

Case Report

Gum hypertrophy in acute myeloid leukemia: an important clinical finding not to be missed

Harshita Narendran¹, Sudeep Gaddam^{2*}, Vimalnath Shanmugam³, Dhaarani Jayaraman²,
Sri Gayathri Shanmugam⁴, Latha M. Sneha², Julius Xavier Scott²

¹Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India

²Department of Pediatric Hematology and Oncology, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India

³Department of Pediatrics, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India

⁴Department of Pathology, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India

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*Correspondence:

Dr. Sudeep Gaddam,

E-mail: sudeepgaddam2@gmail.com

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ABSTRACT

Acute myeloid leukaemia (AML) is characterized by disordered differentiation and proliferation of abnormal hematopoietic stem cells. AML usually presents with symptoms of anemia like pallor and fatigue, recurrent infections, petechiae, and mucosal bleeds. Extramedullary infiltration of leukemic cells is a common finding like proptosis or myeloid sarcoma. The occurrence of gingival hypertrophy in the pediatric age group is uncommon and usually due to inflammation followed by prolonged use of certain drugs like cyclosporin or phenytoin. Gingival infiltration in AML is rare in children, usually associated with subtypes M4/M5 (FAB classification). This case report highlights the importance of considering AML as an important differential diagnosis in cases of gum hypertrophy, as being a less common cause, it is often overlooked. Timely diagnosis and prompt treatment can be lifesaving. Here, we report two cases who presented with gum hypertrophy.

Keywords: AML, Physical examination, Gum hypertrophy

INTRODUCTION

Acute myeloid leukaemia (AML) is characterized by disordered differentiation and proliferation of abnormal hematopoietic stem cells.¹ AML usually presents with symptoms of anemia like pallor and fatigue, recurrent infections, petechiae, and mucosal bleeds. Extramedullary infiltration of leukemic cells is a common finding like proptosis or myeloid sarcoma.² The occurrence of gingival hypertrophy in the pediatric age group is uncommon and usually due to inflammation followed by prolonged use of certain drugs like cyclosporin or phenytoin.³ Gingival infiltration in AML is rare in children, usually associated with subtypes M4/M5 (FAB classification).^{4,5}

This case report highlights the importance of considering AML as an important differential diagnosis in cases of gum hypertrophy, as being a less common cause, it is often overlooked. Timely diagnosis and prompt treatment can be lifesaving. Here, we report two cases who presented with gum hypertrophy.

CASE REPORT

Case 1

A 7-year-old boy presented with high grade intermittent fever, fatigue, and swollen gums for two weeks. He was admitted previously at a private hospital elsewhere and treated with antibiotics. There was no history of chronic

drug intake or any known developmental delays. On examination he was noticed to have pallor, hepatosplenomegaly, cervical lymphadenopathy, and gum hypertrophy (Figure 1). He was not found to have any dysmorphic features or congenital abnormalities. Investigations revealed bicytopenia with hyperleukocytosis-WBC count was 32,550 per cubic millimetre with 26% blasts, haemoglobin was 5.6 gm per decilitre, and the platelet count was 57,000 per cubic millimetre. Bone marrow biopsy revealed 75 % myeloblasts. Flow cytometry showed blasts identified as dim population in CD45 SS plot and positive for HLADR, CD33, CD38, CD64, CD11c and CD13, confirmative of AML M5. Cytogenetics showed absence of t (15;17), t (8;21); inv of Ch16 and karyotyping revealed 46, XY, t (10;11) (p12; q23)-MLL translocation. The child was then treated with induction chemotherapy daunorubicin and cytarabine which resulted in the resolution of gingival lesions, but due to profound neutropenia and disseminated adenoviral disease with bilateral pneumonia, he succumbed to the illness.



Figure 1: Gum hypertrophy in a 7-year-old boy who was diagnosed with AML-M5 subtype.

Case 2

A 6-year-old boy presented with fever, pallor and gum swelling for ten days with no history of chronic drug intake. He received blood transfusion prior to the admission. On examination he was noticed to have hepatosplenomegaly and gum hypertrophy (Figure 2). There were no dysmorphic features or congenital abnormalities. At admission the white blood cell count was 4,430 per cubic millimetre with 4% blasts, haemoglobin was 7.3 gm per decilitre, and the platelet count was 1,46,000 per cubic millimetre. Flow cytometry showed blasts identified as dim population in CD45 SS plot and positive for CD13, CD33, CD34, HLADR4, CD117, with aberrant expression of CD7 and negative for other T cell, B cell markers. Cytogenetics showed absence of t (15;17), t (8;21); inv of Ch16 and karyotyping showed loss of chromosome 7, suggestive of AML-M0 (monosomy 7). Molecular markers including NPM, C KIT, FLT3, and CEBP alpha were negative. The child had induction failure after daunorubicin and

cytarabine chemotherapy with 13% residual blasts on bone marrow examination, but gum hypertrophy resolved. He received once cycle of salvage chemotherapy FLAG-bortezomib regimen, attained remission and planned for bone marrow transplant.



Figure 2: Gum hypertrophy in a 6-year-old boy who was diagnosed with AML-M0 subtype.

DISCUSSION

Gingival enlargement can be attributed to a range of factors, including chronic gingivitis, drug-induced hyperplasia (e.g., nifedipine, verapamil, phenytoin), genetic syndromes, vitamin C deficiency, and idiopathic gingival enlargement. Additionally, systemic causes, such as Wegener's granulomatosis, sarcoidosis, Crohn's disease, neurofibromatosis I, storage disorders, and tuberculosis can also cause gum hypertrophy.³

AML can be classified into 8 subtypes (FAB classification) based on morphology.⁶ Extramedullary involvement in AML is significant in children, particularly in subtypes M4 and M5.^{4,7} WBC precursors infiltrate tissues usually resulting in clinical manifestations such as anemia due to decreased red blood cell production, infections due to neutropenia and excessive bleeding due to thrombocytopenia. Organ infiltration can most commonly be seen in the liver, spleen, skin, lymph nodes, CNS, and testes, often leading to organomegaly, proptosis and myeloid sarcoma. Gum hypertrophy, seen in rarer 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.⁸

Oral manifestations, especially gum hypertrophy occur in most patients with AML and are often one of the first presenting signs. Even though it is not a specific sign, it can help to guide the diagnosis towards a malignancy. It is hypothesized that gingival pervasion is due to the expression of endothelial adhesion molecules which enhance infiltration of leukocytes.⁹ As a result, the patient presents with soft, hemorrhagic, swollen, ulcerative gums. Biopsy can reveal leukemic infiltrates in many

sites including gingival enlargement, mucosal and skin nodules.¹⁰

Gum hypertrophy associated with fever, organomegaly and lymphadenopathy in a child should raise a suspicion of leukemia and prompt further investigations. If left untreated, AML tends to progress rapidly and is typically fatal within a few weeks or months.² Being an early sign, its recognition by a physician is pivotal in the outcome of children with AML.

CONCLUSION

This case report underscores the critical importance of recognizing gum hypertrophy as a potential indicator of AML. Prompt identification and appropriate diagnostic measures are crucial for timely intervention and improved prognosis. These cases emphasize the need for heightened clinical suspicion in the presence of gum enlargement accompanied by systemic symptoms, facilitating early initiation of treatment and ultimately enhancing patient outcomes in pediatric AML management.

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