

**Acute myeloid leukemia
post-allogeneic peripheral
stem cell transplant with
gastric chloroma**

To The Editor: Acute myeloid leukemia (AML) is common among the Saudi Arabian population with a median age at diagnosis of 25 years (range, 0-99 years) among males and 28 years (range, 0-88 years) among females.¹ However, patients with a high risk of relapse, as determined on the basis of cytogenetics, counts on presentation and the presence of an FLT3 mutation, are considered for allogeneic bone marrow transplantation. Extramedullary relapse (EMR) occurs in approximately 2% to 8% of all AML cases. It has been known that relapse can involve the soft tissue of the head and neck, periosteum and bone, as well as the skin and central nervous system (CNS). It also has been reported to involve other sites such as the breast, nasopharynx, paranasal sinusoid, bladder, perineum, testis, chest wall, stomach, intestine, peritoneal cavity and pleural cavity.¹ EMR of AML is not uncommon after allogeneic HSCT. Isolated cases of EMR without evidence of leukemia in the marrow, however, occur rarely. EMR involves the stomach or genitourinary tract very rarely. EMR without coexisting leukemic marrow involvement may occur

because the graft-versus-leukemia (GVL) effect is more pronounced in the marrow than at extramedullary sites.² Donor lymphocyte infusion (DLI), which augments and potentiates the GVL effect, may eliminate the marrow remnants of leukemic cells, but it might not be as effective in eliminating extramedullary leukemic cells.³

We report a case of a 34-year-old female patient diagnosed with AML (FAB M2) with t(8;21). Flow cytometry, which was conducted after she achieved remission for the third time, revealed that the tumor cells were positive for CD13, CD33 and CD56. She received an allogeneic peripheral stem cell transplant from her HLA-identical brother. Two years after the transplant, she presented with odynophagia. On further investigation, she was found to have a mass in the gastric mucosa (Figure 1). An upper gastrointestinal (GI) endoscopy revealed that the mass extended all over and obstructed the gastroesophageal junction. Analysis of the biopsy specimen of this mass confirmed heavy infiltration of the gastric mucosa by malignant cells, i.e., a chloroma (Figure 2). These malignant cells were myeloperoxidase-positive (Figure 3); this confirmed their myeloid lineage. Considering her good performance status, she was treated for the fourth time with standard induction chemotherapy to enable her to achieve remission again and then receive a second bone marrow transplant with another donor. She achieved remission for the fourth time, as confirmed by bone marrow analysis and a chloroma-negative stomach biopsy specimen (Figure 4). However, her post-consolidation phase was complicated by fungal septicemia, which turned out to be fatal.

It has been suggested that the GVL effect derived from chronic



Figure 1. CT scan showing filling defect in the stomach wall.

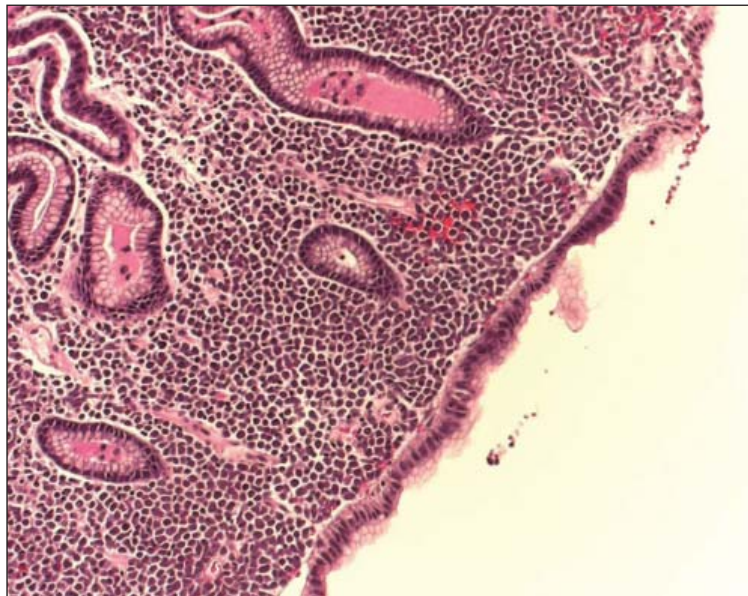


Figure 2. Biopsy showing infiltration of the gastric wall with tumor.

GVHD is effective in preventing only marrow relapse and not extramedullary relapse. EMR following allogeneic HSCT has a dismal prognosis.²⁻⁸ Management of EMR in the post-transplant setting is extremely difficult. Commonly, systemic induction chemotherapy, in addition to localized radiation

to the involved area, has been administered to prevent the overt development of a leukemic relapse. However, most patients who develop EMR have been previously treated with high-dose chemotherapy, and with intense immunosuppressant secondary to GVHD. Thus their risk of toxicity from

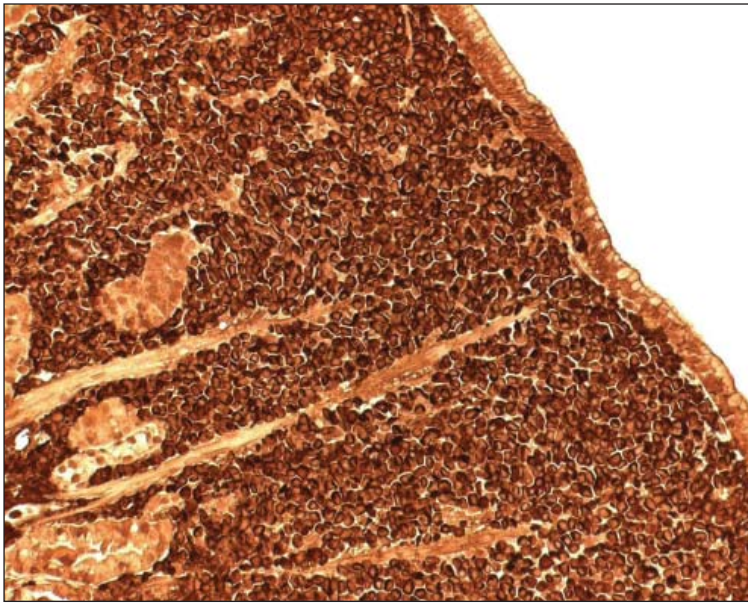


Figure 3. Malignant cells were positive for myeloperoxidase.

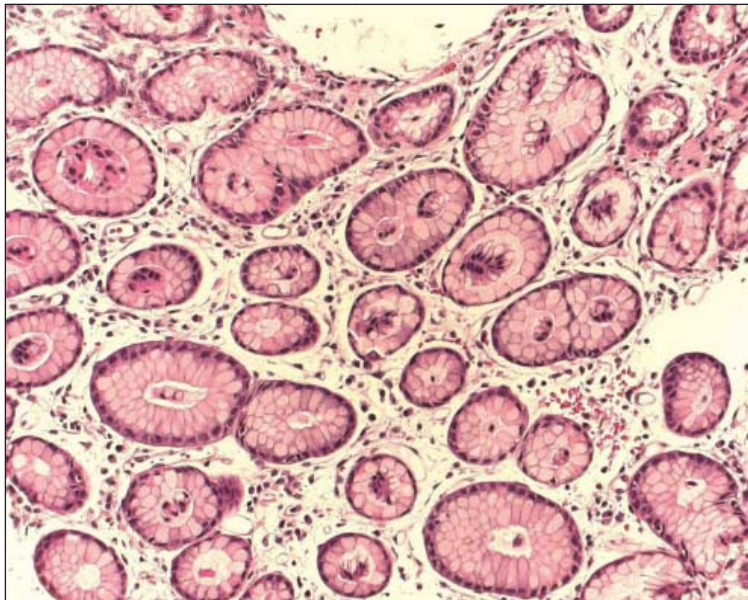


Figure 4. Disappearance of gastric tumor.

further treatment is very high. On the basis of previous experience, it can be said that extensive chronic GVHD has been more frequently observed in patients developing EMR compared to those developing only marrow relapse of leukemia after allogeneic HSCT

in whom chronic GVHD usually does not develop.⁴ In addition, the patient's tumor cells expressed the CD56 antigen, which is reported to be associated with extramedullary involvement of AML⁵⁻⁷ and adhesion of leukemia cells to other extramedullary tissues.

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