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Case Report

Leukemia Cutis as the Initial Presentation of B-Cell Acute Lymphoblastic Leukemia: A Case Report and review of the literature

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Abstract

Background: Leukemia cutis (LC) is uncommon in children and may precede recognition of systemic leukemia. A solitary scalp mass with a normal blood count can be mistaken for a benign lesion.

Case Presentation: A 12-year-old girl had a six-month history of a right frontoparietal scalp mass initially considered a lipoma. The lesion was firm, alopecic, and approximately 2 × 2 cm. Complete blood count and peripheral smear were unremarkable. Skin biopsy showed a TdT-, CD19-, CD10-, CD34-, and PAX5-positive precursor B-cell infiltrate with a high Ki-67 index. Bone marrow contained 30.8% blasts with a B-lineage immunophenotype, establishing B-cell acute lymphoblastic leukemia (B-ALL). The scalp lesion nearly regressed after induction therapy. Although morphologic remission was achieved at end of induction, measurable residual disease (MRD) was 2.05%; MRD declined to 0.0066% at end of consolidation and was 0.0033% at last reported assessment.

Conclusion: Persistent or enlarging scalp lesions in children warrant tissue diagnosis even when peripheral blood findings are normal.

1. INTRODUCTION

Leukemia cutis (LC) is infiltration of the epidermis, dermis, or subcutis by neoplastic hematopoietic cells. It is distinct from nonspecific cutaneous disorders seen in patients with leukemia, such as infection, drug eruption, and inflammatory dermatoses (1). Lesions may be papules, nodules, plaques, or less commonly macules, ulcers, bullae, or purpura; they are often firm and red, violaceous, brown, or hemorrhagic. The head and lower extremities are commonly involved, although presentation is variable (2-4).

LC may be diagnosed concurrently with systemic leukemia, precede it, or less commonly, occur without detectable marrow or peripheral blood disease (aleukemic LC, ALC). ALC represents fewer than 10% of LC cases in the cited literature; skin disease may precede systemic disease by days to months (2, 5). A normal complete blood count or smear therefore does not exclude a cutaneous leukemic infiltrate. The biology of skin tropism is incompletely understood; proposed mechanisms include selective homing involving adhesion molecules and chemokine receptors, and associations with local inflammation and CD56 expression in some acute leukemias (1, 4).

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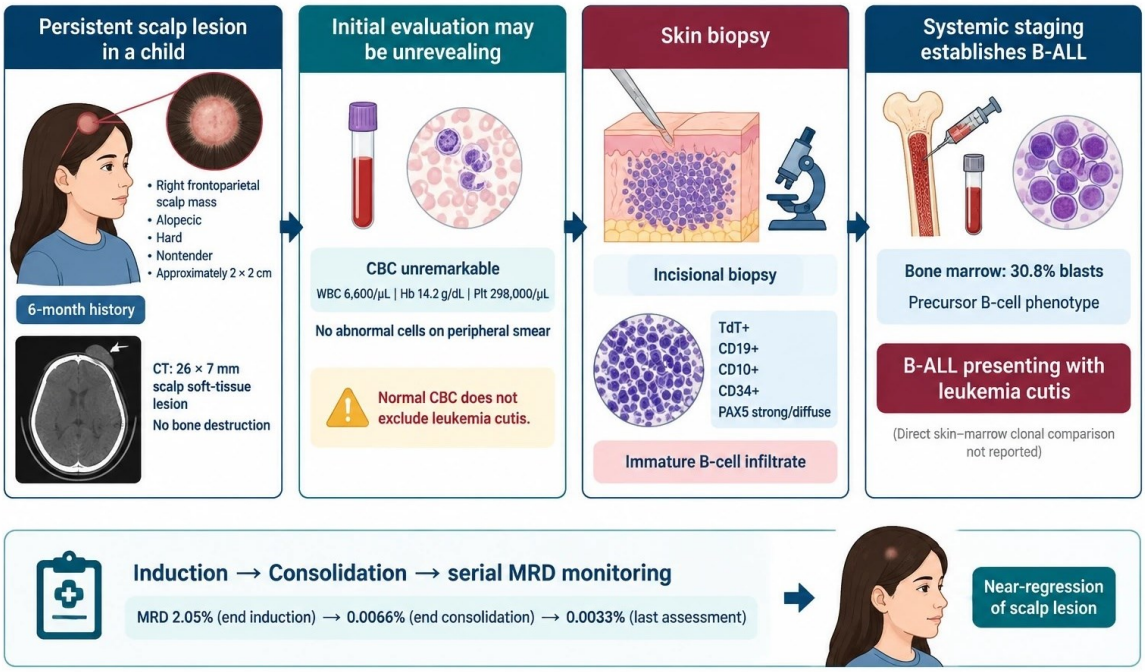

Figure 1. Graphical abstract.

In children, the clinical appearance is nonspecific and can resemble benign tumors, inflammatory eruptions, histiocytic disorders, or metastatic malignancy. A biopsy with appropriate immunohistochemistry is central to diagnosis, followed by hematologic staging. We describe a 12-year-old girl whose solitary scalp mass was initially considered a lipoma and whose skin biopsy suggested a precursor B-cell lymphoblastic neoplasm; bone marrow evaluation established B-cell acute lymphoblastic leukemia (ALL). This case emphasizes the diagnostic value of integrating skin histology with marrow findings.

2. CASE REPORT

A 12-year-old girl presented to dermatology with a six-month history of swelling in the right frontoparietal scalp. The lesion had initially been considered a lipoma. Examination showed a well-circumscribed, hard, nontender, alopecic mass measuring approximately 2 × 2 cm, without erythema or discharge (**Figure 2A**). There was no palpable lymphadenopathy or hepatosplenomegaly. Oral, regional lymph-node, and remaining skin examinations were unremarkable.

Complete blood count showed a white-cell count of 6,600/µL, hemoglobin 14.2 g/dL, and platelet count 298,000/µL. Peripheral blood smear showed no abnormal cells. Erythrocyte sedimentation rate, lactate dehydrogenase (LDH), uric acid, and the reported chemistry panel were described as unremarkable. Cranial computed tomography showed an oval soft-tissue lesion measuring 26 × 7 mm in the frontal scalp, without macroscopic fat or bone erosion/destruction. Because the lesion enlarged, an incisional biopsy was performed.

Skin histology and immunohistochemistry demonstrated an immature B-cell infiltrate. The lesional cells were positive for TdT, CD19, CD10, CD34, and PAX5 (strong and diffuse), with a high Ki-67 index. The skin report favored a precursor B-cell lymphoblastic lymphoma/leukemia.

Bone marrow immunophenotyping identified 30.8% blast cells. Within the reported blast gate, CD19, CD10, and CD22 were each positive in 48%; CD34 in 82%; and HLA-DR in 58%. The blasts were negative for CD7, CD13, CD20, and CD117. These findings supported B-lineage ALL. The reported skin and marrow profiles overlap in precursor B-cell markers (CD19, CD10, CD34, and, in skin, TdT/PAX5), supporting a related B-lymphoblastic population; however, direct clonal comparison was not reported. The marrow blast proportion establishes B-ALL in this case (rather than an isolated primary cutaneous lymphoblastic lymphoma). Accordingly, the integrated diagnosis is B-ALL presenting with LC.

Bone-marrow conventional karyotyping by G-banding showed 46,XX without reported numerical or structural

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Table 1. **Selected pediatric leukemia cutis reports cited in this manuscript**

Reference	Age / presentation	Underlying disease / salient point
(6)	19-month-old; morbilliform eruption	AML; initially treated as drug eruption; skin biopsy established LC
(7)	2-year-old; erythematous-to-violaceous rash and brow tumor	B-ALL; initially unremarkable CBC/smear reported
(2)	10-month-old; lesions mimicking cutaneous mastocytosis	B-ALL with aleukemic cutaneous presentation; diagnosis by skin biopsy
(8)	4-year-old; Sweet syndrome-like papules/plaques	JMML with LC; somatic PTPN11 variant reported
(9)	2-month-old; solitary parietal scalp nodule	Pro-B-ALL with t(9;11)/KMT2A::MLLT3
Present case	12-year-old; six-month solitary lipoma-like scalp mass	B-ALL (marrow 30.8% blasts); normal CBC/smear; skin lesion nearly regressed after induction

abnormalities. Qualitative real-time PCR for ETV6::RUNX1 [t(12;21)], BCR::ABL1 [t(9;22)], TCF3::PBX1 [t(1;19)], and KMT2A::AFF1 [t(4;11)] transcripts showed no detected aberration. No additional molecular testing was reported. Cerebrospinal-fluid analysis showed no CNS involvement.

Treatment was reported as a Children's Oncology Group (COG)-based high-risk B-ALL regimen (AALL0331). The available account lists daily dexamethasone for 28 days, weekly vincristine and doxorubicin for four weeks, peg-asparaginase, and intrathecal methotrexate and cytarabine during induction. The scalp lesion nearly completely regressed after induction (**Figure 2B**). End-of-induction marrow was normocellular with <5% blasts (morphologic M1 remission), while day-29 flow cytometry remained MRD-positive at 2.05%. No fully-matched related or unrelated hematopoietic stem cell transplant (HSCT) donor was identified in the reported donor search. Treatment continued through consolidation, with end-of-consolidation MRD of 0.0066%. During subsequent chemotherapy and serial marrow monitoring, MRD declined; at manuscript preparation, the patient was in maintenance, with medullary and extramedullary remission and last reported MRD of 0.0033%.

3. DISCUSSION

This case illustrates an uncommon presentation of B-ALL as a solitary scalp mass in a child with normal peripheral blood counts and smear. The lesion was clinically labeled a lipoma, but its firmness, alopecia, interval enlargement, and absence of macroscopic fat on CT prompted biopsy. The diagnosis required integration of cutaneous histology, immunophenotype, and marrow evaluation.

Diagnostic classification merits clarification. B-lymphoblastic lymphoma and B-ALL are manifestations of the same precursor B-cell neoplasm and are distinguished chiefly by the extent of marrow involvement; a marrow blast proportion of 25% or greater supports the leukemia designation. Here, marrow contained 30.8% blasts, so the final diagnosis is B-ALL with LC. The initial skin interpretation of lymphoblastic lymphoma/leukemia describes the cutaneous precursor B-cell neoplasm, not a separate final diagnosis of primary cutaneous lymphoma. The overlapping precursor B-cell markers in skin and marrow are concordant, although no molecular/clonal comparison between the sites was reported. The absence of cytopenia or circulating blasts does not negate marrow-defined B-ALL.

Pediatric LC can mimic a broad range of conditions. Published examples include a morbilliform eruption initially treated as a drug reaction in a toddler with AML (6), a rash and brow tumor preceding recognition of B-ALL despite initially unremarkable blood findings (7), lesions mistaken for cutaneous mastocytosis in an infant with ALC who later had B-ALL (2), and a Sweet syndrome-like eruption in a child with juvenile myelomonocytic leukemia (JMML) (8). A particularly relevant report described a healthy-appearing two-month-old infant with a solitary parietal scalp nodule; biopsy and subsequent evaluation established pro-B-ALL with t(9;11)/KMT2A::MLLT3 (9). These reports show that both diffuse eruptions and isolated nodules may precede or accompany pediatric leukemia. The present case adds an older child with a six-month, lipoma-like scalp lesion, normal CBC and smear, and a marrow blast count diagnostic of B-ALL.

Table 1 summarizes the pediatric reports described in the cited literature and in the source manuscript. It is a focused narrative summary, not a systematic review; case details are limited to information reported in those publications and the original manuscript. In a cohort cited in the literature,

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Figure 1A. Leukemia cutis. A dome-shaped, indurated, crusted erythematous-purplish nodule in the scalp surrounded by atrophic and telangiectatic skin.

Figure 1B. The lesion after one month of induction chemotherapy.

24 of 1,359 children with acute leukemia/lymphoblastic lymphoma had skin involvement; lesions preceded diagnosis by a median of six weeks (range, days to eight months), and the head was involved in 21 of 24 cases (1). The source literature also reports that approximately 30% of LC cases coincide with newly diagnosed leukemia and fewer than 10% present as ALC (5). These estimates are drawn from heterogeneous reports and should not be interpreted as precise pediatric-specific incidence estimates.

The differential diagnosis of a solitary scalp mass includes lipoma and dermoid/epidermoid cyst, which are benign lesions but may be distinguished by imaging and histology; the absence of macroscopic fat in this lesion argued against lipoma. Neurofibroma and other soft-tissue tumors, Langerhans-cell histiocytosis and other histiocytic proliferations, and infectious/inflammatory nodules are also considerations. Malignant alternatives include metastatic neuroblastoma, Ewing sarcoma, rhabdoid tumor, and other sarcomas (10-15). Primary cutaneous B-lymphoblastic lymphoma is an especially relevant histologic differential because it may share the same precursor B-cell phenotype; marrow staging and the extent of marrow blasts resolve the classification in this patient. Other lymphomas are assessed by morphology and lineage-specific immunophenotyping.

LC is a manifestation of the underlying hematologic malignancy and is generally treated with systemic therapy directed at the leukemia subtype; cutaneous lesions commonly regress with hematologic response (1). In pediatric ALL, published experience includes prompt skin responses to ALL-directed regimens and durable remissions in individual reports (1, 2, 9). In the present patient, near-complete regression followed induction, while MRD remained positive at end of induction and subsequently declined during therapy. The case therefore demonstrates clinical cutaneous response but also the need to distinguish visible lesion regression from marrow MRD response and to continue risk-adapted leukemia treatment.

Pediatric LC is also reported in AML and JMML. The cited literature describes associations between AML-related LC and KMT2A/MLL abnormalities and discusses outcome data from the ELAM02 study (16, 17); such associations are not universal and do not apply to this patient, whose reported targeted fusion panel was negative.

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While the etiology of childhood leukemia is multifactorial involving an interplay between genetic predisposition and environmental exposures, epidemiological studies have identified several postnatal risk factors, including family history of leukemia, birth order, and household environmental factors (18). This highlights the importance of obtaining a thorough clinical and family history when evaluating pediatric patients with unusual skin lesions, as in the present case. Although rare, LC in children requires a high index of suspicion for timely diagnosis.

The present report is distinctive for the prolonged, solitary, apparently benign scalp presentation in a 12-year-old, the normal peripheral blood evaluation, and marrow-confirmed B-ALL with concordant precursor B-cell markers in the skin. It reinforces that persistent or enlarging scalp masses deserve biopsy and that a normal CBC should not defer hematologic evaluation when pathology suggests a hematolymphoid neoplasm. Early recognition and prompt treatment are essential for improving prognosis and guiding therapeutic strategies.

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Conflict of interest

All authors declare that they have no conflict of interest.

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Ethical statement

An informed written consent was taken from the patient and her parents to publish the report. We assure them that it will be done anonymously.

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