

Acute leukemia presenting as acute coronary syndrome: A rare case report

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Abstract

Coronary heart disease (CHD) occurs due to atherosclerosis of the coronary arteries. Although, other causes of coronary heart disease should always be taken into account. In our case, the patient presented as symptoms of coronary heart disease as a result of Acute Leukemia. The pathophysiological mechanisms of acute leukaemia leading to Coronary Heart Disease are discussed.

Introduction

Acute Leukemias (ALs) carry a major cause of death in the developed and developing countries of the world.¹ Acute Leukemias are of two types-Acute Myeloid and Acute Lymphoid Leukemias., Acute myeloid leukemia (AML) is the most common acute leukemia in adults especially amongst males.² Acute lymphoblastic leukemia (ALL) and Acute Promyelocytic Leukemia (APL) are least encountered in adults.²

Diagnosis of AML can be done by examination of the peripheral blood smear. For confirmation, adequate bone marrow aspiration is necessary. A part of the marrow material can be sent for biopsy also in the neutral buffered formalin.³

The French-American-British classification (FAB) Classification is used for sub-typing of AML.M0 is Acute Myeloid leukemia –undifferentiated, M1 and M2-AML without and with maturation, M3-AML –promyelocytic, M4- AML –myelomonocytic with more than 20% monocytes,M5-AML-monoblastic, M6 and M7-AML – erythroid and megakaryocytic. For confirmation of Myeloblasts, myeloperoxidase should be performed. Complete workup of the diagnosis involves immunophenotyping and cytogenetics.⁴

The Acute Lymphoid Leukemia (ALL) is subdivided into L1, L2 and L3 based on the morphology of blasts according to FAB classification.L1 has smaller blasts with scanty cytoplasm and inconspicuous small nucleoli. L2 are bigger in size than in L1 and have more cytoplasm with

prominent 1-2 nucleoli.L3 are larger and have vacuoles in the basophilic cytoplasm. They can be typed using block positivity property by PAS and L3 can be demonstrated by fat stains like Oil red O and Sudan Black B.L3 carries a dismal prognosis.⁴

Case Report

A 50 years old man came to casualty of Mahatma Gandhi Medical College and Research Institute with sudden onset of chest pain and dyspnea. His complete blood count revealed anaemia, leucocytosis and thrombocytopenia. There was no history of any hematological disorder. On examination; the patient had pallor alone. No generalized lymphadenopathy and hepatosplenomegaly were noted. The blood sample was run in an automatic hemolyser (Horiba Pentra DF Nexus) and peripheral smear was examined.

Peripheral smear examination showed Normocytic normochromic red blood cells, Total leucocyte count is increased with lymphocytic predominance and platelets are reduced on smear. The leucocyte differential count was neutrophils 5%, lymphocytes 55%, eosinophils 0%, monocytes 10% and Blasts 30%. The impression of peripheral smear was given as features suggestive of Acute Myeloid Leukemia, which was confirmed on bone marrow aspiration study. His serum Troponin levels were elevated. His blood glucose levels were 239 mg/dl.ECG shows T wave inversion with ST elevation. Coronary angiography revealed double vessel involvement.

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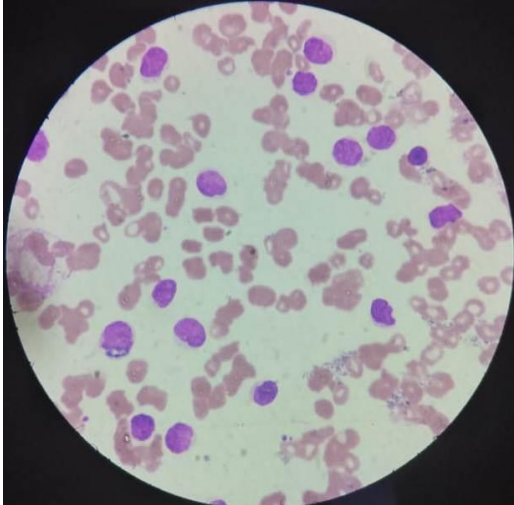


Fig. 1

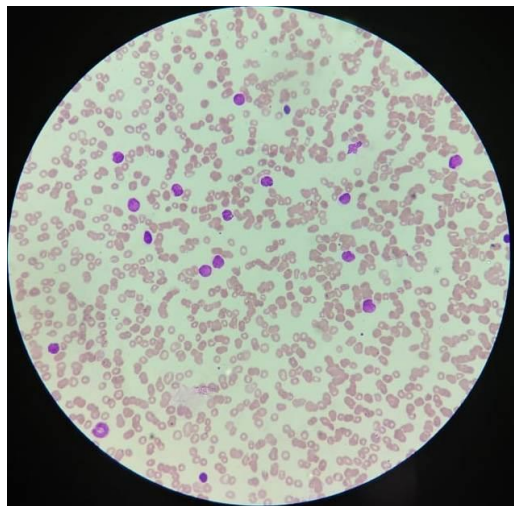


Fig. 2

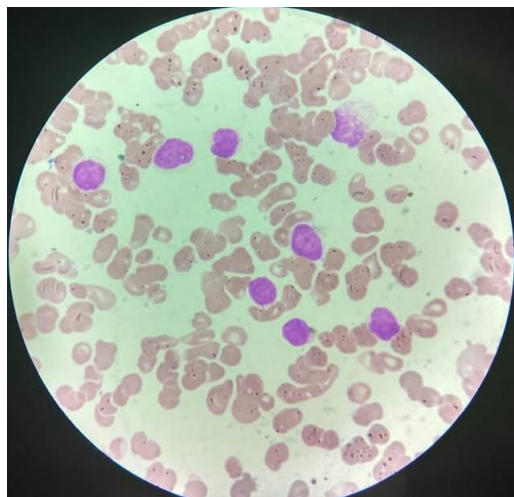


Fig. 3

Discussion

The incidence of acute leukemia is approximately 2.3 per 100000 people per year.

The patient was a 50 years old male. His total count was 27,200, which is uncommon for an Acute Leukemia to present. Cytopenias are usually present but 30% of patients have platelet counts $>100000/uL$ but in our case who had low platelet count ($75 \times 10^9/L$).⁵

Patient presented with symptoms of acute coronary syndrome in our case. Ischaemic heart disease occurs due to atherosclerotic vascular occlusion.⁶ Although, nonatherosclerotic myocardial infarction can occur due to aberrant anatomy of the coronary arteries, congenital or acquired coagulation disorders, embolism and connective tissue disorders.⁷ There are few mechanisms described for the occurrence of cardiovascular events in Acute Leukemia:

1. Leukemic thrombi in major arteries;
2. Leukemic infiltration into the myocardium or pericardium;
3. Disorders of coagulation precipitated by leukemia leading to hypercoagulation state.
4. Antileukemic drugs causing ischemia.⁸

Leukemic thrombi can culminate in Acute Myocardial Infarction. The proposed pathogenic mechanism behind this is large myeloblasts adhere to myocardium, when cell counts are above $100 \times 10^9/l$. Intravenous fluids, prednisolone, radiotherapy and by anticancer drugs are used in this condition. It is recommended that leucapheresis can improve the patients condition in this case.⁹

The antileukemic treatment consisting anthracyclines are known for their deleterious cardiotoxicity. Use of such drugs in myocardial failure patients should be prohibited. Intravenous fluid infusion is recommended seriously to curb the serious side effects of anticancer drugs. In such cases, patients with acute leukemia and Ischemic Heart disease aggravated due to the former, respond poorly to the treatment. Hence, Leukemic patients presenting with ischemic heart disease have an extremely poor prognosis.⁹

Conclusion

The above case sets an example that a nonatherosclerotic cause of coronary heart disease should also be taken into account. Despite there are many anticancer drugs, few exert serious cardiotoxicity. Hence, the symptoms with which patient presented restricts the use of cardiotoxic drugs as chemotherapy regimen. Such cases require immediate attention, diagnosis and treatment.

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