the kidney through mediation by several expressed proteins, such as Rac1 and Cdc42.[21] Other reports claimed long-term consumption of TKI was generally associated with proteinuria and increased serum creatinine level which was reversible upon TKI cessation.[18,19]

Another possible cause of AKI in cancer patients is tumour lysis syndrome (TLS), an oncology emergency that occurs when malignant cells are rapidly destroyed, either spontaneously or after initiation of cytotoxic chemotherapy. The damaged malignant cells released their intracellular ions and metabolites into the extracellular space, causing hyperkalemia, hyperuricemia, hyperphosphatemia, and secondary hypocalcemia with acute renal failure.[22] These

changes could cause a series of severe clinical impacts, one of which being kidney disorders due to either impaired renal vasoconstriction, renal inflammation, or metabolic precipitation obstructing the tubular system.[23,24]

CML is a haematological malignancy with a low risk of TLS.[25] However, there were some reports of CML patients having the occurrence of TLS, particularly the blast crisis phase.[26,27] Although TLS usually occurs within one week after initiation of chemotherapy, there were case reports of TLS in patients who have been on immunotherapy for years; and the patients either had a treatment failure or low compliance.[22,26,28] In this patient, TLS was excluded since the patient did not meet the definition of clinical nor laboratory TLS, due to only the occurrence of hyperuricemia and an increase in serum creatinine despite long-term use of imatinib for approximately six years.[23,29]

Another possible cause of acute anuria in this patient is hyperuricemia, usually in relation to a type of obstructive acute kidney disorder called urate nephropathy or urate uropathy. This condition is caused by supersaturation and precipitation or uric acid crystal of the urine. It frequently happens in collecting tubules where the urine becomes more acidic, a good environment for non-ionized uric acid to crystallize. As the effect to this process, intraluminal obstruction ensues, causing dilatation and inflammation of proximal and distal renal tubule. In a long run, the glomerular filtration rate is reduced and acute kidney injury and fibrosis ensues.[30,31] In urate nephropathy, hyperuricemia and hyperuricosuria also accompany acute kidney failure.[32]

Considering the clinical presentation of this patient, acute anuria, bilateral hydronephrosis and hydroureters, increased serum creatinine, and BUN with very high serum uric acid levels up to 23.2 mg/dL, the most likely cause of acute anuria is urate obstructive nephropathy, which is worsened by adverse effects of long-term use of imatinib. Volume depletion and dehydration may contribute towards the occurrence of urate nephropathy.[33] The relation between TKI use with urate nephropathy is unclear; one case report of AKI in CML patients receiving TKI found slightly increased uric acid level but the relationship between these two conditions was not considered.[34]

The management of Acute Kidney Injury (AKI) involves key principles: regular monitoring of electrolytes (every 24 hours or 4-8 hours for high-risk patients),[35,36] strict management of fluid intake and output (especially for oliguric or anuric patients),[35–37] and balancing fluid administration for obstructive AKI patients to achieve euvolemia and obstruction relief. During the diuretic phase of AKI, fluid, and electrolyte corrections are necessary to maintain euvolemia.[37] Hemodialysis is indicated for patients with oliguria or anuria, fluid overload, refractory hyperkalemia, and metabolic acidosis.[35,36] Continuation of CML therapy is generally advised, with temporary cessation in unstable conditions.[26] The case study involved intravenous fluid resuscitation with sodium bicarbonate for dehydration and metabolic acidosis, careful fluid balance maintenance, continuation of imatinib, and hemodialysis to manage overload syndrome due to acute anuria.

In anticipating and preventing urate nephropathy in cancer patients, maintaining fluid intake is important, with additional consideration to cardiac and renal condition of the patient. Routine monitoring of uric acid level is also important to ensure accurate management of hyperuricemia.[38] In treating urate nephropathy, hypouricemic agents like allopurinol (100 mg daily) can be considered for reducing uric acid levels, especially in symptomatic patients. However, allopurinol's prolonged action, requirement for dose adjustments, and risk of xanthine accumulation in renal impairment are limitations, so its administration is usually limited for symptomatic hyperuricemia.[36,38] Febuxostat, a non-purine selective xanthine oxidase inhibitor, offers an alternative with better tolerance, rapid onset, and fewer adverse effects. Sharma et al[39] reported its superiority over allopurinol in efficacy and side effects. Rasburicase is another option for patients with renal impairment.[39] In this case, due to inadequate response to allopurinol, the patient was switched to 80 mg daily of febuxostat, leading to a significant reduction in serum uric acid levels (2.7 mg/dL) within 14 days, without requiring renal dose adjustment.

Urine alkalinization, as recommended to patients by drinking soda or using sodium bicarbonate, can also reduce uric acid crystallization and increase uric acid excretion.[35] However, this strategy may cause phosphate-related nephropathy, considering that alkaline urine can trigger calcium phosphate precipitation in the renal tubules. Therefore, apart from reconsidering urine alkalinization strategies, strategies for reducing phosphate also need to be considered, such as by administering phosphate binding agents and limiting phosphate intake through diet.[40] In this patient, intravenous sodium bicarbonate (100 mEq/daily) was given gradually while monitoring daily arterial blood gas with the target of urinary pH of 6.4 – 6.8 for optimal uric acid clearance. To prevent phosphate-related nephropathy after urine alkalinization, calcium carbonate was given at 500 mg every 8 hours orally.

CONCLUSION

A 23-year-old man was diagnosed with anuria acute kidney injury due to urate obstructive uropathy with hydronephrosis and hydroureters in chronic phase of CML. The diagnosis was based on history taking, physical examination, and supporting examinations. The patient was treated with careful fluid therapy, hypouricemic agents, urinary alkalinization, hemodialysis, and blood product transfusion. The use of imatinib was continued. The patient was discharged after being able to urinate well, having improved edema, and upon normalization of blood urea nitrogen, serum creatinine, and uric acid levels.

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CONFLICT OF INTEREST

All of the authors declare that there is no conflict of interest.

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