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Original Research Paper

Pathology

HYPERTENSIVE EMERGENCY UNMASKING ACUTE MYELOID LEUKEMIA- A RARE PRESENTATION

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ABSTRACT We report a rare case presentation of acute myeloid leukemia (AML) in a 40-year-old female presenting with hypertensive emergency, acute pulmonary edema and acute febrile illness. The case highlights the importance of considering hematological malignancies in patients presenting with severe hypertension and respiratory distress with unexpected leukocytosis.

KEYWORDS : Hypertensive emergency, Leukocytosis, Acute Myeloid Leukemia

INTRODUCTION:

Acute myeloid leukemia (AML) is a malignant disease of bone marrow characterized by the abnormal proliferation and differentiation of myeloid precursors. AML is the most common form of leukemia in adults, with most cases occurring in individuals over the age of 60 years. AML presenting as hypertensive emergency is rare, seen in certain cases. We present such a case to emphasize the atypical clinical presentation.[1]

CASE REPORT:

A 40 year old female presented to the emergency department with complaints of shortness of breath(SOB), fever for 5-6 days, chest pain and palpitations. On admission her blood pressure(BP) was 210/100mmhg, and she appeared dyspneic with basal crepitations. Radiological findings revealed pulmonary edema, bilateral mild pleural effusion and no hepatosplenomegaly. A provisional diagnosis of hypertensive emergency with acute pulmonary edema and acute febrile illness under evaluation was made. The patient was started on antihypertensive therapy with subsequent normalization of blood pressure.

Laboratory Investigations :

CBC :

WBC – 97,750/cumm
Hb – 8.2g/dl
Platelets – 2.35lakhs/cumm
LFT- Normal
Blood urea – 34mg/dl
Serum creatinine – 0.83mg/dl

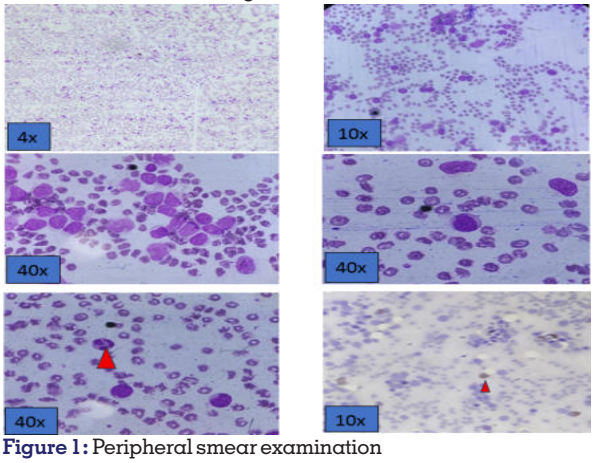

Figure 1: Peripheral smear examination

Normocytic normochromic rbcs, marked leukocytosis with shift to left upto myeloblasts,Blasts-52%,promyelocytes-8%, myelocytes-10%, metamyelocytes-10%, bandforms_9%, neutrophils_6%, lymphocytes-5%,adequate platelets. MPO cytochemical stain - <10% blasts are positive

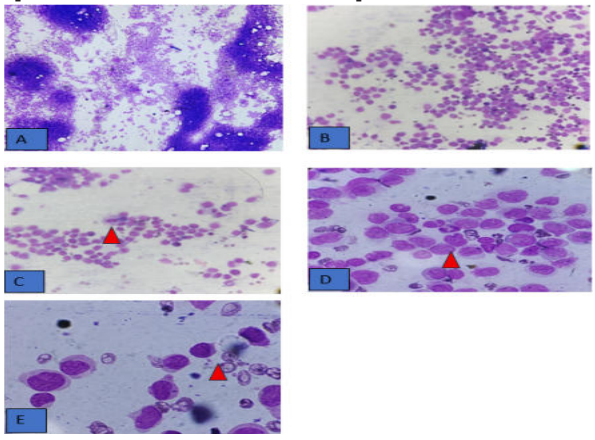

Figure 2: Bone marrow aspiration smear of the patient showing A. Hypercellular marrow 4x B. Showing myeloid hyperplasia C. Mitotic figure D. Myeloblasts and neutrophil E. Myeloblasts showing round nuclei with diffuse chromatin,1-2 nucleoli, moderate basophilic cytoplasm with cytoplasmic granules. Myeloblasts –55%, Promyelocytes-9%, myelocytes-10%, metamyelocytes- 8%, bandforms-45, neutrophils- 9%, lymphocytes-5%

DISCUSSION:

AML results from the clonal expansion of myeloid blasts in the peripheral blood, bone marrow or other tissues. It is a heterogeneous disease clinically, morphologically, and genetically and can involve a single or all myeloid lineages.[1]

The present case represents an unusual presentation of Acute Myeloid Leukemia (AML) manifesting as hypertensive emergency with acute pulmonary edema. AML typically presents with features related to cytopenias- such as anemia, bleeding, or infection-but cardiovascular manifestations and hypertension are uncommon.

In this patient, marked leukocytosis prompted consideration of the differential diagnoses of AML-M2 (acute myeloblastic leukemia with maturation), AML with MECOM rearrangement, and Chronic Myeloid Leukemia (CML) in

blast transformation.

AML M2 can be confirmed by doing flow cytometry with Morphological and cytochemical findings favoured a diagnosis of AML[figure 1&2] and molecular analysis to identify MECOM rearrangement was considered given the patient's age (40 years) and female predominance, as this subtype often occurs in this demographic and is associated with poor prognosis.[4]

CML blast crisis transforming to AML can be considered as platelets are adequate in this case.

The mechanism underlying hypertension in AML remains multifactorial.

Several possible pathophysiologic explanations have been proposed:
1. Leukostasis – severe leukocytosis can lead to microvascular obstruction, endothelial dysfunction, and increased vascular resistance.[5]
2. Infection or sepsis – systemic inflammation may cause dysregulated vascular tone and transient hypertension.
3. Leukemic infiltration of renal vasculature or adrenal glands – resulting in secondary hypertension due to altered renin-angiotensin activity.
4. Tumor lysis syndrome (TLS) – rapid cell turnover and release of intracellular contents can impair renal function and elevate blood pressure.[6]
To exclude the above pathophysiological explanations investigations like ESR,CRP,RFT [Renal function tests], Serum uric acid levels can be done respectively.

A study by Zhang et al. and Park CH et al. demonstrated that low platelet count at presentation is associated with improved survival in AML, possibly reflecting less proliferative disease burden.[2&3] However, despite initial hemodynamic stabilization in our case, the patient succumbed within one month of diagnosis, emphasizing the aggressive course and poor prognosis of AML, particularly in variants associated with MECOM rearrangement.[4]AML with MECOM rearrangement is known to have resistance to conventional chemotherapy, high relapse rates, and shorter overall survival, underscoring the need for early molecular characterisation and consideration of hematopoietic stem cell transplantation where feasible.

In summary, this case underscores the necessity of broad diagnostic consideration when encountering hypertensive emergencies with leukocytosis. Recognition of rare presentations such as AML manifesting with sever hypertension is vital for timely diagnosis and management.

CONCLUSION:

AML can present with hypertension, hence for any acute febrile illness cases with symptoms of SOB, chest pain, palpitations with leukocytosis – leukemia should be ruled out and simple peripheral smear examination plays a vital role in diagnosis.

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