Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

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Plasmacytosis in Acute Myeloid Leukemia: A Diagnostic Challenge

Mona Jabrane[1*], Maria Jaimi[1], Wafaa Quiddi[1], Sanaa Sayagh[1]

[1]Biological Hematology Laboratory, Arrazi Hospital, Mohamed VI University Hospital Center, Marrakech, Morocco

DOI: https://doi.org/10.36347/sjmcr.2025.v13i05.069 | **Received:** 03.04.2025 | **Accepted:** 09.05.2025 | **Published:** 20.05.2025

***Corresponding author:** Mona Jabrane
Biological Hematology Laboratory, Arrazi Hospital, Mohamed VI University Hospital Center, Marrakech, Morocco

Abstract | **Case Report**

***Introduction*:** Bone marrow plasmacytosis is an uncommon finding at the time of acute myeloid leukemia (AML) diagnosis and may mimic plasma cell dyscrasias, potentially leading to diagnostic confusion. ***Case Presentation*:** We report the case of a 43-year-old man who presented with pancytopenia and was found to have 35% myeloid blasts and 22% mature plasma cells on bone marrow aspirate. No monoclonal protein was identified, and flow cytometry revealed co-expression of plasma cell and myeloid markers. Cytogenetic analysis revealed t(8;21). A diagnosis of AML with marked reactive plasmacytosis was established. ***Discussion*:** Marked plasmacytosis at AML onset is rare. It may result from cytokine-mediated immune activation. Distinguishing reactive from clonal plasmacytosis is critical to avoid misdiagnosis with plasma cell neoplasms such as multiple myeloma. ***Conclusion*:** This case highlights the diagnostic complexity posed by AML with associated plasmacytosis and underscores the importance of a multimodal diagnostic approach incorporating morphology, immunophenotyping, cytogenetics, and protein studies.

Keywords: Acute Myeloid Leukemia (AML), Plasmacytosis, Bone Marrow, Diagnostic Confusion, Reactive Plasmacytosis.

INTRODUCTION

Acute myeloid leukemia (AML) is a clonal hematopoietic malignancy characterized by the accumulation of immature myeloid precursors in the bone marrow, leading to hematopoietic failure. While AML typically presents with symptoms related to cytopenias, it can sometimes be associated with atypical bone marrow findings that complicate diagnosis.

One such rare finding is plasmacytosis, defined as an increased percentage of plasma cells in the bone marrow. Plasmacytosis is usually observed as a reactive phenomenon during marrow regeneration post-chemotherapy, triggered by immune or inflammatory stimuli. However, its presence at the time of initial AML diagnosis is uncommon and may resemble plasma cell disorders, such as multiple myeloma or monoclonal gammopathy of undetermined significance (MGUS).

In this report, we present a rare case of newly diagnosed AML associated with marked plasmacytosis, illustrating the diagnostic challenges and the importance of a comprehensive diagnostic workup.

CASE PRESENTATION

A 43-year-old male was referred to our institution for evaluation of suspected megaloblastic anemia. He reported progressive fatigue and generalized weakness. On physical examination, he appeared pale but had no palpable lymphadenopathy or organomegaly. There was no history of hematological disorders in the patient or his family.

Initial laboratory findings showed pancytopenia: hemoglobin 3.1 g/dL, VGM :130 fl, white blood cell count 8.4 × 10^9/L, and platelet count 45 × 10^9/L. Peripheral blood smear revealed 8% circulating blasts. C-reactive protein was elevated (55 mg/dL). Serum vitamin B12 and folate levels were reduced (140 pg/mL and 2 µg/L, respectively). Viral serologies, including HIV, hepatitis B and C, were negative.

Bone marrow aspirate demonstrated hypercellular marrow with 35% myeloblasts and 22% mature plasma cells, some showing binucleation and cytoplasmic vacuolation. These findings raised the possibility of concurrent AML and plasma cell dyscrasia.

Serum protein electrophoresis (SPE) revealed a polyclonal increase in gamma globulins without

evidence of monoclonal bands in serum or urine. Myeloperoxidase (MPO) staining confirmed myeloid lineage in over 3% of blasts. Cytogenetic analysis showed a complex karyotype, including the recurrent translocation t(8;21) (q22;q22). Flow cytometry demonstrated that the blasts were positive for CD34, CD117, and HLA-DR. A subset also expressed plasma cell-associated markers CD38 and CD138.

Given the polyclonal nature of immunoglobulin production, absence of monoclonal protein, and the immunophenotypic and cytogenetic profile, a final diagnosis of AML with marked reactive plasmacytosis was made.

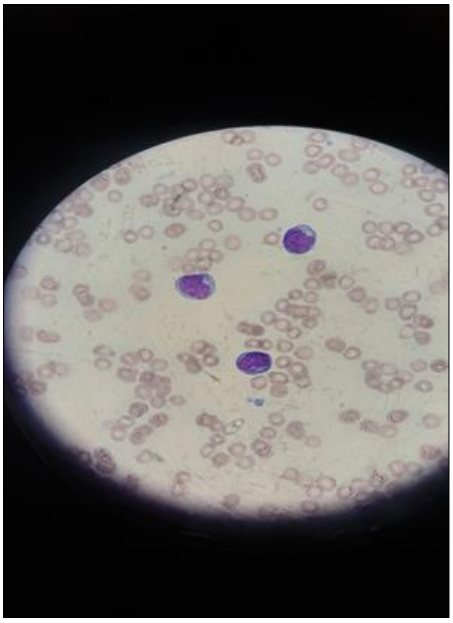

Figure 1: Peripheral blood smear showing blast cells (wright x100)

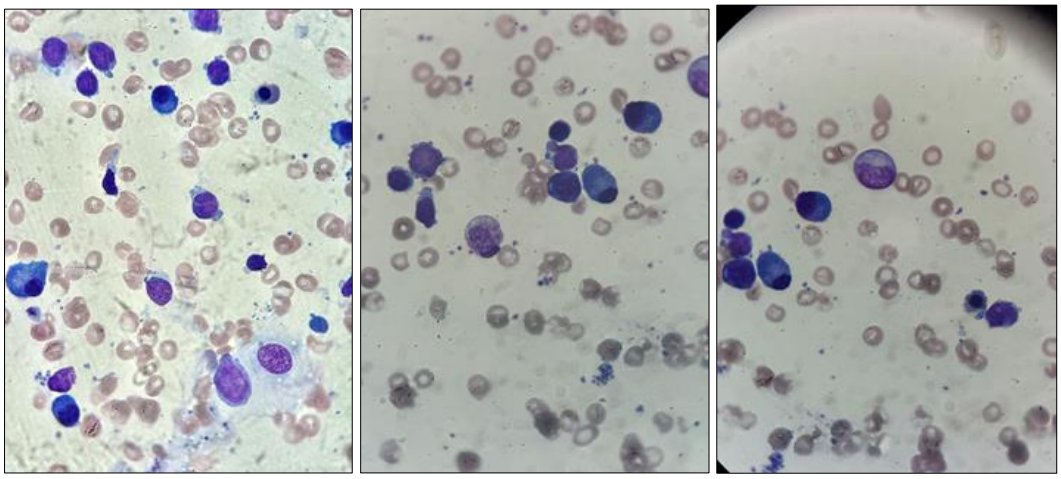

Figure 2,3,4: Bone marrow aspirate: Blast cells are seen along with many plasma cells (Giemsa stain, x100)

DISCUSSION

Plasmacytosis is defined as the presence of >10% plasma cells in the bone marrow. While this finding is characteristic of plasma cell neoplasms, it may also occur in a wide range of reactive conditions, such as viral infections, autoimmune diseases, liver cirrhosis, and other malignancies. In AML, plasmacytosis is typically encountered during marrow recovery following chemotherapy and is seldom seen at diagnosis.

In our case, the marked plasmacytosis (22%) initially raised concern for a concomitant plasma cell neoplasm. However, the absence of monoclonal gammopathy, polyclonal elevation of gamma globulins, and lack of end-organ damage (renal impairment, hypercalcemia, or bone lesions) argued against multiple myeloma. Additionally, the plasma cells did not display atypical morphology often seen in clonal processes.

Similar cases have been rarely reported in the literature. When present, plasma cell infiltration in AML generally remains below 10%. Cases with >20% plasma

cells at presentation, like ours, are exceedingly rare and underreported. This emphasizes the need for careful interpretation of marrow findings to avoid misdiagnosis.

The exact mechanism behind reactive plasmacytosis in AML is unclear. A likely explanation involves cytokine production by leukemic blasts—particularly interleukin-6 (IL-6), which promotes B-cell differentiation into plasma cells. Some studies have shown elevated IL-6 levels in AML patients with plasmacytosis, supporting a cytokine-driven paracrine effect. Other cytokines, including IL-1 and TNF-α, may also contribute.

Another layer of complexity arises from aberrant antigen expression. In our patient, flow cytometry revealed co-expression of plasma cell markers (CD38, CD138) in some blasts. This may reflect lineage promiscuity or antigen acquisition, a phenomenon occasionally observed in AML. However, without evidence of clonal immunoglobulin gene rearrangement or monoclonal protein production, these findings do not support a diagnosis of plasma cell neoplasm.

This case underscores the critical role of a multimodal diagnostic approach—including cytomorphology, flow cytometry, cytogenetics, and serum/urine protein studies—to accurately classify hematological malignancies. Misinterpreting reactive plasmacytosis as a plasma cell disorder could delay AML-specific treatment and compromise patient outcomes.

Regarding prognosis, the clinical significance of reactive plasmacytosis in AML remains uncertain. Some reports suggest it may reflect a preserved immune response and confer a better prognosis, while others associate it with aggressive disease. Further research is needed to clarify its prognostic impact.

CONCLUSION

Plasmacytosis at the time of AML diagnosis is rare and diagnostically challenging. When present in high proportions, it can mimic plasma cell neoplasms and mislead clinicians. Differentiating between reactive and clonal plasmacytosis requires a thorough workup, integrating morphological, immunophenotypic, cytogenetic, and protein studies.

This case illustrates the importance of maintaining a broad differential diagnosis and highlights the value of combining multiple diagnostic modalities. Awareness of this rare presentation is essential to avoid misclassification and ensure timely and appropriate therapy. Further studies are warranted to explore the pathophysiological basis and prognostic implications of reactive plasmacytosis in AML.

REFERENCES

- Dodhy MA, Habib M, Shaheen M, Abbasi M. Acute Monocytic Leukaemia with Reactive Plasmacytosis. Int J Pathol. 2018:94–5.
- Döhner H, Estey E, Grimwade D, et al. Diagnosis and management of AML in adults: 2017 ELN recommendations. Blood. 2017;129(4):424–447.
- Hameed M, Raziq F. Reactive Plasmacytosis: A Diagnostic Conundrum In Acute Myeloid Leukaemia. J Ayub Med Coll Abbottabad. 2021;33:335–8.
- Jurczyszyn A, Radocha J, Davila J, et al. Prognostic indicators in primary plasma cell leukaemia: a multicentre retrospective study of 117 patients. Br J Haematol. 2018;180:831–9.
- Stul MS, Bergmans GB, Logghe GN, Vanvuchelen JK, Louwagie AC. Bone Marrow Plasmocytosis in Acute Leukemia. Acta Haematol. 2009;77:146–9.
- Wulf GG, Jahns-Streubel G, Hemmerlein B, et al. Plasmacytosis in acute myeloid leukemia: two cases and IL-6 production in AML blasts. Ann Hematol. 1998;76:273–7.