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Oral and Maxillary Mucormycosis in a Patient with Acute Myeloid Leukemia: A Rare Case Report

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Introduction: Mucormycosis is an opportunistic fungal infection with rapid progression and high mortality, typically occurring in patients with hematologic malignancies, diabetes mellitus, organ transplantation, or prolonged immunosuppression. Clinically, the most common forms are rhinocerebral, pulmonary, cutaneous, and gastrointestinal involvement. Rhino-cerebral mucormycosis often originates in the paranasal sinuses and may extend to the orbit and brain. Oral mucormycosis is less common and usually presents with maxillary bone necrosis and palatal perforation. Early diagnosis and appropriate antifungal therapy are critical for improving prognosis. In this report, we present a case of newly diagnosed acute myeloid leukemia (AML) who developed rhino-orbito-cerebral mucormycosis involving the maxilla following chemotherapy. Case Report: A 52-year-old male patient was admitted to the hematology outpatient clinic with complaints of epistaxis and fatigue. Laboratory evaluation revealed pancytopenia, and peripheral smear, bone marrow aspiration, and flow cytometry confirmed the diagnosis of acute myeloid leukemia (AML). The patient received induction chemotherapy with daunorubicin (60 mg/m² for 3 days) and cytarabine (100 mg/m² for 7 days). In the second week of treatment, the patient developed pain in the left maxillary region and was referred to the Faculty of Dentistry, Inönü University. Oral and radiological examination (Figure 1)revealed a fixed dental bridge with good marginal adaptation. The prosthetic device was removed, and no pathology was observed in the teeth or surrounding mucosa, Türkiye.

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A CASE OF COVID-19 PNEUMONIA DEVELOPING DURING ALLOGENEIC STEM CELL TRANSPLANTATION IN A PATIENT WITH ACUTE MYELOID LEUKEMIA

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Objective: Allogeneic hematopoietic stem cell transplantation (HSCT) is a curative therapeutic modality for patients with

acute myeloid leukemia (AML)1. Patients undergoing HSCT are highly susceptible to SARS-CoV-2 infection due to profound immunosuppression, delayed immune reconstitution, and graft-versus-host disease (GVHD) prophylaxis2,4. Covid-19 infection in HSCT recipients, particularly during the early post-transplant period, has been associated with high morbidity and mortality5. Studies have demonstrated increased mortality in patients diagnosed with Covid-19 during HSCT, especially in the early phase⁶. Herein, we present a case of Covid-19 pneumonia that developed during allogeneic HSCT with myeloablative conditioning in a patient with high-risk AML. Case report: Our patient was a 33-year-old female diagnosed with AML in March 2025. Following diagnosis, she received induction therapy with the 3+7 regimen (Idarubicin+Cytarabine). As remission was not achieved, she was administered FLAG-Mito as reinduction therapy. Despite two cycles of induction, no response was obtained, and the patient was considered refractory AML; thus, allogeneic transplantation was planned. During the pre-transplant period, she received two cycles of azacitidine + venetoclax. On July 21, 2025, myeloablative conditioning (Fludarabine + Treosulfan) was initiated. During conditioning, her caregiver developed upper respiratory tract infection symptoms and tested positive for Covid-19. Since the patient was asymptomatic, the transplantation procedure was continued. On July 28, 2025, she underwent allogeneic transplantation from her HLA-matched sibling donor. GVHD prophylaxis consisted of CsA + MTX, and voriconazole was given for antifungal prophylaxis. On day +3 post-transplant, the patient developed fever (38°C) and was treated as febrile neutropenia with broad-spectrum antibiotics (Meropenem). SARS-CoV-2 PCR testing was positive. Initially, she presented with mild symptoms, but one week after positivity, chest imaging revealed diffuse pulmonary infiltrates consistent with Covid-19 pneumonia (Figure-1). Despite broad-spectrum antibiotics, she required high-dose corticosteroids and a single dose of tocilizumab (400 mg) for cytokine release syndrome. Oxygen support was initiated. Ten days after pneumonia diagnosis, respiratory distress worsened, and she was admitted to the intensive care unit. On day +18, CPAP was initiated. Neutrophil engraftment was achieved on day +19. However, despite non-invasive respiratory support, progressive respiratory failure necessitated intubation. Shortly after intubation, the patient developed cardiac arrest and, despite CPR, she passed away. DISCUSSION: Patients undergoing allogeneic HSCT develop profound immunosuppression, predisposing them to opportunistic infections with high mortality. COVID-19, which caused a global pandemic in 2020, has also emerged as a life-threatening infection in HSCT recipients⁵. Even after neutrophil engraftment, severe viral pneumonia-related mortality has been reported in transplant patients diagnosed with Covid-197. When SARS-CoV-2 infection occurs in the very early post-transplant period, such as the first week, rapid clinical deterioration may occur due to insufficient immune response and cytokine dysregulation8. Multicenter studies have demonstrated that early post-HSCT Covid-19 infection is associated with high non-relapse mortality (NRM)⁵, 9. In our case, Covid-19 symptoms began in the early post-transplant phase and rapidly progressed to pneumonia. Despite neutrophil engraftment, the pulmonary disease worsened. This

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finding is consistent with results reported during the pandemic period. Although immunosuppressive and cytokine-blocking therapies (steroids, tocilizumab) were administered, they failed to prevent mortality. **Conclusion**: Patients undergoing allogeneic HSCT and their caregivers should undergo comprehensive pre-transplant infectious disease screening. In addition, early initiation of cytokine-targeted therapies may play a role in reducing mortality in this high-risk patient population.

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Autologous Transplant Alone Achieving Decade-Long Remission in AML: A Single-Center Two-Patient Case Report

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Introduction: Allogeneic HSCT is the conventional curative strategy in fit AML patients lacking favorable biology. Autologous HSCT (auto-HSCT) is rarely curative and is typically considered consolidation in selected complete remissions (CR). We report two adults with de novo AML who achieved and maintained extraordinarily long complete remissions after auto-HSCT as the sole transplant approach—highlighting potential patient-selection signals and the under-recognized curative potential of auto-HSCT in carefully chosen cases. Methods: This single-center, retrospective two-patient case report summarizes baseline features, induction/consolidation regimens, transplant details, and long-term outcomes from clinic records. Both patients received standard 7+3 induction, high-dose cytarabine (HiDAC) consolidation, and auto-HSCT due to the absence of suitable allogeneic donors. Follow-up included serial clinical assessment, complete blood counts, and routine biochemistry. Results: Case A (FM, male): Diagnosed 2017-2018 with AML; marrow blasts >20%. Immunophenotype at diagnosis included MPO positivity with CD34 -/CD117- profile. He achieved CR after 7+3 and completed four cycles of HiDAC. In 2008, he underwent auto-HSCT (G-CSF-mobilized peripheral blood). Engraftment was uneventful. Over serial evaluations he has remained in continuous first remission without relapse, secondary malignancy, or organ dysfunction. Current remission duration: 7+ years (2018→2025). Case B (MA,male): Diagnosed in 2010 with AML (marrow blasts \sim 40%). After CR with 7+3 and HiDAC consolidation, lack of a matched donor prompted auto-HSCT the same year using mobilized peripheral blood stem cells. Early and late post-transplant courses were uncomplicated. He remains in continuous first remission with stable counts and no major late toxicities. Current remission duration: \sim 15 years (2010 \rightarrow 2025). **Discussion:** These two cases share three notable features. First, both achieved rapid CR to anthracycline-cytarabine induction and completed HiDACconditions associated with deeper molecular remissions and lower relapse risk. Second, in the absence of a donor, auto-HSCT alone consolidated remission and, in these patients, appears functionally curative across one and a half decades. Third, neither patient developed clinically significant late toxicities or second cancers during long follow-up. Although contemporary risk genomics were unavailable, the CD34negative/MPO-positive phenotype in Case A and the brisk chemo-sensitivity in both suggest favorable disease biology. These observations reinforce that, for rigorously selected AML patients in high-quality CR—particularly when allografting is not feasible—auto-HSCT may deliver durable disease control approaching cure. The report is limited by the small sample size and absence of uniform molecular profiling; nonetheless, the remission lengths (\geq 15 and \geq 17 years) are exceptional and educational. Conclusion: Two adults with AML achieved very long, ongoing first remissions (≈15 and >17 years) after autologous HSCT following 7+3 and HiDAC, without allogeneic rescue. In carefully selected CR patients lacking donors, auto-HSCT can be a valid, potentially curative strategy that merits consideration within individualized treatment algorithms.

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Adult Hematology Abstract Categories

Chronic Leukemias

PP 22

Richter Transformation with Spontaneous Splenic Hematoma: A Life-threatening Complication in Chronic Lymphocytic Leukemia

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Introduction: Richter transformation occurs when chronic lymphocytic leukemia transforms into aggressive lymphoma, most commonly diffuse large B-cell lymphoma (DLBCL), in approximately 5-10% of CLL patients. While typically presenting as rapidly enlarging lymph nodes, extranodal involvement can occur. Splenic transformation is uncommon, and spontaneous splenic hemorrhage represents an extremely rare, life-threatening complication requiring immediate recognition and intervention. Case Report: A 71-year-old female with established CLL presented with progressive abdominal pain, fatigue, and anorexia. She had previously received CLLdirected therapy with initial lymph node regression during follow-up. Physical examination revealed poor general condition, left upper quadrant tenderness with fullness, and minimal peripheral edema. No palpable lymphadenopathy was detected. Laboratory evaluation demonstrated cytopenias: hemoglobin 9.1 g/dL, leukocytosis $13.6 \times 10^3/\mu$ L (lymphocyte- $83 \times 10^{3}/\mu L$. predominant), thrombocytopenia