

Case Report

Gingival hypertrophy in a child: Expect the unexpected

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Abstract

Acute leukemia is the most common malignant disorder of childhood. Acute leukemia is characterized by marrow failure due to the replacement of marrow elements by leukemic blasts. Gingival hypertrophy (GH) due to acute myeloid leukemia (AML) in a child is extremely rare. Here, we report a case of AML (subtype M5 of FAB classification) in a 3-year-old male child wherein the disease primarily presented as GH.

Key words: Acute myeloid leukemia, child, gingival hypertrophy

INTRODUCTION

In the field of dentistry, gingival hypertrophy (GH) is a common presenting complaint, especially among patients belonging to pediatric age group. Gingivitis is a commonly occurring phenomenon since the oral cavity is a potential site for active infection as it is constantly exposed to pathogenic microorganisms. The prevalence of GH among school going children is nearly 14%, inflammatory origin being the most common cause followed by chronic drug intake.^[1] Hence, there is a tendency among us to consider GH as a mere inflammatory response to local infection rather than considering systemic illnesses; this can eventually lead to delay in diagnosis and initiating appropriate treatment. GH due to acute myeloid leukemia (AML) in a child is extremely rare. Here, we report a case of AML (subtype M5 of FAB classification) in a 3-year-old male child wherein the disease primarily presented as GH.

CASE REPORT

A 3-year-old male child presented to us with fever and body pain for 2 weeks, gum swelling for 1-month duration, initially presented to a pediatrician and a dentist, for which symptomatic treatment was given. The child was again reviewed with the

pediatrician after 1 week with persisting symptoms. This time, the child was treated with antibiotics. He then presented to us. There was no history of chronic disease or similar illness in the family. The child had attained normal developmental milestones appropriate for his age. No history of halitosis or dental caries.

On general examination, he had pallor; there was no icterus, cyanosis, clubbing, pedal edema, or generalized lymphadenopathy. Oral examination revealed the enlargement of maxillary and mandibular gingiva covering two-third of the crown structure in buccal, lingual, and palatal aspects. Gingiva was reddish with the loss of stippling, soft, spongy, bleeding on touch but was nontender and not warm [Figure 1]. On abdominal examination, the liver was palpable 2.5 cm below the right costal margin, and the spleen was palpable 2 cm beyond the left costal margin. His hemoglobin was 78 g/L, total white blood cell count was $5.6 \times 10^9/L$ with 90% blast cells and 10% lymphocytes, and platelet

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Figure 1: Gum hypertrophy

count was $0.9 \times 10^{11}/L$. His renal and liver function tests were within the normal range; uric acid and lactate dehydrogenase levels were also normal. Bone marrow aspirate smear showed blasts which are large with moderate amount of agranular to granular cytoplasm and round to irregular nucleus with fine chromatin and 1–3 nucleoli. Few of the blasts showed Auer rods [Figure 2]. In flow cytometric analysis, the blasts were positive for CD33, CD117, human leukocyte antigen-DR, and CD13. Parents were counseled about his condition, treatment, and outcome of the disease. Due to long distance commuting issues, the parents opted for the child to receive treatment elsewhere; the child was lost to follow-up.

DISCUSSION

Most common causes of GH in children are poor dental hygiene and chronic drug intakes such as phenytoin, cyclosporine, and calcium channel blockers. Systemic illnesses such as Wegener's granulomatosis, sarcoidosis, Crohn's disease, infantile systemic hyalinosis, tuberculosis, and hereditary gingival hyperplasia should also be considered in the differential diagnosis.^[1-3]

Acute leukemia, which is the most common malignant disorder in children, is characterized by neoplastic proliferation of blast cells causing accumulation of >20% blast cells in the bone marrow, interfering hematopoiesis resulting in fatigability, bleeding tendency, and infections. Acute leukemia is subdivided into acute lymphoblastic leukemia and acute myeloid leukemia (AML), based on the phenotype of the blast. AML in a child generally presents with fatigue, pallor, abnormal bleeding and infections with or without splenomegaly and lymphadenopathy; however rarely,

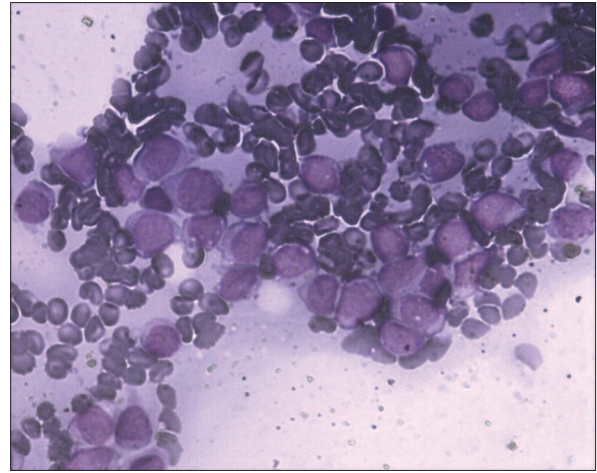


Figure 2: Bone marrow aspirate, Leishman stain, x40 showing myeloblasts

it can present with infiltration of blasts cells in tissues such as skin, mucosa, and gums.

Literature review has shown only few studies that have reported cases of AML presenting with oral lesion as a primary manifestation. Oral manifestations in leukemia are GH, oral ulcers, pale mucosa, herpes, and candida infections.^[4] Gingival infiltration in AML is rare, usually associated with subtypes M4 and M5 (FAB classification). GH represents 5% frequency as the initial presenting complaint of AML.^[5] It is due to infiltration of tissues with neoplastic precursors of myeloid cells or secondary to thrombocytopenia, neutropenia, or impaired granulocyte function. GH in extremely rare cases can also be a manifestation of isolated recurrence. Hence, gingival tissue, although unusual, is one of the sites that should be monitored for relapse.^[6] Gingival hyperplasia due to AML resolves with effective chemotherapy.

CONCLUSION

Although confirmatory diagnosis and treatment of AML is primarily done by the pediatric hemato-oncologist, it is almost always a dentist or a pediatrician who first attend to cases of GH, and hence increasing awareness among them will aid in early detection and prompt referral and drastically improve the outcome of the disease. Finally, any GH, particularly when associated with hepatosplenomegaly and abnormal blood counts should raise the suspicion of possible underlying hematological malignancy in the child.

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Conflicts of interest

There are no conflicts of interest.

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