Case Report

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FROM MICROCYTIC ANEMIA TO ACUTE LYMPHOBLASTIC LEUKAEMIA: A CASE REPORT OF RAPID HEMATOLOGIC TRANSFORMATION

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ABSTRACT

This case study outlines the clinical progression of a 22-year-old male patient who initially reported symptoms of fever, severe fatigue and loss of appetite. Physical examination revealed mild splenomegaly accompanied by hematological abnormalities including moderate anemia and leukocytosis. General blood picture showed microcytic hypochromic red blood cells and neutrophilic leukocytosis with total leucocyte count of 16800/cu mm having 76% neutrophils, 22% lymphocytes, 1% eosinophils and 1% monocytes. Platelet count was within normal range and approximately 1.5 lac/cu mm. There was no significant past medical history or family history nor any history of smoking or alcohol abuse. Chest X-ray revealed only mild prominent broncho vascular marking. Patient was treated with antibiotics and came for follow up within a month with symptoms of fatigue and mild breathlessness on exertion. Examination revealed hepatosplenomegaly and on ultrasonogram hepatosplenomegaly with left sided gross pleural effusion and abdominal lymphadenopathy was revealed. On repeating a complete blood count, it showed marked leukocytosis with 90% lymphocytes. The peripheral blood smear examination revealed 80% lymphoblasts 10% neutrophils 8%lymphocytes and 2% eosinophils. This case shows the transformation of blood picture to acute lymphoblastic leukemia within a month's time in a 22 years old male.

KEYWORDS: L2-Acute Lymphoblastic Leukemia (ALL), Anemia, Young Adult, Total Leucocyte Count.

INTRODUCTION

Acute lymphoblastic leukemia (ALL) is a form of blood cancer that develops from lymphopoietic stem cells, which are located in the bone marrow, lymph nodes, and thymus. This condition is marked by chromosomal abnormalities and genetic mutations that influence the growth and development of lymphoid progenitor cells. In adults, about 75% of ALL cases arise from B-cell precursors, while the rest stem from malignant T-cell precursors. ALL is the most prevalent cancer among children under 15 years old. Nearly 80-85% of children who survive 10 years without relapse, may be considered cured. (1) The highest incidence of ALL occurs in children between the ages of 1 and 5, with a slight male predominance. The signs and symptoms arise either from the involvement of extramedullary sites or from bone marrow failure due to the replacement of normal blood-forming cells by proliferating leukemic blasts. Common symptoms are fatigue, bone/ joint pain, fever, weight loss, purpura and bleeding manifestations while signs include lymphadenopathy, splenomegaly, hepatomegaly, sternal tenderness or mediastinal mass. (2)

The development of ALL involves the uncontrolled growth and maturation of a particular group of lymphoid cells. Research in children has pinpointed various genetic syndromes that increase the risk of ALL in a small number of cases, such as Down syndrome, Fanconi anaemia, Bloom syndrome, ataxia-telangiectasia, and Nijmegen breakage syndrome.(3,4,5,6) However, in most cases, it emerges as a new malignancy in individuals who were previously healthy. While chromosomal abnormalities are a significant feature of ALL, they are not sufficient by themselves to cause the leukemia. Notable translocations include t(12;21) (ETV6-RUNX1), t(1;19) (TCF3-PBX1), t(9;22) (BCR-ABL1), and MLL rearrangements. The majority of clinical symptoms of ALL arise from the accumulation of cancerous, immature lymphoid cells in the bone marrow, peripheral blood, and other tissues outside the bone marrow. The presentation can be nonspecific, often involving a combination of general symptoms

and signs of bone marrow failure (anemia, thrombocytopenia, leukopenia). Typical symptoms include 'B symptoms' such as fever, weight loss, and night sweats, as well as easy bleeding or bruising, fatigue, shortness of breath, and infections.Extramedullary involvement occurs in approximately 20% of patients, leading to symptoms such as lymphadenopathy, splenomegaly, or hepatomegaly. Also, central nervous system (CNS) involvement is observed at diagnosis in 5-8% of cases and may present as cranial nerve deFicits or signs of meningitis. T-cell acute lymphoblastic leukemia (ALL) can also present with a mediastinal mass. Diagnosis is usually conFirmed when 20% or more lymphoblasts are detected in the bone marrow or peripheral blood. (8)

DISCUSSION

This patient reported was 22 years old male who came to the OPD with the complaints of fever, severe fatigue and slight and loss of appetite or two weeks. Physical examination was done which revealed mild palor cervical lymphadenopathy and splenomegaly. The complete blood count was advised which revealed hematological abnormalities including moderate anemia and leukocytosis, a hemoglobin of 9.0mg/dl and total leucocyte count of 16800/cu mm. which included 76% neutrophils, 22% lymphocytes, 1% eosinophils and 1% monocytes. Platelet count was within normal range and approximately 1.5 lac/cu mm. General blood picture showed microcytic hypochromic red blood cells and neutrophilic leukocytosis. There was no significant past medical history or family history nor any history of smoking or alcohol abuse. Chest Xray was also done in view of severe fatiguability and mild breathlessness on exertion which revealed mild prominent broncho vascular marking. Patient was treated with antibiotics and was asked to come for follow up after two weeks. However, he returned after a month with severe fatiguability and worsening of breathlessness on exertion. The examination revealed hepatosplenomegaly and an ultrasonogram was advised which showed hepatosplenomegaly with left sided gross pleural effusion and abdominal lymphadenopathy was revealed. On repeating a hemogram it showed hemoglobin (Hb) of 8.8 gm/dl with marked leukocytosis with total leucocytic count (WBC) of 3,28,000/cu mm with 90% lymphocytes along with thrombocytopenia with platelet count of 35,000/cu mm. The peripheral blood smear examination revealed 80% lymphoblasts 10% neutrophils 8%lymphocytes and 2% eosinophils along with few nucleated RBCs. Red blood cell count (RBC) count was 4.50 million/cu mm, Hematocrit (HCT) 32.5%, Mean corpuscular volume (MCV) 72.2 fl and Platelet (PLT) was found to be 35000/cu mm. The patient had no other symptoms and was otherwise physically doing well. This case shows the transformation of blood picture to acute lymphoblastic leukemia within a month's time in a 22 years old male.

Bone marrow examination was done and it revealed a hypercellular marrow with 80% of lymphoblasts. Those lymphoblasts had high nuclear to cytoplasmic ratio with prominent nucleoli. There was myeloid suppression that is suppression of normal erythropoiesis leading to anemia, neutropenia and thrombocytopenia. The diagnosis was confirmed by flowcytometry and the patient was referred to a higher center.

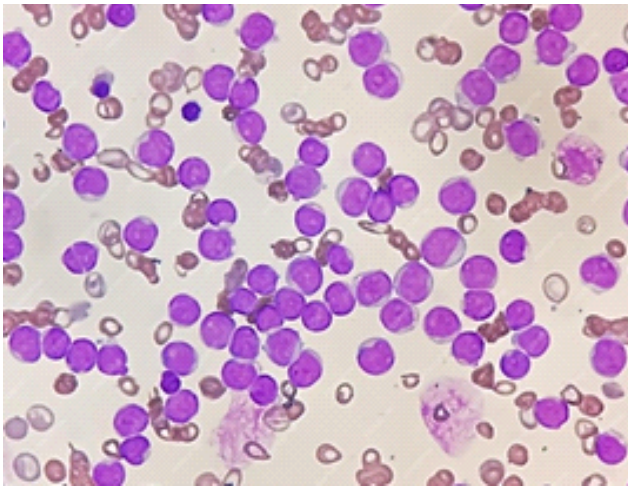

Fig.1: Acute Lymphoblastic Leukaemia (ALL): Blood Picture Shows Proliferation of Large and Heterogeneous Lymphoblasts.

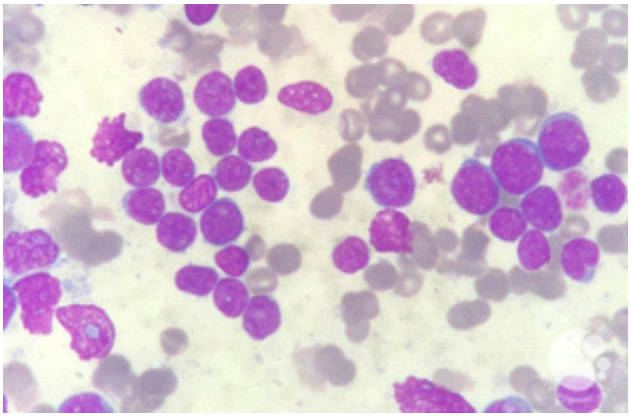

Fig.2: Bone Marrow Field with Lymphoblasts

CONCLUSION

This case highlights the sudden transformation of an unremarkable blood picture to Acute lymphoblastic leukaemia in a young adult within a period of one month. Thus underscoring the urgency of early evaluation in cases with meagre symptoms of easy

fatigability in young adults and starting the rapid management. Continued research and education are imperative to enhance clinical practices and improve outcomes by early diagnosis and management.

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