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An Overview Of B- Cell Lymphoma Subtypes and Their Classification

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Abstract:
B-cell lymphomas are a heterogeneous group of malignancies derived from B lymphocytes, exhibiting diverse clinical, morphological, immunophenotypic, and genetic features, classified according to lineage, stage of differentiation. The most widely used classification systems, such as the World Health Organization (WHO) and the International Consensus Classification, are regularly updated to reflect advances in molecular and genetic understanding. B-cell lymphomas represent the majority of non-Hodgkin lymphomas (NHLs) and can occasionally present in the oral cavity, often posing diagnostic challenges due to their mimicry of benign odontogenic or inflammatory lesions. This review synthesizes current literature and World Health Organization (WHO) hematolymphoid neoplasm classifications (5th edition, 2022) to summarize key B-cell lymphoma subtypes encountered in the oral and maxillofacial region.
B-cell lymphoma classification is increasingly driven by molecular and genetic insights, Genetic subtyping guides targeted therapy and predicts response to immunochemotherapy, especially in DLBCL with the WHO-5 and ICC systems providing updated frameworks. Accurate subtyping is essential for prognosis and treatment, but some areas remain under active investigation as new discoveries continue to refine the field.
Despite these advances, challenges remain in subclassifying certain entities and in translating molecular findings into routine clinical practice.
Ongoing research and consensus efforts continue to shape the evolving landscape of B-cell lymphoma classification, aiming to enhance personalized management and therapeutic outcomes for affected patients.

Keywords: B-cell lymphoma**,** World Health Organization (WHO), Classification.

نظرة عامة على الأنماط الفرعية لورم الغدد الليمفاوية البائية

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الملخص
الأورام اللمفاوية البائية هي مجموعة غير متجانسة من الأورام الخبيثة المشتقة من الخلايا اللمفاوية البائية، وتتميز بخصائص سريرية ومورفولوجية ومناعية وجينية متنوعة. تُحدِّث أنظمة التصنيف الأكثر شيوعًا، مثل منظمة الصحة العالمية (WHO) والتصنيف الدولي للإجماع، بانتظام لتعكس التطورات في الفهم الجزيئي والوراثي. تُمثل الأورام اللمفاوية البائية غالبية الأورام اللمفاوية غير هودجكينية (NHLs)، وقد تظهر أحيانًا في تجويف الفم، مما يُشكل غالبًا تحديات تشخيصية نظرًا لتشابهها مع الآفات السنية الحميدة أو الالتهابية. تُلخص هذه المراجعة الأدبيات الحالية وتصنيفات منظمة الصحة العالمية لأورام الدم اللمفاوية (الطبعة الخامسة، 2022) لتلخيص الأنواع الفرعية الرئيسية للأورام اللمفاوية البائية التي تُصادف في منطقة الفم والوجه والفكين. يعتمد تصنيف ليمفوما الخلايا البائية بشكل متزايد على الرؤى الجزيئية والجينية. يُرشد التصنيف الفرعي الجيني العلاج المُستهدف ويتنبأ بالاستجابة للعلاج الكيميائي المناعي، خاصةً في ليمفوما الخلايا البائية الكبيرة (DLBCL)، حيث يوفر نظاما WHO-5 وICC أطرًا مُحدثة. يُعد التصنيف الفرعي الدقيق ضروريًا للتشخيص والعلاج، ولكن لا تزال بعض المجالات قيد البحث النشط مع استمرار الاكتشافات الجديدة في تحسين هذا المجال.

الكلمات المفتاحية: ليمفوما الخلايا البائية، منظمة الصحة العالمية والتصنيف.

Introduction

A primary goal of lymphoma classification is to guide clinical management by grouping patients with similar disease characteristics and outcomes, enabling consistent diagnosis and treatment across institutions [1].

Initial classifications distinguished lymphomas based on the presence of Reed-Sternberg cells, separating Hodgkin lymphoma from non-Hodgkin lymphoma. This distinction remains clinically important, as staging is more critical for Hodgkin lymphoma management [2]. Rappaport Classification (1960s) introduced a modern approach using cytologic features, but was limited by incorrect assumptions about cell origin. Lukes-Collins and Kiel Classifications (1970s) linked lymphoma subtypes to specific stages of lymphoid differentiation, using cytology and grading (low vs. high grade). The Kiel system gained popularity in Europe, but lacked robust clinical validation [3]. Working Formulation (1982) Sought to standardize terminology and facilitate research, but was largely morphologic and less biologically informed.

REAL Classification (1994): Marked a shift to defining distinct disease entities using morphology, immunophenotype, genetics, and clinical features, laying the groundwork for modern systems. WHO Classifications (2001, 2008, 2016, 2017) built on the REAL system, integrating molecular and genetic data, and achieving international consensus. The latest editions define over 80 lymphoma entities, reflecting advances in understanding and enabling more precise diagnosis and therapy

B-cell lymphomas with features intermediate between DLBCL and CHL:

This lymphoma subtype most frequently affects young males between the ages of 20 and 40. Its cause remains unknown, and the mediastinum is the most common site of involvement, which may occur with or without associated lymph node disease [4].

Diffuse large B-cell lymphomas DLBCLs:

About 30–40% of cases of non-Hodgkin lymphoma worldwide are (DLBCL). There is considerable variation in the disease's presentation, prognosis, and response to treatment, making it a clinically and genetically diverse condition [4].

Tumor cells resemble immunoblasts, with prominent nucleoli, eccentric nuclei and high mitotic activity. They typically lack (CD20, CD79a) expression but express plasma cell markers (CD138, MUM1, VS38c) [5]. EBV positivity is reported in 50–74% of oral PBL cases, particularly in HIV-infected individuals. PBL shows genetic heterogeneity, with frequent mutations in pathways such as JAK/STAT and MAPK/ERK. The ST2 genetic subtype is seen in about 23–27% of cases [6].

Mucosa-associated lymphoid tissue (MALT) lymphomas:

(MALT) lymphomas, also known as extranodal marginal zone B-cell lymphomas, are among the most common extranodal lymphomas in the head and neck. They typically present as slow-growing, localized masses or submucosal thickenings, often mimicking benign or inflammatory conditions, and have an indolent clinical course [5]. In Waldeyer's ring, they are the second most common lymphoma type after DLBCL [5]. Patients often present with painless, persistent swelling or mass. Symptoms are usually mild or absent, leading to delayed diagnosis. In the oral/maxillofacial region, asymptomatic swelling is typical [5]. MALT lymphomas rarely disseminate. Microscopically characterized by small to medium-sized lymphocytes with centrocyte-like appearance, frequent lymphoepithelial lesions, and sometimes plasmacytoid differentiation. Dutcher bodies may be seen in cases with plasmacytoid features. Tumor cells often display a distinct immunophenotypic profile and harbor specific chromosomal translocations, which can drive oncogenesis and influence diagnostic strategies. A typical pattern is positivity for pan–B-cell markers and BCL-2, with negativity for CD5, CD10, CD23, and IgD, alongside recurrent translocations that activate the NF-κB pathway [5].

Follicular lymphomas (FL):

In western population, FL is the second most common NHL. These lymphoma types frequently relapse and have a slow clinical course. It could develop into an aggressive lymphoma. FL's biology and morphology are similar to those of a typical germinal-center (GC) reaction [6]. Centrocytes, which are small cleaved follicular center cells, centroblasts, which are large non-cleaved follicular center cells, and tumors originating from germinal center B cells are all included in FL, a diverse clinical pathological disorder. The discovery of somatic mutations in the variable region of the immunoglobulin (IgVH), which function as a marker of germinal center transit, primarily supports the germinal center ancestry of these cells. FL cells express (CD19, CD20, CD22, CD79a, and CD79b) and surface immunoglobulins (IgM>IgD >IgG >IgA)., and CD10 positivity or negativity [6]. Follicular lymphoma generally expresses CD10, bcl-2 and bcl-6 [6].

Mantle cell lymphomas (MCL):

Despite being the second most common malignant neoplasm in the head and neck area overall, MCL rarely manifests as its primary form in the oral cavity. Small-to-medium-sized lymphoid cells are the hallmark of MCL [7]. Microscopic diagnosis of MCL can be difficult because of its rarity and histologic resemblance to other small cell lymphomas, especially in the oral cavity where other lymphomas are more common. The term "mantle cell lymphoma," was accepted by the WHO in 2001 and is currently the accepted nomenclature [8].

Based on the previous study, MCLs accounts for between 6% and 10% of all B-cell lymphomas. The majority of these cases occur in lymph nodes, but many patients have extranodal involvement, and the waldeyers ring is the most affected site [9]. Microscopically Extranodal MCL is a B-cell lymphoma subtype that often disrupts normal tissue architecture and displays distinctive morphological and molecular features. The hallmark is a monomorphic lymphocytic infiltrate, often with the t (11;14) (q13; q32) translocation, but this is not universal**.**which results in the juxtaposition of the (Bcl-1) gene locus to the Ig heavy chain promoter. Both the pleomorphic variant (PVMCL) and the blastoid variant (BV-MCL) are aggressive subtypes of MCL. The WHO has identified these entities as separate subtypes, despite the fact that some people think they are variations along the same spectrum. Cells that resemble lymphoblasts and have abnormally high mitotic rates are characteristics of BV-MCL.

In contrast to normal MCL, BV-MCL is characterized by extra copies of the CCND1 gene, and its proliferation rate is measured by the expression of the Ki67 antigen, which is greater than 50% [9]. This variant has a shorter response duration following first-line therapy and is more likely to affect older patients. Similar to BV-MCL, PV-MCL has a distinct histology with large pleomorphic cells with pale cytoplasm, round to oval nuclear contours, and, in the case of at least some of the cells, conspicuous nucleoli [7].

Small lymphocytic lymphoma (SLL):

In all regions, SLL accounts about 7% of all NHL cases. A proliferation of tiny, mature-looking lymphocytes is the morphological hallmark of SLL. SLL closely resembles other small B-cell lymphomas like MCL, FL, and MZL under a microscope [10]. Only the clinical picture distinguishes SLL from chronic lymphocytic lymphoma, which have similar morphological, phenotypic, and genotypical features. In order to recognize that CLL and SLL are distinct expressions of the same disease entity, the International Lymphoma Study Group suggested the designation CLL/SLL [10].

A diffuse and monotonous pattern of small lymphocytes with proliferation centers made up of pro-lymphocytes or paraimmunoblasts was visible under a microscope in the incisional biopsy specimen. The neoplastic lymphoid cells were immunohistochemically positive for LCA, CD5, CD20, CD79a, CD23, CD43, Bcl-2, and Bcl-6, and negative for smooth-muscle actin, immunoglobulin light chains, CD10, Bcl-1 (cyclin D1), CD3, CD30, and CD68. B-cell CLL/SLL is a disease that affects middle-aged and older people. The majority of Bcell CLL/SLL patients present with widespread lymphadenopathy. It can be challenging to distinguish B-CLL/SLL from other small B-cell lymphomas. Genetic research, immunophenotyping, and clinicopathological correlation can all yield incredibly helpful data. Conservative treatment is an option for CLL/SLL, an indolent lymphoproliferative disease, particularly in its early stages [10].

B-cell lymphoblastic lymphoma (B-LBL):

A high-grade tumor arises from precursor lymphocytes. Despite having the same morphology, it is thought to be a different clinical manifestation of B-cell acute lymphoblastic leukemia (B-ALL). Small to medium-sized cells with a high rate of mitosis, numerous apoptotic figures, finely stippled chromatin, and irregular nuclear contour, and inconspicuous nucleoli [11].

described a case of B-LBL in childhood that showed up as a painless swelling in the mandible. In other locations, migratory pain was the most prevalent early sign of BLBL. MRI and radiographic imaging are useful for identifying these tumors. Since B-LBL histologically resembles Ewing's sarcoma, immunohistochemistry may be the most accurate diagnostic tool. TdT, CD43, and CD79a are helpful in ruling out a diagnosis in the event of a positive BLBL identification of Ewing’s sarcoma.

Despite being a rare high-grade cancer with a poor prognosis for relapsed cases, B-LBL appears to be curable, particularly in children, with prompt diagnosis and vigorous chemotherapy. (B-LBL) typically affects extranodal sites, mostly the skin, in contrast to T-cell lymphoblastic lymphoma (T-LBL), which typically affects the lymph nodes and mediastinum. Intraoral B-LBL was first reported in 2007 [12].

Plasmablastic lymphoma PBL:

Plasmablastic lymphoma is categorized as a subtype of diffuse large B-cell lymphoma. It can be mistaken for a straightforward tooth abscess due to its location and frequently subtle appearance. Patients who present with an expanding oral lesion should have a formal dental evaluation [13]. PBL is categorized by the World Health

Organization as a non-Hodgkin's B-cell lymphoma that primarily affects individuals with HIV. According to reports, PBLs are accountable for 2.6% of non-Hodgkin's lymphoma cases linked to HIV [14].Plasmablastic lymphoma typically has a monomorphic histological appearance with a cohesive growth pattern and diffuse lymphoid infiltration. Neoplastic cells have a single, noticeable, central nucleolus or multiple peripheral ones, and they resemble plasmablasts, which are large cells with eccentric round or oval nuclei with fine chromatin. The cells have a lot of cytoplasm, which shows up as deeply basophilic when stained with Giemsa [15]. On a physical examination, the neoplastic infiltrate often causes superficial ulceration in the surrounding soft tissue [16]. Typically, plasmablastic lymphoma exhibits a distinctive immunophenotype that includes positivity for plasma cell markers like CD38, CD138, and VS38c and negativity for common B-cell antigens like CD20 [16].

Burkitt's Lymphoma (BL):

Approximately one-third of AIDS-related cancers are NHL, with 2.4–20% of HIV-associated NHL being Burkitt's lymphoma (BL). Children are frequently affected by BL, the incidence peaks between ages 3 and 8 years with males affected about twice as often as females [17]. HIV-associated BL, endemic BL, and sporadic BL are the three clinical forms of BL. These clinical variations can be distinguished in part by their geographic location and share similar histological characteristics [18]. In Africa and Papua New Guinea, endemic BL is frequently observed in children as an orbital or jaw mass. Sporadic BL typically involves the abdomen or nodes and has no particular age or geographic predilection. HIV-positive people have immunodeficiency-associated BL. There are very few reports of HIV-associated Burkitt's lymphoma presenting intraorally [17].

The jaws and abdomen are the most frequently involved sites. In endemic BL, jaw involvement occurs in up to 60–75% of cases, while in sporadic BL, it is seen in 12–30% of cases [15]. Common oral symptoms include swelling, tooth loosening or displacement, pain, and paraesthesia. These may be mistaken for dental infections or other benign conditions [16]. BL lesions appear as radiolucent areas with poorly defined, irregular margins, often leading to bone destruction and "floating teeth. Classic BL is characterized by sheets of uniform, intermediate-sized lymphoid cells with round nuclei, multiple nucleoli, scant cytoplasm, and numerous mitotic figures. The presence of tingible body macrophages creates a distinctive "starry-sky" pattern. Dentists and oral health professionals play a crucial role in early detection, which is vital for improving outcomes [16]. Intensive multiagent chemotherapy is the standard treatment, with high remission and survival rates (75–95%) when diagnosed early [17]. Prognosis depends on the stage at diagnosis; early treatment is associated with better outcomes.

B-cell lymphomas with features intermediate between DLBCL and BL:

Aggressive tumors that do not neatly fall into either category are B-cell lymphomas with characteristics halfway between Burkitt lymphoma (BL) and diffuse large B-cell lymphoma (DLBCL). These "unclassifiable" lymphomas have a poor prognosis and share overlapping clinical, morphological, and genetic traits [18]. Usually intermediate in size, tumor cells are bigger than BL but smaller than classic DLBCL. With atypical characteristics like high BCL2 expression or a Ki-67 index below 90%, the immunophenotype frequently resembles BL (e.g., CD10+, BCL6+, high Ki-67) [19]. MYC rearrangements, occasionally in conjunction with BCL2 and/or BCL6 rearrangements, are present in many cases and contribute to their aggressive behavior [20].

These lymphomas are rare in adults, often present with extranodal involvement, and are more likely to be diagnosed at advanced stages [19]. Accurate classification requires integration of morphology, immunophenotype, and genetic studies. Markers like Pax-5 and Ki-67 can help distinguish these from other B-cell neoplasms

Epstein Barr virus associated lymphomas:

Epstein-Barr virus (EBV) is linked to several aggressive oral tumors, particularly in EBV-positive lymphomas often express MUM1 and lack CD10, and show markers like LMP1 and CD30, with reduced Bcl-6 and immunoglobulin expression. These features make them more similar to Hodgkin lymphoma than to EBV-negative DLBCL [20]. EBV-positive oral lymphomas are typically aggressive and associated with poor prognosis, especially in immunocompromised patients [19]. Standard therapies are often unsatisfactory. Adoptive cellular immunotherapy has shown promise in some cases, particularly for lymphomas expressing more immunogenic EBV latency programs.

EBV is implicated in Burkitt lymphoma, Hodgkin lymphoma, diffuse large B-cell lymphoma (DLBCL), plasmablastic lymphoma (PBL), post-transplant lymphoproliferative disease, nasopharyngeal carcinoma, and some gastric and smooth muscle tumors [19]. In the oral cavity, EBV-related tumors most often affect the tongue, parotid gland, and sometimes the periodontium. PBL is especially common in HIV-positive patients, with nearly all oral mucosal PBLs in this group being EBV-positive. The risk and aggressiveness of EBV-associated oral tumors are heightened in immunosuppressed individuals, such as those with HIV/AIDS or post-transplant status [20].

EBV-positive DLBCL of the Elderly:
Epstein-Barr virus (EBV)-positive diffuse large B-cell lymphoma (DLBCL) of the elderly is a distinct, aggressive lymphoma recognized as a provisional entity in the 2008 WHO classification [20].
Most patients are older than 50, with a male-to-female ratio of about 1.4–1.5:1. Median age is typically in the 60s–70s. Higher prevalence in Asia, lower in Western countries, suggesting ethnic or geographic factors [21]. Frequently extranodal, with polymorphic or monomorphic large B-cell infiltrates, sometimes with Reed-Sternberg-like cells. B-symptoms and advanced stage are common. Tumor cells are positive for CD20, CD79a, EBER, and LMP1, and often negative for CD15. Most cases show an activated B-cell (non-germinal center) phenotype and high CD30 expression [21].
EBV latency type III is typical, reflecting immune senescence rather than overt immunodeficiency [21]. The disease is clinically aggressive, with a median overall survival of less than one year and significantly worse outcomes compared to EBV-negative DLBCL.

DLBCL Associated with Chronic Inflammation:
Diffuse large B-cell lymphoma associated with chronic inflammation (DLBCL-CI) is a rare, aggressive lymphoma that develops in sites of long-standing inflammation and is strongly linked to (EBV) infection. The classic example is pyothorax-associated lymphoma (PAL), which typically arises in the pleural cavity after many years of chronic pyothorax, often related to prior tuberculosis treatment [23]. DLBCL-CI can also develop in bones, periarticular tissues, pseudocysts, and other areas with chronic inflammation or foreign material.
These lymphomas often present as large masses (over 10 cm in more than half of cases) and may directly invade adjacent structures, though they are usually confined to the affected cavity at diagnosis [24]. Most patients are older adults, but cases can occur in individuals over 10 years old. About 70% present at early clinical stages (I or II). Nearly all DLBCL-CI cases are EBV-positive, with the lymphoma cells exhibiting type III EBV latency. EBV is central to the pathogenesis, promoting B-cell transformation in the context of local immune dysregulation [24]. PAL cells express chemokine's (CXCL9, CXCL10, CCL17, CCL22) that attract cytotoxic and regulatory T cells, contributing to tissue necrosis and immune evasion [22].

Primary Effusion Lymphoma (PEL):
It is important to differentiate PEL from DLBCL, which is linked to persistent inflammation. The clinical feature of PEL is diffusion to one of the body cavities (pleural, pericardial, or peritoneal cavity), without lymph node enlargement or lymphadenopathy. HIV+ individuals, patients with severe immunodeficiency and EBV infection, people with HHV-8 co-infection, and transplant recipients are all diagnosed with this condition [24].

Lymphomatoid Granulomatosis:
This is a rare disease referred to as EBV-associated angiocentric and angiodestructive lymphoproliferation. The incidence in men is twice as high as in women. Although the condition is typical for adults' pediatric cases were also reported. Lymphomatoid granulomatosis is characterized by the presence of polymorphic infiltrate of cells [25].

Plasmablastic Lymphoma (PBL):
This is a rare lymphoma, originally described on oral and nasal mucosal membranes of HIV+ persons [22]. Nearly 100% of the cases turn out to be associated with EBV infection. PBLs can also affect HIV (-) individuals with immunosuppression after organ transplantation [23].

Primary DLBCL of Central Nervous System (PCNSL):
A rare and aggressive type of NHL which is limited to the brain, spinal cord, leptomeninges, and eyes. It rarely or never spreads to the bone marrow or outside the central nervous system (CNS). When systemic recurrence does occur, it usually affects the breasts or testicles [22]. Immunocompromised people, such as those with HIV/AIDS, are more likely to develop PCNSL, which is often linked to Epstein-Barr virus (EBV) infection and tends to manifest later in the course of infection. Large lymphoma cells are the pathological hallmark of PCNSL, which is frequently accompanied by activated astrocytes, microglial cells, and small reactive lymphocytes. Necrotic areas may also be present within the tumor. Ocular involvement can occur, and the disease may recur within the CNS even after treatment [22].

Conclusion
B-cell lymphomas of the oral maxillofacial region comprise a heterogeneous group of malignancies with distinct biological behaviors and therapeutic responses. Updated classification systems integrating histology, immunophenotype, and genetics are essential for accurate diagnosis and personalized treatment planning.

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