Leukemia Cutis at the Site of Trauma in a Patient With Burkitt Leukemia

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Leukemia cutis is an infrequent finding in patients with acute lymphocytic leukemia (ALL). We present a patient with Burkitt ALL (L3ALL) who developed leukemia cutis at the site of trauma.

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Burkitt leukemia is a rare form of acute lymphocytic leukemia (ALL) that accounts for only 1% to 3% of all ALL cases.[1] This leukemia is characterized by a chromosomal translocation (most commonly between chromosomes 8 and 14) affecting the proto-oncogene c-*myc*. Burkitt ALL (also called L3ALL in the French-American-British classification) is the leukemic variant of Burkitt lymphoma and is considered to have a poor prognosis.[2]

Leukemia cutis is an uncommon feature of systemic leukemia that also portends a poor prognosis. The incidence of leukemia cutis is highest in acute granulocytic or monocytic leukemia and is believed to be uncommon in ALL.[3] A MEDLINE search using the keywords *leukemia cutis* revealed no prior reported case of leukemia cutis in Burkitt ALL.

Leukemic skin infiltration is typically multifocal,[3] but there are multiple reports of preferential localization to scars, sites of trauma, burns, and herpetic infections.[4-6] We report a case of leukemia cutis at the site of minor trauma in a patient with Burkitt ALL.

Case Report

In January 2002, a 46-year-old man had 5 days of flulike symptoms. He was found to have a white blood cell count of 41,000/μL with 21% bands. Results of a bone marrow biopsy showed 80% to 90% blasts, morphologically consistent with lymphoblasts. Immunophenotyping was consistent with acute lymphoblastic lymphoma, B-precursor type. The patient was started on chemotherapy with cyclophosphamide, vincristine, doxorubicin, and dexamethasone, as well as intrathecal methotrexate, intrathecal cytarabine, and rituximab. Results of karyotype analysis of his bone marrow revealed a translocation of chromosomes 8 and 14 (t[8;14][24;q32]), and his diagnosis was changed to Burkitt ALL. After 4 cycles of chemotherapy, the patient's bone marrow showed a normal karyotype and no evidence of leukemia. He received 2 additional cycles of chemotherapy.

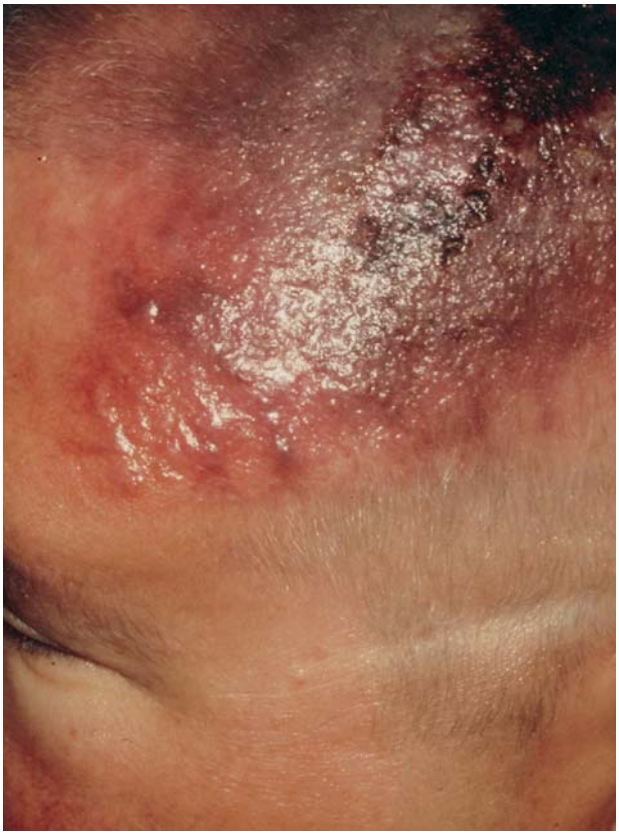

Figure 1. Left frontal parietal scalp with 12×8-cm violaceous plaque.

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In July 2002, the patient scraped the left side of his scalp on a pine tree while mowing the lawn and received a minor abrasion. The following day, he bumped the same area of his head while doing electrical work. One day after this second injury, he noticed redness and swelling at the site of trauma. An oncologist evaluated him one week after the initial injury. A computed tomography scan of the head showed no abnormalities, and the patient was started on dicloxacillin for presumed cellulitis. The lesion continued to grow and became more red and raised. After one week with no improvement, ciprofloxacin was added to his therapeutic regimen. He had no fever or chills. His white blood cell count was within reference range at 7500/μL with a normal differential.

The patient presented to dermatology 2½ weeks after the initial injury. He reported new swelling on the left side of his face and neck in addition to the enlarging scalp lesion. On physical examination, he had a firm violaceous plaque measuring 12×8 cm on the left frontal parietal scalp (Figure 1). The lesion had dusky pigmentation centrally with hemorrhagic crusting, and there were multiple shiny red papules peripherally. He had marked left preauricular and anterior cervical lymphadenopathy. Results of a biopsy showed a malignant lymphoid infiltrate consistent with leukemia/lymphoma (Figures 2 and 3). Immunostaining was not performed in light of the patient's known diagnosis of leukemia. Results of a tissue culture for bacteria, fungi, and acid-fast bacilli were negative.

Results of a computed tomography scan of the head, face, and neck showed an extracranial soft tissue mass in the left frontal and lateral regions, as well as left cervical and supraclavicular lymphadenopathy. Bone marrow biopsy results showed recurrent leukemia, and karyotype analysis again showed a translocation in the region of the c-*myc* oncogene. The patient received salvage ifosfamide, carboplatin, etoposide chemotherapy, as well as intrathecal methotrexate, cytarabine, rituximab, and dexamethasone. His scalp lesion resolved after 2 cycles of chemotherapy. He underwent a bone marrow transplant but died of multisystem organ failure one year after his initial diagnosis.

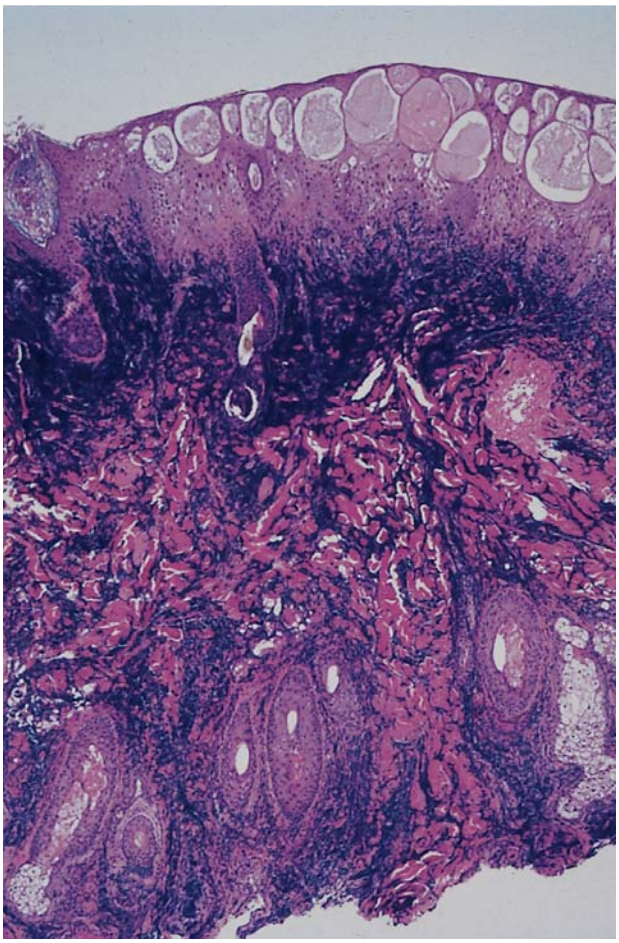

Figure 2. Skin biopsy specimen from the scalp shows a diffuse infiltrate of leukemic cells with extensive crush artifact (H&E, original magnification ×20).

Comment

A MEDLINE search of the literature using the keywords *leukemia cutis* revealed that the incidence of leukemia cutis varies with leukemic subtype, though reported incidences vary widely. Leukemic skin infiltration is most common in monocytic leukemia, with a reported incidence ranging from 10% to 50%.[7] The incidence in acute lymphocytic leukemia is lower, ranging from 1.3% to 3%.[7,8] Leukemia cutis is typically a harbinger of systemic relapse but has been reported as an isolated phenomenon with no evidence of bone marrow involvement.[9]

Burkitt lymphoma was initially reported as an Epstein-Barr virus–associated jaw sarcoma of children in East Africa and was believed to be endemic to Africa.[2] Sporadic cases were subsequently reported in the United States and Europe. These nonendemic forms are less closely associated with Epstein-Barr virus and most often present with an abdominal extramedullary tumor mass.[1,2] In contrast, Burkitt leukemia typically lacks an extramedullary tumor mass and instead presents with signs of marrow failure due to massive bone marrow infiltration by blasts.[1] Despite the different clinical presentations, Burkitt lymphoma and Burkitt leukemia are both characterized by a chromosomal translocation affecting the

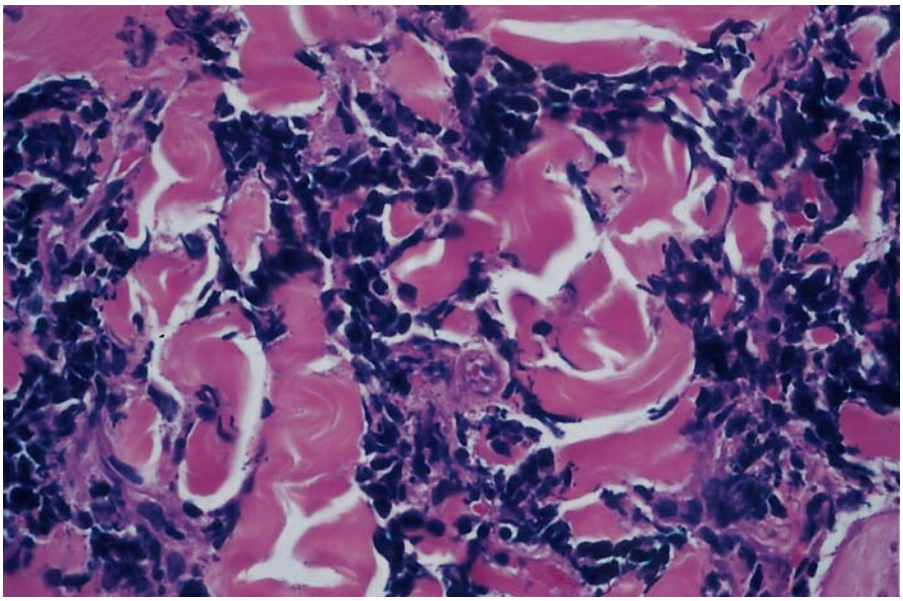

Figure 3. Leukemic cells characterized by a high nuclear-to-cytoplasmic ratio infiltrating collagen bundles (H&E, original magnification ×40).

c-*myc* oncogene.[2] To our knowledge, this is the first report in the literature of Burkitt leukemia with skin infiltration by leukemic cells.

Nonspecific skin lesions associated with systemic leukemia are more common than leukemia cutis and include lesions due to marrow failure (ie, bleeding diatheses, infections) and reactive and paraneoplastic lesions (eg, erythema multiforme, pruritus, nonspecific eczematous eruptions).[8] Although leukemia cutis most often presents as widely disseminated papules, plaques, or nodules, localization to sites of minor trauma, scars, and Hickman catheter sites has been reported.[4,5] The presumed mechanism involves increased production of cytokines by injured keratinocytes leading to proliferation, chemotaxis, and diapedesis of leukocytes.[4] Our patient's diagnosis was delayed by several weeks because he was initially thought to have cellulitis related to trauma. He came to the attention of the dermatology department after seeing an infectious disease specialist who requested a biopsy for cultures.

As this case illustrates, dermatologists should have a low threshold for performing biopsies of persistent skin lesions in patients with a history of leukemia.

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