Characteristics of Cutaneous Marginal Zone Lymphomas With Marked Plasmacytic Differentiation and a T Cell–Rich Background

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Abstract

Primary cutaneous marginal zone lymphoma (MZL) is a common B-cell lymphoma of skin and is characterized by an infiltrate of neoplastic marginal zone B cells typically within the marginal zones of reactive lymphoid follicles and the interfollicular region. However, in our experience, many cases have underemphasized features such as marked plasmacytic differentiation and/or a prominent T-cell component, which may obscure the neoplastic B cells and lead to misdiagnosis. We wanted to draw attention to these features and have studied 15 cases of MZL with marked plasmacytic differentiation, 10 of which had numerous T cells, some with cytologic atypia, and few B cells in the interfollicular region. Plasma cells were monotypic in all cases by in situ hybridization. By polymerase chain reaction, 6 of 8 T cell–rich cases had an IgH gene rearrangement, and none were clonal for T-cell receptor gene. We discuss the terminology, morphologic features, molecular profile, behavior, and differential diagnosis of cutaneous MZL.

Primary cutaneous marginal zone B-cell lymphoma (MZL) is a recently described entity that can present diagnostic difficulty. MZL has been historically diagnosed as “primary cutaneous immunocytoma” or “primary cutaneous plasmacytoma” if the lesion was considered malignant and as “lymphocytoma cutis” if the lesion was considered benign. These terms persist in medical and pathology literature and contribute to confusion regarding this diagnostic entity. Primary cutaneous plasmacytoma was first described in the English literature by Agarwal in 1956.1 A first series of cutaneous lymphoplasmacytic lymphomas has been published by Rijlaarsdam et al2 in 1993 under the name of immunocytoma. The term immunocytoma was subsequently used in the updated Kiel classification and in the European Organization for Research and Treatment of Cancer (EORTC) Cutaneous Lymphoma Project Group classification.3,4 At the same time, similar lesions were being diagnosed as MZL by other groups, who argued that their morphologic features were analogous to those of lymphoma of mucosa-associated lymphoid tissue (MALT) at other extranodal sites and noted that some cases of cutaneous lymphoma were concurrent with MALT lymphomas at extracutaneous sites.5–10 Following consensus meetings of the World Health Organization (WHO) and EORTC, the term primary cutaneous MZL has been accepted as the favored diagnostic term, incorporating the entities of primary cutaneous immunocytoma and primary cutaneous plasmacytoma.11,12 This change has been reflected in the latest WHO classification of skin tumors.13

Primary cutaneous MZL is defined as lymphoma composed of small B cells, including marginal zone (centrocyte-like) or monocytoid cells, lymphoplasmacytoid cells, and...
plasma cells. It is considered to be part of the broad group of extranodal MALT lymphomas. The monotypic lymphoplasmacytoid and plasma cells are usually located at the periphery of the infiltrates, while central areas of the infiltrates may contain variable numbers of reactive B and T cells with reactive lymphoid follicles. These definitions allow for a relatively straightforward recognition of classic cases of cutaneous MZL. However, atypical cases may be quite challenging for a pathologist owing to their rarity and the confusing terminology.

We recently encountered an unusual case with a prominent component of monotypic plasma cells, numerous T cells, and few extrafollicular B cells. We searched our files for primary cutaneous MZLs with similar features.

Materials and Methods

A computer-assisted search of the pathology files of the Massachusetts General Hospital, Boston, and review of identified cases disclosed 15 patients with primary cutaneous MZL characterized by marked plasmacytic differentiation, defined as focal solid aggregates of plasma cells occupying at least one ×20 power field on an excision specimen or a ×40 power field on a small biopsy specimen as determined by histologic and light chain restriction studies. Lymphomas were defined as primary if there was no evidence of extranodal involvement at the time of diagnosis and completion of initial staging evaluation, based on the WHO-EORTC classification.

The total number of cases diagnosed as primary cutaneous MZL was 33. Cases had been classified according to the criteria of the WHO classification of skin tumors using a combination of morphologic and immunophenotypic criteria. The remaining 18 cases not described in this report were typical cutaneous MZL as described. Of 15 cases, 2 were retrieved from the department of pathology archive and 13 from the consultation files of two of us (J.A.F. and N.L.H.).

Immunohistochemical analysis and in situ hybridization were performed on paraffin-embedded tissue as part of the diagnostic evaluation, including immunostaining for pan–B and pan–T antigens (CD20, DAKO, Carpinteria, CA; CD3, Novocastra, Newcastle upon Tyne, England), CD21 (Novocastra), CD23 (Novocastra), bcl-2 (DAKO), bcl-6 (DAKO), CD10 (DAKO), CD5 (Novocastra), CD2 (Novocastra), CD4 (Novocastra), CD8 (Novocastra), CD7 (Novocastra), CD138 (Serotec, Raleigh, NC), MUM-1 (DAKO), CD79a (DAKO), and immunoglobulin heavy chains (BioGenex, San Ramon, CA), γ (BioGenex), μ (DAKO), δ (DAKO) and assessment of in situ hybridization for κ and λ immunoglobulin light chains (Becton Dickinson, Mountain View, CA).

Based on immunohistochemical analysis of B- and T-cell markers, cases were further subdivided in 2 groups: a T cell–rich group (10 cases) with rare extrafollicular B cells and numerous extrafollicular T cells (B cells < T cells, using a semiquantitative analysis) and a B cell–rich group (5 cases) with conspicuous extrafollicular B cells, equal in number or more frequent than the T cells (B = T or B > T cells).

Cases from the T cell–rich group were analyzed for immunoglobulin heavy chain (IGH) and T-cell receptor γ chain (TCR) rearrangements. DNA was isolated from deparaffinized tissue using QIAGEN QIAamp DNA micro kits (QIAGEN, Valencia, CA). The B- and T-cell clonality assays were performed using polymerase chain reaction (PCR) according to the manufacturer’s instructions with reagents purchased from InVivoScribe Technologies, San Diego, CA (IgH Gene Clonality Assay and TCRG Gene Clonality Assay for ABI Fluorescence Detection). The kits use the primer sequences published by BIOMED-2, and had all the necessary reagents except for sample DNA and Taq polymerase (AmpliTaq Gold, Applied Biosystems, Foster City, CA). PCR products were analyzed by capillary gel electrophoresis (3100xl, Applied Biosystems).

Clinical information was available in all cases through the pathology report, the electronic medical record, or by contacting the referring physician. Institutional review board approval was obtained from Partners Healthcare System, Boston.

Results

Clinical Findings

The clinical features are summarized in Table 1. Overall, 11 patients were men and 4 were women with a median age of 55 years (range, 20-83 years).

T Cell–Rich Group

There were 8 men and 2 women with a median age of 55 years (range, 20-74 years). All patients had solitary cutaneous nodules involving the leg (4 cases), trunk (2 cases), shoulder (2 cases), arm (1 case), and scalp (1 case).

Two patients had a history of lymphoma. Case 1 first manifested 17 years earlier with a lesion on the left upper arm, diagnosed at the time as lymphocytoma cutis (original material not available for review). Thirty years later, the patient had a new skin lesion on the right upper thigh diagnosed as cutaneous MZL and treated with local radiation. He developed a recurrence in the right buttocks area 6 months later, was again treated with electron beam radiation therapy, and is currently free of disease.

Case 7 first manifested with a solitary scalp lesion 15 years earlier, diagnosed as “granulomatous mycosis fungoides,”
based on numerous atypical-appearing T cells and rare aggregates of cytologically benign B cells. Fourteen years later, the patient developed a new, morphologically similar-appearing scalp lesion, consistent with MZL owing to evidence of clonal plasma cells by in situ hybridization and PCR. TCR-γ gene analysis showed a polyclonal pattern. The patient received external beam radiation to a total dose of 30 Gy and is currently doing well.

The remaining patients had no significant history. Three patients had 1 relapse, and 1 patient had 2 relapses occurring 3 months to 16 years (mean, 96 months) after the initial presentation. In 2 of 4 patients, the recurrent skin lesions appeared at a different site, whereas the other 2 patients had local recurrence at the same location.

B Cell–Rich Group

There were 3 men and 2 women with a median age of 54 years (range, 28-83 years). Patients had solitary skin lesions involving trunk (2 cases), shoulder and upper arm (2 cases), and neck (1 case).

Two patients had a history of lymphoma or atypical lymphoid proliferation. Case 12 manifested 5 years earlier with skin lesions on the chest and left arm, diagnosed at the time as atypical lymphoid infiltrate, “suspicious” of but not diagnostic for lymphoma. After 2 years, he developed a new lesion on the back, which was diagnostic for cutaneous MZL with marked plasmacytic differentiation. On review, the prior biopsy specimens had similar morphologic features except for a much less conspicuous presence of plasma cells. Case 12 had a history of “diffuse small cleaved cell lymphoma” present in lymph nodes and in paratrabecular bone marrow diagnosed 19 years earlier and subsequently of diffuse large B-cell lymphoma of the stomach. Fourteen years later, she developed an isolated nodule on the right shoulder, diagnosed as cutaneous MZL, most likely unrelated to the prior lymphoma.

The remaining patients had no significant history. One patient developed a local recurrence 2 months after the initial diagnosis, followed by a distant recurrence after 6 months.

Pathologic Findings

The cases with numerous T cells and few B cells were indistinguishable from those with many B cells by light microscopy. Relevant findings are summarized in Table 2.

<table>
<thead>
<tr>
<th>Case No./Sex/Age (y)</th>
<th>Site of Examined Skin Lesion*</th>
<th>Additional Lesions*</th>
<th>Treatment After Excision</th>
<th>Follow-up*</th>
</tr>
</thead>
<tbody>
<tr>
<td>T cell–rich MZL</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/M/55</td>
<td>Right thigh (13 y)</td>
<td>Left arm†, right buttoc (16 y)</td>
<td>RT</td>
<td>ANED (17 y)</td>
</tr>
<tr>
<td>2/M/72</td>
<td>Left knee</td>
<td>Left knee (10 mo)</td>
<td>RT</td>
<td>ANED (12 mo)</td>
</tr>
<tr>
<td>3/M/41</td>
<td>Left shoulder</td>
<td>Left upper back (3 mo)</td>
<td>RT</td>
<td>ANED (11 mo)</td>
</tr>
<tr>
<td>4/M/20</td>
<td>Right lower leg</td>
<td>None</td>
<td>RT</td>
<td>ANED (45 mo)</td>
</tr>
<tr>
<td>5/F/29</td>
<td>Right breast</td>
<td>None</td>
<td>RT</td>
<td>ANED (26 mo)</td>
</tr>
<tr>
<td>6/F/72</td>
<td>Back</td>
<td>None</td>
<td>None</td>
<td>ANED (26 mo)</td>
</tr>
<tr>
<td>7/M/60</td>
<td>Scalp (15 y)</td>
<td>Scalp†</td>
<td>RT</td>
<td>ANED (16 y)</td>
</tr>
<tr>
<td>8/M/74</td>
<td>Right arm</td>
<td>None</td>
<td>RT</td>
<td>ANED (3 mo)</td>
</tr>
<tr>
<td>9/M/67</td>
<td>Right thigh</td>
<td>None</td>
<td>None</td>
<td>ANED (3 mo)</td>
</tr>
<tr>
<td>10/M/70</td>
<td>Right shoulder</td>
<td>None</td>
<td>None</td>
<td>ANED (2 mo)</td>
</tr>
<tr>
<td>B cell–rich MZL</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11/M/44</td>
<td>Back</td>
<td>Back x3 (2 mo); right arm (6 mo)</td>
<td>RT</td>
<td>ANED (12 mo)</td>
</tr>
<tr>
<td>12/M/28</td>
<td>Back (3 y)</td>
<td>Chest and left arm†</td>
<td>None</td>
<td>ANED (48 mo)</td>
</tr>
<tr>
<td>13/F/83</td>
<td>Right shoulder</td>
<td>SCL lymph node; DLBCL stomach‡</td>
<td>None</td>
<td>AWD (15 mo)</td>
</tr>
<tr>
<td>14/F/83</td>
<td>Left neck</td>
<td>None</td>
<td>None</td>
<td>ANED (5 mo)</td>
</tr>
<tr>
<td>15/M/81</td>
<td>Right upper arm</td>
<td>None</td>
<td>None</td>
<td>ANED (2 mo)</td>
</tr>
</tbody>
</table>

ANED, alive with no evidence of disease (lymphoma); AWD, alive with disease (lymphoma); DLBCL, diffuse large B-cell lymphoma; MZL, marginal zone lymphoma; RT, radiation therapy; SCL, “small cleaved cell–type” lymphoma. Values in parentheses are the time after initial diagnosis.

† Site of initial presentation.
‡ The DLBCL occurred 14 years before the skin lesion; the SCL occurred 17 years before the skin lesion (slides not available for review).
bodies (Image 1B). Small lymphoid cells with irregular nuclei resembling marginal zone B cells were present in all cases (Image 2B). Lymphoid follicles were small and compact or expanded and infiltrated by atypical lymphocytes and plasma cells. The epidermis was uninvolved in all cases. Prominent lymphoepithelial lesions were not present.

**Immunohistochemical Studies**

**T Cell–Rich Group**

CD20+ B cells were predominantly confined to the centers of lymphoid aggregates, while the vast majority of the lymphoid cells surrounding the follicles and in diffuse areas were CD3+ T cells, including 3 cases with conspicuous atypical-appearing lymphocytes with irregular nuclear contours and clear cytoplasm morphologically resembling marginal zone cells (cases 1, 2, and 7) Image 3A, Image 3B, Image 3C, and Image 3D. In cases in which lymphoid follicles were not identified, the B cells were scattered throughout the infiltrate, predominantly in the most superficial portion. B cells in follicle centers were bcl-2- and bcl-6+. The extrafollicular B cells were negative for CD10 in all cases. CD3+ T cells commonly surrounded the B-cell aggregates and expressed CD2, CD5, CD7, and CD43. Of 10 cases, 6 had small, compact, nodular meshworks of CD21+...
and CD23+ follicular dendritic cells confined to the B-cell aggregates without evidence of expansion or disruption. Two cases had expanded and irregular follicular dendritic cell meshworks, and in 2 cases, there was minimal and focal staining with CD21 and CD23. CD79a immunostain expression mirrored the distribution of CD20+ B cells and also highlighted the increase in plasma cells, further confirmed by the CD138 and immunoglobulin stains Image 3E and Image 3H. Both CD20 and CD79a immunostains showed that the B-cell population was decreased in relationship to the T-cell population. CD4 and CD8 stains were performed on 3 cases and showed a predominance of CD4+ T cells with few scattered CD8+ T cells. All cases showed monotypic plasma cells with immunostains or in situ hybridization for κ and λ; 8 cases showed monotypic κ expression and 2 cases showed monotypic λ expression Image 3G, Image 3H, Image 3I, and Image 3J.

**B Cell–Rich Group**

CD20+ B cells were present in the centers of the lymphoid aggregates and in the extrafollicular region and were equal to or more numerous than CD3+ T cells Image 4A, Image 4B, Image 4C, and Image 4D. Of 5 cases, 3 had expanded nodular meshworks of follicular dendritic cells Image 4E. The remaining 2 cases had minimal to no staining with CD21 and CD23. CD79a immunostain highlighted B cells and plasma cells. All cases showed monotypic plasma cells with immunostains or in situ hybridization for κ and λ; 8 cases showed monotypic κ expression and 1 case had monotypic λ expression Image 4F.

**Molecular Analysis**

In 8 of the 10 T cell–rich cases, material was available for IGH and TCR-γ gene rearrangement analysis Table 3. Of 8 examined cases (including patients with numerous atypical-appearing lymphocytes), 7 had a polyclonal pattern of TCR-γ chain. One case had suboptimal DNA quality and showed minimal TCR amplification. Of 8 cases, 6 had evidence of a clonal rearrangement of IGH: 4 of 8 showed IGH VH-JH rearrangement (frameworks 1 and 2 with a polyclonal framework 3 pattern), while the 2 remaining cases were polyclonal with VH-JH PCR analysis but showed a clonal PCR product with the IgH D H-JH (BIOMED-2 tube D) fluorescent-labeled primer.

**Table 3**

Molecular Profile of T Cell–Rich Cutaneous Marginal Zone Lymphoma

<table>
<thead>
<tr>
<th>Case No.</th>
<th>IGH VH-JH</th>
<th>IGH D H-JH</th>
<th>TCR-γ Chain</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Clonal</td>
<td>Polyclonal</td>
<td>Polyclonal</td>
</tr>
<tr>
<td>2</td>
<td>Polyclonal</td>
<td>Clonal</td>
<td>Minimal</td>
</tr>
<tr>
<td>3</td>
<td>Clonal</td>
<td>Clonal</td>
<td>Polyclonal</td>
</tr>
<tr>
<td>4</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>Polyclonal</td>
<td>Polyclonal</td>
<td>Polyclonal</td>
</tr>
<tr>
<td>6</td>
<td>Polyclonal</td>
<td>Clonal</td>
<td>Polyclonal</td>
</tr>
<tr>
<td>7</td>
<td>Clonal</td>
<td>—</td>
<td>Polyclonal</td>
</tr>
<tr>
<td>8</td>
<td>Clonal</td>
<td>—</td>
<td>Polyclonal</td>
</tr>
<tr>
<td>9</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>10</td>
<td>Polyclonal</td>
<td>—</td>
<td>Polyclonal</td>
</tr>
</tbody>
</table>

IGH, immunoglobulin heavy chain; TCR, T-cell receptor.
the diagnostic possibility of plasmacytoma. Extraosseous plasmacytoma is a neoplasm of plasma cells in patients without histologic and radiologic evidence of bone marrow plasma cell myeloma. It constitutes 3% to 5% of all plasma cell neoplasms, with 80% occurring in the upper respiratory tract.14 Solitary plasmacytoma of the skin is unusual, with approximately 35 reported cases.17-23 The clinical course in most cases was indolent with or without local recurrences, although a few patients subsequently developed overt plasma cell myeloma and died of disease. Our experience, in conjunction with the indolent behavior of many of the previously reported cases, suggests that many of them may be examples of MZL.

Discussion

Plasmacytic differentiation is a common feature in MZL in the skin and in extracutaneous locations and likely represents a recapitulation of normal MALT, with reactive lymphoid follicles surrounded by neoplastic marginal zone cells and interfollicular plasma cells. The observation of a mixture of small lymphoid cells, marginal zone cells, and monotypic plasma cells is an extremely helpful feature in diagnosing MALT lymphomas. However, as our study illustrates, occasionally the plasma cells are unusually prominent with arrangement in monotonous sheets or nodules, raising
Image 4L (Case 9) Immunohistochemical profile of a B cell–rich cutaneous marginal zone lymphoma with numerous plasma cells. A and B, Abundant CD20+ B cells in nodular aggregates (×4 and ×20, respectively). C and D, Numerous CD3+ T cells surrounding and infiltrating the lymphoid follicles (×4 and ×20, respectively). E, Expanded and colonized lymphoid follicles highlighted by CD21 immunostain (×20). F, Monotypic expression of κ light chain in neoplastic plasma cells (×20).
with marked plasmacytic differentiation, while rare cases may be cutaneous presentations of plasma cell myeloma. In the absence of history of plasma cell myeloma, the diagnosis of cutaneous plasmacytoma should be made with great caution. The presence of any lymphoid follicles, B cells, or follicular dendritic cells would tend to exclude plasmacytoma.

We also observed that two thirds of the cases with prominent plasma cells had numerous T cells and few extrafollicular marginal zone B cells. The B cells that were present were mostly confined to reactive germinal centers. In addition, 3 of these cases appeared to have numerous atypical small lymphocytes, resembling the marginal zone B cells; however, by immunohistochemical analysis, most if not all of these cells were expressing T-cell markers. It has been previously described that lesions in some patients may have a significant number of reactive T cells, designated by Magro et al25 as T-cell–rich plasmacytic MZL. This phenomenon is not particular to MZL, as, in general, cutaneous B-cell lymphomas tend to have many admixed T cells. This may cause diagnostic difficulty, as illustrated by case 7 that was probably initially misdiagnosed as mycosis fungoides based on the paucity of B cells and the presence of numerous atypical-appearing T cells.

Another entity that needs to be distinguished from MZL is primary cutaneous small-medium CD4+ T-cell lymphoma. Similar to MZL, it presents with nodular or diffuse infiltrate of dermis and subcutaneous tissue and contains atypical small to medium-sized lymphoid cells admixed with small reactive lymphocytes and histiocytes. Faced with this differential diagnosis, heavy chain and light chain analysis should be performed to evaluate clonality of the marginal zone cells and plasma cells. Molecular study of IGH gene and TCR-γ gene rearrangements may be warranted to confirm the results.

Conversely, peripheral T-cell lymphomas (PTCLs), in particular angioimmunoblastic T-cell lymphoma, may have an extensive nonneoplastic component, including a prominent B-cell proliferation.28-30 The expansion of B cells ranges from small clusters of large activated B cells to focally confluent B cells, which may obscure the T-cell proliferation.31 In 1 study, up to 40% of these cases demonstrated oligoclonal and clonal B-cell rearrangements.28 The majority of B-cell proliferations have been attributed to expansion of an Epstein-Barr virus–positive B cell population.30,32,33 However, cases with Epstein-Barr virus–negative clonal B-cell proliferations coexisting with PTCL have also been reported, some with large sheets of monotypic plasma cells that displaced the PTCL component.34 The distinction between a primary B-cell cutaneous lymphoma and a T-cell cutaneous lymphoma is clinically relevant because the latter overall has a poor prognosis and requires more aggressive treatment compared with the indolent nature of MZL.

To address this differential diagnosis, we performed IGH and TCR PCR on all cases with abundant T cells. No cases showed TCR-γ gene rearrangements, and 6 (75%) of 8 had clonal IGH. This finding is similar to that previously reported (64% in a study of 22 cutaneous MZLs).24 All of our clonal cases showed a polyclonal pattern with framework 3 primers, as may be seen with postsomatic hypermutation, and supports the use of BIOMED-2 primers for frameworks 1, 2, and 3, as opposed to older studies that typically used framework 3 analysis only.24 In addition, we found that additional testing with IGH DH-JH primers increases PCR sensitivity even further. Thus, based on our findings, D-tube analysis appears to be of added value in clonality assessment of MZL.

The abundance of reactive T cells in cutaneous B-cell lymphomas may be indicative of an as yet unknown pathogenic environmental insult affecting the skin. Borrelia burgdorferi infection has been linked to occasional cases of MZL in European patients34,35; however, these findings have not been confirmed in other series.36 Extramedial MZLs typically arise in a background of Th1-type chronic inflammation, related to infection (Helicobacter pylori) or autoimmune disease (Sjögren syndrome). In contrast, most primary cutaneous MZLs appear to be associated with a Th2-type cytokine environment,37 making them somewhat unique among extranodal MZLs. In this context, primary cutaneous MZLs also undergo class-switch, as confirmed in our series, with expression of IgG and IgA by the neoplastic plasma cells, as opposed to the non–class-switched IgM+ extranodal MZLs.37 In short, although the cause of the inflammation that may lead to cutaneous MZLs is not known in most cases, it is possible that the initiating agent may be responsible for eliciting a Th2-type cytokine environment and numerous admixed T cells in some cases.

According to the literature, MZL of the skin affects middle-aged men and women.7,24,26 Cutaneous lesions most commonly involve upper extremities, trunk, and head and neck. The rate of recurrence ranges from 30% to 70% and appears independent of the treatment modality used.5,10,24,26 In our study, patients in the T cell–rich group were comparable to patients from the B cell–rich group and the literature in terms of age, sex, and location of the lesions. Clinical course was also similar, with relapsing disease present in 4 (40%) of 10 patients in the T cell–rich group and 2 (40%) of 5 patients in the B cell–rich group. These results indicate that the T cell–rich variant of MZL has no prognostic implications.

We have found that cutaneous MZLs often show marked plasmacytic differentiation (about half of the cases) with sheets and aggregates of neoplastic plasma cells. In addition, two thirds of such cases are associated with a predominance of T cells. These 2 features may cause diagnostic difficulty and lead to misdiagnosis of plasmacytoma or cutaneous T-cell
lymphoma. However, these features are not associated with clinical or prognostic differences.

References


