

Multiple bone lytic lesions in a pregnant patient with Acute Lymphoblastic Leukemia: A Rare Presentation

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ABSTRACT

Osteolytic bone lesions are a common feature of several diseases, rarely observed in adult acute lymphoblastic leukemia (ALL). We report a case of a pregnant woman with osteolytic lesions, in whom the diagnosis of ALL was difficult. We discuss the osteolytic lesions mechanism and treatment in the ALL.

KEY WORDS

Acute lymphoblastic leukemia ; osteolytic lesions ; pregnant malignancies

INTRODUCTION

The majority of acute lymphoblastic leukemia patients present blood count abnormalities and tumor syndrome made of polyadenopathy and splenomegaly. However, Bone lesions can induce a wrong diagnosis of ALL in adult.

Herein we describe a clinical case of a pregnant patient admitted for ALL, presented diffuse osteolytic lesions in the absence of leukocytosis.

This clinical case aims at avoiding misdiagnosis of this fatal pathological entity and delay patient management.

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I. CASE PRESENTATION

A 31 year-old 11 weeks pregnant patient, admitted to our hospital with 3-months history of generalized weakness and arm pain. This pain worsened resulting difficulty moving upper limb. For the last month, patient's symptoms were progressive worsening of dyspnea. Without any fever, weight loss.

At the Physical examination, she was afebrile. There was remarkable pain for palpation along left arm and shoulder with limited Flexion.

There was multiple cervical, supraclavicular and axillary bilateral lymphadenopathies. The biggest one measuring 3-cm. splenomegaly and sallow, the skin examination revealed three subcutaneous nodules on the scalp measuring 4 cm. Cardiovascular and pulmonary exam were normal.

The haemogram showed white blood cells at 2.5 G/L also a hemoglobin level of 5.4 g/dL and platelet at 135 G/L. blood smear showed anisochromia and 10% blasts. Bone marrow aspiration showed infiltration by 99% blasts.

The blast cells were positive for CD19, CD79a, CD33, and TDT, at immunophenotyping consistent with B-lymphoblastic leukemia. Conventional cytogenetics analysis was normal. Uremia was 0,22 g/l and creatinine at 5,0 mg/l while the ionogram was normal including specially calcium.

Albumin level at 93 mg/l also total proteins were 61 g/dL. The urine analysis was negative for proteins. Liver function tests and clotting profile were normal. C - reactive protein was 35,0 mg/l, PTH blood test was 5.0, with a normal level of vitamin D3.

Urine electrolytes were normal, following a suspicion of hyperparaproteinemia, additional tests were done including electrophoresis and immunofixation, showed no monoclonal gammopathy.

The ethics committee recommended the therapeutic abortion of the fetus in order to start the chemotherapy according to protocol GRAALL-2014.

Then, arm X-rays revealed multiple osteolytic lesions (figure 1), and CT-scan determined several lytic lesions of the sternum, the left clavicle, the left scapula, the left humerus and some homolateral ribs, (figure 2)

II. DISCUSSION

We do not normally find osteolytic lesions in acute leukemia compared to other diseases like multiple myeloma. [1]

There are not many studies related to bone involvement in acute leukemia in children and even fewer in adults, the prevalence determined in children is 13%, nevertheless, the prevalence in adults still not known.

Diffuse osteolytic lesions in adults often seen in association to other malignant blood diseases, notably multiple myeloma and tumors metastases. Therefore, patients with ALL and osteolytic lesions may their diagnostic be challenging.[2]

Most cases of ALL associated with skeletal manifestations are B cell-ALL. Generally, T cell ALL has better prognosis than B cell-ALL has worse prognosis at adult age.[3][4]

Many cytogenetic abnormalities can be found among adults ALL.

The most common one is the Philadelphia chromosome/BCR-ABL1 indicates poor prognosis and 11-29% of patients express this translocation, it is also a recurrent cytogenetic abnormality in childhood ALL featuring osteolytic lesions.[5][6]

The osteolysis mechanism in ALL induced by the activation of osteoclasts; this phenomenon is due especially to the production of PTHrP by lymphoblasts. Thus in case of normal PTHrP, inflammatory cytokines can contribute to this bone lysis process.[7]

Bisphosphonates and an antibody against RANKL as denosumab, targeting osteoclasts. They are known capable of reducing the bone loss in adjunction to chemotherapy in patients with ALL and osteolysis.

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Figure 1: Multiple osteolytic lesions in the skull



Figure 2: lesions the left clavicle, the left scapula, and the left humerus fracture

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