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– CASE REPORT –

Plasma Cell Dyscrasia Masquerading Acute Leukemia: A Diagnostic Challenge

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Abstract

Introduction*: Increase in bone marrow plasma cells can be seen in many conditions including reactive as well as neoplastic. High marrow plasma cell count can present a diagnostic dilemma.*

Case report*: We present a case of acute T-cell lymphoblastic leukemia initially presenting as bone marrow plasmacytosis (60%) and reported as plasma cell dyscrasia on morphology on bone marrow aspiration. Reason for reactive plasmacytosis may be due to release of paracrine growth factor especially interleukin (IL-6) from the leukemic cells.*

Conclusion*: We reported this case to highlight that transient plasmacytosis can be present in hematological malignancies and therefore, proper workup should be done to avoid the diagnosis of plasma cell dyscrasisas, which can predispose the patient to wrong treatment and hamper/ delay the treatment required.*

Keywords*: Acute leukemia, Cytokines, Plasma cells*

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Introduction

Increase in bone marrow plasma cells can be seen in many conditions including reactive as well as neoplastic.[1,2] Reactive bone marrow plasmacytosis usually consist of 10% to 20% of the nucleated cells.[3,4] It is important to differentiate reactive plasmacytosis in non-neoplastic conditions from the malignant ones. High marrow plasma cell count can present a diagnostic dilemma.[5] Here, we present the case of acute T-cell lymphoblastic leukemia initially presenting as bone marrow plasmacytosis comprising plasma cells around 60% of the nucleated cells, which was reported as plasma cell dyscrasia on bone marrow aspiration.

Case presentation

A 32 year old female patient presented with complaint of sore throat in medicine outpatient department 5 months back. She was a known case of diabetes mellitus type 2, on oral antidiabetic drugs. Patient received antibiotic for sore throat. After 2 months she had an episode of acute suppurative otitis media, for which she was admitted in a private hospital and investigated and found to have bicytopenia. Due to no obvious cause of bicytopenia bone marrow aspiration (BMA) was done which was suggestive of dyserythropoiesis and presence of 10% mononuclear cells of undetermined significance. Following this the patient was referred to our institute and investigated further. Peripheral blood film showed bicytopenia with presence of 13% atypical lymphoid

cells/blasts (Table 1) and repeat BMA revealed hypercellular marrow with reduced myeloid and erythroid precursors and presence of more than 50% plasma cells. The plasma cells were mostly mature, but a small number of binuclear and atypical forms were also observed. Hence based on morphology on BMA case was reported as plasma cell dyscrasia. Patient had given supportive treatment and advised for further work up including bone scan and serum protein electrophoresis, which patient refused and lost to follow up.

	PBF and 1st BMA findings	PBF and 2nd BMA Findings	PBF and 3rd BMA Findings
CBC			
Hb(gm/dl)	6.7	6.7	9.4
TLC(/cumm)	2650	1600	4200
DLC(%)			
• Blasts	00	00	85
• Neutrophils	13	20	10
• Lymphocytes	80	76	05
• Monocytes	06	02	00
• Eosinophils	01	02	00
• Basophils	00	00	00
Platelet Count(/microltr)	125	160	200
PBF	Dimorphic blood picture	Dimorphic blood picture.Few atypical cells.	Dimorphic blood picture.
BMA Findings	Dyserythropoeisis There is presence of mononuclear cells(10%)?lymphoid?plasma ?primitive erythropoeitic cells	Plasma cell dyscrasia	Acute Leukemia
Immunophenotyping findings	Not performed	Not performed	CD45: Dim positive Tdt: Positive CD34: Positive CD4: Positive CD5: Positive CD117: Positive HLA-DR: Positive CD13: Positive CD33: Positive cCD3: Positive

Table 1. *Peripheral Blood Findings (PBF), Bone Marrow Aspirate (BMA) and Immunophenotyping findings*

Again, after one-month patient presented with the complaints of fever, cough, lymphadenopathy (multiple, including cervical and retroperitoneal) and hepatosplenomegaly. Complete hemogram revealed presence of 85% atypical lymphoid cells/blasts. (Table 1). Bone marrow aspirate and biopsy showed complete replacement of marrow by blast cells pertaining to morphology of lymphoid cells with presence of 10% plasma cells. The blasts on immunophenotyping were positive for T-cell markers and immunohistochemistry of bone marrow biopsy was also positive for CD34, CD7 and CD117 (focal). Fine needle aspiration of cervical lymph node revealed reactive hyperplasia. Skeletal survey did not show any lytic lesions. Urine examination for Bence Jones proteins was negative. Serum electrophoresis revealed hypoalbuminemia and a polyclonal increase in gamma globulins conforming the reactive nature of plasma cells.

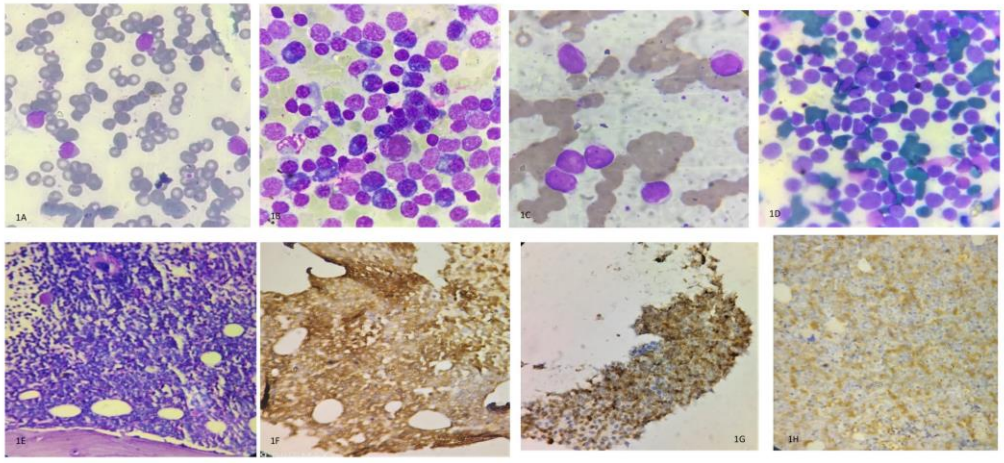

Figure 1A & 1B: *Atypical cells on peripheral blood film and plasma cells on Bone Marrow Aspirate (IInd BMA) Figure 1C & 1D : Presence of blasts on peripheral blood film and on BMA (IIIrd BMA) Figure 1E & 1F : Bone marrow biopsy revealed replacement of marrow by blast cells and on IHC: CD 7 positive in blasts (10X(H&E) and 40X(CD7)) Figure 1G & 1H : On IHC: Blasts positive for CD34(10X) and CD117(40X)*

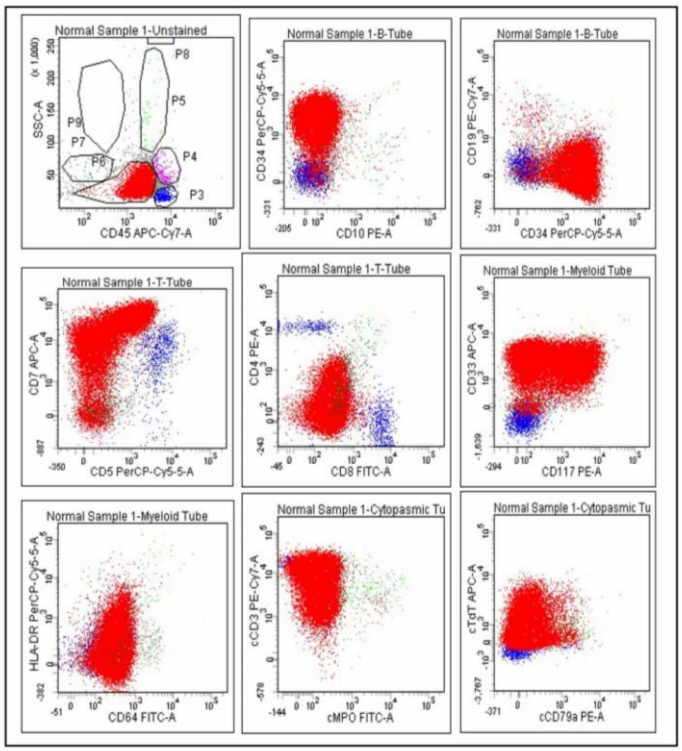

Figure 2: *Immunophenotyping findings. SSC/CD45 gating showed CD45 (Dim), CD34, CD7, CD5, CD4 (Dim), CD117, CD33, HLA-DR, cCD3 and Tdt positive while CD10, CD19, CD64, CD8, CD79a and cMPO negative*

he previous BMA were reviewed in view of recent marrow findings and found to have 5-6% mononuclear hematolymphoid cells of undermined significance which were overlooked due to marked plasmacytosis. Patient was on induction therapy (vincristine, prednisolone , daunorubicin and asparaginase). However, Patient expired during induction therapy .

Discussion

Reactive BM plasmacytosis is characterized by an increase in the percentage of plasma cells above the normal, polyclonal in nature and distributed diffusely in the marrow. The mechanism of polyclonal proliferation of plasma cells may be a defense response to the underlying disease process, infection or abnormal immune response which secrete cytokines such as IL-6 or IL-10 which are known to stimulate plasma cell generation.[6]

Wolf et al suggest that paracrine growth stimulation of plasma cells by paraneoplastic IL-6 production of the leukemic blast cells contributes to the plasmacytosis observed in patients with AML. [7]

Gupta et al [8] represent the data related to degree of plasmacytosis in various conditions showing less than 10% plasmacytosis in various types of anemia, 10- 30% plasma cells in infections and hypoplastic marrows. In reactive conditions, the percentage range was 5%- 24% and in cases of plasma cell dyscrasias the range varied from 20- 40%. Plasma cells were chiefly mature in non-neoplastic conditions while in plasma cell dyscrasias mature and immature plasma cells were found.

In our case patient presented with bicytopenia with presence of 60% plasma cells in bone marrow. Diagnosis of plasma cell dyscrasias was suggested based on morphological findings only and other relevant investigations were advised. Generally, reactive plasma cell proliferation seen in bone marrow does not exceed 20%. In the present case, 50-60% plasma cells were observed which is unusual for reporting as reactive plasmacytosis and leads to diagnostic dilemma. However, later on these plasma cells turn out to be reactive in nature. As patient was known diabetic with recent history of acute suppurative otitis media, which may be responsible for reactive plasmacytosis in our case.

Ozdemırkıran et al have shown similar findings in a patient who presented with pancytopenia and hypoplasia of bone marrow with presence of 96% plasma cells, it was reported as plasma cell dyscrasias based on morphological findings but protein electrophoresis and bone marrow immunohistological examination revealed polyclonal protein restriction. They concluded that when pancytopenia and B-cell proliferation present together it may be a sign of lymphoproliferative disease or other hematological malignancies, and such patients should be followed up closely. [6]

Plasmacytosis has also been observed in few cases in literature associated with AML at the time of diagnosis. Plasma cells usually do not exceed 10% in these cases. However, there are very few cases where the plasma cell count is found to be higher than 20% in newly diagnosed acute leukemias.[7][9,10]

Literature searched also revealed, two pediatric cases of Acute Lymphoblastic Leukemia (ALL) having pancytopenia, bone marrow aplasia and polyclonal B-cell proliferation initially. Later on presented with ALL, may be attributed to newly developed chromosomal anomalies which may explain both pancytopenia and ALL. [11] However; association between B-cell proliferation and ALL has not been clear. Another possible explanation, could be persistent EBV infection leading to ALL with pancytopenia and polyclonal B-cell proliferation had been reported by Finlay et al. [12]

Jamal [13] and Ali et al [14] suggested that the etiology of reactive bone marrow plasmacytosis may be due to release of paracrine growth factor, interleukin (IL-6) from the leukemic cells in AML.

Jabrane et al [15] also supported the role of cytokine (IL-6) along with aberrant antigen expression on blasts (CD38, CD138) which reflect lineage promiscuity or antigen acquisition, a phenomenon occasionally observed in AML.

Studies related to association between T ALL and reactive plasmacytosis are scarce. However; we also came to the conclusion that in our case, presence of chronic disease (diabetes), recent ASOM and leukemic blasts may contribute in the process by releasing the cytokines which play an important role in reactive plasmacytosis.

Conclusion

Marked proliferation of plasma cells can be observed in various infections and immune reactions, which needs to be investigated to differentiate the etiology into infectious, immunological and neoplastic. As in our case, initially presented as bicytopenia with features of dyserythropoiesis and reported as a plasma cell dyscrasia which finally diagnosed as acute T-cell lymphoblastic leukemia. Such cases need to be reported to understand that transient plasmacytosis can be present in hematological malignancies and therefore, proper workup should be done to avoid the diagnosis of plasma cell dyscrasisas, which can predispose the patient to wrong treatment and hamper/ delay the treatment required.

Ethics Statement and Conflict of Interest Disclosures

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Ethics Consideration: The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as well as the national laws. Written informed consent was provided by the patient participant in this study.

Conflict of interest: No known conflict of interest correlated with this publication.
Availability of data and materials: The data used and/ or analyzed throughout this study are available from the corresponding authors upon reasonable request.
Competing interests: The authors declared that they have no competing interests.
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