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Acquired Platelet Dysfunction in Atypical Chronic Myeloid Leukemia: A Case Report

Mohamed Ilyass Tazi [1], Hassane Mamad [1], Naoufal Benlachgar [2], Jalila Zirar [1], Mohamed Ifleh [1], Souad Benkirane [1], Azlarab Masrar [1]

1. Central Laboratory of Hematology, Ibn Sina Hospital, Faculty of Medicine and Pharmacy, Mohammed V University, Rabat, MAR 2. Department of Clinical Hematology, Ibn Sina Hospital, Faculty of Medicine and Pharmacy, Mohammed V University, Rabat, MAR

Corresponding author: Mohamed Ilyass Tazi, tazimi.dr@gmail.com

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Abstract

Atypical chronic myeloid leukemia (aCML), now classified as myelodysplastic/myeloproliferative neoplasm with neutrophilia, is a rare myeloid neoplasm characterized by neutrophilia, dysgranulopoiesis, and the absence of BCR::ABL1. Hemorrhagic manifestations may be related to acquired platelet dysfunction despite a platelet count only marginally below the reference range.

We report the case of an 86-year-old woman with aCML who presented with diffuse spontaneous ecchymoses and a history of hemopericardium. Laboratory evaluation revealed marked leukocytosis of 43.7 × 10^9/L, bone marrow dysgranulopoiesis with 4% blasts, absence of BCR::ABL1, and pathogenic TET2 and KRAS mutations. Platelet aggregometry revealed acquired platelet dysfunction, with severely impaired responses to arachidonic acid and collagen despite a platelet count of 145 × 10^9/L, only marginally below the reference range. Renal function was normal, and she was not taking aspirin or NSAIDs. Low-dose hydroxyurea and close monitoring resulted in clinical and hematological stabilization.

This case highlights the importance of functional platelet testing in patients with aCML who present with bleeding manifestations that cannot be explained by the platelet count alone, where it may be a contributing factor.

Categories: Hematology, Oncology
Keywords: acquired thrombopathy, atypical chronic myeloid leukemia, case report, next-generation sequencing, platelet dysfunction

Introduction

Atypical chronic myeloid leukemia (aCML), BCR::ABL1-negative, is a rare myeloid neoplasm of older adults with a poor prognosis, belonging to the myelodysplastic/myeloproliferative neoplasm (MDS/MPN) group. It is primarily characterized by neutrophil-predominant leukocytosis, marked dysgranulopoiesis, bone marrow hypercellularity, and the absence of the BCR::ABL1 rearrangement. Its diagnosis relies on the integration of clinical, morphological, cytogenetic, and molecular findings, with exclusion of other BCR::ABL1-negative myeloid neoplasms [1-6].

Hemorrhagic manifestations are less well characterized than the proliferative complications of this disease. In myeloid neoplasms, bleeding may occur in the presence of thrombocytopenia, but it may also develop despite a relatively preserved platelet count due to qualitative abnormalities of platelet function [7-10]. Acquired platelet dysfunction has been described in various myeloid neoplasms and may be responsible for sometimes severe bleeding diathesis [1,8,11]. It is well documented in classical myeloproliferative neoplasms, particularly primary myelofibrosis [12], whereas in aCML the evidence is limited to isolated reports [1].

The mechanisms underlying these functional abnormalities remain incompletely understood and appear to be multifactorial. Platelet aggregometry is an important tool for identifying these abnormalities and characterizing the platelet activation pathways involved. However, data on acquired platelet dysfunction specifically associated with aCML remain limited. A recent review highlighted the paucity of available data on bleeding manifestations and platelet functional abnormalities in aCML and chronic neutrophilic leukemia [1].

We report the case of an 86-year-old woman with aCML who presented with spontaneous bleeding manifestations, including diffuse ecchymoses and a history of hemopericardium, in whom acquired platelet dysfunction was demonstrated by platelet aggregometry. This case highlights the value of functional platelet testing in the evaluation of bleeding manifestations disproportionate to the platelet count in patients with myeloid neoplasms.

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Case Presentation

An 86-year-old woman was admitted for evaluation of spontaneous bleeding, characterized by diffuse cutaneous ecchymoses (Figure *1*), with a recent history of hemopericardium. Her medical history included type 2 diabetes mellitus for more than 30 years, controlled hypertension, chronic rheumatism, a hysterectomy performed in 2004 for significant uterine bleeding, and lower-limb varicose veins. She was not taking aspirin or other non-steroidal anti-inflammatory drugs (NSAIDs) before hemostatic testing.

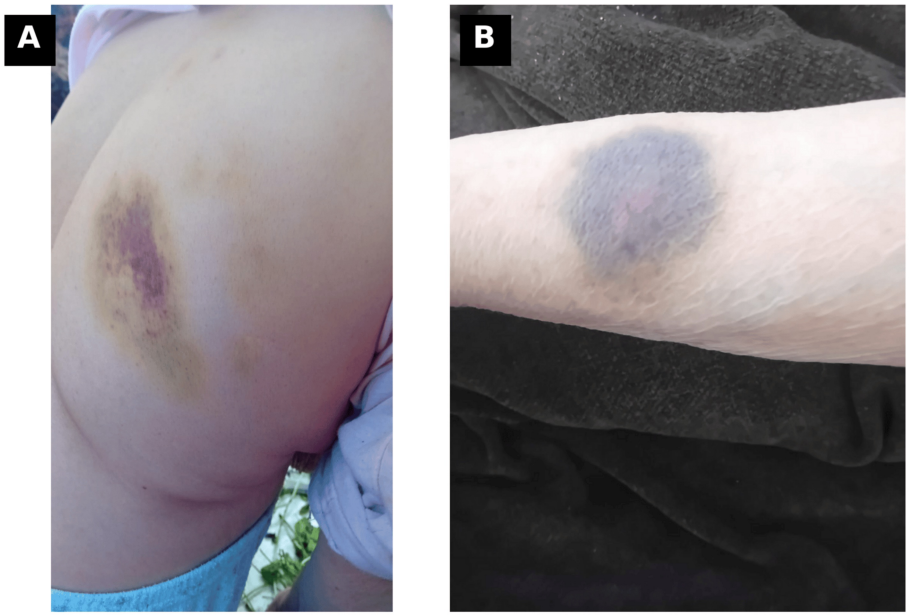


FIGURE 1: Extensive spontaneous ecchymoses in the patient.

A: Ecchymosis on the dorsal region. B: Ecchymosis on the forearm.

In May 2023, she developed post-influenza pericarditis complicated by hemopericardium. Pericardial drainage yielded serohemorrhagic fluid. Persistent leukocytosis was identified during this episode.

At admission, physical examination revealed multiple ecchymoses involving the forearms, legs, and back, with no lymphadenopathy or splenomegaly. The patient was hemodynamically stable. Complete blood count showed leukocytosis of 43.7 × 10^9/L, predominantly neutrophilic, with a hemoglobin level of 11.2 g/dL and a platelet count of 145 × 10^9/L, only marginally below the reference range. The absolute neutrophil count was 36.27 × 10^9/L (83%), with no basophilia. Serological tests for hepatitis B virus, hepatitis C virus, and human immunodeficiency virus were negative. Renal function was normal (serum creatinine 8 mg/L, urea 0.3 g/L, eGFR 75 mL/min/1.73 m^2).

Bone marrow aspiration showed granulocytic hyperplasia with a predominance of granulocytic precursors. Promyelocytes, myelocytes, and metamyelocytes accounted for 42% of the marrow cells, with a blast count of 4% (Figure *2*). The karyotype was normal (46,XX), and reverse transcription polymerase chain reaction (RT-PCR) for BCR::ABL1 was negative. Next-generation sequencing (NGS) using a 40-gene myeloid panel identified two distinct frameshift TET2 mutations (p.Ser817fs, VAF 47%; p.Pro980fs, VAF 41%) and one KRAS mutation (p.Gly12Ser, VAF 43%). Integration of the clinical, hematological, morphological, cytogenetic, and molecular findings supported the diagnosis of BCR::ABL1-negative aCML according to the 2022 World Health Organization (WHO) criteria (Table *1*) [13].

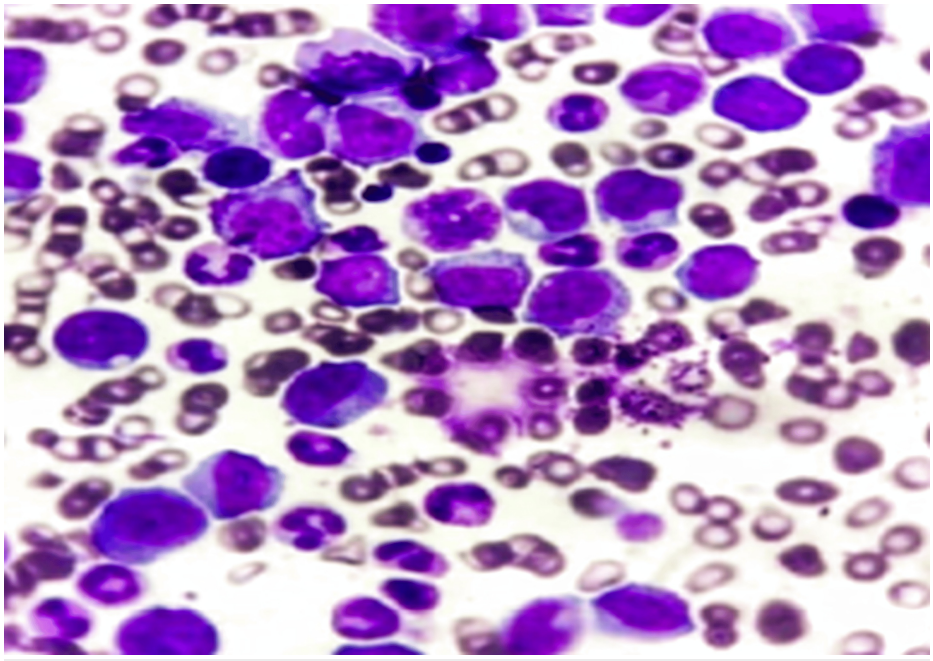


FIGURE 2: Bone marrow aspirate showing marked granulocytic hyperplasia, prominent dysgranulopoiesis, and 4% blasts, consistent with atypical chronic myeloid leukemia (aCML).

2022 WHO diagnostic criterion	Expected findings	Findings in our patient
Peripheral blood leukocytosis	WBC ≥13 × 10^9/L	WBC = 43.7 × 10^9/L
Neutrophilia	Neutrophil predominance	Neutrophils = 36.3 × 10^9/L
Blasts	<20% in peripheral blood	Not reported
	<20% in bone marrow	Bone marrow blasts = 4%
Dysgranulopoiesis	Marked dysgranulopoiesis	Marked dysgranulopoiesis observed
Monocytes	<10% of peripheral blood leukocytes	Monocytes = 4.8%
Eosinophils	<10% of peripheral blood leukocytes	Eosinophils = 2%
Bone marrow cellularity	Hypercellular bone marrow	Hypercellular bone marrow
Granulocytic proliferation	Granulocytic hyperplasia	Marked granulocytic hyperplasia
Granulocytic dysplasia	Granulocytic dysplasia	Dysgranulopoiesis observed
BCR::ABL1	Absence of BCR::ABL1 fusion	BCR::ABL1 = Negative
Other tyrosine kinase rearrangements	Absence of PDGFRA rearrangement	No other kinase rearrangement was reported in the available workup
	Absence of PDGFRB rearrangement	-
	Absence of FGFR1 rearrangement	-
	Absence of PCM1::JAK2 rearrangement	-
Molecular findings	Clonal molecular abnormality	TET2 p.Ser817fs
		TET2 p.Pro980fs
		KRAS p.Gly12Ser
Cytogenetics	No specific abnormal karyotype is required	Karyotype = 46,XX
Exclusion of other myeloid neoplasms	No evidence of CMML	CMML excluded
	No evidence of classic CML	Classic CML excluded
	No evidence of CNL	CNL excluded (prominent dysgranulopoiesis)

TABLE 1: 2022 World Health Organization (WHO) and International Consensus Classification (ICC) diagnostic criteria for myelodysplastic/myeloproliferative neoplasm with neutrophilia (formerly atypical chronic myeloid leukemia) and their application to the present case.

Adapted from References [13,14].

CML: chronic myeloid leukemia; CMML: chronic myelomonocytic leukemia; CNL: chronic neutrophilic leukemia

The hemostasis workup showed normal prothrombin time (PT) and fibrinogen levels, with a prolonged activated partial thromboplastin time (aPTT) (ratio, 1.58). Factors VIII, IX, XI, and XII were normal or increased; factor XIII was mildly decreased (62.6%), and no mixing study was available. Evaluation of von Willebrand factor (VWF) showed no evidence of deficiency, with preserved functional activity and increased VWF antigen levels. Platelet aggregometry demonstrated acquired platelet dysfunction, characterized by markedly reduced responses to arachidonic acid (3.5%) and collagen (16.5%), a normal response to ADP (54.7%), and a preserved response to ristocetin (63.6%). Given the patient's age, comorbidities, and clinical stability, symptomatic management combined with low-dose cytoreduction with hydroxyurea was initiated, with complete blood count monitoring every three weeks. After three months of follow-up, leukocytosis remained stable, and no further episodes of hemopericardium were observed.

A summary of the main hematological and functional findings is presented in Table *2*.

Parameter	Results	Reference range (normal values)	Interpretation/clinical comments
Age/sex	86 years/female	-	Elderly patient with multiple comorbidities
Hemoglobin (g/dL)	11.2 g/dL	12.0-16.0	Decreased
Hematocrit (%)	38.10%	36-46	Normal
Mean corpuscular volume (MCV) (fL)	80.7 fL	80-100	Normal
Mean corpuscular hemoglobin (MCH) (pg)	26.5 pg	27-32	Mildly decreased
Mean corpuscular hemoglobin concentration (MCHC) (g/dL)	32.8 g/dL	32-36	Normal
Total leukocyte count (×10³/µL)	43.7	4.0-10.0	Marked leukocytosis
Neutrophils (×10³/µL)	36.27	1.5-7.0	Neutrophilia
Lymphocytes (×10³/µL)	4.37	1.0-4.0	Mildly increased
Monocytes (×10³/µL)	2.19	0.2-1.0	Monocytosis
Eosinophils (×10⁹/L)	0.87	0.04-0.50	Mild absolute eosinophilia
Basophils (×10⁹/L)	0	0-0.10	Normal
Platelets (×10³/µL)	145	150-400	Marginally below the reference range
Mean platelet volume (MPV) (fL)	10.9	7.0-12.0	Normal
Platelet distribution width (PDW) (fL)	17.8	9.0-16.0	Increased platelet size variability
Creatinin (mg/L)	8	5.7-12.5	Normal
Urea (g/L)	0.3	0.15-0.55	Normal
eGFR (mL/min/1.73 m²)	75	Average 75	Normal for age
Bone marrow karyotype	46,XX	Normal	No chromosomal abnormality detected
BCR::ABL1 transcript (PCR)	Negative	Negative	Excludes classic BCR::ABL1-positive CML
TET2 mutation 1	p.Ser817fs; VAF 47%	No pathogenic mutation expected	Pathogenic clonal molecular abnormality
TET2 mutation 2	p.Pro980fs; VAF 41%	No pathogenic mutation expected	Pathogenic clonal molecular abnormality
KRAS mutation	p.Gly12Ser; VAF 43%	No pathogenic mutation expected	Pathogenic clonal molecular abnormality
Prothrombin time (PT)	99-100%	70-100%	Normal
International normalized ratio (INR)	1	0.8-1.2	Normal
Activated partial thromboplastin time (aPTT)	Ratio: 1.58	0.8-1.2	Prolonged, not explained by factor VIII, IX, XI or XII levels; no mixing study
Fibrinogen	3.1 g/L	2.0-4.0 g/L	Normal
Factor VIII activity	170.50%	50-145%	Increased, possibly reactive
Factor IX activity	147.90%	50-145%	Mildly increased
Factor XI activity	135%	50-145%	Normal
Factor XII activity	69.20%	50-145%	Normal
Factor XIII activity	62.60%	70-140%	Mildly decreased
von Willebrand factor antigen (VWF:Ag)	200%	50-150%	Increased, possibly reactive

von Willebrand factor activity	170%	50-150%	Increased, with preserved functional activity
ADP-induced platelet aggregation (10 µM)	54.70%	50-100%	Normal
Collagen-induced platelet aggregation (5 µg/mL)	16.50%	50-100%	Markedly reduced response
Arachidonic acid-induced platelet aggregation (0.5 mg/mL)	3.50%	50-100%	Markedly reduced response, also seen with aspirin/NSAIDs
Ristocetin-induced platelet aggregation (0.5 mg/mL)	63.60%	50-100%	Preserved response

TABLE 2: Main laboratory, platelet function, and hemostatic parameters observed in the patient.

NSAIDs: non-steroidal anti-inflammatory drugs

Discussion

MDS/MPN with neutrophilia, formerly known as aCML, BCR::ABL1-negative, is a rare myeloid neoplasm characterized by both myelodysplastic and myeloproliferative features. In the 2022 WHO classification, this entity was renamed MDS/MPN with neutrophilia, whereas the International Consensus Classification (ICC) retained the term aCML [13,14]. Diagnosis relies on the integration of clinical, hematological, morphological, cytogenetic, and molecular findings, including neutrophil-predominant leukocytosis, marked dysgranulopoiesis, the presence of circulating granulocytic precursors, and the absence of BCR::ABL1 fusion. Molecular abnormalities, which are frequently observed in this entity, contribute to clonal characterization and differentiation from other myeloid neoplasms [1-7,11,15,16].

In our case, the clinical presentation was dominated by unusual bleeding manifestations, including hemopericardium complicating post-influenza pericarditis and diffuse cutaneous ecchymoses. This discrepancy between the severity of the bleeding manifestations and the platelet count (145 × 10^9/L, only marginally below the reference range) suggests that a qualitative abnormality of platelet function may have contributed to the bleeding phenotype. Light transmission aggregometry effectively demonstrated acquired platelet dysfunction, highlighting that bleeding risk in myeloid neoplasms depends not only on platelet count but also on platelet functional quality [1,8,10,12,17].

The mechanisms underlying acquired platelet dysfunction in myeloid neoplasms remain incompletely understood. Several mechanisms have been proposed, including abnormalities in megakaryopoiesis and platelet production, as well as disturbances in intracellular pathways involved in platelet activation and aggregation. Increased platelet cyclic adenosine monophosphate (cAMP) levels have been reported in certain other myeloid malignancies associated with acquired platelet dysfunction, suggesting a possible role for intracellular inhibitory pathways of platelet activation [8,17]; this pathway was not investigated in our patient. These findings suggest that platelet functional abnormalities may result from complex mechanisms involving both clonal hematopoiesis and intrinsic disturbances of platelet signaling pathways.

Light transmission aggregometry is considered a reference method for the investigation of acquired platelet function disorders [10]. In our patient, light transmission aggregometry demonstrated markedly impaired responses to arachidonic acid (3.5%) and collagen (16.5%), whereas the response to ADP (54.7%) was relatively better preserved. The response to ristocetin was 63.6%. This profile demonstrates an abnormality of platelet function, particularly pronounced in response to arachidonic acid, compatible with impairment of the arachidonic acid-thromboxane pathway [8,10,17]. Aspirin and other NSAIDs produce the same pattern, and uremia also impairs platelet function [10]; neither was applied here, as the patient was not taking these drugs and her renal function was normal. This abnormality provides one plausible contributor to the bleeding manifestations observed in our patient. However, the absence of complementary data regarding intracellular platelet mechanisms prevents precise identification of the underlying mechanism, and a causal relationship cannot be established from a single case.

The prolonged aPTT remains unexplained. Factors VIII, IX, XI, and XII were normal or increased, and no mixing study was available to look for an inhibitor. The mild factor XIII decrease (62.6%), the pericardial inflammation, and the previous uterine bleeding may also have contributed. Platelet dysfunction is therefore a possible contributory factor rather than the established cause of the bleeding.

Molecular analysis identified two TET2 mutations and one KRAS mutation. These abnormalities are consistent with the complex molecular profile described in myeloid neoplasms and are compatible with the genetic diversity observed in aCML/MDS/MPN with neutrophilia [11,16,18,19], but they support clonality without being specific to this entity. TET2 mutations are involved in the epigenetic regulation of

hematopoiesis, whereas KRAS mutations affect the RAS/MAPK signaling pathway. However, in our case, these mutations should primarily be considered as components of the clonal context of the disease. Although an indirect influence of molecular abnormalities on hematopoietic cell differentiation and platelet function may be hypothesized, no direct causal relationship between the identified TET2/KRAS mutations and the observed platelet dysfunction can be established based on this single case [11,16,19].

Comparison with the available literature shows that bleeding manifestations associated with myeloid neoplasms may result from qualitative abnormalities of platelet function and may occur despite a normal or only moderately decreased platelet count [1,8]. In our case, the presence of objectively demonstrated platelet dysfunction despite a platelet count of 145 × 10^9/L, only marginally below the reference range, further supports this concept. In contrast, hemopericardium represents an unusual and potentially severe bleeding manifestation. A PubMed search (inception to 28 October 2026, no language restriction) using the query ("atypical chronic myeloid leukemia" OR "atypical chronic myeloid leukaemia" OR "atypical chronic myelogenous leukemia" OR "atypical CML" OR "MDS/MPN with neutrophilia" OR "myelodysplastic/myeloproliferative neoplasm with neutrophilia") AND (hemopericardium OR haemopericardium OR pericardial OR pericardium OR "platelet dysfunction" OR "platelet function" OR "platelet aggregation" OR aggregometry) retrieved four records. None described hemopericardium or aggregometry-documented platelet dysfunction in this entity; the only pericardial involvement reported was extramedullary hematopoiesis with tamponade [20]. To our knowledge, this is the first such report.

Management of MDS/MPN with neutrophilia remains challenging, particularly in elderly patients with comorbidities. Allogeneic hematopoietic stem cell transplantation is currently the only potentially curative approach, but its use is limited by age, general condition, comorbidities, transplantation-related risk, and disease characteristics [1,3,4,7]. In our patient, given her advanced age and comorbidities, low-dose cytoreductive therapy with hydroxyurea was initiated, combined with regular clinical and laboratory monitoring. After three months of follow-up, leukocytosis remained stable, and no further episode of hemopericardium was observed. However, this limited follow-up does not allow any conclusion regarding a specific effect of hydroxyurea on platelet dysfunction or bleeding risk. Targeted therapeutic strategies and approaches based on molecular abnormalities remain areas of investigation, but their role in preventing or correcting hemostatic abnormalities remains to be clarified [1,3,4].

In conclusion, our case documents acquired platelet dysfunction in MDS/MPN with neutrophilia presenting with severe bleeding, including hemopericardium. It emphasizes that bleeding risk in myeloid neoplasms depends not only on platelet count but may also be related to qualitative impairment of platelet function. When the severity of bleeding is disproportionate to the platelet count, functional platelet testing, particularly by aggregometry, may therefore provide important diagnostic information. Further studies are needed to better understand the mechanisms linking clonal hematopoiesis, molecular abnormalities, megakaryopoiesis, and platelet function in this rare hematological malignancy.

Conclusions

This case highlights the importance of investigating acquired platelet dysfunction in patients with MDS/MPNs with neutrophilia (MDS/MPN with neutrophilia), particularly when bleeding manifestations appear disproportionate to a platelet count only marginally below the reference range. In our patient, light transmission aggregometry demonstrated markedly impaired platelet aggregation, a possible contributory factor alongside the unexplained aPTT prolongation, low factor XIII, and prior uterine bleeding.

A comprehensive hemostatic evaluation, including platelet function testing, medication history and renal function, may therefore provide additional diagnostic information in patients presenting with unexplained or disproportionate bleeding. Further studies are needed to elucidate the underlying mechanisms of platelet dysfunction in MDS/MPN with neutrophilia and to determine whether these abnormalities have prognostic or therapeutic implications.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Mohamed Ilyass Tazi, Hassane Mamad, Naoufal Benlachgar, Souad Benkirane, Azlarab Masrar, Jalila Zirar, Mohamed Ifleh

Acquisition, analysis, or interpretation of data: Mohamed Ilyass Tazi, Hassane Mamad, Naoufal Benlachgar, Souad Benkirane, Azlarab Masrar, Jalila Zirar, Mohamed Ifleh

Drafting of the manuscript: Mohamed Ilyass Tazi, Hassane Mamad, Naoufal Benlachgar, Souad Benkirane, Azlarab Masrar, Jalila Zirar, Mohamed Ifleh

Critical review of the manuscript for important intellectual content: Mohamed Ilyass Tazi, Hassane Mamad, Naoufal Benlachgar, Souad Benkirane, Azlarab Masrar, Jalila Zirar, Mohamed Ifleh

Supervision: Mohamed Ilyass Tazi, Naoufal Benlachgar, Souad Benkirane, Azlarab Masrar

Disclosures

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References

1. Szuber N, Orazi A, Tefferi A: Chronic neutrophilic leukemia and atypical chronic myeloid leukemia: 2024 update on diagnosis, genetics, risk stratification, and management. Am J Hematol. 2024, 99:1360-87. 10.1002/ajh.27321
2. Danyal K, Devitt K, Gardner JA: Solving the puzzle: the diagnosis of atypical chronic myeloid leukemia, BCR-ABL1-negative (aCML). J Assoc Genet Technol. 2021, 47:127-31.
3. Diamantopoulos PT, Viniou NA: Atypical chronic myelogenous leukemia, BCR-ABL1 negative: diagnostic criteria and treatment approaches. Front Oncol. 2021, 11:722507. 10.3389/fonc.2021.722507
4. Castellino A, Santambrogio E, Rapezzi D, Massaia M: Atypical chronic myeloid leukemia: new developments from molecular diagnosis to treatment. Medicina (Kaunas). 2021, 57:10.3390/medicina57101104
5. Sadigh S, Hasserjian RP, Hobbs G: Distinguishing atypical chronic myeloid leukemia from other Philadelphia-negative chronic myeloproliferative neoplasms. Curr Opin Hematol. 2020, 27:122-7. 10.1097/MOH.0000000000000565
6. Sun Y, Wang Q, Zhang Z, et al.: Distinct clinical profiles and patient outcomes in aCML and CNL . Ann Hematol. 2024, 103:5325-32. 10.1007/s00277-024-06032-z
7. Dhakal P, Gundabolu K, Amador C, Rayamajhi S, Bhatt VR: Atypical chronic myeloid leukemia: a rare entity with management challenges. Future Oncol. 2018, 14:177-85. 10.2217/fon-2017-0334
8. Lecchi A, Femia EA, La Marca S, Onida F, Artoni A: Acquired platelet dysfunction and overproduction of platelet cyclic AMP in two patients with myeloid malignancies. Platelets. 2019, 30:1053-6. 10.1080/09537104.2019.1636020
9. Mair Y, Zheng Y, Cai D: Dysmegakaryocytopoiesis and maintaining platelet count in patients with plasma cell neoplasm. N Am J Med Sci. 2013, 5:316-9. 10.4103/1947-2714.112474
10. Brennan Y, Levade M, Ward CM: Acquired platelet function disorders . Thromb Res. 2020, 196:561-8. 10.1016/j.thromres.2019.06.009
11. Li B, Gale RP, Xiao Z: Molecular genetics of chronic neutrophilic leukemia, chronic myelomonocytic leukemia and atypical chronic myeloid leukemia. J Hematol Oncol. 2014, 7:93. 10.1186/s13045-014-0093-1
12. Linnik YA, Salvatore LT, Lowrey CH, Ornstein DL: Severe acquired platelet dysfunction because of primary myelofibrosis with full functional and morphological recovery after allogeneic hematopoietic cell transplantation. Blood Coagul Fibrinolysis. 2019, 30:419-22. 10.1097/MBC.0000000000000850
13. Khoury JD, Solary E, Abla O, et al.: The 5th edition of the World Health Organization Classification of Haematolymphoid Tumours: myeloid and histiocytic/dendritic neoplasms. Leukemia. 2022, 36:1703-19. 10.1038/s41375-022-01613-1
14. Arber DA, Orazi A, Hasserjian RP, et al.: International Consensus Classification of myeloid neoplasms and acute leukemias: integrating morphologic, clinical, and genomic data. Blood. 2022, 140:1200-28. 10.1182/blood.2022015850
15. Schwartz LC, Mascarenhas J: Current and evolving understanding of atypical chronic myeloid leukemia . Blood Rev. 2019, 33:74-81. 10.1016/j.blre.2018.07.004
16. Faisal M, Stark H, Büsche G, et al.: Comprehensive mutation profiling and mRNA expression analysis in atypical chronic myeloid leukemia in comparison with chronic myelomonocytic leukemia. Cancer Med. 2019, 8:742-50. 10.1002/cam4.1946
17. Rao AK: Molecular and biochemical basis for the platelet dysfunction in myeloproliferative disorders . Semin Hematol. 2004, 41:6-9. 10.1053/j.seminhematol.2004.02.010
18. Seth N, Brody J, Hsu P, Kolitz J, Deb PQ, Zhang X: Expanding the spectrum of CSF3R-mutated myeloid neoplasm beyond chronic neutrophilic leukemia and atypical chronic myeloid leukemia: a comprehensive analysis of 13 cases. J Clin Med. 2025, 14:10.3390/jcm14155174
19. Gao T, Yu C, Xia S, Liang T, Gu X, Liu Z: A rare atypical chronic myeloid leukemia BCR-ABL1 negative with concomitant JAK2 V617F and SETBP1 mutations: a case report and literature review. Ther Adv Hematol. 2020, 11:2040620720927105. 10.1177/2040620720927105
20. Toms DR, Cannick L, Stuart RK, Jenrette JM, Terwiliger L: Helical tomotherapy for extramedullary hematopoiesis involving the pericardium in a patient with chronic myeloid leukemia. Jpn J Radiol. 2010, 28:476-8. 10.1007/s11604-010-0452-y