

Hyperleukocytotic Presentation Worked Up for Suspected Atypical Chronic Myeloid Leukaemia, BCR-ABL1-Negative: A Case Report and Review of the Diagnostic Biochemical Clues

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Abstract:

➤ *Background:*

Atypical chronic myeloid leukaemia (aCML), BCR-ABL1-negative, is a rare clonal myelodysplastic/myeloproliferative neoplasm accounting for roughly 1–2% of BCR-ABL1-positive CML cases. It typically affects patients in the seventh–eighth decade, presenting with marked leukocytosis, dysgranulopoiesis, organomegaly, and variable cytopenias in the absence of the BCR-ABL1 fusion.

➤ *Case Presentation:*

We report a 40-year-old man with a reported but undocumented prior diagnosis of "chronic myeloid leukaemia," admitted with a two-week history of intermittent high-grade fever, productive cough, oral ulcers, and loose stools, on a background of heavy chronic alcohol use. Investigations revealed extreme leukocytosis ($295.15\text{--}309.40 \times 10^9/\text{L}$), macrocytic anaemia (haemoglobin 5.8 g/dL; MCV 107.8 fL), and thrombocytopenia ($98\text{--}122 \times 10^3/\mu\text{L}$), with peripheral/marrow smears showing a full spectrum of myeloid maturation. Biochemistry showed marked hyperuricemia (17.98 mg/dL), renal impairment (creatinine 2.79 mg/dL), mild hyponatraemia (131.5 mEq/L), pronounced hypocholesterolaemia (total cholesterol 65 mg/dL; LDL 31 mg/dL; HDL 16 mg/dL), hypoproteinaemia with low globulin, and mildly elevated GGT — a pattern consistent with a high myeloid cell-turnover state. He was managed with supportive care, hydration, and antimicrobials for a concurrent lower respiratory tract infection, with referral to nephrology and haematology for further work-up.

➤ *Result:*

Atypical chronic myeloid leukaemia (aCML).

➤ Conclusion:

This case highlights how routine biochemical derangements — hyperuricemia, renal impairment, hyponatraemia, and hypocholesterolaemia — accompanying extreme leukocytosis can serve as an early clue to an underlying high-turnover myeloproliferative process, warranting urgent haematology referral.

Keywords: Atypical Chronic Myeloid Leukaemia; BCR-ABL1-Negative; Myelodysplastic/Myeloproliferative Neoplasm; Hyperleukocytosis; Hyperuricemia.

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I. INTRODUCTION

Atypical chronic myeloid leukaemia (aCML), BCR-ABL1-negative, occupies the myelodysplastic/myeloproliferative neoplasm (MDS/MPN) overlap category in the World Health Organization (WHO) classification of myeloid neoplasms, sharing clinical features with classic Philadelphia-chromosome-positive CML — leukocytosis, dysgranulopoiesis, and organomegaly — but defined by the absence of BCR-ABL1 and, usually, of PDGFRA/B, FGFR1, and PCM1-JAK2 rearrangements [1,2]. Historically grouped under Philadelphia-negative “atypical” myeloid disorders long before molecular tools existed [3], aCML has since been characterised by recurrent somatic mutations in SETBP1, CSF3R, ASXL1, and ETNK1, which both aid diagnosis and carry prognostic weight [4,9–12].

Because aCML is a diagnosis of exclusion requiring integration of morphology, cytogenetics, and molecular studies, published case reports remain an important means of describing its heterogeneous presentations and of refining the differential diagnosis from classic CML, chronic myelomonocytic leukaemia (CMML), chronic neutrophilic leukaemia (CNL), and reactive leukaemoid reactions [5–8,13]. We report a case in which extreme hyperleukocytosis and a distinctive biochemical profile prompted a comprehensive diagnostic evaluation for suspected atypical CML, and we use the case to review the biochemical correlates of high myeloid cell turnover that can accompany such presentations.

Method: this case report was prepared in accordance with the CARE (CAse REport) guidelines. Clinical information was obtained through a detailed review of the patient’s medical records, including demographic characteristics, medical history, physical examination findings, laboratory investigations, imaging studies, and other relevant diagnostic procedures. The diagnosis was established based on the patient’s clinical presentation and the results of standard

diagnostic investigations interpreted according to current clinical guidelines.

Patient’s identifiers were removed to maintain confidentiality. Written informed consent for publication of the clinical details and accompanying images was obtained from the patient.

II. CASE PRESENTATION

➤ History

The patient reported being apparently well until two weeks before admission, when he developed intermittent, high-grade fever with an evening rise in temperature, partially relieved by antipyretic medication. This was accompanied by a two-week history of cough with whitish, non-blood-stained expectoration, and by oral ulcers. He also reported intermittent loose stools, the last episode two days before admission. He was a reported known/suspected case of chronic myeloid leukaemia, although prior haematology records were not available for review at the time of this admission. There was no personal history of diabetes mellitus, hypertension, dyslipidaemia, asthma, or chronic obstructive pulmonary disease, and family history was non-contributory. He gave a history of heavy alcohol use since the age of 18 year with the most recent binge one week before admission; he was not a smoker. He also reported a reduced sleep pattern and reduced appetite in the preceding weeks.

III. RESULT

On examination the patient was conscious, oriented, and afebrile at the time of assessment. Vital signs were: blood pressure 110/70 mmHg, pulse rate 90/min, and oxygen saturation 98% on room air. Cardiovascular examination revealed normal first and second heart sounds without added sounds; the respiratory system showed bilateral equal air entry without adventitious sounds; the abdomen was soft and non-tender; and the neurological examination was unremarkable, with a Glasgow Coma Scale score of 15/15.

Table 1 Timeline

Day	Event
Day 1 (admission)	History and examination; CBC with ESR, LFT, and RFT with electrolytes sent; working impression of lower respiratory tract infection with query CML recorded; empirical IV antibiotics, antipyretics, IV fluids, and supportive medications started.
Day 1 (afternoon/evening)	Critical-value CBC reported: haemoglobin 5.8 g/dL, total WBC count $295.15 \times 10^9/L$ (critical alert), platelet count $98 \times 10^3/\mu L$; LFT and lipid profile reported; peripheral smear reviewed under the microscope showing marked myeloid hyperplasia.
Day 2 (early morning)	Repeat CBC: total WBC count $309.40 \times 10^9/L$ (critical alert), platelet count $122 \times 10^3/\mu L$; nephrology referral made in view of renal impairment; extended work-up ordered (viral serology, blood culture, thyroid profile, HbA1c, chest X-ray, ECG, 2D echocardiogram, ultrasound abdomen, sputum for acid-fast bacilli/culture).
Day 2 (ongoing)	Oral antimicrobial regimen adjusted; supportive medications continued; strict input/output charting and four-hourly temperature monitoring instituted; further haematology work-up (bone marrow evaluation, cytogenetics, BCR-ABL1 testing) pending at the time of the records reviewed.

➤ *Diagnostic Assessment — Haematology*

Complete blood count on admission and on repeat testing the following morning are summarised in Table 2. The patient had severe macrocytic anaemia, extreme leukocytosis with a critical-value flag on two consecutive days, and mild thrombocytopenia, with a markedly elevated red cell distribution width and a normal erythrocyte sedimentation rate.

Table 2

Parameter	Day 1 value	Day 2 value	Reference range	Interpretation
Haemoglobin (g/dL)	5.8	—	13.0–17.0	Severe anaemia
Haematocrit/PCV (%)	15.8	—	40.0–50.0	Markedly reduced
RBC count (millions/ μL)	1.47	—	4.5–5.5	Markedly reduced
MCV (fL)	107.8	—	83–101	Macrocytic
MCH (pg)	39.6	—	27–32	Elevated
MCHC (%)	36.7	—	31.5–34.5	Mildly elevated
RDW-CV (%)	19.4	—	11–16	Elevated (anisocytosis)
RDW-SD (fL)	76.3	—	35.0–56.0	Elevated
Total WBC count ($\times 10^9/L$)	295.15*	309.40*	4.0–10.0	Extreme leukocytosis (critical value)
Platelet count ($\times 10^3/\mu L$)	98	122	150–410	Thrombocytopenia
ESR (mm/1st hour)	03	—	0–15 (male)	Normal

*Flagged as a critical alert value by the reporting laboratory.

➤ *Diagnostic Assessment — Biochemistry*

Renal function, electrolytes, liver function, and a fasting lipid profile were obtained; results are summarised in Tables 3

Table 3

Parameter	Value	Reference range	Interpretation
Urea (mg/dL)	49	17.12–49.2	Upper limit of normal
Creatinine (mg/dL)	2.79	0.9–1.3 (male)	Markedly elevated (renal impairment)
Uric acid (mg/dL)	17.98	4.4–7.6 (male)	Markedly elevated
Sodium (mEq/L)	131.5	136–145	Mild hyponatraemia
Potassium (mEq/L)	4.31	3.5–5.1	Normal
Chloride (mEq/L)	100.3	96–106	Normal
Total bilirubin (mg/dL)	0.58	0.3–1.1	Normal
Direct bilirubin (mg/dL)	0.34	0.0–0.2	Mildly elevated
Indirect bilirubin (mg/dL)	0.24	0.2–0.9	Normal
AST/SGOT (IU/L)	22.8	<35 (male)	Normal
ALT/SGPT (IU/L)	7.3	<45 (male)	Normal
Alkaline phosphatase (IU/L)	116.2	53–128 (male, 19–50y)	Normal

Parameter	Value	Reference range	Interpretation
Gamma-glutamyl transferase (IU/L)	61.6	<55 (male)	Mildly elevated
Total protein (g/dL)	5.82	6.0–7.8	Reduced
Albumin (g/dL)	3.5	3.35–5.2	Normal
Globulin (g/dL)	2.32	2.5–3.5	Reduced
A/G ratio	1.51	1.2–2.1	Normal
Lipid parameter	Value (mg/dL)	Reference (NCEP ATP III)	Interpretation
Total cholesterol	65.0	Desirable <200	Markedly low
Triglycerides	120	Normal <150–199	Normal
HDL cholesterol	16	Low <40	Markedly low
LDL cholesterol	31	Optimal <100	Markedly low
VLDL cholesterol	24	<30	Normal
Total cholesterol/HDL ratio	3.49	<4.5	Normal
LDL/HDL ratio	0.99	2.8–4.1	Reduced

➤ *Peripheral Blood/Bone Marrow Smear*

Photomicrographs obtained from the reporting laboratory (Leishman/Wright-stained smear, oil-immersion) showed marked myeloid hyperplasia with cells distributed across multiple stages of maturation — myelocytes, metamyelocytes, and band forms — admixed with occasional larger, more immature-appearing mononuclear cells, against a background of reduced platelet clumping. This pattern is compatible with a proliferative myeloid process.

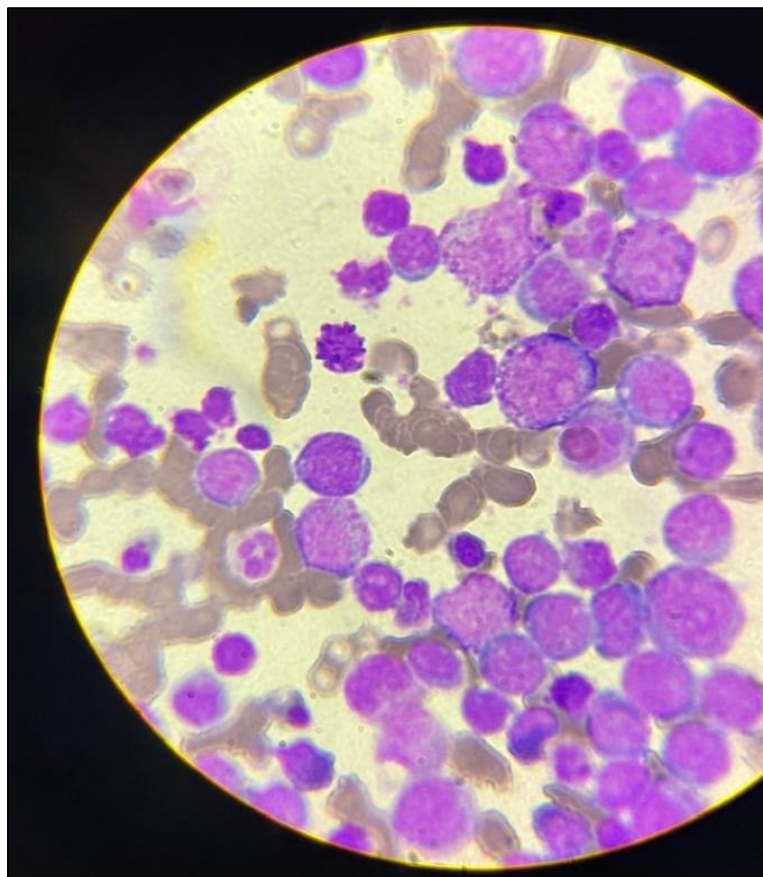


Fig 1. Peripheral blood/Bone Marrow Aspirate Smear (Leishman/Wright Stain, Oil-Immersion, $\times 1000$) Showing Marked Myeloid Hyperplasia with Cells Spanning Multiple Stages of Maturation (Myelocytes, Metamyelocytes, and Band Forms) Mixed with Occasional Larger Immature-Appearing Mononuclear Cells, Against a Background of Platelet Clumping (Patient's Original Sample)

➤ Working/Differential Diagnosis

The treating team's working impression, as documented in the case notes, was lower respiratory tract infection with a query of underlying chronic myeloid leukaemia. Given the degree of leukocytosis, macrocytic anaemia, thrombocytopenia, and the biochemical picture described above, the differential diagnosis for the underlying haematological process included: (i) atypical CML, BCR-ABL1-negative; (ii) classic BCR-ABL1-positive CML, including accelerated or blast phase; (iii) chronic myelomonocytic leukaemia; and (iv) a severe leukaemoid reaction secondary to infection, which is less likely given the extent and persistence of the leukocytosis on two consecutive days. Because BCR-ABL1 fluorescence in-situ hybridisation/polymerase chain reaction, karyotype/cytogenetics, and a mutation panel (SETBP1, CSF3R, ASXL1, ETNK1) were not available in the documentation reviewed, aCML could be raised as a leading working diagnosis on clinical and biochemical grounds but could not be formally confirmed against WHO 2016/2022 criteria at the time of this report [1,2].

➤ Therapeutic Intervention

Management was initially supportive and syndromic: intravenous fluids (dextrose normal saline at 75 mL/hour), empirical intravenous antibiotics for the presumed lower respiratory tract infection, an antipyretic, a proton-pump inhibitor, an antiemetic, and a bronchodilator/expectorant syrup with a leukotriene-receptor antagonist. On the second hospital day, the antimicrobial regimen was revised to an oral combination agent along with a nitroimidazole, a multivitamin/B-complex preparation, a probiotic, and a topical oral gel for the ulcers, with continuation of supportive care. A nephrology referral was made for further evaluation of the renal impairment, and blood culture, viral serology, thyroid function tests, glycated haemoglobin, chest imaging, electrocardiography, echocardiography, and abdominal ultrasound were requested as part of a broader systemic and infective work-up alongside the planned haematological evaluation.

IV. DISCUSSION

Atypical CML is a rare BCR-ABL1-negative MDS/MPN overlap neoplasm defined by persistent peripheral blood leukocytosis with $\geq 10\%$ circulating immature granulocytes, prominent dysgranulopoiesis, minimal or absent monocytosis and basophilia, and $< 20\%$ blasts in blood and marrow, in a patient without a PDGFRA/B, FGFR1, or PCM1-JAK2 rearrangement [1,2]. Historically, such Philadelphia-negative, BCR-ABL1-negative myeloproliferative pictures were grouped together as “chronic myeloid disorders” before the semi-molecular reclassification of the 2000s separated them from classic CML [3]. Patients typically present in the seventh or eighth decade of life with marked leukocytosis (reported medians of approximately $23\text{--}97 \times 10^9/\text{L}$), moderate-to-severe anaemia (median haemoglobin $8.6\text{--}11.7 \text{ g/dL}$), organomegaly

in roughly half to three-quarters of cases, and a poor prognosis, with median survival estimates in the range of 12–37 months and a substantial risk of transformation to acute myeloid leukaemia [6,8].

The present patient's leucocyte count of $295\text{--}309 \times 10^9/\text{L}$ considerably exceeds the leukocytosis typically reported in aCML case series and is more in keeping with the range classically described in florid, BCR-ABL1-positive CML; this discrepancy is an important reason that BCR-ABL1 testing is indispensable before an aCML label can be applied, since the two entities can otherwise appear clinically similar [1,6,7]. Molecular studies over the past decade have identified recurrent somatic mutations that assist in this distinction and carry independent prognostic value: SETBP1 mutations, clustered in the SKI-homologous domain, are found in roughly a quarter of aCML cases and are associated with a higher presenting white-cell count and shorter survival [4,12]; CSF3R mutations, first described as oncogenic drivers in chronic neutrophilic leukaemia, are also found in a substantial minority of aCML cases and may predict sensitivity to JAK or SRC-family/TNK2 kinase inhibition [9–11]. None of these studies were available in the documentation reviewed for the present case, and their absence is the principal reason this report frames the diagnosis as “suspected” rather than confirmed.

The biochemical picture in this patient is, in our view, the most instructive aspect of the case. Marked hyperuricemia (17.98 mg/dL , roughly 2.4 times the upper limit of normal) together with an elevated creatinine is consistent with a high nucleic-acid/purine turnover state, of the kind classically described in tumour-lysis physiology, even though frank tumour lysis syndrome by the Cairo–Bishop criteria (which requires ≥ 2 of hyperuricemias, hyperkalemia, hyperphosphatemia, and hypocalcaemia, plus a clinical criterion) was not met here, since potassium and clinical criteria were not deranged [17,18]. This pattern is better described as a sub-clinical, high-turnover hyperuricemia state accompanying massive myeloid proliferation than as established tumour lysis syndrome, but it should prompt the same vigilance — hydration, urate-lowering therapy, and monitoring of renal function — recommended in that literature [17,18]. The mild hyponatraemia is also noteworthy: extreme leukocytosis is well described as a cause of laboratory electrolyte artefacts (for example pseudo hyperkalemia from ex-vivo cell lysis during sample handling) [19], and this possibility should be considered and, where feasible, excluded by paired serum/plasma or point-of-care testing; however, because this patient's total protein and lipid values were low rather than elevated, a dilutional or hypovolemic (from reduced intake and loose stools) mechanism for the hyponatraemia is more plausible here than a spurious laboratory artefact.

The pronounced hypocholesterolaemia — total cholesterol 65 mg/dL , LDL 31 mg/dL , and HDL 16 mg/dL , all

well below the lower limits typically reported in healthy adults — together with hypoproteinaemia and a low globulin fraction, is compatible with the increased metabolic consumption and altered hepatic synthetic priorities that can accompany a high-turnover proliferative marrow process, superimposed on the patient's reduced appetite and chronic alcohol use. Low cholesterol and low total protein/globulin are non-specific findings across several haematological malignancies and cannot, on their own, support or refute a diagnosis of aCML; we highlight them here mainly to draw attention to a biochemical pattern that, in combination with extreme leucocytosis, macrocytic anaemia, and thrombocytopenia, should lower the threshold for urgent haematology referral and marrow evaluation, and to flag them as candidates for more systematic study in future case series.

Chronic heavy alcohol use is an important confounder in this case. Alcohol use disorder can independently cause macrocytosis (through direct marrow toxicity and folate/B12

depletion), mild transaminase and gamma-glutamyl transferase elevation, and reduced total protein/albumin from poor nutrition and hepatic synthetic impairment [3]. These mechanisms plausibly contribute to the macrocytosis and the mild GGT elevation seen here, but they do not account for the magnitude and left-shifted pattern of the leucocytosis, which points to a primary marrow proliferative disorder rather than an alcohol-related phenomenon alone. Nutritional B12/folate deficiency should nonetheless be actively excluded as a contributor to the macrocytic anaemia before attributing it solely to marrow infiltration.

➤ Descriptive Comparison with Reported Literature Values

As this is a single-patient case report, no inferential statistical testing is applicable or presented; the following table is a purely descriptive comparison of this patient's laboratory values against the patient's own reference ranges and against literature-reported medians for aCML case series, included for contextual interpretation only [6].

Table 1

Parameter	Patient Value	Local Reference Range	Deviation from ULN/LLN	Literature Median in aCML [6]
WBC ($\times 10^9/L$)	295.15–309.40	4.0–10.0	$\sim 30\text{--}31\times$ the ULN	23–97 (range of reported medians)
Haemoglobin (g/dL)	5.8	13.0–17.0	$\sim 55\text{--}65\%$ below LLN	8.6–11.7 (range of reported medians)
Platelet count ($\times 10^3/\mu L$)	98–122	150–410	$\sim 19\text{--}35\%$ below LLN	Variable; often reduced
Creatinine (mg/dL)	2.79	0.9–1.3	$\sim 2.1\text{--}3.1\times$ the ULN	Not routinely reported
Uric acid (mg/dL)	17.98	4.4–7.6	$\sim 2.4\times$ the ULN	Not routinely reported

V. CONCLUSION

Extreme hyperleukocytosis accompanied by hyperuricemia, renal impairment, mild hyponatraemia, and pronounced hypocholesterolaemia should raise suspicion of an underlying high-turnover myeloproliferative process and prompt urgent haematology referral, even before marrow or molecular results are available.

DECLARATIONS

➤ Ethics and Consent:

Written informed consent for publication of de-identified clinical and molecular data was obtained from the patient. All identifying information (name, hospital/case identification numbers, and exact dates) has been removed in accordance with journal and institutional requirements for patient confidentiality.

➤ Conflicts of Interest

The authors declare no conflicts of interest relevant to this manuscript.

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REFERENCES

- [1]. Arber DA, Orazi A, Hasserjian R, Thiele J, Borowitz MJ, Le Beau MM, et al. The 2016 revision to the World Health Organization classification of myeloid neoplasms and acute leukemia. *Blood*. 2016;127(20):2391–2405.
- [2]. WHO Classification of Tumours Editorial Board. *Haematolymphoid Tumours*. 5th ed. Vol. 11. Lyon: International Agency for Research on Cancer; 2024. Section: Myelodysplastic/myeloproliferative neoplasms, p.102–104.
- [3]. Tefferi A, Gilliland DG. Classification of chronic myeloid disorders: from Dameshek towards a semi-molecular system. *Best Pract Res Clin Haematol*. 2006;19(3):365–385.
- [4]. Piazza R, Valletta S, Winkelmann N, Redaelli S, Spinelli R, Pirola A, et al. Recurrent SETBP1 mutations in atypical chronic myeloid leukemia. *Nat Genet*. 2013;45(1):18–24.

- [5]. Belkhair J, Raissi A, Elyahyaoui H, Ait Ameer M, Chakour M. Atypical chronic myeloid leukemia BCR-ABL1 negative: a case report and literature review. *Leuk Res Rep.* 2019;12:100172.
- [6]. Diamantopoulos PT, Viniou NA. Atypical chronic myelogenous leukemia, BCR-ABL1 negative: diagnostic criteria and treatment approaches. *Front Oncol.* 2021;11:722507.
- [7]. Sadigh S, Hasserjian RP, Hobbs G. Distinguishing atypical chronic myeloid leukemia from other Philadelphia-negative chronic myeloproliferative neoplasms. *Curr Opin Hematol.* 2020;27(2):122–127.
- [8]. Schwartz LC, Mascarenhas J. Current and evolving understanding of atypical chronic myeloid leukemia. *Blood Rev.* 2019;33:74–81.
- [9]. Maxson JE, Gotlib J, Pollyea DA, Fleischman AG, Agarwal A, Eide CA, et al. Oncogenic CSF3R mutations in chronic neutrophilic leukemia and atypical CML. *N Engl J Med.* 2013;368(19):1781–1790.
- [10]. Gotlib J, Maxson JE, George TI, Tyner JW. The new genetics of chronic neutrophilic leukemia and atypical CML: implications for diagnosis and treatment. *Blood.* 2013;122(10):1707–1711.
- [11]. Pardanani A, Lasho TL, Laborde RR, Elliott M, Hanson CA, Knudson RA, et al. CSF3R T618I is a highly prevalent and specific mutation in chronic neutrophilic leukemia. *Leukemia.* 2013;27(9):1870–1873.
- [12]. Damm F, Itzykson R, Kosmider O, Droin N, Renneville A, Chesnais V, et al. SETBP1 mutations in 658 patients with myelodysplastic syndromes, chronic myelomonocytic leukemia and secondary acute myeloid leukemias. *Leukemia.* 2013;27(6):1401–1403.
- [13]. Elliott MA, Dewald GW, Tefferi A, Hanson CA. Chronic neutrophilic leukemia (CNL): a clinical, pathologic and cytogenetic study. *Leukemia.* 2001;15(1):35–40.
- [14]. Cazzola M, Malcovati L, Invernizzi R. Myelodysplastic/myeloproliferative neoplasms. *Hematology Am Soc Hematol Educ Program.* 2011;2011(1):264–272.
- [15]. Gagnier JJ, Kienle G, Altman DG, Moher D, Sox H, Riley D, for the CARE Group. The CARE guidelines: consensus-based clinical case reporting guideline development. *J Clin Epidemiol.* 2014;67(1):46–51.
- [16]. Riley DS, Barber MS, Kienle GS, Aronson JK, von Schön-Angerer T, Tugwell P, et al. CARE guidelines for case reports: explanation and elaboration document. *J Clin Epidemiol.* 2017;89:218–235.
- [17]. Cairo MS, Bishop M. Tumour lysis syndrome: new therapeutic strategies and classification. *Br J Haematol.* 2004;127(1):3–11.
- [18]. Howard SC, Jones DP, Pui CH. The tumor lysis syndrome. *N Engl J Med.* 2011;364(19):1844–1854.
- [19]. Menakuru SR, Priscu A, Beirat A, Dhillon VS, Salih A, Emran J. Hyperleukocytosis and pseudohyperkalemia in a patient with severe SARS-COVID-19 unmasks chronic lymphocytic leukemia. *Cureus.* 2021;13(12):e20826.
- [20]. National Cancer Institute, Surveillance, Epidemiology, and End Results (SEER) Program. SEER Hematopoietic and Lymphoid Neoplasm Database: C92.2, Atypical chronic myeloid leukemia, BCR/ABL-negative. Bethesda, MD: National Cancer Institute; 2024. Available from: <https://seer.cancer.gov/seertools/hemelymph/>