International Journal of Research in Medical Sciences
Anandan J et al. Int J Res Med Sci. 2023 May;11(5):1851-1854
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20231373

Case Report

Pulmonary infarct as a initial presentation of acute myeloid leukemia: a diagnostic difficulty

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Received: 23 March 2023
Revised: 16 April 2023
Accepted: 18 April 2023

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ABSTRACT

The incidence of acute myeloid leukemia (AML) is age dependent and increasing among the patient groups aged above 60 years. The overall presentations in a case of AML is due to marrow infiltration of leukemic cells which results in fever and chills due to neutropenia, bleeding due to thrombocytopenia, fatigue and weakness due to anemia. Incidence of venous thromboembolism is very rare in hematological malignancies. We describe a case of adolescent male presented with features of lung consolidation later found to be a pulmonary infarct and finally diagnosed to have acute leukemia (AL).

Keywords: Acute myeloid leukemia, Pulmonary thromboembolism, Consolidation, Pneumonia, Pulmonary infarction

INTRODUCTION

Acute myeloid leukemia (AML) is defined based on the morphological inspection revealing a myeloblast count of >20% out of 500 bone marrow cells.[1] AML may be associated with life-threatening complications that are specific to phenotypes which include infection, anemia, and bleeding. In general solid tumors such as pancreatic, ovarian, and brain cancers carry a much higher risk of thromboembolism than hematologic malignancies such as lymphoma and leukemia.[2-5] Among the phenotypes described, acute promyelocytic leukemia (AML-M3) has been associated with higher rates of thrombosis. The incidence of venous thromboembolism as such is quite rare in acute leukemia and usually doesn't alter the one-year mortality. But in our case, what we found was quite different. Here we represent a case of acute leukemia (AL) presented initially as a pulmonary infarct due to pulmonary thromboembolism.

CASE REPORT

A 17-year-old male student with no known co-morbidities or addictions presented to emergency with the complaints of breathlessness, cough with expectoration occasionally blood-tinged, fever, and vomiting for 3 days. Breathlessness was sudden in onset, progressive in nature associated with orthopnea, aggravated on exertion without any specific relieving factors. He had no leg swelling, abdominal distension, facial puffiness. Cough with expectoration was present with sputum being white coloured, mucoid in consistency, blood tinged and non-foul smelling. Fever was low grade, continuous, with chills and rigors without any rash or evening rise in temperature associated with vomiting immediately after food intake which was non bilious and non-projectile. There was no history of chest pain, burning micturition, headache, and abdominal pain. On examination, he had pallor, desaturation (90% in room air), tachycardia, and was normotensive. There

were no palpable peripheral lymph nodes or hepatosplenomegaly. On auscultation, he had absent breath sounds in the left infraaxillary area. Furthermore, electrocardiography (ECG) showed sinus tachycardia. Chest X-ray showed left lower zone haziness and distended bowel loops (Figure 1).

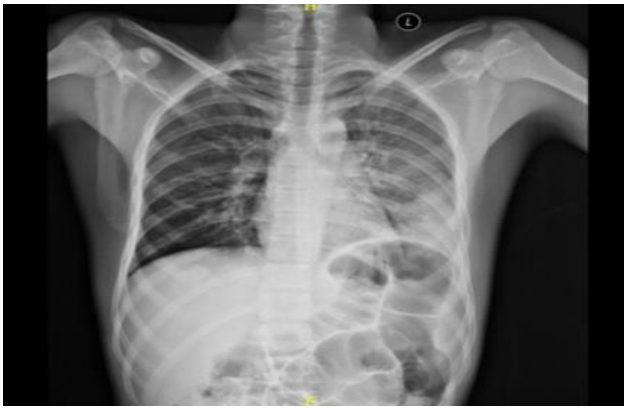

Figure 1: Chest X-ray showing left lower zone haziness.

Ultrasonography of thorax showed left minimal pleural effusion. Ultrasonography (USG) abdomen showed sluggish peristalsis, and no organomegaly. After initial evaluation, diagnoses of: community-acquired pneumonia (CAP) with parapneumonic effusion and acute gastroenteritis (AGE) were made and started on oxygen support and empirical antibiotics. Because of elevated JVP, echocardiography was done which showed – right atrial and ventricular mass suggestive of infective endocarditis (IE). Renal function parameters were normal. Liver enzymes and lactate dehydrogenase enzyme were elevated (aspartate transaminase – 471 IU/L, alanine transaminase – 623 IU/l, LDH – 1012 IU/l). Hemogram showed haemoglobin of 6.5 gm/dl, platelets 85,000 cells/microlitre, mild leukocytosis-11,890 cells/ microlitre. Peripheral smear revealed 72% blasts but no Auer rods, suggestive of acute leukemia (AL) (Figure 4a). Then, the diagnosis was as follows: AL with IE, and CAP with acute hepatitis (atypical pneumonia/tropical infection). Work up for all tropical infections was negative. Hence we arrived at diagnoses of AL with IE and CAP with acute hepatitis probably atypical pneumonia and started azithromycin. As patient had blood stained sputum and peripheral patch in X-ray with base towards pleura and was worsening in terms of saturation with Modified wells score was 5 (pulmonary embolism - likely), a computed tomographic pulmonary angiogram (CTPA) with contrast enhanced computed tomography (CECT) of chest was done which showed left descending pulmonary artery thrombus and right multiple sub-segmental arterial thrombosis and multiple peripheral wedge shaped opacities suggestive of infarcts over the bilateral lung fields**,** along with liver, spleen**,** and renal infarcts (Figures 2 and 3). Bone marrow aspirate revealed 62% blasts with evidence of maturation which had coarse granular cytoplasm and were positive for Sudan black B stain. No definitive Auer rods were seen. Flow cytometry on CD45/SSC gating showed 60.78% of all events fell in the blast window which were positive for CD117, cMPO, CD13, CD33, CD11b (weak positive) and CD11c (weak positive); negative for CD34, nTdT, CD64, B-lymphoid and T-lymphoid markers (Figure 4b). Finally reported as AML with maturation by French American British (FAB) classification i.e., AML-M2. Hence, the final diagnosis of AML-M2 with multiple organ vascular thrombosis and secondary infarcts was made.

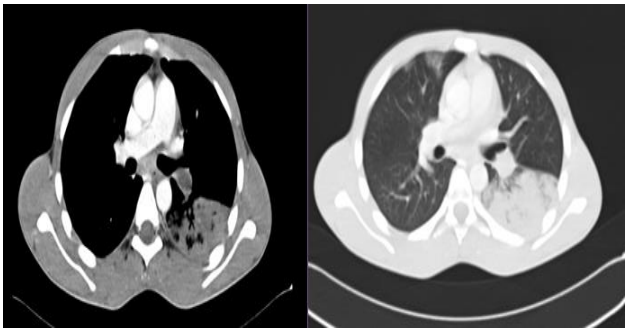

Figure 2: CECT thorax showing left descending pulmonary artery thrombosis and infarcts, right lung parenchyma showing peripheral wedge shaped opacities.

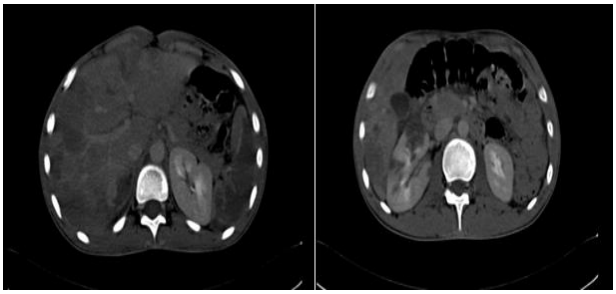

Figure 3: CECT abdomen showing liver, spleen and renal infarcts.

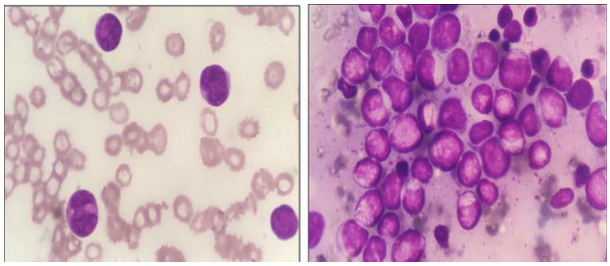

Figure 4: (a) PS showing blasts which are 1.5 to 2 times the size of small mature lymphocyte with high N:C ratio, nuclear folding in some with opened up chromatin, scant amount of coarse granular chromatin. No definitive Auer rods were seen (Leishman x 400), and (b) bone marrow imprint showing many blasts with evidence of maturation, erythropoiesis, megakaryopoiesis are suppressed (Leishman x 400).

DISCUSSION

Presentation of an AML is mostly due to marrow infiltration, including fever and chills due to neutropenia, bleeding due to thrombocytopenia, fatigue, and weakness due to anemia and constitutional symptoms like fever, weight loss and fatigue. It can also present due to infiltration and enlargement of the organs. In cases where white blood cell counts being more than 50,000 cells/microlitre, vascular occlusions can occur resulting in infarction. This patient had breathlessness and cough with expectoration as major complaints. Since the clinical presentations were not typical of leukemia with a total WBC count in the normal range there were considerable difficulties in establishing the diagnosis. Initially, we thought that the consolidation with hepatitis was due to tropical infection or atypical infection but related workups were negative finally the consolidation of lung and all other radiological abnormalities were found to be infarcts due to pulmonary, hepatic, cardiac, splenic, and renovascular thrombosis without any hyperleukocytosis or leukostasis.

Astonishing to see the right heart chambers being filled with huge thrombotic masses as evidenced in echocardiography. Such a widespread systemic hypercoagulability in haematological malignancies is rare and if occurs will account for a significant percentage of mortality and morbidity. Thrombosis usually results due to dysregulated hemodynamics resulting in one of the components of Virchow's triad that includes stasis of blood or hypercoagulable state or vascular endothelial damage. Thrombosis in malignancy has multiple causes which include thrombophilia due to tumor-induced activation of the clotting system, immobilization due to morbidity, compression of veins by the tumor resulting in stasis, surgery, and intravascular catheters.[6] In leukemia-associated thromboembolism, activation of the clotting system is the most important pathogenetic factor which is likely to be in our case, as none of the other above-mentioned factors were associated.[7] Leukemic cells, in particular promyelocytes, contain procoagulants that are released into the bloodstream such that thrombosis occurs even in the presence of severe thrombocytopenia.[6] Reported rates of venous thrombosis were similar in patients with AML and acute lymphoblastic leukemia (ALL) with the highest incidence noted in patients with AML-M3. But this case was finally diagnosed as AML with maturation (AML-M2) which made this case more interesting.

Important risk factors for the development of thrombosis in patients with AML include insertion of a central venous catheter, female sex, and the presence of any two chronic comorbid medical conditions.[8] But in this case, none of the above-mentioned risk factors predisposing to clot activation were present, yet he presented with such an extensive thrombotic event which made this case stand alone. Even though there were considerable difficulties in establishing the complete diagnosis, all the workups were completed within 2 days and the patient was planned for chemotherapy. Studies showed that the diagnosis of venous thrombosis or pulmonary embolism is not a significant predictor of death within 1 year of diagnosis of AML.[8] But unfortunately, the patient expired within 2 days of diagnosis, even before initiating chemotherapy making it an unusually fatal disease. Hence, this scenario added up to the fact that the diagnosis and treatment of patients with leukemia and associated thromboembolism requires considerable skill as their presentation varies enormously and poses challenges in multiple ways to the treating physician.

CONCLUSION

An adolescent male, without any comorbidities, had presented with features of a lung consolidation and had normal leukocyte count, was found to have pulmonary infarct, and finally diagnosed to have AML-M2. This explains the fact that irrespective of the phenotypes described and hyperleukocytosis, hypercoagulability can result as a complication of acute myeloid leukemia. Hence, in a young individual who doesn't have any risk factor for hypercoagulability if found to have focal or widespread systemic vascular thrombosis even without typical symptoms and signs of leukemia, and without abnormal laboratory parameters like hyperleukocytosis and resulting leukostasis, acute leukemia with thrombotic potential that are readily fatal should be considered a differential for related work up and initiating appropriate therapy as early as possible.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Anandan J, Upadhya P, Babu VM, Basu D, Rangarajan V. Pulmonary infarct as a initial presentation of acute myeloid leukemia: a diagnostic difficulty. Int J Res Med Sci 2023;11:1851-4.