Primary cutaneous diffuse large B-cell lymphoma of the scalp: A case report and brief review of the literature

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Abstract. Primary cutaneous lymphomas (PCLs) are a heterogeneous group of extranodal lymphoid neoplasms confined to the skin at diagnosis. Primary cutaneous diffuse large B-cell lymphoma, not otherwise specified (PCDLBCL-NOS), is a rare and aggressive subtype, and the involvement of the scalp is exceptionally uncommon. The present study describes the case of a 45-year-old female patient with PCDLBCL-NOS presenting with atypical alopecic scalp lesions. The patient presented with a 4-month history of localized scalp hair loss associated with mild erythema and pruritus, without systemic symptoms. A histopathological examination of a punch biopsy revealed a dense dermal and perifollicular infiltrate of atypical large lymphoid cells. Immunohistochemistry demonstrated positivity for CD20, CD10, CD79a and BCL-2, with negativity for CD3 and MUM1. Comprehensive imaging revealed no extracutaneous disease, and a diagnosis of stage IA(E) PCDLBCL-NOS was established. The patient was treated with six cycles of R-CHOP chemotherapy, achieving complete clinical and radiological remission at the 6-month follow-up. Subsequently, the patient developed pancytopenia and was diagnosed with B-cell acute lymphoblastic leukemia. Despite the initiation of HYPER-CVAD chemotherapy, her course was complicated by severe neutropenic sepsis; thus, the patient succumbed. In addition, a review of the literature identified only five previously reported cases of scalp PCDLBCL, all occurring in male patients. The case described herein highlights an unusual clinical presentation, underscores the importance of considering lymphoma in the differential diagnosis of atypical alopecic scalp lesions, and illustrates both the potential for initial therapeutic response and the risk of severe hematologic complications.

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Introduction

Primary cutaneous lymphomas (PCLs) are a diverse group of lymphoproliferative malignancies restricted to the skin at diagnosis without involving the lymph nodes, bone marrow, or other organs. While non-Hodgkin lymphoma (NHL) is relatively common, PCLs account for only 4% of all NHL cases annually. These lymphomas primarily originate from T-lymphocytes (65%), B-lymphocytes (25%), or natural killer cells (10%) (1).

The incidence of primary cutaneous B-cell lymphomas (PCBCLs) is estimated to be <1 per 100,000 individuals per year, with a rise in frequency as with the increase in age (2). The World Health Organization-European Organization for Research and Treatment of Cancer classifies PCBCLs into three main subtypes: Primary cutaneous marginal zone lymphoma (PCMZL), primary cutaneous follicle center lymphoma (PCFCL) and primary cutaneous diffuse large B-cell lymphoma, leg type (PCDLBCL-LT) (3).

PCDLBCL-LT is rare and aggressive, marked by the abnormal growth of B-cells in the skin (4). This condition typically involves the dermis and subcutaneous layers, resulting in rapidly growing, red to blueish nodular tumors, usually on the lower limbs. However, it can also occur in other parts of the body. PCDLBCL-LT accounts for 4% of all newly diagnosed PCLs annually. It primarily affects older women and is associated with a worse prognosis (4).

Cases of PCDLBCL that were not categorized as PCDLBCL-LT have long been termed primary cutaneous diffuse large B-cell lymphomas, other (PCLBCL/other). In the WHO classification for skin tumors, these were renamed as primary cutaneous diffuse large B-cell lymphomas, not otherwise specified (PCDLBCL-NOS) (3).

The present study describes the case of a 45-year-old female patient with PCDLBCL on the scalp. PCDLBCL on the scalp is extremely rare, with only a few cases documented in the literature to date (4-8).

Case report

Patient information. A 45-year-old female patient presented to Smart Health Tower (Sulaymaniyah, Iraq) in January, 2025 with localized hair loss on the scalp lasting for 4 months, accompanied by mild redness and itching. No notable systemic symptoms were reported.

Clinical findings. Upon a clinical examination, irregular patches of hair thinning, each a few centimeters in size, were observed on an erythematous base, accompanied by slight swelling but no palpable mass (Fig. 1). There was no lymphadenopathy or similar lesions elsewhere on the body.

Diagnostic approach. Laboratory investigations at initial presentation revealed anemia with a hemoglobin level of 7.7 g/dl (reference range, 12-16 g/dl) and leukopenia with a white blood cell count of $2.1x10^9$/l (reference range, $4.0\text{-}11.0x10^9$/l), while the platelet count was within normal limits at $167x10^9$/l (reference range, $150\text{-}400x10^9$/l). The erythrocyte sedimentation rate was elevated at 38 mm/h (reference range, 0-20 mm/h). A peripheral blood smear examination demonstrated normochromic anemia with neutropenia, with no circulating atypical lymphoid cells or blast forms. Liver and renal function test results were within normal ranges: Alanine aminotransferase, 22 U/l (reference range, 7-56 U/l); aspartate aminotransferase, 25 U/l (reference range, 10-40 U/l); alkaline phosphatase, 86 U/l (reference range, 44-147 U/l); total bilirubin, 0.8 mg/dl (reference range, 0.2-1.2 mg/dl); serum creatinine, 0.9 mg/dl (reference range, 0.6-1.3 mg/l); blood urea nitrogen, 14 mg/dl (reference range, 7-20 mg/dl). Serological testing for human immunodeficiency virus, hepatitis B surface antigen and hepatitis C virus yielded negative results. A 4-mm punch biopsy was obtained from an active erythematous alopecic lesion on the parietal region of the scalp. The specimen was fixed in 10% neutral buffered formalin at room temperature for 24 h, embedded in paraffin, and sectioned at a thickness of 5 μm. A histological examination was performed using hematoxylin and eosin (H&E) staining (Bio Optica Co.), applied for 1-2 min at room temperature, and evaluated under a light microscope (Leica Microsystems GmbH). Microscopic analysis revealed multiple sheets of atypical cells with scant cytoplasm and large vesicular nuclei with prominent nucleoli, infiltrating the reticular dermis and subcutaneous fat and surrounding the hair follicles, while the overlying epidermis remained uninvolved. In our institution, immunohistochemical analysis for BCL2 was performed on formalin-fixed, paraffin-embedded tissue sections cut at a thickness of 4-5 μm and mounted on positively charged glass slides. The sections were incubated in an oven at 60˚C overnight, followed by deparaffinization in xylene and rehydration through graded alcohol solutions to distilled water. Heat-induced antigen retrieval was carried out using the Dako PT Link system (Agilent Technologies, Inc.) at 100˚C for 20 min using alkaline buffer (pH 9.0). Endogenous peroxidase activity was quenched using 3% hydrogen peroxide at room temperature. The slides were then rinsed in Tris-buffered saline containing 0.05% Tween-20 (pH 7.6), and a hydrophobic barrier was created using a Dako Pen (Agilent Technologies, Inc.). The primary antibody used was a mouse monoclonal anti-BCL2 antibody (clone 124; cat. no. M0887; Dako/Agilent Technologies, Inc.), applied at a dilution of 1:100 and incubated at room temperature for 60-80 min. Immunoreactivity was detected using a horseradish peroxidase-labeled polymer-based secondary detection system (EnVision™+ System-HRP, anti-mouse) obtained from Dako/Agilent Technologies, Inc. (cat. no. K5007), with 3,3'-diaminobenzidine (DAB) used as the chromogen. Finally, the sections were counterstained with Gill II hematoxylin for 30 sec, dehydrated, cleared, and mounted for light microscopic examination. All slides were evaluated using a standard light microscope (Leica Microsystems GmbH). Immunohistochemistry subsequently demonstrated diffuse BCL2 positivity (Fig. 2). These findings were consistent with a diagnosis of non-Hodgkin's lymphoma involving the hair follicles and subcutaneous fat, for which a comprehensive immunohistochemical panel was required for definitive confirmation and subtyping. Another punch biopsy was performed at an external hospital (Shorsh General Hospital, Sulaymaniyah). A histopathological examination demonstrated a diffuse dermal and perifollicular infiltrate of atypical intermediate- to large-sized lymphoid cells with vesicular to hyperchromatic nuclei, prominent nucleoli, and a moderate amount of pale cytoplasm. Extension of the infiltrate into the subcutaneous tissue was also observed. Immunohistochemistry revealed positive CD20, CD10 and CD79a. At the same time, CD3 and MUM1 were negative. Representative immunohistochemistry images were not available, as this component of the diagnostic evaluation was performed at an external institution and the original histopathology and immunohistochemistry image files could not be retrieved. These findings were diagnostic of non-Hodgkin lymphoma, specifically diffuse large B-cell lymphoma of germinal center type. Computed tomography (CT) scans of the neck, chest, abdomen and pelvis revealed no evidence of lymphadenopathy, hepatosplenomegaly, or visceral organ involvement. The corresponding CT images were not available for review, as the imaging was performed at an external radiology facility. Based on the localized cutaneous disease, negative systemic imaging, histopathological findings and immunophenotypic profile, the diagnosis of PCDLBCL-NOS, stage IA(E), was established. Viral screening was negative, and bone marrow aspiration and biopsy demonstrated lymphoma involvement.

Therapeutic intervention. The patient received six cycles of standard-dose R-CHOP chemotherapy administered every 21 days, consisting of rituximab 375 mg/m^2 intravenously on day 1, cyclophosphamide 750 mg/m^2 intravenously on day 1, doxorubicin (hydroxydaunorubicin) 50 mg/m^2 intravenously on day 1, vincristine (Oncovin) 1.4 mg/m^2 intravenously on day 1 (maximum dose 2 mg), and prednisone 100 mg orally on days 1-5. Prophylactic antibiotics and granulocyte colony-stimulating factor (G-CSF) were administered according to the institutional protocol. During treatment, the patient developed chemotherapy-induced neutropenia, which was managed with supportive care, including the administration of G-CSF,

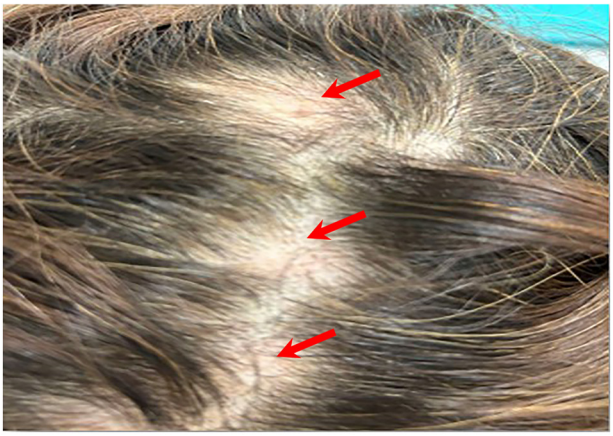

Figure 1. Irregular erythematous plaques (red arrows), each measuring a few centimeters in diameter, accompanied by mild hair thinning.

the close monitoring of complete blood counts and prophylactic broad-spectrum antibiotics. Antibacterial prophylaxis consisted of oral levofloxacin 500 mg once daily. No treatment interruptions were required, and subsequent chemotherapy cycles were continued with G-CSF support.

Follow-up and outcome. The patient was followed-up for a total duration of 6 months following the completion of chemotherapy. Follow-up assessments included regular clinical examinations, laboratory investigations and contrast-enhanced CT scans of the chest and abdomen, as well as positron emission tomography imaging. At the 6-month follow-up, all scalp lesions were completely resolved, with no evidence of residual or recurrent disease on imaging and no signs of lymph node or internal organ involvement (Fig. 3). The patient continued to undergo clinical and radiological surveillance.

Subsequently, the patient developed pancytopenia. Repeat bone marrow aspiration and biopsy, along with flow cytometry, led to the diagnosis of B-cell acute lymphoblastic leukemia.

Molecular analysis for the BCR-ABL fusion gene was conducted at an external medical facility (Hiwa Cancer Hospital, Sulaymaniyah, Iraq) using reverse transcription polymerase chain reaction (RT-PCR) on RNA extracted from bone marrow aspirate specimens. Primers specific for the major (b2a2, b3a2) and minor (e1a2) BCR-ABL fusion transcripts were used. No amplification of BCR-ABL transcripts was detected, confirming the absence of the Philadelphia chromosome. Detailed technical information regarding the RNA extraction buffer and manufacturer, the reverse transcription reagents, as well as the exact temperature and cycling protocols, was not available to the authors. In addition, cytogenetic evaluation was also performed at the same external facility using standard G-banding techniques on short-term cultured bone marrow aspirate cells, with a minimum of 20 metaphase spreads analyzed. The karyotype revealed a normal chromosomal complement without evidence of recurrent or complex cytogenetic abnormalities. The patient was commenced on the HYPER-CVAD chemotherapy protocol, consisting of alternating courses of hyperfractionated cyclophosphamide, vincristine, doxorubicin (Adriamycin), and dexamethasone (course A), and high-dose methotrexate with cytarabine (course B). Course A included cyclophosphamide at 300 mg/m^2 intravenously every 12 h on days 1-3, vincristine at 2 mg intravenously on days 4 and 11, doxorubicin 50 mg/m^2 intravenously on day 4, and dexamethasone at 40 mg orally or intravenously on days 1-4 and 11-14. Course B consisted of methotrexate 1 g/m^2 intravenously over a period of 24 h on day 1, followed by cytarabine at 3 g/m^2 intravenously every 12 h on days 2 and 3. Cycles were planned to alternate every 21-28 days according to hematologic recovery. During treatment, she developed severe pancytopenia complicated by neutropenic sepsis. Despite appropriate supportive and antimicrobial management, the patient unfortunately succumbed to sepsis.

Discussion

Systemic lymphomas can affect the skin as secondary cutaneous lymphomas exhibit distinct clinical behaviors and prognoses compared to PCL, necessitating different treatment approaches (6). PCBCLs often manifest as smooth, well-defined, erythematous to non-ulcerated violaceous nodules, primarily appearing on the trunk, head and neck regions. Among cutaneous sites, the scalp is a rare location for these lymphomas (6). Čolović *et al* (8) described a case of scalp PCDLBCL characterized by regional hair loss, a scalp rash and itching that had been present for 16 years. By contrast, Khatib *et al* (5) reported a case involving a solitary scalp swelling that progressively enlarged over a period of 5 months. In the case presented herein, the patient exhibited localized scalp hair loss, mild redness and itching for 4 months. Viral screening was negative, and bone marrow aspiration and biopsy demonstrated lymphoma involvement.

PCDLBCL-LT is most frequently diagnosed in elderly women, with a median age of 70 years at diagnosis. Typically, PCDLBCL-LT presents as solitary or multiple asymptomatic, rapidly growing red or purple dermal nodules, primarily affecting one or both legs. However, in 15-20% of cases, the initial tumor arises at sites other than the legs (1,4). A literature search was performed using PubMed and Google Scholar to identify previously reported cases of PCDLBCL involving the scalp. Combinations of the terms 'primary cutaneous diffuse large B-cell lymphoma', 'scalp', 'cutaneous lymphoma', 'non-Hodgkin lymphoma' and 'case report' were used, with no time restrictions applied. All available articles up to the time of submission were screened based on the relevance of the title and abstract, followed by full-text review of potentially relevant reports. Through this process, five previously reported cases of scalp PCDLBCL were identified, all occurring in male patients aged 46-92 years (4-8) (Table I). By contrast, the patient in the present case report was a 45-year-old female.

The defining features of PCDLBCL-NOS are not yet well-established. Recent research indicates that this condition is more common in younger individuals and typically manifests as a gradually enlarging plaque exceeding 5 cm at diagnosis, often due to delayed medical attention. The PCDLBCL-NOS subtype usually involves multiple lesions confined to one or two adjacent body areas. A notable difference in tumor location on the lower leg has been identified between the PCDLBCL-LT and PCDLBCL-NOS subtypes, although no significant difference in ulceration rates has been observed. Nevertheless, tumor location alone cannot provide a definitive diagnosis (4). In a review of

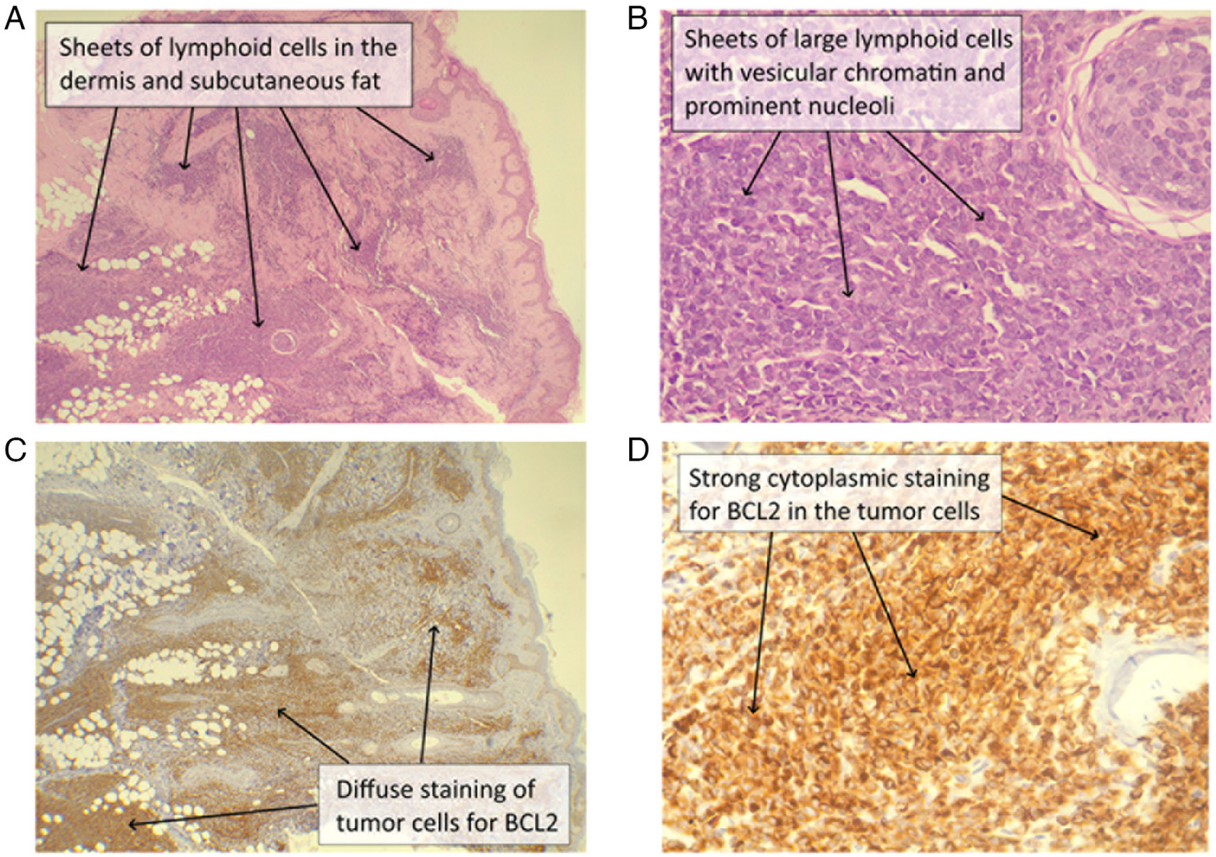

Figure 2. (A) There are sheets of lymphoid cells diffusely infiltrating the dermis, around the adnexa and into the subcutaneous fat. The overlying epidermis is unremarkable. (B) There are sheets of atypical, large cells that have scant cytoplasm with large nuclei, vesicular chromatin, irregular nuclear outlines, and prominent nucleoli. (C and D) The tumor cells are diffusely positive for BCL2 in a cytoplasmic pattern with strong intensity. (A and B) Hematoxylin and eosin staining, and (C and D) immunohistochemistry for BCL2 using diaminobenzidine chromogen. Original magnification (A and C) x40, and (B and D) x400.

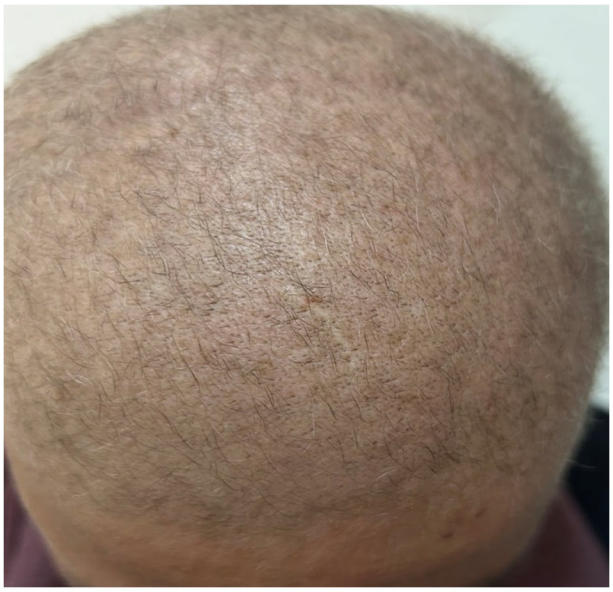

Figure 3. Complete resolution of all scalp lesions following six cycles of R-CHOP.

2,831 cases of B-cell lymphomas conducted by Kim *et al* (9), only 21 patients were identified with PCLBCL, of which only two cases belonged to the PCLBCL-NOS subtype.

The exact pathogenesis of PCBCL is not yet fully understood. It is hypothesized that PCBCL may arise as a lymphoproliferative response to antigenic stimuli in the skin, similar to the process observed in mucosa-associated lymphoid tissue in the gastrointestinal tract (5). Molecular analyses on PCBCL support this theory, revealing a characteristic pattern of somatic hypermutation and intraclonal diversity in B-cell immunoglobulin genes, suggesting an antigen-driven germinal center origin (5).

Both variants of DCLBCL are aggressive, with a 5-year survival rate ranging from 20 to 60% (4). It is crucial to differentiate DCLBCL from PCFCL, particularly those with a diffuse growth pattern, as PCFCL requires a different therapeutic approach and has a highly favorable prognosis, with a 90% 5-year survival rate (4).

Histopathological features of PCDLBCL include a non-epidermotropic, diffuse infiltration of the dermis by large cells, predominantly composed of centroblasts and immunoblasts, which may extend into the subcutis. The infiltration is characterized by its monotonous nature and prominent mitotic activity, with few inflammatory or reactive T-cells present (4). Tumor cells exhibit B-cell-related antigens, including CD19, CD20, CD22, CD79a and Pax-5. The majority of PCBCLs express germinal center-associated antigen BCL6 and post-germinal center antigen mum-1/IRF-4, while CD5 and CD10 are generally negative, and the t(14;18) (q32;q21) translocation is absent. The apoptosis-regulating protein BCL2 is strongly expressed in 100% of PCDLBCL-LT cases, but only in

Table I. Review of 5 cases of PCBCLs affecting the scalp identified in the literature.

First author	Year of publication	Age, years	Sex	Presentation	Examination	Imaging	IHC		Treatment	Outcome	(Refs.)
							Positive	Negative			
Behera	2022	62	Male	7-month history of a gradually enlarging, asymptomatic swelling on the right temple.	An erythematous lobulated plaque (5x2 cm) with telangiectasia a satellite papule on the right temporal area, and two arcuate plaques on the occipital scalp.	A CT of the scalp showed bone erosion up to the dura beneath the temporal lesion.	CD20 Bcl-2 Bcl-6	CD10 CD5 CD30 CD138 CD21 CyclinD1 ALK1 MUM1	The patient underwent six cycles of R-CHOP and intrathecal methotrexate	At 1 year post-therapy, the patient remains well, with no recurrence or recurrence or	(4)
Khatib	2017	57	Male	A solitary scalp swelling gradually increased over 5 months.	A nontender scalp swelling (2x2 cm) with a bosselated surface, irregular margins, and firm consistency.	An axial CT of the brain showed an ill-defined hyperdense lesion in the right parietal scalp with irregular margins extending into the deeper layers, with no bony erosion.	CD20 Bcl-6[a]	CD3 Bcl-2 CD10 CD30 MUM-1 CD23 CD4	The patient received three cycles of R-CHOP chemotherapy	The skin lesion fully regressed after treatment.	(5)
Liao	2017	92	Male	Multiple large nodular scalp masses on the right parieto-occipital region for 7 months.	A firm, painless, non-pulsating subcutaneous scalp mass measuring approximately 8x12 cm, with multiple nodules	An MRI showed a large, multiple-nodular scalp mass with heterogeneous signals, low on T1-weighted imaging and variable on T2-weighted imaging.	CD20 Bcl-2	N/A	underwent surgery for partial removal of the scalp mass.	The tumor recurred *in situ* after 6 months, and the patient died from dyscrasia 2 years later.	(6)
Ochiai	2010	72	Male	The patient presented with a slow-growing mass in the	A 7 cm, firm, slow-growing mass was noted in the left	A CT scan revealed a subcutaneous	CD 20 CD79α	CD3 CD45R0	The patient had a craniotomy,	The patient was discharged without any	(7)

Table I. Continued.

First author	Year of publication	Age, years	Sex	Presentation	Examination	Imaging	IHC		Treatment	Outcome	(Refs.)
							Positive	Negative			
				left temporoparietal area of the scalp.	temporoparietal region.	mass in the left temporoparietal region and an extra-axial mass beneath it, without skull bone invasion.			revealing a gray, hard, hypovascular tumor then he received whole-brain radiation and CHOP chemotherapy	neurological deficits.	
Čolović	2008	46	Male	Regional baldness, scalp rash, and itching occurred 16 years prior.	N/A	The head tomographic scan was normal, except for a tumorous mass in the scalp's soft tissue.	CD 20 CD79α	Bcl-2 CD30 CD3 CD5 Bcl-10	The patient was treated with the R-CHOP regimen, completing 8 cycles, which resulted in a notable local response.	Follow-up skin biopsy revealed normal skin histology with no evidence of lymphomatous infiltration.	(8)

[a]Weakly positive. CT, computed tomography; R-CHOP, rituximab, cyclophosphamide, doxorubicin, vincristine and prednisone; N/A, non-available; PCDLBCL-LT, primary cutaneous diffuse large B-cell lymphoma, leg type; PCDLBCL-NOS, primary cutaneous diffuse large B-cell lymphoma, not otherwise specified.

50% of PCDLBCL-LT cases (10). CD10 is expressed in 28-40% of patients with DLBCL and is a follicular center cell origin marker. Research indicates that CD10-positive patients generally have improved survival outcomes. Furthermore, ~25% of patients with DLBCL exhibit the expression of CD43, which has been identified as an independent poor prognostic factor for the disease (11). In the case in the present study, immunohistochemistry revealed positive staining for CD20, CD10 and CD79a, along with diffuse BCL2 staining, while CD3 and MUM1 were negative. Considering the clinical presentation, imaging findings, histopathology and immunohistochemistry results, the diagnosis was confirmed as PCDLBCL-NOS.

The differential diagnosis of patchy alopecia with a dermal lymphoid infiltrate includes alopecia areata, PCFCL, metastatic carcinoma or secondary systemic lymphoma involving the skin, and inflammatory/scarring alopecias with lymphoid infiltrate. Alopecia areata is a non-scarring autoimmune hair loss characterized histologically by a peribulbar lymphocytic infiltrate of mainly T-cells around anagen hair bulbs and features, such as increased catagen/telogen hairs and follicular miniaturization; it lacks sheets of atypical B-cells and a clonal B-cell immunophenotype, making it distinct from a neoplastic infiltrate observed in lymphoma (12). Primary cutaneous follicle center lymphoma is an indolent B-cell lymphoma presenting as plaques or nodules often on the scalp or trunk with a follicular and/or diffuse architecture composed mainly of centrocytes and centroblasts; unlike diffuse large B-cell lymphoma, PCFCL exhibits fewer confluent sheets of large cells and a different immunophenotype, and distinguishing features were absent in the present study (13).

The standard front-line treatment for PCDLBCL is the R-CHOP regimen, with or without involved-site radiation therapy. Despite this approach, relapses are common, occurring in ~70% of patients (1). Liao *et al* (6) reported the case of a 92-year-old patient with scalp PCDLBCL. The mass was partially resected surgically, but no additional treatment was provided due to the patient's advanced age and poor physical condition. The tumor recurred at the same site after 6 months, and the patient passed away 2 years thereafter (6). By contrast, the other four reported cases by Behera *et al* (4), Khatib *et al* (5), Ochiai *et al* (7) and Čolović *et al* (8) were successfully treated with the R-CHOP regimen, with no recurrences. Similarly, the patient in the present report was treated with the R-CHOP regimen and exhibited no recurrence after 6 months.

The present case report provides several key teaching points that add to the limited existing literature on scalp PCDLBCL. First, unlike the majority of previously reported cases that presented as solitary or multiple nodular masses (5-7), the patient in the present case report exhibited patchy alopecia with mild erythema and pruritus, a presentation that may mimic inflammatory or autoimmune scalp disorders and delay diagnosis. Second, the immunohistochemical profile in the present case, CD10 positivity with MUM1 negativity, is relatively uncommon in reported scalp PCDLBCL cases and may suggest biological heterogeneity within the PCDLBCL-NOS category. Third, despite the aggressive nature traditionally associated with diffuse large B-cell lymphomas, early-stage disease confined to the skin responded well to R-CHOP chemotherapy, resulting in complete clinical remission at six-month follow-up. Collectively, these findings highlight the need for heightened clinical suspicion, timely biopsy of atypical alopecic scalp lesions, and the potential effectiveness of standard systemic chemotherapy in selected cases.

The present case report has several limitations. First, although molecular analysis for the BCR-ABL1 fusion gene and conventional cytogenetic evaluation were performed, both investigations were conducted at an external medical facility. Detailed methodological parameters for the RT-PCR assay, including reagent specifications, primer sequences, and cycling conditions, were not available, and representative cytogenetic (G-banding) images could not be retrieved from the external laboratory's archival system; therefore, these data could not be illustrated or described in greater technical detail. Second, although histopathological and immunohistochemical confirmation was achieved, complete immunohistochemistry image documentation was not available, as part of the diagnostic evaluation was performed at an external institution and the original histopathology and immunohistochemistry image files could not be retrieved. Third, representative CT scan images could not be included, as the imaging studies were performed at an external radiology facility and the original Digital Imaging and Communications in Medicine (DICOM) files were not accessible. Fourth, some potentially informative prognostic markers, such as serum lactate dehydrogenase levels and the Ki-67 proliferation index, were not reported, which may have provided further insight into disease activity and prognosis. Finally, comprehensive molecular and cytogenetic profiling relevant to lymphoma biology, such as fluorescence *in situ* hybridization (FISH) for MYC, BCL2 and BCL6 rearrangements, next-generation sequencing, and formal cell-of-origin classification using the Hans algorithm, were not performed due to the limited availability of advanced molecular diagnostic facilities. Only basic targeted molecular testing (RT-PCR for BCR-ABL) and conventional karyotyping were available as part of routine clinical diagnostics.

In conclusion, PCDLBCL should be considered in the differential diagnosis of scalp lesions, and the R-CHOP regimen could be an effective treatment option for managing PCDLBCL cases.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

FHK and SSO were major contributors to the conception of the study, as well as to the literature search for related studies. RSA, DOK, HAN and KAN contributed to the clinical management of the patient, assisted in data acquisition and interpretation, and participated in the literature review and manuscript preparation. SQH, SOS and SSA contributed to the conception and

design of the study, the literature review, the critical revision of the manuscript, and the processing of the table. RMA was the pathologist who performed the diagnosis of the case. FHK and RSA confirm the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

Ethical approval was not required by the institutional review board for this single case report. Written informed consent was obtained from the patient for participation in the study.

Patient consent for publication

Written informed consent was obtained from the patient for the publication of the present case report and any accompanying images.

Competing interests

The authors declare that they have no competing interests.

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