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Cutaneous manifestations of mantle cell lymphoma: an extensive literature review

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Abstract

Mantle cell lymphomas account for about 2 to 10% of non-Hodgkin B-cell lymphomas. Despite the cellular maturity of B-cell lymphomas, the disease is aggressive in the majority of cases and its course is unpredictable. The clinical presentation is variable, and multiple nodal and extranodal manifestations have been described. Cutaneous infiltration is an uncommon (2–6%) location of the disease. An extensive review of the literature was performed, and 24 case reports and five case series were found describing cutaneous locations. These data were thoroughly studied in order to present their clinical and laboratory characteristics in this review.

Keywords: B-cell lymphomas, non-Hodgkin lymphomas, mantle cell lymphoma, cutaneous manifestations

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Introduction

The incidence of mantle cell lymphoma (MCL) is much lower than other forms of B lymphomas. It accounts for about 6% of non-Hodgkin B lymphomas occurring mainly in males, with a median age of 60 years. It is moderately aggressive despite belonging to the mature B-cell lymphomas (1). The overall survival of the patients ranges from 4 to 5 years after diagnosis. Recent progress in the understanding of the biology of MCL has led to substantial improvements in patient outcomes and to the development of several novel targeted therapies such as Bruton's tyrosine kinase (BTK) inhibitors, ibrutinib, and acalabrutinib (2). A typical immunophenotype consists of cells that overexpress CD5 and cyclin D1. The t(11;14)(q13;q32) translocation, which juxtaposes the CCDN1 gene encoding cyclin D1 to the immunoglobulin heavy chain (IgH), results in the overexpression of cyclin D1, a protein that plays an important role in the cell cycle and especially in the process of cell proliferation (3). Alternatively, less-frequent alterations in CCND2 and CCND3 genes, encoding cyclin D2 and D3, respectively, have been identified in MCL lacking the t(11;14)(q13;q32) translocation. However, over the past decade, further investigation into the pathogenesis of MCL has defined other molecular abnormalities that define cyclin D1-negative MCL. Among the several types of MCL described in the literature, the blastic and pleomorphic variants show a high proliferation rate and rapid deterioration in the clinical course (4). Patients usually present at stage III or IV with typical involvement sites affecting the lymph nodes, spleen, gastrointestinal tract, and bone marrow. Although the disease may spread to all organs, skin involvement remains an uncommon location. This study presents a comprehensive review of the literature regarding the skin involvement of MCL.

Materials and methods

A comprehensive search was performed in medical databases, including Pubmed, Embase, and Google Scholar. The keywords used for the search were *mantle*, *lymphoma*, *mantle cell*, and every keyword referring to the skin, such as *cutaneous*, *dermal*, and *skin*. All studies describing patients with MCL located in the skin were included. There was no limitation regarding the type, language, or year of publication.

Results

Twenty-four case reports published from 2008 to 2018 were identified. Most of them were published between 2010 and 2016. Table 1 illustrates the clinical characteristics of these cases. In the majority of cases, the diagnosis of MCL was demonstrated by a skin biopsy. The lesions were mostly located in the head and neck region or were on multiple skin locations throughout the body, mainly on the extremities. Stage III or IV was the most common disease stage when a patient first presented with a cutaneous MCL (Table 1). In addition, five case series with a total of 40 patients were identified published between 2002 and 2016. Among them, the most common location was the extremities or other multiple cutaneous sites (Table 2). Only four out of 40 patients had stage I disease, and in about half of the patients the diagnosis of MCL was made by a skin biopsy. Overall survival was applicable in 15 patients and varied from 14.67 to 74.7 months. Immunohistochemistry was positive for CD20 and cyclin D1 in the vast majority of the studies. Histology usually revealed blastoid or pleomorphic features (Table 3). For all published cases, cutaneous location, disease stage, clinical appearance, and prior MCL history if any is summarized and compared in Table 4.

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Table 1 | **Summary of published case reports.**

Report (Year; reference)	Age (sex)	Location	Interval from lesion to diagnosis	Stage of disease	Follow-up after diagnosis	Treatment	Comments
Leduc et al. (2015; 10)	76 (M)	Pruritic maculopapular rash on trunk, extremities	1 year	Intrabdominal and axillary lymphadenopathy, stage IV	4 years	Yes, prior to skin diagnosis	
Phelps et al. (2013; 21)	71 (M)	Papule on cheek	5	MCL of left tonsil with multiple recurrences on tongue	NG	NG	Skin and tonsil biopsies consistent with MCL
Yoo et al. (2008; 12)	58 (M)	Tarsal conjunctivae	0	Bone marrow infiltration and hepatosplenomegaly	NG	CVP	
Canpolat et al. (2010; 23)	49 (F)	Papules on face, shoulders, back, chest for a month	0	Positive axillary nodes and bone marrow	Died 4 months after skin lesion	Yes, after skin lesion biopsy confirmation	
Li et al. (2012; 24)	53 (M)	Dark purple nodules and plaques on left temporal region, right palpebra, abdomen, lower extremities	0	Diffuse lymphadenopathy, stage IVB	Died 1 month later	Yes, R-Hyper-CVA after diagnosis, with skin lesion improvement	
Hrgovic et al. (2016; 25)	55 (F)	Mass on infraorbital region extending to underlying bone	0	Axillary, tonsillar, cervical and intraabdominal lymphadenopathy, bone marrow infiltration, stage IV	4 years	Yes, multiple treatments due to frequent relapses	
Moody et al. (2001; 7)	47 (M)	1-year history of red swollen earlobes	0	Cervical lymphadenopathy and hepatosplenomegaly	3 years	R-CHOP	Recurrence on earlobes
Jawed et al. (2014; 22)	50 (M)	Petechial maculopapular rash on entire body	0	Peripheral lymphadenopathy	NG	Yes, chemotherapy and stem cell transplantation	
Paludo et al. (2014; 8)	78 (M)	Conjuctival mass	9 months	Intrabdominal lymphadenopathy and B-symptoms	NG	Yes, six cycles of bendamustine and rituximab	
Cao et al. (2013; 26)	53 (M)	Multiple nodules on head, neck, limbs	0	Diffuse lymphadenopathy	NG	NG	
Shaikh et al. (2018; 27)	53 (F)	Erythematous nodular rash on left breast	4 weeks	Stage IVB	NG	Yes, chemotherapy plus intrathecal infusions	Improvement of skin lesions after treatment
Guerra et al. (2018; 16)	76 (F)	Subcutaneous papules on extremities, face, neck; large left supraorbital mass	1 year	Co-lesions in spleen and gastrointestinal tract	NG	NG	
Ishibashi et al. (2010; 28)	68 (M)	Subcutaneous nodules on left thigh	9 months	Diffuse cervical lymphadenopathy, history of CLL before MCL	Died 3 months after skin diagnosis	Yes, CVP treatment	
Shimada et al. (2016; 29)	71 (M)	Subcutaneous chest lesions	0	Multiple chest and intraabdominal masses	Died after 2 months	No, patient refused treatment	Concurrent squamous cell carcinoma on face
Estrozi et al. (2009; 30)	72 (M)	Cutaneous nodule in right temporal region	0	No evidence of other pathology	Alive 6 months after diagnosis	Localized radiotherapy	Primary MCL
Cesinaro et al. (2014; 31)	75 (F)	Purplish nodule on left lower leg	0	No evidence of other pathology	Died after 40 months	Initially radiotherapy but chemotherapy started after recurrence	Primary MCL with recurrence at same and another site
Lynch et al. (2012; 32)	83 (M)	Red-pink nodular plaque on right thigh	0	No evidence of other pathology	Died from treatment-related issues	Chemotherapy due to extent of dermal lesions	Primary dermal MCL
Zattra et al. (2010; 33)	77 (M)	Diffuse erythematous nodules	0	No evidence of other pathology	Alive	Chemotherapy	Complete remission 22 months after treatment
Hamad et al. (2014; 34)	68 (F)	Violaceous nodular lesions on right calf	0	No evidence of other pathology	Alive	Yes, R-CHOP	Recurrence in same region after 15 months and second recurrence after another 16 months

Table 1 | **Continued.**

Report (Year; reference)	Age (sex)	Location	Interval from lesion to diagnosis	Stage of disease	Follow-up after diagnosis	Treatment	Comments
Mishchenko et al. (2014; 35)	81 (M)	Red plaques on left leg	18 months	Stage III	Alive	Yes, chemotherapy and topical radiotherapy without improvement	Finally, response with lenalidomide
Borras Perera et al. (2014; 36)	57 (M)	Right occipital area	0	Lymphadenopathy in cervical area	Alive 4 years after diagnosis	Yes, R-CHOP and transplantation	
Hjira et al. (2013; 37)	46 (–)	Multiple nodules on abdomen and upper extremity	0	Also lymphadenopathy and B-symptoms	NG	NG	
Markiewicz et al. (2017; 38)	55 (–)	Erythema, local bruising in auricular region, multiple face lesions	0	B-symptoms and multiple lymphadenopathy, stage III	Died	Received R-CHOP but unable to tolerate treatment	
Mancebo et al. (2014; 39)	78 (M)	Multiple nodules on lower extremities	7 years	No residual disease from initial diagnosis	NG	Yes, rituximab	Ibrutinib caused significant improvement lasting 2 months

NG = not given (data not available); M = male; F = female; MCL = mantle cell lymphoma; CLL = chronic lymphocytic leukemia; R-CHOP = rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisolone; CVP = cyclophosphamide, vincristine, and prednisolone; R-Hyper-CVAD = rituximab, cyclophosphamide, vincristine, theprubicin, and dexamethasone.

Table 2 | **Summary of published case series.**

Report (Year; reference)	No. of patients	Disease stage at diagnosis			Relapse on skin	Location	MCL diagnosed from skin lesion	Survival in months, mean (SD)	Mean follow-up in months for living patients at study end (SD)	Treatment, comments
		I	III	IV						
Wehkamp et al. (2015; 9)	14	2/11	1/11	8/11	6/14	Limbs 9/14, back 2/14, scalp 1/14	6/14	Applicable in 7/14 patients; 65.86 (45.32)	Living: 4/14; 21.25 (26)	Surgery 2/14, R-CHOP / R-DHAP 4/14, other 6/14
Gru et al. (2016; 41)	10	0	0	10/10	6/10	Limbs 3/10, eyelids and conjuctivae 3/10, trunk 3/10, face 1/10	2/10	Applicable in 5/10 patients; 74.4 (61.05)	Living: 2/10; 36.5 (14.84)	
Hsi et al. (2016; 17)	8	NG	NG	NG	NG	NG	0/8	NG	NG	Skin lesions secondary 8/8, but no disease stage mentioned
Sen et al. (2002; 15)	5	1	0	4	NG	Limbs 3/5, chest 1, multiple lesions 1	4/5	Applicable in 3/5 patients; 14.67 (5.5)	Living: 2/5; 25.5 (6.36)	All received treatment; recurrent lesions treated with paclitaxel and topotecan
Dubus et al. (2002; 41)	3	1	0	2	1/3	Multiple lesions 3/3	1/3	Only one death after diagnosis	Living: 2/3; 48 (12)	

SD = standard deviation; MCL = mantle cell lymphoma; NG = not given (data not available); R-CHOP = rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisolone; R-DHAP = rituximab, dexamethasone, cytarabine, and cisplatin.

Table 3 | **Immunohistochemistry findings in published studies.**

Study	Histology	CD20	Cyclin D1	CD10	CD5	CD23	Bcl2	Bcl6	Mum-1	CD79	Ki67	Sox 11	FISH for cyclin D1
Wehkamp et al.	12/14 blastoid, 2/14 small	13/14	13/14	2/14	10/14	0/14	11/14	0/ 14	3/14			5/ 14	12/14
Gru et al.	2/10 blastoid, 4/10 pleomorphic, 4/10 classic	3/10	9/10		5/ 10					2/10	1/10	1/ 10	
Hsi et al.	1/8 blastoid features, 7/8 classic morphology		7/8		8/8			0/3				7/8	
Sen et al.	4/5 blastoid features, 1/5 typical features	5/5	5/5		4/5						3/5		4/5
Dubus et al.	1/3 blastoid features	3/3	1/3	0/3	2/3								1/3
Leduc et al.	Two distinct lymphoid populations: one MCL pleomorphic, one C-ALCL	+	+		+	–				+			
Phelps et al.	Blastoid variant	+	+	+	+	–	+	–			+		
Yoo et al.	Not specified	+	+	–	–	–	+				+		
Canpolat et al.	Not specified	+	+		+		+			+			
Li et al.	Blastoid MCL		+	–	–	–	+	–	+	+	+	+	
Hrgovic et al.	Blastoid MCL	+	+	–	+	–				+	+		
Moody et al.	Not specified	+	+	–	–/+			–			+		
Jawed et al.	Not specified	+	+	–	+	–							+
Paludo et al.	Not specified	+	+	–	+								
Cao et al.	Blastoid features	+	+	–	–	–	+	–	+	+	+		+
Shaikh et al.	Small to medium-sized lymph cells	+	+	+	+		+	+		+			
Guerra et al.	Small to medium-sized lymph cells	+	+		+						+		
Ishibashi et al.	Blastoid features	+	+		–						+		
Estrozi et al.	Blastoid features	+	+	–	+	–		–			+		+
Cesinaro et al.	Blastoid features	+	+	+	+		+	+	+		+		
Lynch et al.	Blastoid features	+	+	–	+	–				+	+		+
Zattra et al.	Not specified	+	+	–	+	–	+						
Hamad et al.	Pleomorphic features	+	+	–	+	–	+	+			+		+
Mishchenko et al.	Blastoid features		+						+	+	+		
Perera et al.	Not specified		+										+
Hjira et al.	Not specified	+			+	–							
Markiewicz et al.	Not specified	+		–	+	+/–	+	–	–	+			
Mancebo et al.	Blastoid morphology		+										

FISH = fluorescence in situ hybridization; MCL = mantle cell lymphoma; C-ALCL = anaplastic large cell lymphoma.
Empty spaces indicate data not available for this staining.

Table 4 | **Summarized data of published mantle cell lymphoma skin manifestations.**

		Case reports	Case series
Location:	Head and neck	7/24	5/32
	Trunk	2/24	6/32
	Extremities	5/24	17/32
	Multiple lesions	10/24	4/32
Type of lesion:	Papule	9/24	–
	Maculopapular rash	6/24	–
	Other	9/24	–
Disease stage:	Localized cutaneous (stage I, II)	6/24	4/29
	Systemic (stage III, IV)	18/24	25/29
Mantle cell lymphoma diagnosis prior to skin manifestation:	Yes	7/24	27/40
	No	17/24	13/40

Discussion

Skin involvement of MCL is typically found in 2 to 6% of patients suffering from MCL. Male patients are affected three times more frequently than women. Although localized disease is the first manifestation, in the majority of cases the condition almost always becomes systemic as time passes (5, 6). In addition, the response to treatment is usually transient, with frequent relapses. As a result, most studies report a disease-free period of approximately 20 months (7, 8).

Currently, more than 50 cases of skin MCL have been described in the literature. Most of the published studies are case reports, but also there are a few case series. Areas of the skin affected can be everywhere, with a probable preference toward the upper and lower limbs (Table 4). Other areas commonly described are the face or multiple locations. The skin lesions vary from papules and plaques to subcutaneous nodules and widespread erythematous rash.

Figure 1 shows a case of cutaneous MCL in a 66-year-old male patient that presented to our department complaining of an ulcerated lump on his upper torso. He had a history of stage IV MCL managed with chemotherapy. A punch biopsy confirmed cutaneous MCL with immunohistopathology positive for CD20 and cyclin D1, and negative for CD3, CD4, CD8, and CD30 (Fig. 2).

The skin lesion may last from 1 month up to 2 years (Tables 1 and 2). A small percentage of studies used PET-CT, which almost always showed increased uptake on the skin lesion, and this may be an additional route to differential diagnosis, especially in patients with a history of lymphoma. Mortality is often related to disease progression or systemic treatment ineffectiveness. Skin lesions often represent a disease flare and the need for systemic treatment. Wehkamp et al. report 14 cases of MCL skin involvement. In that study, most of the patients had systemic disease concurrently with skin involvement, and apparently most of them were diagnosed from a skin lesion. As a result, careful staging must be performed when a patient is diagnosed with skin MCL (9). Leduc et al. report a case of a concomitant MCL and anaplastic large cell lymphoma (ALCL) of the same skin lesion. In that case, the two distinct cell populations did not share the same mutation. The MCL cell had the t(11;14) mutation, and the ALCL had no evidence of any mutation (10). On the other hand, although marginal cell lymphomas are the most common lymphomas in ocular tissues, cases of MCL in this region have also been reported. Yoo et al. report a case of conjunctival MCL in a male patient. The patient had a progressively enlarging mass in his tarsal conjunctiva. Biopsy of the mass confirmed the diagnosis, and a diagnostic workup was initiated for disease staging. Differential diagnosis of marginal zone cell lymphomas and MCL is essential because the treatment options and overall survival differs between these two conditions. For example, MCL is usually diagnosed at stage III or IV. In contrast, marginal cell lymphomas generally present at stage I with good overall prognosis (11, 12).

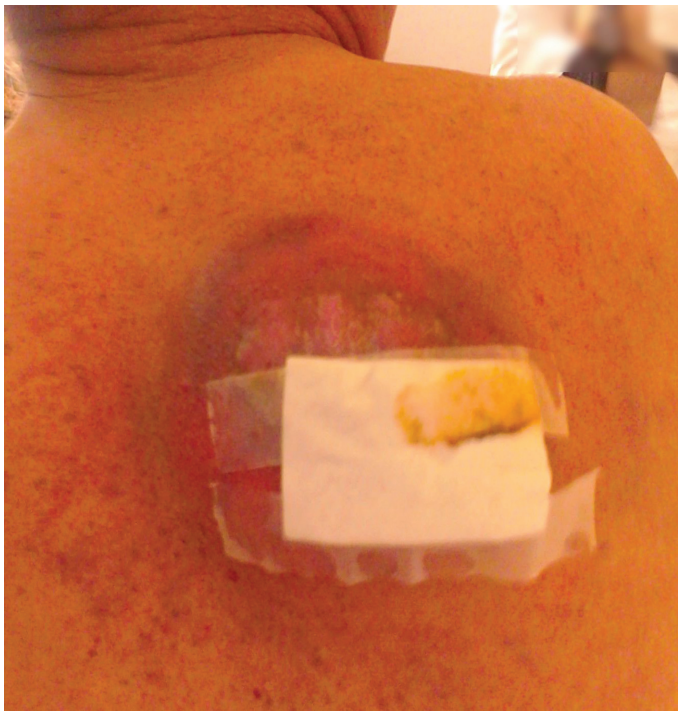

Figure 1 | Skin lesion on the upper back. The mass is 3 cm × 2 cm, immotile, with a central ulceration.

Other causes of skin lymphoid tissue infiltration may be cutaneous lymphoid hyperplasia (CLH) or pseudolymphoma. Several antigens may trigger this kind of reaction. Among them, the most common are traumas, injections, and insect bites (13). The main pathophysiological principle is that the immunologic response is incomplete and as a result a remaining lymphocytic population remains at the site of inflammation. In these situations, the differential diagnosis between benign and malignant disease may be difficult. Furthermore, primary cutaneous follicular lymphoma (PCFL) is another benign disease that must be ruled out. Goteri et al. report a concomitant case of MCL in a lymph node and skin PCFL. They stress the need to carefully investigate all cases of skin lymphomas for possible underlying aggressive disease (14). Finally, other causes of primary skin lymphoma are marginal zone B-cell lymphoma, intravascular large B-cell lymphoma, and plasmatocytoma. Each of these has its own immunohistochemistry profile that can easily be differentiated (15, 16). In addition, cutaneous manifestation of chronic lymphocytic leukemia (CLL) has been described in the literature. However, CLL cases are advanced diseases, and immunohistochemistry can effectively differentiate MCL and CLL. CLL, for instance, usually lacks cyclin D1 and SOX11 expression (17).

In MCL immunochemistry, the neoplastic cell population exhibits strong positivity for pan-B-cell markers such as CD79a, CD19, CD20, and CD22, possible positivity for T cell markers CD5 and CD43, and finally strong nuclear staining for cyclin D1 (18). Interestingly, cyclin D1, or rarely cyclin D2 and D3, can be used to dif-

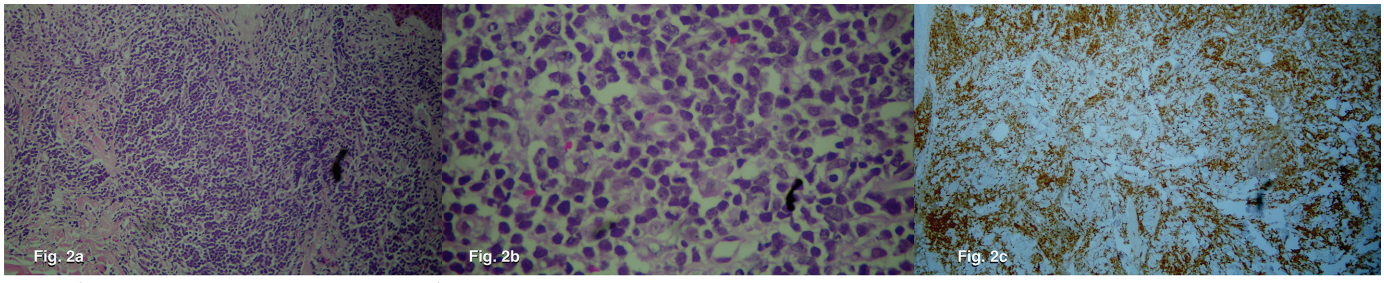

Figure 2 | Skin biopsy from an upper back lesion: a) H&E sections ×40 show that the epidermis is uninvolved and there is diffuse infiltration of the dermis by medium-sized lymphoid cells with enlarged hyperchromatic nuclei and little eosinophilic cytoplasm; b) H&E ×400; c) immunohistochemistry stain for cyclin D1 ×40.

ferentiate MCL from other types of lymphoma (19, 20). Phelps et al. report a case of CD10-positive skin MCL. In their case, they discuss the significance of CD10 antigen positivity. They conclude that CD10-positive skin MCL must follow an extensive workup, including cyclin D1, BCL-1, and molecular testing to exclude other forms of aggressive cutaneous lymphomas (21). In addition, studies have shown that overexpression of SOX11 may be implicated with highly aggressive disease (7, 17). The results, however, are still controversial. Other studies suggest the use of SOX11 as a specific marker for MCL if cyclin D1 is not applicable or nondiagnostic (7, 17). Hsi et al. conclude that SOX11 can be used when fluorescence *in situ* hybridization (FISH) techniques are unable to detect the MCL mutation or the specimen contains a large number of reactive lymphocytes (17). In addition, endothelial cells, fibroblasts, or histiocytes maybe sometimes be positive for cyclin D1, and so a marker like SOX11 may help a definite diagnosis for the type of lymphoma (7). On the other hand, Jawed et al. discuss the importance of L-selectin in skin metastasis of MCL. L-selectin is preferably expressed on B lymphocytes that migrate into secondary lymphoid tissues via the bloodstream. Jawed et al. report overexpression of this cell molecule in their case, and they suggest that this may be associated with increased incidence of skin metastasis and disease progression (22). However, we were unable to identify any studies investigating the expression of L-selectin extensively in cutaneous lymphomas.

In conclusion, MCL has an unpredictable clinical course. Although it is rare, MCL may infiltrate the skin. There is a need for new treatment options because highly refractory cases exist. Immunohistochemistry is usually diagnostic, but currently it cannot provide further information about disease aggressiveness. Further studies are required to investigate the role of skin lesions in relation to the disease course. Increased awareness among clinicians regarding patients with a refractory undiagnosed skin lesion and history of blood neoplasms is therefore necessary.

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