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B-Cell Non-Hodgkin's Lymphomas of the Oral Maxillofacial Region: Narrative Review

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ABSTRACT

B-cell non-Hodgkin lymphoma in the oral and maxillofacial region is a rare but serious condition that requires prompt diagnosis and aggressive treatment. Understanding the clinical features, staging, and treatment options is crucial for improving patient outcomes in this challenging area of oncology. This review aims to highlight the clinical presentation, diagnostic approach, etiology, and management of B-cell NHL in the oral and maxillofacial region, emphasizing the importance of early recognition in improving patient outcomes.

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INTRODUCTION

B-cell lymphomas make up 80% to 85% of non-Hodgkin lymphomas (NHLs) at all anatomic sites. Most oral cavity lymphomas are B-cell lymphomas, and their frequency ranges from 41% to 100%. The predominant histologic type appears to be diffuse large B-cell lymphoma. A lymphoma that develops in the oral cavity is often a component of the spreading process of a disease, which may involve regional nodes, or it may represent primary extranodal disease confined to the oral cavity or jaws. Epstein–Barr virus (EBV), a DNA virus, has been associated with a variety of human cancers, including B and T-cell lymphomas. EBV can infect both epithelial cells of the oropharynx and B lymphocytes.

The virus is secreted in the saliva, and human infection occurs through oral transmission. An oropharyngeal infection causes a lytic (productive) infection, which is followed by infection of B cells in circulation. This causes the viral DNA to persist in the nucleus as an episome, resulting in a latent infection. This subset of latently infected lymphocytes is thought to give rise to EBV-containing lymphomas, particularly when immunodeficiency prevents the body from identifying and eliminating infected cells.

Lymphoproliferative disorders

Nasopharynx, nasal, paranasal sinuses, and salivary glands are the most affected parts of the head and neck by lymphoproliferative disorders. The nasopharynx and Waldeyer's ring are functionally similar to the mucosa-associated lymphoid tissue (MALT) of the gastrointestinal tract. B-cell lymphomas, most commonly mantle cell lymphoma, affect the nasopharynx and Waldeyer's ring. The most common lymphoid lesion of the salivary gland is lymphoepithelial sialadenitis, which is associated with Sjögren's syndrome. Patients with Sjögren's syndrome are at increased risk of developing B-cell lymphomas, most commonly MALT lymphomas. The nasal and paranasal sinuses are the prototypical site for the development of extranodal natural killer (NK) /T-cell lymphoma, nasal type [1].

Head and neck B-cell lymphoma overview

Lymphomas are a heterogeneous group of clonal malignant diseases that share the single characteristic of arising as the result of a somatic mutation in a lymphocyte progenitor. The progeny of the affected cell usually carries the phenotype of a B, T-, or natural killer-cell as determined by immunophenotyping and/or gene rearrangement studies.

A lymphoma may arise within the lymph nodes or any organ, either by spread from lymphatic sites or as a manifestation of primary extra-nodal disease [2]. Histologically, they are characterized by the presence or absence of Reed-Sternberg cells. Reed-Sternberg cells are pathognomonic of Classic Hodgkin's lymphoma (CHL), and all other lymphoid neoplasms that lack these cells are characterized as NHLs. They mainly involve lymph nodes, spleen, and other non-haemopoietic tissues, with painless enlargement of lymph nodes being the most common presenting symptom. However, an important feature of NHL, which distinguishes it from Hodgkin's disease, is that NHL can arise in an extranodal site, either as a primary or occult lesion [3]. CHL rarely shows extranodal disease (1% cases) in contrast to NHL (23-30% cases) [4]. Typical locations of extra nodal NHL include gastrointestinal tract, Waldeyer's ring, skin, and others. Waldayer's ring is the second most common area after the gastrointestinal tract to be involved in NHL. The oral cavity as a primary site constitutes only 2% of all extranodal NHL [5]. Approximately 85% of these lesions are located submucosally on

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the gingivae, the hard or soft palate, and the tongue. Lymphomas of the salivary glands represent 2% to 5% of all salivary gland neoplasms. The parotid gland is most commonly affected, comprising 80% of cases, followed by the submandibular gland (16%), sublingual gland, and minor salivary glands (2%) [6].

Oral cavity lymphomas are rare, representing the third most common malignancy in the oral cavity, after squamous cell carcinoma and salivary gland malignancies, accounting for only 3% to 5% of all oral malignancies [7].

Soft tissue lymphomas of the oral cavity and primary lymphomas of the jaw are often misdiagnosed. Clinically and radiographically, the manifestation is usually similar to squamous cell carcinoma or an odontogenic tumor, cyst, or infection [6].

Among the NHLs that occur in the oral cavity, about 15-45% arise in the maxilla and Mandible [7]. The maxilla is most commonly affected (11%), followed by the mandible (8%), the palatal soft tissue (8%), and the gum (7%). Isolated mandibular NHL accounts for only 0.6% of all NHLs [8]. NHLs are more common in males. The male/female ratio ranges from 1.3:1 to 2.3:1.

A predominance of females was found only in mucosa-associated lymphoid tissue (MALT) lymphomas in which the female/male ratio was close to 2:1 [11]. As a rule, NHLs of the jawbone arise during the fourth to fifth decade of life [11]. Clinical or radiographic features are not pathognomonic for NHL of the jaws, and for cases without concurrent lymph node involvement, this creates a diagnostic challenge. Incision biopsy of the deep portion of a mass provides the optimal opportunity to obtain the correct diagnosis and the subtype of these lesions. Surface biopsy specimens often show only chronic inflammation or necrosis. Histologic interpretation may be further complicated by superimposed inflammation or secondary infection [12].

Although all subtypes of non-Hodgkin lymphomas can occur in the oral cavity, lymphomas composed of large cells are the most common, with a nuclear size more than twice the size of a normal lymphocyte or larger than a normal macrophage, and usually have a B-cell phenotype [13]. However, large B-cell lymphomas include several subtypes of lymphoma that are important to recognize because of differences in clinical course [13].

The majority of adult NHLs are of B-cell origin. Low-grade B-NHL comprises several well-defined disease entities, including small lymphocytic lymphoma/chronic lymphocytic lymphoma (SLL/CLL), follicular lymphoma (FL), and extra-nodal marginal cell lymphoma (ENMZL). These tumors are characterized and distinguished by a combination of clinical, morphological, and immunophenotypical features and distinctive genetic abnormalities. They show a range of cytologic differentiation and can transform into high-grade neoplasms at variable frequency [14].

Epidemiology

The incidence of Non-Hodgkin lymphomas has increased dramatically from the 1970s to the middle of the 1990s [15]. During this period, the steady increase of 3% to 4% per year in incident cases of lymphoma and diffuse large B-cell lymphoma occurred in both sexes, among all racial categories, and all age groups except the very young. Incidence rates remained stable in the late 1990s and from 1992 to 2006. The age-adjusted incidence rate for DLBCL increased approximately 1% per [15].

The incidence of NHL varies markedly from country to country. In Europe and the United States, B-NHL represents the largest class of NHL, whereas lymphoblastic lymphoma (LL) and anaplastic large cell lymphoma (ALCL) represent 20–25% and 10% respectively, of NHL in children and adolescents. In Africa, up to 50% of childhood cancers are lymphomas [16].

Based on the epidemiological features of Hodgkin lymphomas, its aetiology may differ by age of patients at presentation [17]. In developing countries, children acquire HL at an earlier age compared to developed countries, and usually their Reed-Sternberg cells demonstrate Epstein-Barr virus (EBV) genomic sequences.

There is also a slight overall male predominance in the incidence of HL, which is marked in children less than 10 years old, whereas in adolescents, the incidence is approximately equal between males and females in industrialised countries [16].

Aetiology

The exact aetiology of non-Hodgkin lymphoma is not clear. The potentiality of viruses as a causative agent has been suggested. It has also been observed that the incidence of lymphomas is higher in congenitally immunocompromised patients and patients who receive immunosuppressive treatment [17].

According to Jarrett (2003), infectious agents may play a role in the aetiology of Hodgkin's lymphoma (HL), with EBV as one of the major causes of many cases of HL. This has been confirmed by immunohistochemical studies and molecular biology, where 40–50% of HL cases are associated with EBV in developed countries [18].

There is a high incidence rate of HL among children with immunodeficiency, HIV
patients, Wiskott Aldrich syndrome, ataxia-telangiectasia, children with a previous history of chemotherapy, and Bloom’s syndrome [19].

DLBCLs may originate de novo or as a result of progression from a less aggressive lymphoma. There are some factors which is thought to be potentially associated with the risk of developing DLBCLs. These factors include immunosuppression (including AIDS and iatrogenic etiologies in the setting of transplantation or autoimmune diseases), ultraviolet radiation, pesticides, hair dyes, and diet. A subset of diffuse large B-cell lymphoma, including immunoblastic and primary CNS disease, is strongly associated with EBV [20].

The occurrence of HL within families or races suggests genetic predisposition to the disease. According to studies of affected families with HL there is an association of HL with specific HLA antigens. EBV has been considered to be the aetiological agent of many cases of HL [20].

Epstein-Barr virus

EBV belongs to the Herpes viridae family, the gamma subfamily, and the Lymphocryptovirus genus. Two genetically different forms of EBV have been detected in the latent-cycle genes, termed EBV type 1, which is more predominant in Western countries, and EBV type 2.

Both types are common in sub-Saharan Africa and Papua New Guinea, and encode the nuclear antigens 2 and 3 (EBNA 2 and EBNA 3). Both EBV types can simultaneously infect the same person. EBV type 1 is more potent than type 2 in achieving B-lymphocyte transformation in vitro [21]. In vitro EBV latent antigens can transform immunoglobulin-secreting B lymphocytes into immortal, dividing lymphoblasts. EBV LMP-1, which is an integral membrane protein, appears to comprise the most important oncoprotein in mediating EBV-related malignant cell transformation.

In vitro, LMP-1 mimics receptors of the tumor necrosis factor receptor superfamily (TRAFs) and activates numerous signaling pathways, including nuclear factor kappaB (NF-KB), c-Rel, p38, c-Jun N-terminal kinase (JNK), and phosphatidylinositol 3-kinase (PI3K)/ Akt [22].

The life cycle of EBV involves two compartments: the peripheral blood and the oral cavity. EBV resides in B lymphocytes. Latently infected memory B lymphocytes circulate in the peripheral blood and are believed to constitute the main reservoir for EBV persistence [21].

EBV evades the host by altering its pattern of gene expression. The EBV genome normally codes for nearly 100 viral proteins, but EBV-infected resting memory cells evade immune recognition by limiting the gene expression to nine viral latent proteins in varying patterns. The six nuclear antigens EBNAs-1, -2, -3a, -3b, -3c, and -LP are responsible for maintaining the viral genome as well as controlling the expression of three latent membrane proteins: LMP-1, -2a, and -2b. EBV-infected NHLs variably express proteins associated with latent infection, some of which have been shown to have oncogenic potential, both in vitro and in vivo. Latent viral gene products with oncogenic potential include Epstein-Barr nuclear antigen-2 (EBNA2), a specific transcriptional trans- activator of viral and cellular genes, the latency membrane protein-1 (LMP-1), and the transmembrane phosphoprotein. LMP-1, EBV's main transforming protein, induces B-cell lymphomas in transgenic mice, proving its oncogenic potential. It binds to B-cells' CD40 receptor because it resembles the CD40 ligand. Thus, LMP-1 expression activates the NF-kB pathway constitutively without ligand and signals B-cell growth. B-cell immortalization requires the JAK/STAT, ERK, MAPK, IRF4, and Wnt pathways, all of which are modulated by LMP-1. By upregulating the expression of antiapoptotic proteins like Bcl-2 and A20, LMP-1 can also prevent p53-mediated apoptosis in B-cells. Additionally, LMP-1 stimulates cytokines like IL-10 to promote B-cell proliferation. By keeping the EBV-infected cell from entering its replicative cycle and being eliminated by the immune system, LMP-1 also seems to inhibit the plasma cell differentiation of B-cells, which may aid in lymphomagenesis [23].

Latency patterns of EBV infection

EBV-encoded genes replicate specific virion components, including viral proteins and DNA, during lytic infection. Six nuclear antigens (EBNA 1, 2, 3A, 3B, 3C, and LP), three latent membrane proteins (LMP 1, 2A, and 2B), two small non-coding RNAs (EBER1 and EBER2), and BamHI-A rightward transcripts are among the genes encoded by EBV that shield the viral genome from immune surveillance during latent infection. Three latency programs equivalent to B cell differentiation have been proposed following analysis of EBV-infected B cells in cell lines and seropositive individuals [22]. EBV-infected naive B cells that express all latent antigens are impacted by type III latency (growth program). The EBV-infected pool of cells grows as the naive infected B cells move into the germinal center (GC), where they multiply and clonally expand.

These GC-infected cells show a restricted EBV gene expression pattern (EBNA-1, LMP-1, LMP-2), which is referred to as the type II latency. The infected GC cells then differentiate into memory B cells, which function as the long-term reservoir for EBV. Most of the infected memory B cells in the periphery seem not to express any of the viral antigens, which is considered by some as type 0 latency; but some only express EBNA-1, which has been referred to as type I latency [23].

Pathophysiology

Cell proliferation is controlled by proto-oncogenes. Activation of these protooncogenes by genetic abnormalities like chromosomal translocations, gene mutations, or amplifications results in malignant transformation. In contrast, tumor suppressor genes promote cell differentiation and decreased cell proliferation. In the malignant transformation process, oncogene activation and inhibition of the tumor suppressor genes usually occur [19]. During the germinal center reaction, at least two DNA modifications, somatic hypermutation (SHM) and class switch recombination (CSR), occur. These two reactions are mediated by the B-cell-specific enzyme activation-induced cytidine deaminase (AID) [20]. SHM modifies the Ig variable region by inducing mutations, small deletions, or insertions to produce antibodies with increased affinity for the immunizing antigen. However, CSR is the process

by which the heavy chain class changes from IgM to IgG, IgA, or IgE. CSR occurs by DNA recombination within highly repetitive switch regions located at 5 ′ of each constant region [24]. After the germinal center reaction, the controlled steps in B-cell development can go awry, and lymphomas may develop. V (variable), D (diversity), and J (joining) V(D)J recombination, SHM, and CSR represent critical processes that might predispose to malignancies. For instance these are some of the translocations occurring during V(D)J recombination, t(14;18) and t(11;14). The t(14;18), which is detected in virtually all cases of follicular lymphoma and a fraction of diffuse large B-cell lymphoma (DLBCL) cases, involves the BCL2 gene and the IgH locus, leading to dysregulation of BCL2 [24].

Chromosomal translocations involving the BCL6 gene disorders found in 30%-40% of tumors stem from DNA sequence disorders in the promoter region. B-cells in the germinal center normally express the BCL6 gene on chromosome 3q27. Recently, the BCL6 gene's role in DLBCL pathogenesis was explained. Abnormal BCL6 protein expression causes cell proliferation and activation without differentiation. Since it increases cell survival and genetic instability, it promotes malignant transformations [23].

Clinical presentation

Oral lymphomas are difficult to diagnose because there are no specific clinical features. Most cases present with nonspecific clinical signs and symptoms such as local swelling, pain, and ulcer. The oral NHL may resemble benign, oral and dental pathologic lesions such as pyogenic granuloma, periodontal disease, osteomyelitis, and other malignancies [24]. The clinical presentation varies with their site of origin and tumor type. In the oral cavity, it commonly presents as an asymptomatic localized or diffuse soft tissue swelling with or without ulceration that may rapidly grow and may be accompanied by paresthesia and loosening of teeth [24].

The most common presenting symptoms of extranodal NHL in the head and neck region include local swelling, pain, and discomfort in the region of involvement. Clinical signs included unilateral enlargement of the face, localized swelling, destruction of hard and soft tissue, cavitation and ulceration, and/or sessile soft tissue mass.

Diagnosis and staging

Diagnosis of lymphoma includes a combination of physical examination, blood tests, diagnostic imaging, and selective biopsies [22]. Early diagnosis can allow the treatment of the disease in its early stages, providing better patient prognosis (Epstein et al, 2001). Diagnosis should be made on the basis of a surgical excision of the lymph node or extranodal tissue biopsy providing enough material for formalin-fixed samples. Patients requiring emergency treatment are advised to undergo a core biopsy diagnostic test, which involves a large needle with a cutting tip that is used during core needle biopsy to draw a column of tissue out of a suspicious area. Minimal immunohistochemistry (CD45, CD20, and CD3) is mandatory. The histological report should provide the diagnosis according to the current World Health Organization classification [19]. Immunohistochemistry is required to confirm the diagnosis. Some difficulties arise in the differential diagnosis of lymphoma clinically and histologically from other lesions. Lymphomas can mimic inflammation [24].

the chances of underdiagnosing (eg, periapical granuloma, or pyogenic granuloma or granulation tissue) or overdiagnosing (eg, intraosseous carcinoma, salivary gland carcinoma, or neurogenic tumors) are high. The need to differentiate from the other neoplasms or conditions of the oral cavity is necessary, as they differ in the treatment modalities. It is therefore important to diagnose accurately at the early stage, as it may result in either an aggressive treatment modality or simple excision. For diagnosis, ultrasonography in combination with Doppler flow or fine needle aspiration biopsy (FNAB) or open biopsy is useful. Along with these methods, various molecular methods can be used, such as polymerase chain reaction, fluorescence in situ hybridization, microarray, and special karyotyping.

The most sensitive technique for localizing EBV in latently infected cells is in situ hybridization to EBV-encoded RNA (EBER). Virtually all EBV-infected tumors express EBER 1 [21]. In comparison to other techniques, in situ hybridization is quicker and simpler. It also provides simultaneous morphological information. It allows the investigation of the role of EBV in neoplastic conditions of lymphoid and epithelial cells, and may prove valuable in determining the sites of latent virus in healthy subjects [21]. Once the diagnosis has been established, the first critical step is the pre-treatment evaluation and staging. This staging system represents the number of sites involved and their relation to the diaphragm, the presence of B symptoms (fevers >38◦C for at least three consecutive days, night sweats, body weight loss >10% during the 6 months prior to diagnosis), and the presence of extra nodal disease. A careful history and physical examination are the most important factors in the patient's evaluation. Physical examination includes evaluation of all lymph node enlargements, recording the site and size of all abnormal lymph nodes, inspection of Waldeyer's ring, evaluation of the presence or absence of hepatosplenomegaly, inspection of the skin, and detection of palpable masses. The presence or absence of B symptoms should be noted, and other symptoms may show specific sites of involvement. An assessment of performance status according to the Eastern Cooperative Oncology Group ECOG scale is important in all patients, and especially for those entering into clinical research trials. Laboratory studies that

should be routinely performed in NHL patients include a complete blood count to assess bone marrow reserves and a white blood cell differential with careful examination of the peripheral blood to look for the presence of circulating lymphoma cells. Serum chemistry should include an assessment of hepatic and renal function. Lactic dehydrogenase (LDH) is also an important indicator of tumour activity and is included in the International Prognostic Index [22].

A bone marrow aspirate and biopsy should be performed in all patients. Bilateral bone marrow biopsies have been recommended because they increase the sensitivity of detection of NHL involvement by 10–20%. However, an adequate (>2 cm) unilateral bone marrow specimen is generally sufficient [23]. Fluoro-deoxyglucose positron emission tomography (PET) is now a standard procedure both for staging and response assessment. Many studies showed that PET at the end of treatment is highly predictive of progression free survival (PFR) and overall survival (OS) in aggressive lymphomas with or without residual masses detected with CT scan. A PET scan can distinguish between lymphoma and necrosis or fibrosis in residual masses [25].

Treatment and management

Chemotherapy is the most common form of treatment given to the patients diagnosed with lymphoma. The 5-year survival rate of DLBCLs is 67% and the 10-year survival rate is 55% [24].

Chemotherapy with radiotherapy was the treatment of choice in 41 cases from the 15 studies describing the treatment of non-Hodgkin lymphomas located in head and neck region. For localized lymphoma, it was suggested that radiotherapy was an adequate treatment, while chemotherapy was preferable for patients with systemic complications. A retrospective study of 92 patients with intermediate- and high-grade lymphoma showed a good response to combined chemotherapy and radiotherapy [25]. For isolated lesions, surgical enucleation was possible even though typically surgery was combined with radiation or chemotherapy [26].

Conclusion

Lymphomas consist of malignant neoplastic mutations of normal lymphocytes, and they may occur in any region containing lymph tissue. Many factors may result in these mutations, such as infectious agents, for instance (Epstein-Barr virus, human immunodeficiency virus, Helicobacter pylori infection, hepatitis C virus infections). Host susceptibility factors can also contribute to these mutations.

Conflict of interest. Nil

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