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Case Report

Hemophagocytosis in Acute Monoblastic Leukemia with a Complex Karyotype: A Case Report

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Abstract: Hemophagocytosis is a rare feature in leukemic blasts, occurring in approximately 1% of acute myeloid leukemias (AML), mainly in the monoblastic or monocytic subtypes [1,2]. Associations with chromosomal abnormalities, particularly *t(8;16)*, have been reported, and the presence of hemophagocytic blasts can help suggest such cytogenetic alterations [2]. Unlike macrophage activation syndrome (MAS), no specific treatment is usually indicated for patients presenting hemophagocytic blasts [3]. We report a rare case of hemophagocytosis occurring in acute monoblastic leukemia with a complex karyotype associated with a poor prognosis according to the European LeukemiaNet (ELN) 2022 risk classification [4]. The clinical course was complicated by disseminated intravascular coagulation (DIC), and the patient died on the fourth day of induction therapy, emphasizing the adverse nature of this entity.

Keywords: Monoblast Hemophagocytosis, AML-M5, Complex Karyotype, Disseminated Intravascular Coagulation.

INTRODUCTION

Hemophagocytosis, defined as the engulfment of hematopoietic cells by leukemic myeloid blasts, is a rare finding in acute myeloid leukemia (AML) [1]. It is mainly described in acute monocytic or monoblastic leukemia. The t(8;16) (p11;p13) translocation, although uncommon, is frequently associated with monocytic AML presenting hemophagocytosis [2]. Here, we report a case of acute monoblastic leukemia (AML-M5) showing blast hemophagocytosis and a complex karyotype harboring a t(5;21) (q13;q22) translocation, a cytogenetic abnormality rarely reported in myeloid malignancies [4, 5].

CASE PRESENTATION

A 34-year-old man, with no significant past medical history, was admitted to the emergency department for abdominal pain. Clinical examination revealed a fever of 39°C, gingival bleeding, petechiae on the trunk, and hepatosplenomegaly.

The complete blood count revealed leukocytosis at 68.4×10^9/L with monocytosis at 5×10^9/L, hemoglobin at 13.1 g/dL, and thrombocytopenia at 16×10^9/L.

The peripheral smear, stained with May–Grünwald–Giemsa, showed 80% large blasts with abundant, often vacuolated cytoplasm, along with promonocytes and monocytes. Myeloperoxidase (MPO) staining was positive in 60% of blasts.

Coagulation studies showed prothrombin time (PT) 43%, INR 1.82, and activated partial thromboplastin time (aPTT) ratio 1.41.

A bone marrow aspiration was performed for cytologic, immunophenotypic, and cytogenetic analysis. The marrow was hypercellular, infiltrated by 73% blasts and monoblasts, with a monocytic component representing 20% (promonocytes and monocytes). MPO reaction was positive in 62% of blasts.

Notably, several monoblasts were observed engulfing erythrocytes and platelets, consistent with hemophagocytosis. These findings supported a diagnosis of AML-M5 with hemophagocytosis, according to the French-American-British (FAB) classification.

Flow cytometry (Beckman Coulter Navios) revealed 21% of cells expressing HLADR$^+$, CD13$^+$,

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$CD117^+$, $CD14^+$, $CD11^+$, partial $CD7^+$, MPO^+, and $CD34^-$.

Cytogenetic analysis demonstrated a complex karyotype with multiple numerical and structural abnormalities, including a *t(5;21) (q13;q22)* translocation—a rare alteration reported in myeloid proliferations.

The clinical course was marked by the development of biological DIC (D-dimers 65,000 ng/L, Factor V deficiency at 22%, PT 43%) and leukostasis (WBC 142×10^9/L). The patient received standard induction chemotherapy (7+3 regimen: cytarabine continuous IV for 7 days and daunorubicin IV for 3 days), combined with hydration and allopurinol to prevent tumor lysis syndrome. Platelet and fresh frozen plasma transfusions were also given to correct DIC. Unfortunately, the patient died on day 4 of induction following sudden respiratory distress of unexplained origin.

DISCUSSION

Blast hemophagocytosis is most often described in monocytic leukemias (AML-M4/M5), and rarely in other AML subtypes such as M1, M2, or M7. It has also been sporadically reported in non-hematologic malignancies [1, 2].

The specificity of blast hemophagocytosis remains debated. In contrast to macrophage activation syndrome (MAS), no specific investigations or treatments are generally required. Both phenomena can, however, coexist in the same patient.

The underlying pathophysiological mechanism remains poorly understood, although several hypotheses involve aberrant monocytic differentiation and cytokine dysregulation [3].

Monocytic leukemias with hemophagocytosis are frequently associated with the *t(8;16) (p11;p13)* translocation. Other variants have been reported, including *t(3;8;17) (q27;p11;q12)*, *inv(8) (q11q13)*, *t(16;21) (p11;q22)*, and *t(10;17) (p13;q12)*.

Some studies have demonstrated that leukemic cells with *t(16;21)* express stem cell antigens such as CD34 and c-Kit, with partial monocytic differentiation allowing phagocytic activity. Moreover, the presence of hemophagocytic monoblasts can be an early morphological clue to the presence of *t(8;16)* or *t(16;21)* translocations, emphasizing the diagnostic value of cytology before immunophenotypic or cytogenetic confirmation [2-5].

These chromosomal rearrangements are generally associated with a poor prognosis, with nearly half of patients dying within 10 months of diagnosis, highlighting the importance of early recognition [2-5].

Our patient's complex karyotype, involving more than three structural and numerical abnormalities, corresponds to an adverse-risk AML according to the European LeukemiaNet (ELN) 2022 classification [4]. The *t(5;21) (q13;q22)* translocation is extremely rare; only two cases of AML with *t(5;21) (q13;q21)* have been reported, none with hemophagocytosis [6].

Blast phagocytosis may occur at disease onset, as in our case, or during treatment. The emergence of cytogenetic abnormalities in patients initially presenting with a normal karyotype may result from genetic instability or chemotherapy-induced toxicity [7].

Unlike MAS, blast hemophagocytosis is usually observed before treatment initiation. No specific therapy is recommended, and the phenomenon is considered a morphologic expression of leukemic behavior rather than an independent pathological process.

Despite its frequent association with poor outcome, blast hemophagocytosis is not yet included in prognostic scoring systems, although the repeated observation of unfavorable evolution in reported cases suggests that it deserves further study [6].

CONCLUSION

Although hemophagocytosis is associated with an unfavorable prognosis in acute monoblastic leukemia, no specific treatment is currently recommended for patients presenting hemophagocytic blasts, distinguishing this feature from macrophage activation syndrome. Early recognition of this cytological finding is essential for diagnostic and prognostic orientation. However, the absence of this feature in current prognostic classifications underscores the need for further studies to better understand its impact on therapy and disease progression.

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