

Photoclinic



Figure 1. Oral orifice and both jaws of a 23-year-old man (frontal view)

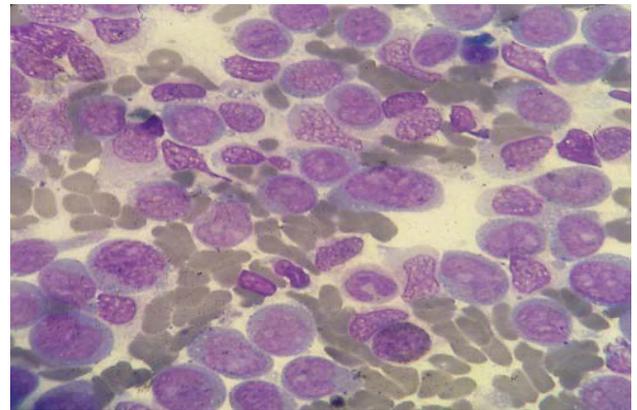


Figure 2. Patient's bone marrow aspirate

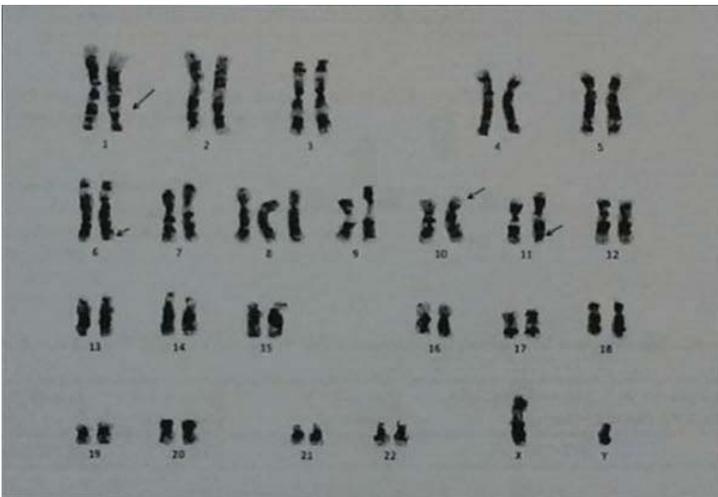


Figure 3. Patient's karyotype

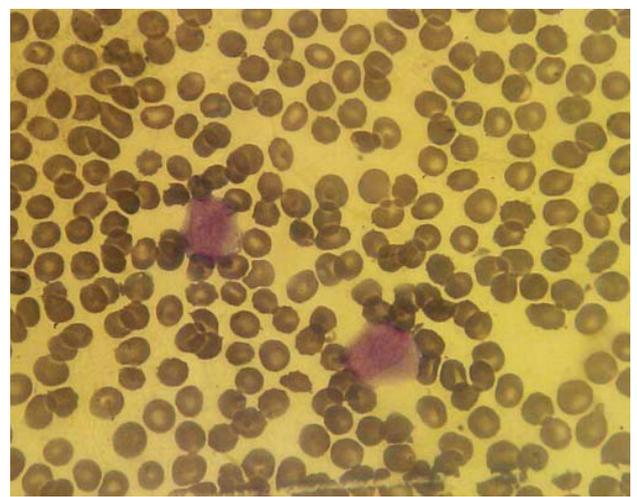


Figure 4. Patient's peripheral blood smear

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A 23-year-old man was referred to our clinic on suspicion of gingivitis from dentistry. He presented with malaise, weakness, generalized musculoskeletal pain and complaint of gingival pain. He had no history of tobacco or drug use. He did not recall any bleeding

Rambod Mozafari MD¹, Ali Asadollahi-Amin MD²

Author's affiliations: ¹Department of Hematology, Imam Khomeini Hospital, Tehran University of Medical Sciences, Tehran, Iran, ²Department of Infectious Disease, Imam Khomeini Hospital, Tehran University of Medical Sciences, Tehran, Iran.

•Corresponding author and reprints: Ali Asadollahi-Amin MD, Department of Infectious Disease, Imam Khomeini Hospital, Tehran University of Medical Sciences, Tehran, Iran. Tel: +98-21-61192811, E-mail: amiyaneh@gmail.com. Accepted for publication: 21 July 2017

from the gums. On examination, oral hygiene was fair and no ulcer was seen. Gingival hypertrophy (Figure 1) with mild tenderness, two small, non-tender, firm lymph nodes in the left posterior cervical chain and splenomegaly were noted. His temperature was 38° C, and other examinations were unremarkable. White cell count was 20,000 per cubic millimeter. Hemoglobin was 10 milligrams per deciliter, and platelet count was 100,000 per cubic millimeter.

**What is your diagnosis?
See the next page for diagnosis**

Photoclinic Diagnosis:**Acute myeloid leukemia**

Based on history, physical examination, and bi-cytopenia with leukocytosis, bone marrow aspiration and biopsy were performed, revealing about 80% cellularity with more than 90% blasts, normal megakaryocytes, increased ratio of myeloid to erythroid cells and large monoblasts with cytoplasm containing fine granules (Figure 2). Immunophenotyping analysis showed more than 95% monoblasts that were positive for CD117, CD11b, CD14, CD64, and HLA-DR, which supported AML-M5a based on French-American-British (FAB) classification. Cytogenetic analysis revealed abnormal male karyotype with 47, XY, inv(1)(p21q43), t(6;11:10)(q25;q23;p12),+8[20] (Figure 3). Results for FLT3 (ITD) and WTI were negative. In addition, peripheral blood smear showed two monoblasts with abundant cytoplasm and numerous azurophilic granules, containing nuclei with delicate folds (Figure 4).

Abnormal overgrowth of gingival tissues can be classified under four groups: I) inflammatory gingival hypertrophy owing to poor hygiene, II) medication-induced (e.g. nifedipine, phenytoin) gingival hypertrophy, III) hereditary gingival fibromatosis, and IV) systemic causes of gingival hypertrophy, such as pregnancy, hormonal imbalances, and leukemia.¹ Among systemic causes, acute myeloid leukemia (most commonly, acute monocytic leukemia or FAB M5) might manifest only with gingival hypertrophy.² Therefore, the dental practitioner should be aware of this diagnosis.^{3,4}

Acute myeloid leukemia (AML) is a neoplasm of the myeloid line in the bone marrow, which can infiltrate to other tissues. According to FAB classification, in AML-M5, bone marrow monocytic cells constitute more than 80% of non-erythroid cells. Moreover, the bone marrow aspirate contains greater than 30% myeloblasts.

Some risk factors include exposure to pesticides, herbicides, benzene, petroleum, aplastic anemia, myelodysplastic syndrome, paroxysmal nocturnal hemoglobinuria, radiation, and some congenital disorders. Patients may present with fatigue, persistent fever, severe infections, and disorders of hemostasis (such as epistaxis, oral or gingival bleeding, menorrhagia, or cerebral

hemorrhage). Thrombocytopenia may be associated with Disseminated Intravascular Coagulopathy (DIC). Decreased hemoglobin level can lead to pallor, dizziness, headache, collapse, dyspnea and/or congestive heart failure. Infiltration of monoblasts into soft tissues can form masses called "chloroma." Gingival hyperplasia may be seen, but is not the classic sign of AML-M5 aggressive multidrug chemotherapy regimens, and allogeneic bone marrow transplantation (alloBMT) in some patients remain the mainstay of treatment. Without alloBMT, overall survival is 35% – 60%.⁵ However, the overall prognosis in patients with AML-M5 does not differ from other types of AML.⁶

Acknowledgments

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Conflict of Interests: *Authors have no conflict of interests.*

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