Literature review

CD20 expression in plasma cell neoplasm and B-cell non-Hodgkin lymphoma with plasmacytic differentiation: insights into aberrant markers

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Introduction

B-cell Non-Hodgkin lymphoma (B-NHL) arises from a clonal proliferation of B-cells, exhibiting a wide spectrum of manifestations ranging from immature B-cells to terminally differentiated plasma cells. According to the World Health Organization (WHO) 2022 classification, B-NHL is categorized into four groups: precursor B-cell neoplasms, mature B-cell neoplasms, Hodgkin lymphoma, and other diseases characterized by paraproteins.[1] In 2024, the estimated prevalence of NHL was approximately 4% of all cancer cases.[2] B-NHL typically presents with lymphadenopathy; however, many cases may lack nodal involvement and instead present with extranodal disease. Given the heterogeneity of its clinical features, correlation with pathological findings and/or genetic abnormalities is essential for establishing a definitive diagnosis. In pathological assessments, Hematoxylin and Eosin (H&E)-stained slides are first evaluated following receipt of tissue specimen. The morphological findings in B-NHL demonstrate abnormal lymphoid cells infiltration with diverse patterns, including diffuse, paratrabecular, interstitial and nodular. Plasmacytic differentiation (PD) and large cell transformation, such as immunoblastic or plasmablastic transformation, may occur in B-NHL. In addition, cccasional fibrosis or amyloid deposition may be observed.[3-5]

In contrast to B-NHL, plasma cell neoplasms (PCN) or multiple myeloma (MM), account for approximately 1.8% of all cancer cases, according to the Surveillance, Epidemiology, and End Results (SEER) Program in 2024.[2] PCN is characterized by a clonal proliferation of plasma cells within the bone marrow or extramedullary tissues, with diagnosis often confirmed by the identification of plasmacytomas. At diagnosis, patients with PCN or MM commonly present with classical clinical manifestations, including anemia, renal insufficiency, hypercalcemia and bone pain. The presence of myeloma-defining events (MDEs)–such as clonal bone marrow plasma cells ≥ 60%, a serum free light chain (FLC) ratio ≥ 100 with an involved FLC concentration > 100 mg/L, and/or the detection of more than one focal bone lesion on magnetic resonance imaging (MRI)–indicates a significant tumor burden and is strongly associated with the progression to end-organ damage. Plasmacytomas may manifest as either intraosseous or extraosseous lesions, although nodal involvement is exceedingly rare. Beyond the localized symptoms caused by plasmacytomas or MDEs in MM, systemic disorders linked to monoclonal gammopathy, such as amyloidosis and POEMS syndrome, are also encompassed within the broader spectrum of PCN.[6,7]

In addition to the classical clinical manifestations observed in PCN and B-cell non-Hodgkin lymphoma (B-NHL), the expression of CD20 on plasma cells has emerged as an important diagnostic and therapeutic consideration. CD20, a pan-B-cell marker, is predominantly associated with B-cell lymphomas; however, its expression in MM is relatively uncommon. While B-NHL encompasses a spectrum of morphological variants,

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lymphoplasmacytoid or plasmacytic differentiation is frequently observed, particularly in marginal zone lymphoma (MZL) and lymphoplasmacytic lymphoma (LPL). A major diagnostic challenge lies in distinguishing PCN exhibiting CD20 expression from B-NHL with plasmacytic differentiation (BCN-PD), as accurate differentiation is critical for guiding appropriate treatment strategies. Currently, treatment regimens for B-NHL primarily involve CD20-targeted monoclonal antibody-based immunochemotherapy (ICT), whereas PCN is managed with CD38-directed ICT or novel targeted therapies that act on B-cell or plasma cell markers (Table 1). Furthermore, Moreau et al. reported that the use of rituximab in PCN with CD20 expression has produced variable outcomes[8], and the recommendation of CD20-targeted ICT in this setting remains controversial. Given the complexities surrounding CD20 expression in PCN and BCN-PD, immunophenotyping plays a central role in distinguishing these entities. Immunophenotypic analysis provides critical insights into the cellular characteristics and surface markers that define different plasma cell populations and mature B-cells, thereby aiding accurate diagnosis and informing therapeutic strategies. The purpose of this review article is to elucidate the immunophenotypic and molecular characteristics of PCN with CD20 expression and BCN-PD, thereby facilitating the effective selection of diagnostic markers to accurately differentiate between these two entities.

Plasma cell neoplasm

PCN is characterized by the clonal proliferation of plasma cells. According to the International Myeloma

Table 1 National Comprehensive Cancer Network (NCCN) 2024 recommendation: immunotherapy-based drugs used for the treatment of B-cell non-Hodgkin lymphoma and plasma cell neoplasm[9,10]

Targeted marker	Agent	Mechanism of action
CD19	Tafasitamab	mAb
	Axicabtagene ciloleucel	CART
	Lisocabtagene maealeucel	
	Tisagenleleucel	
	Brexucabtagene autoleucel	
	Loncastuximab tesirine	ADC (CD19-SG3199 cytotoxic agent)
CD20	Rituximab	mAb
	Obinotuzumab	
	Glofitamab	BiTE
	Epcoritamab-bysp	
	Mosunetuzumab	
CD79a	Polatuzumab vedotin	mAb (CD79b)
CD38	Daratumumab	mAb
	Isatuximab	
	Ciltacabtagene autoleucel	CART
CD269 (BCMA)	Idecabtagene vicleucel	CART
	Belantamab mafodotin	ADC (BCMA-MMAE cytotoxic agent)
	Elranatamab	BiTE
	Teclistamab	
SLAMF7 (CD319)	Elotuzumab	mAb

mAb, monoclonal antibodies; CAR-T, chimeric antigen receptor-modifier T-cells; BiTE, bispecific T-cell engager; ADC, antibody-drug conjugates; MMAE, monomethyl auristatin E

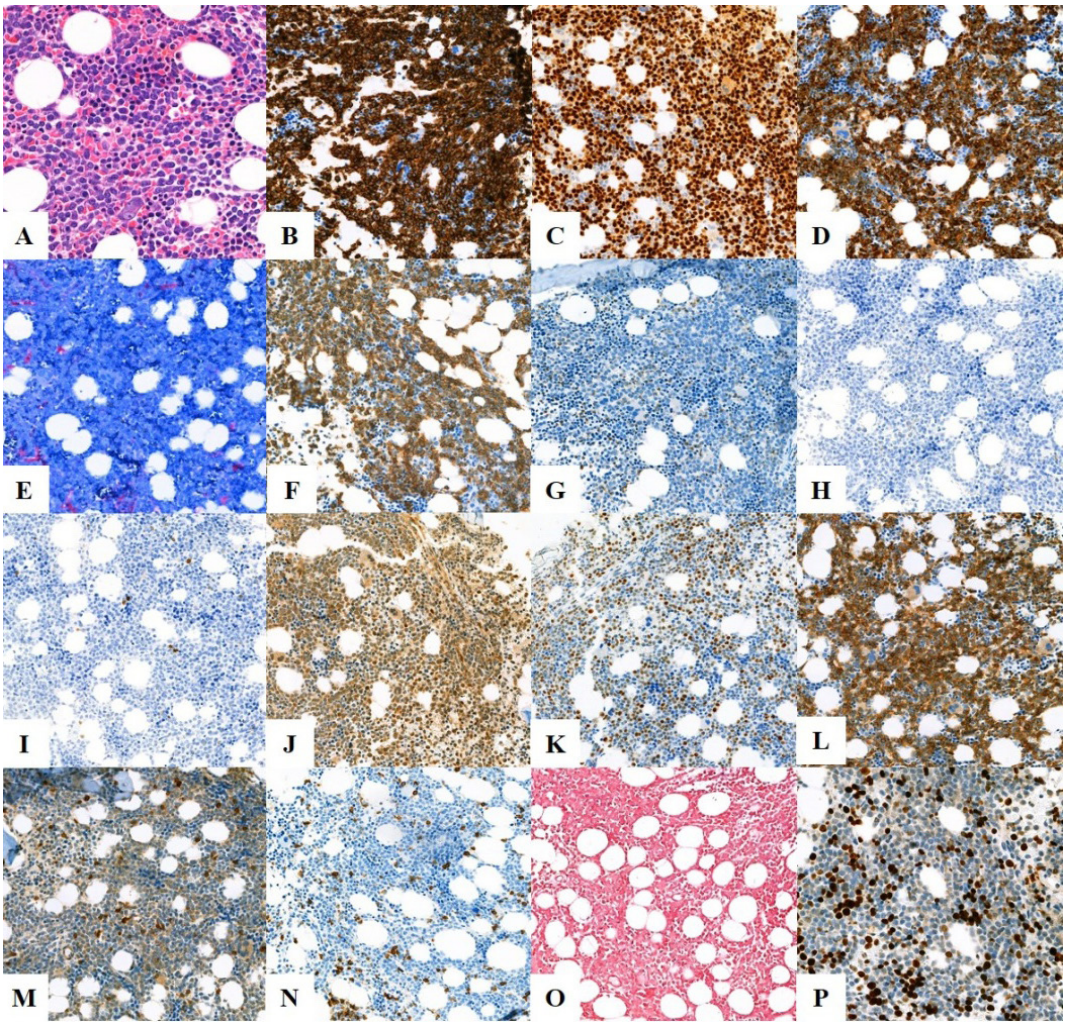

Figure 1 Plasma cell neoplasm with aberrant CD20 expression. (A) A case of progressive anemia and positive IgG kappa immunofixation. The myeloma cells co-express CD138 (B), MUM1 (C) and kappa light chain restriction: kappa+ (D) but lambda- (E). Among B-cell markers, only CD20 is positive (F), but other B-cell markers are negative including CD19 (G), CD22 (H) and CD79a (I). These myeloma cells also express cyclin D1 (J), c-myc (K) and CD117 (L), but they are negative for CD5 (M), CD45 (N) and EBER (O). The proliferative index by Ki-67 is 30% (P).

Working Group (IMWG) criteria, a diagnosis of MM or PCN requires the presence of at least 10% clonal plasma cells in bone marrow specimens or evidence of plasmacytoma accompanied by MDEs.[11,12] Pathological findings play a crucial role in establishing this diagnosis. Morphologically, PCN can display a spectrum of features on H&E-stained sections, ranging from normal plasma cell morphology to immature forms resembling plasmablasts.[13] Moreover, immunoglobulin inclusions, such as Goblet and Mott cells, may occasionally be observed in both normal reactive plasma cells and myeloma cells (Figure 1).

Immunophenotyping is essential for differentiating normal plasma cells from neoplastic plasma cells. Normal plasma cells typically express CD38, CD19 and CD45, while lacking expression of CD56, CD20, CD10 and CD200. Studies have demonstrated considerable heterogeneity in the immunophenotypic profiles of human bone marrow plasma cells. Furthermore, flow cytometry analyses have identified a wide panel of markers, including CD10, CD11b, CD11c, CD13, CD14, CD15, CD16, CD20, CD22, CD33, CD35, CD45, HLA-DR and surface immunoglobulin.

According to the WHO 2022 classification, neoplastic plasma cells characteristically express markers such as CD38, CD138 and MUM1. Additional markers frequently expressed include CD79a, BCMA, SLAMF7 and Blimp-1.[1] In contrast, neoplastic plasma cells typically lack expression of CD19, CD22 and CD24.[13-16] The International Clinical Cytometry Society (ICCS) recommends a standardized flow cytometry panel for the evaluation of multiple myeloma, which includes markers such as CD117, CD56, CD27, CD81 and assessment of cytoplasmic light chain restriction.[17] Aberrant expression of various markers has also been documented in neoplastic plasma cells, with reported frequencies of CD45 (approximately 10%), CD20 (10-15%), CD13 (25%), CD33 (25%), CD117 (25-30%), cyclin D1 (30-45%), CD56 (50-70%) and CD200 (75-80%).[13,18,19] Given that CD20 expression occurs in approximately 10 to 15% of PCN cases, clinicians may frequently encounter diagnostic challenges related to this marker.[20,21] Both normal and neoplastic plasma cells express markers such as CD27, CD28 and CD81; however, the intensity of expression differs between these populations.

Consequently, consensus guidelines advocate for the inclusion of CD27 and CD28 as part of the aberrant marker panel in flow cytometry analyses to enhance diagnostic accuracy.

New markers from the SLAM family have demonstrated multiple positive expressions in PCN, including SLAMF2, SLAMF3, SLAMF6 and SLAMF7.[22] However, these markers can also be expressed in normal plasma cells, though with varying intensity. Hosen, et al. reported that SLAMF2 is strongly expressed in PCN but weakly expressed in normal plasma cells.[23]

Cytogenetic abnormalities are detected using fluorescence in situ hybridization (FISH), which is crucial for prognostic stratification in conditions such as MM. Key abnormalities include t(4;14), t(14;16), t(14;20), del(1p), del(13q), del(17p), gain(1q) and t(6;14).[11,12,24]

Among these, the chromosomal abnormality t(11;14) is associated with a favorable prognosis and is also found in other B-cell neoplasms, particularly mantle cell lymphoma (MCL). Nevertheless, due to its distinctive morphological and immunophenotyping features, t(11;14) support the diagnosis of MCL when accompanied by CD5 and cyclin D1 or SOX11 expression. In contrast, t(11;14)(q13;q32) is present in approximately 10-20% of PCN cases,[11] in which the morphology may resemble lymphoplasmacytoid cells, thereby mimicking BCN-PD. Moreover, the t(11;14) abnormality is associated with expressions of CD20, CD79a and cyclin D1, but with absent CD56 expression.[11,25-29]

Currently, no pathognomonic molecular mutation has been identified for PCN, although mutations involving *MYC*, *FGFR3*, *RAS*, *p16* and *p53* have been reported in MM.[13] In IgM-secretory PCN, *MYD88* mutations–hallmark findings in lymphoplasmacytic lymphoma–have been invesgigated, but most cases are reported as *MYD88* wild type (WT). Notably, Treon et al. found that *MYD88* WT was present in 71.43% (5 of 7) of IgM multiple myeloma cases with the t(11;14) abnormality,[30] while Sebastien et al.'s study reported no *MYD88 L265P* mutations among patients with IgM multiple myeloma (0 of 3).[31]

B-cell non-Hodgkin lymphoma with plasmacytic differentiation (BCN-PD)

In B-NHL, the most common entities exhibiting plasmacytic differentiation are MZL and LPL. Other lymphomas, such as follicular lymphoma (FL), small lymphocytic lymphoma/chronic lymphocytic leukemia (SLL/CLL) and MCL, infrequently show plasmacytic differentiation. Each of these lymphomas possesses unique immunophenotypic markers: CD10 for FL, CD5 and CD23 for SLL/CLL and both CD5 and cyclin D1/SOX11 for MCL. Therefore, this review will primarily focus on MZL and LPL due to their prevalence and clinical significance.

Morphologically, lymphoplasmacytoid cells are identified as small cells with round nuclei and an eccentric nucleus or perinuclear halo. Immunoglobulin production in these cases can lead to misdiagnosis as PCN. To distinguish between MZL and LPL, the latter more frequently exhibits a focal paratrabecular distribution.[32] Both entities express B-cell markers including CD19, CD20, CD22, CD79a and PAX5.[1] In the marrow, LPL shows positive expression of CD19, CD20, CD22, CD38 and surface immunoglobulin light chain in 100, 91, 100, 70 and 100% of cases, respectively.[33] Aberrant expression patterns have been reported in MZL showing positivity for SLAMF3 (CD229), BLIMP1, CD43, CD11c, MUM1, myeloid cell nuclear differentiation antigen (MNDA) and IRTA1 (FCRL4), whereas LPL is characterized by expression of MUM1 alone.[15,34-36] The expression of MNDA and IRTA1 decreases in cases with PD.[37-39] Flow cytometry may also assist in distinguishing these entities, as MZL is typically positive for CD39, CD43 and CD95, while LPL is positive for CD31.[40]

Chromosomal abnormalities in BCN-PD are also useful for diagnosis. According to NCCN guidelines, the recommended work-up panel for chromosomal abnormalities in MZL includes t(14;18), t(11;18), t(1;14), del(13q) and del(7q). In contrast, no specific chromosomal work-up is indicated for LPL.[9,41] Braggio, et al. reported several abnormalities detected in both LPL and MZL using array-based comparative genomic hybridization, including deletion on chromosome 6q (including 6q21, 6q22.31, 6q23.3-q24.1 and 6q24.2) and/or gain of chromosome 9 in LPL.[42]

From a molecular perspective, *MYD88* mutations–including both L265P and non-L265P variants–are found in 90-100% of LPL cases, whereas nodal MZL exhibits these mutations in only a small proportion of cases.[31,43,44] The *MYD88 L265P* mutation has also been identified in cases of IgM monoclonal gammopathy of undetermined significance (MGUS), which may progress to LPL. Xu, et al. reported that the *MYD88 L265P* was present in 54.16% of IgM MGUS cases using allele-specific polymerase chain reaction (AS-PCR) and Sanger sequencing,[45] while Varettoni, et al. detected this mutation in only 5 of 84 IgM MGUS cases.[46] This variability highlights differences in detection methods and patient cohorts across studies.

Evaluating aberrant markers

Given the complexities in distinguishing between PCN and BCN-PD, immunophenotyping plays a crucial role in the differential diagnosis. Certain markers serve as aberrant markers and/or indicators of minimal residual disease. The following section outlines markers categorized within these two groups.

CD45

CD45 (common leukocyte antigen) is expressed on normal plasma cells as well as other hematopoietic cells. It is typically positive in B-NHL but may also stain other hematopoietic cells when bone marrow sample are examined. In contrast, myeloma cells frequently lack CD45 expression. Rosado, et al. reported immunophenotypic features differentiating BCN-PD from PCN using flow cytometry, noting CD45 expression during B-cell lymphoid neoplasm progression.[47] Carulli et al. observed similar mean fluorescent indices for CD45 expression between LPL and MZL cases.[48]

CD19

CD19 is a marker expressed at all stages of B cell development, from pro-B cells to plasma cells. In PCN, neoplastic plasma cells typically lack CD19, in contrast to normal plasma cells which primarily express this marker.[49] Lin, et al. demonstrated that CD19 was absent in CD20-positive myeloma.[50] Moreover, Marafioti et al. reported CD19 expression rates of 92.3% in MZL and 66.67% in LPL.[51]

PAX5

PAX5 is a transcription factor that serves as a crucial marker for B cells, as it remains expressed throughout B cell development but is absent in plasma cells. Thus, PAX5 expression is useful for confirming B-cell lineage in CD20-negative B-NHL, including cases treated with

monoclonal antibodies targeting CD20. Moreover, PAX5 expression is generally absent in PCN. These findings highlight the utility of PAX5 as a distinguishing marker for assessing the progression of B-cell lymphoid neoplasms.[52] Zhang, et al. demonstrated PAX5 positivity in 93.8% of MZL cases but absence in MM. However, it is important to note that although PAX5 is typically not expressed in PCN,[53] cases exhibiting CD20 expression often show concurrent PAX5 expression. Lin, et al. reported PAX5 co-expression with CD20 in 72% of cases.[54] Therefore, PAX5 should not be used in isolation to differentiate between PCN and BCN-PD.

MUM1

MUM1, also known as IRF4 (interferon regulatory factor 4), is a transcription factor that plays a crucial role in B cell differentiation and maturation. It is primarily expressed during the later stages of B cell development, particularly within the germinal centers of lymphoid tissues. MUM1 expression begins at the centrocyte stage, marking a critical transition in the differentiation of B cells into mature plasma cells. Consequently, both normal plasma cells and myeloma cells are positive for MUM1.[55] In addition, MUM1 positivity has been observed in approximately 20% of MZL cases.[36] Notably, aberrant MUM1 expression can also occur in both MZL and LPL, independent of CD138 expression.[56]

VS38c

VS38c serves as a marker for plasma cell differentiation applicable in both immunohistochemistry (IHC) and flow cytometry methods; however, it does not differentiate between normal and neoplastic plasma cells.[57] Turley, et al. reported VS38c positivity in cases of LPL, as well as in bone tumors.[58,59] Moreover, VS38c is positive in EMZL with plasmacytic differentiation but negative in cases without plasmacytic differentiation.[60]

BCMA

B-cell maturation antigen (BCMA, CD269), also known as TNFRSF17, is a surface protein that plays a significant role in B cell development, particularly during plasma cell maturation. BCMA can be detected through serum levels or immunostaining (CD269) and is typically expressed by plasma cells, including normal plasma cells, as well as those associated with MGUS and MM. In addition, BCMA expression has been observed in plasmablastic lymphoma, DLBCL and Burkitt lymphoma.[61-63] Serum BCMA levels can also be detected in WM or LPL, highlighting its potential as a biomarker for these diseases.[64] Moreover, immunotherapeutic strategies that selectively induce BCMA-specific T-cell responses have been demonstrated.[65]

CD79a

CD79a has been reported as positive in 100% of LPL and MALT lymphoma cases, but only 50% of PCN cases.[66] However, another study reported a 63% positivity rate in PCN (including both strong and weak staining), with cyclin D1 co-expression in 26% of cases, although no significant correlation with CD20 co-expression was found.[67]

BLIMP1

The *PRDM1* gene, associated with BLIMP1 expression, plays a role in plasma cell differentiation. In contrast to the persistent expression in PCN, PRDM1-B expression has been reported in 67% of MZL and 44% of LPL, with expression exceeding 5% being more common in MZL.[68]

Vimentin

Vimentin can be expressed in both PCN and B-NHL;[69,70] therefore, this marker is not recommended for distinguishing between PCN and BCN-PD.

CD200

CD200 is typically expressed in chronic lymphocytic leukemia/small lymphocytic lymphoma (CLL/SLL) and is negative in MCL. However, CD200 positivity has also been reported in some cases of MZL and PCN, which limits its utility in distinguishing PCN from BCN-PD.[19,71-73]

CD13

CD13, also known as aminopeptidase N, is a myeloid lineage marker involved in immune cell regulation. In normal plasma cells, CD13 expression is typically absent or present at very low levels. However, in clinical practice, CD13 is often assessed during minimal

residual disease evaluations due to its association with adverse prognosis.[74] Aberrant CD13 expression has been reported in LPL or WM in 31 of 111 cases, but not in MZL (0 of 155 cases) or MM (0 of 7 cases).[75]

SLAM

The SLAM family receptors consist of nine members found on both normal and neoplastic plasma cells. Among them, SLAMF2, SLAMF3, SLAMF6 and SLAMF7 have been confirmed in PCN. The role of SLAM family receptors in B-cell neoplasm progression remains unclear; however, SLAMF3 expression has been confirmed in MZL.[76]

Amyloid

AL amyloid deposition can occur in both PCN and BCN-PD, particularly in MZL and LPL, where it is present in approximately 34.28% of cases. In MZL, amyloid deposition most common occur in the peritumoral area, whereas LPL is more commonly associated with systemic AL amyloidosis.[77,78] Only 1 of 10 cases of BCN-PD with extensive amyloid deposition showed the *MYD88* L265P mutation, suggestive MZL rather than LPL.

CD43

CD43 is normally expressed by T cells, natural killer (NK) cells and myeloid lineages, but it is not typically found in B cells. However, recent studies have demonstrated its diagnostic utility in detecting B-NHL. Treasure, et al. reported CD43 expression in LPL and MALT lymphoma (13 of 131 cases and 9 of 131 cases, respectively).[79] Andriko, et al. found CD43 expression in only 2 of 17 cases of LPL.[80] Ning et al. reported CD43 expression in 77 of 109 cases of MM, with a possible prognostic role.[81]

IRTA1

IRTA1, also known as FCRL4, is a member of the immunoglobulin superfamily of transmembrane receptors predominantly expressed on B cells, particularly in MZL and MALT lymphoma. However, IRTA1 expression is generally absent in terminally differentiated plasma cells.[82,83] In a study by Ikeda, et al., PCN and LPL were negative for IRTA1 expression (0/10 and 0/7 cases, respectively), whereas MZL cases showed a high rate of positivity (23/25 cases). Notably, this study did not specify whether the MZL cases exhibited PD.[84]

WT1

Wilms tumor 1 (WT1) expression in the cytoplasm has been described in several hematologic neoplasms, including plasmacytoma, multiple myeloma and certain types of high-grade NHL, but not in indolent B-NHL.[85,86]

MNDA

MNDA is a nuclear protein predominantly expressed in myeloid lineage cells, including monocytes and granulocytes. MNDA expression is absent in normal plasma cells and PCN (0 of 8 cases). However, weak MNDA expression has occasionally been observed in MZL and MCL, reported in 8 of 15 and 6 of 8 cases, respectively, although associated morphological features were not described.[87]

Cyclin D1, SOX11

Cyclin D1 is a key regulator of the cell cycle that promotes cell proliferation and is overexpressed in most cases of MCL.[88] However, some MCL cases lack CyclinD1 expression, complicating diagnosis. SOX11, a neural transcription factor, is highly and specifically expressed in both Cyclin D1-positive and Cyclin D1-negative MCL, serving as a sensitive and specific biomarker for identifying MCL, particularly in Cyclin D1-negative cases.[89] Both Cyclin D1 and SOX11 are essential markers used to diagnose MCL and hairy cell leukemia (HCL). Normally, plasma cells do not express Cyclin D1 or SOX11. In a study by Padhi, et al., Cyclin D1 positivity was observed in 8 of 14 PCN.[90] Mansoor, et al. reported three cases of Cyclin D1-positive MZL, but none showed PD on H&E staining or immunostaining for plasma cell markers such as CD138 or MUM1.[91] Ribera-Cortada, et al. demonstrated that SOX11-negative MCL cases exhibited PD in 7 of 19 cases.[92] Additionally, Chen, et al. found that all PCN were negative for SOX11 regardless of Cyclin D1 status (0 of 30).[93]

Table 2 Comparison of plasma cell neoplasm with CD20 expression and B-cell non-Hodgkin lymphoma with plasmacytic differentiation

Feature	PCN	BCN-PD
CD45, CD19	-[13] (+ in normal plasma cells)	+[47,48]
PAX5	+/-[53,54]	+[1,52]
CD79a	+/-[66,67]	+[1]
MUM1, VS38c	+[55,57]	(LPL)+[32,36,56,58,59]
BLIMP1	+[1]	+[68]
MNDA	-[87]	+[87] (MZL)
CD200	+[15,19]	+ (SMZL)[71-73]
CD43	+[81]	+/-[40,79,80]
IRTA1	-[82,83]	+ (MZL)[84]
CyclinD1	+/-[90]	+/-[91]
SOX11	-[93]	- (MCL with PD)[92]
BCMA	+[1]	+ (LPL)[64]
SLAM Family		
SLAMF2	+[23]	ND
SLAMF3	+[23]	+ (MZL)[75]
SLAMF6	+[23]	ND
SLAMF7	+[1,23]	ND
CD13	+/-[13,74]	+ (LPL), - (MZL)[75]
Vimentin	+/-[69]	+[70]
WT1	+[85]	-[86]
Cytogenetic FISH[9,11,12,24,38,41,42]	t(11;14) t(4;14) t(6;14) t(14;16) t(14;20) del(1p) del(17p) gain(1q)	t(14;18) t(11;18) t(1;14) del(7q) gain(9p21.32-q34.3)(LPL) del(6q)(MZL)
MYD88[28,31,43,44]	*MYD88* WT	*MYD88* L265P (90% in LPL) *MYD88* WT

LPL, lymphoplasmacytic lymphoma; MCL, mantle cell lymphoma; MZL, marginal zone lymphoma; ND, no detail; PD, plasmacytic differentiation; SMZL, splenic marginal zone lymphoma

Conclusion

Distinguishing between PCN with CD20 expression (observed in approximately 10-15% of PCN cases) and BCN-PD remains a subject of ongoing debate and primarily depends on clinical context. Immunophenotypic analysis provides valuable diagnostic support, particularly when classical clinical and pathological features are present (Table 2). In addition, cytogenetic abnormalities and specific molecular mutations offer distinct profiles for each entity, aiding in differential diagnosis. At present, members of the SLAM family in BCN-PD represent promising biomarkers, although the evidence is limited due to small patient cohorts. As research advances, these markers may become key tools for establishing more definitive and precise diagnostic criteria in the future.

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Conflict of Interest

The authors declare that they have no conflict of interest.

Ethics approval

This article does not contain any studies with human participants performed by any of the authors.

Informed consent

For this type of study informed consent is not required.

Consent for publication

For this type of study consent for publication is not required.

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