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Oral Pathology

HIV AND T-CELL NON-HODGKIN'S LYMPHOMA: A RARE CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT Lymphomas are malignant neoplasms arising from the clonal proliferation of lymphoid cells, and are classified into Hodgkin's lymphoma (HL) and non-Hodgkin's lymphoma (NHL). NHLs predominate and may originate from B-cells, T-cells, or natural killer cells, with B-cell variants being more frequent. Extranodal T-cell NHL is rare, often seen in immunocompromised individuals, and may present as tumours or ulcerated lesions in the oral cavity. We report a rare case of T-cell NHL involving the palate in a 38-year-old HIV-positive (human immunodeficiency virus) patient. A definitive diagnosis was established by combining patient history, clinical and radiographic evaluation, histopathologic features and immunohistochemical (IHC) markers analysis, specifically CD45, CD20, and CD3. HIV/AIDS-associated NHLs spread rapidly and are a leading cause of death in HIV patients, making early detection crucial for improving survival and recovery.

KEYWORDS : Human Immunodeficiency Virus, Immunohistochemistry, Lymphomas, T-cell Non-hodgkin's Lymphoma

INTRODUCTION

Lymphomas, a heterogeneous group of lymphoid neoplasms, are malignancies originating from lymphocytes. The World Health Organisation (WHO) classifies them into Hodgkin's lymphoma (HL) and multiple distinct subtypes of non-Hodgkin's lymphoma (NHL) based on genetic and immunohistochemical criteria.[1] The NHL contributes to about 5% of head and neck malignancies. According to GLOBOCAN 2022, ~5.4 lakhs new cases and ~2.6 lakhs deaths were documented, with NHL ranking 10th among the most common cancers worldwide. Its development is influenced by multiple risk factors, with studies demonstrating strong associations with environmental exposures such as radiation, electromagnetic fields, solvents, etc.[2] Its incidence is notably higher in immunocompromised individuals, including those with HIV/AIDS, autoimmune diseases, and celiac disease.[3] Diagnosing NHL poses significant challenges, as it represents not a single disease but a diverse spectrum of diseases. Each subtype has distinct biological behaviour, requiring precise classification for appropriate treatment. Because of its diagnostic complexity, NHL is often associated with delayed diagnosis, classification discrepancies, and inadequate therapeutic intervention.[4]

We present a rare case of T-cell NHL in an HIV-positive patient, which may clinically simulate features of both benign and malignant neoplasms. Recognising NHL in the differential diagnosis is crucial, as reliance on histopathology alone is insufficient for definitive diagnosis. The application of immunohistochemistry (IHC) in this study highlights its indispensable role in accurately classifying NHL, given that therapeutic strategies and prognostic outcomes vary significantly across different subtypes.

Case History

A 38-year-old male presented with the chief complaint of pain and swelling in the left posterior palatal region for the past 15 days. The patient had been HIV-positive for the past 10 years and was receiving highly active antiretroviral therapy (HAART). On extraoral examination, diffuse swelling was observed over the left midface, corresponding with the intraoral findings. Intraorally, a solitary, reddish, ulceronodular growth was evident on the left palate, extending mediolaterally from the palatal gingiva i.r.t. 24, 25, 26, 27, and 28 up to the midline and anteroposteriorly from the mesial aspect of 24 to the distal aspect of 28 (Figure 1).

Palpation revealed a firm, non-tender, non-compressible swelling, accompanied by mobility of 25, 26, and 27 teeth. In addition, a mobile, non-tender left submandibular lymph node was palpable.

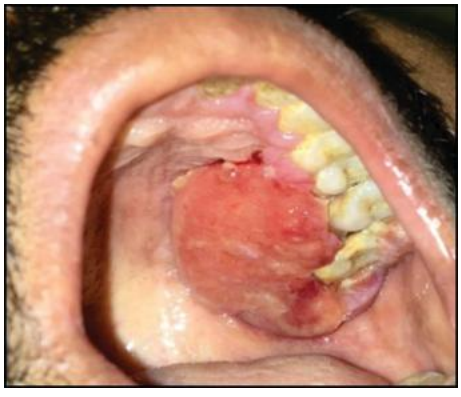

Figure 1: Ulceronodular Growth Seen on the Left Side of Palate Measuring About 5 × 4 cm in Size.

Radiographic evaluation with an orthopantomogram (OPG) revealed irregular horizontal bone loss from teeth 25 to 27, with complete alveolar bone loss around 26 and 27, producing a characteristic “floating tooth” appearance (Figure 2). Taken together, these clinical and radiographic features suggested a provisional diagnosis of carcinoma of the alveolus, prompting an incisional biopsy for histopathological evaluation.

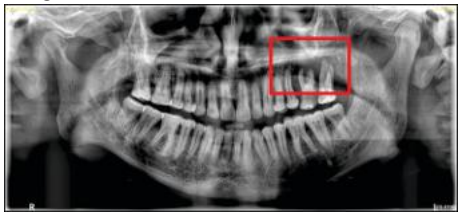

Figure 2: Orthopantomogram Reveals Complete Alveolar Bone Loss Around 26 and 27 Exhibiting Floating Tooth Appearance.

Histopathological examination of H&E-stained soft tissue sections revealed a highly cellular stroma comprised of sheets of uniform, monotonous round cells separated by thin connective tissue septa (Figure 3). These diffusely arranged cells displayed pleomorphic, hyperchromatic nuclei with a thin rim of cytoplasm. The intervening stroma demonstrated moderate vascularity, along with foci of extravasated red blood cells and chronic inflammatory infiltrates (Figure 4). Collectively, these features suggested a differential diagnosis of a round cell tumour.

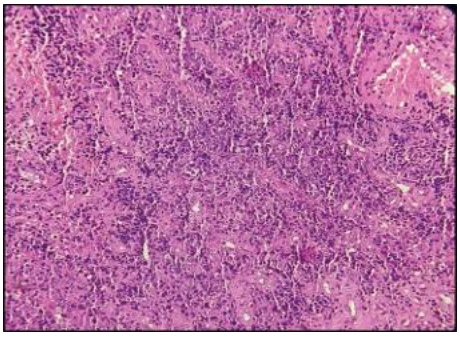

Figure 3: Highly Cellular Stroma with Sheets of Uniform, Monotonous Round Cells (H&E stain, 20x).

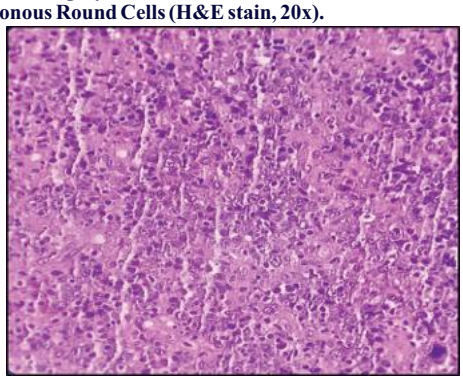

Figure 4: Diffusely Arranged Lesional Round Cells with Pleomorphic and Hyperchromatic Nuclei Along with a thin Rim of Cytoplasm (H&E stain, 40x).

To further characterise the lesion, immunohistochemical analysis was performed using a panel of markers (CD45, CD20, and CD3). The tumour cells expressed CD45 and CD3, with no CD20 immunoreactivity (Figure 5). These IHC findings supported the diagnosis of T-cell NHL.

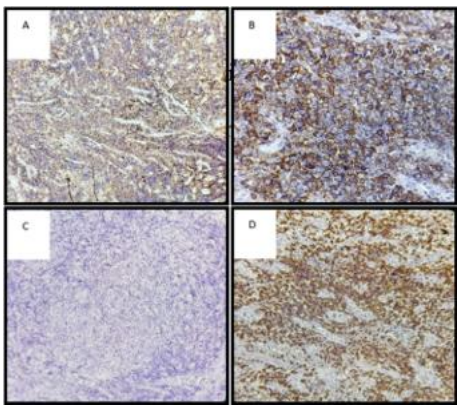

Figure 5: Photomicrographs of IHC Showing Positivity for A) CD45, B) Showing Cells with Cell Membrane Positivity (10x), C) Negativity for CD20 (10x) and D) Positivity for CD3 (20x).

After diagnosis, the patient was sent to the cancer institute for comprehensive multidisciplinary management. To assess disease extent, FDG PET/CT imaging was performed, revealing metabolically active lymphadenopathy in multiple regions spanning both diaphragmatic regions, along with an extralymphatic lesion in the left palatal region measuring approximately 5.6 cm (AP) × 4.2 cm (ML) × 4.6 cm (CC). According to the Lugano staging system, the disease was categorised as Stage III, indicating nodal involvement on either side of the diaphragm with extranodal extension.[5] Histopathological examination revealed diffusely arranged large, pleomorphic cells with marked mitotic activity. Coupled with its rapid growth and systemic involvement, these features are consistent with an aggressive, high-grade T-cell NHL subtype.[6]

Following staging, the patient underwent chemotherapy with the CHOP regimen, a standard combination of Cyclophosphamide, Doxorubicin, Vincristine, and Prednisone, administered at four-week intervals for approximately six months. At 18 months of follow-up, the patient had succumbed to the disease. This outcome highlights the aggressive clinical course and limited therapeutic success in advanced stages of NHL, particularly when complicated by patient-specific factors.

DISCUSSION

HIV/AIDS-associated lymphomas are haematological neoplasms that develop from T and B lymphocytes at various stages of differentiation. If the lymphomas are left untreated, they can lead to the death of an individual within weeks or months after diagnosis.[7] Extranodal involvement occurs much more frequently in NHL compared to HL.[8] It is regarded as the most common malignancy associated with HIV, and the risk is about 60 times greater than in otherwise healthy individuals.[3]

Immune dysfunction contributes to lymphomagenesis by impairing immunosurveillance, thereby facilitating the persistence of malignant mutations in lymphoid cells. Such mutations may develop under conditions of chronic antigenic stimulation or environmental influences, including the mutagenic effects of chemotherapeutic or immunosuppressive agents. Recent research suggests that HIV might contribute to cancer development not just by weakening the immune system but also through certain proteins it produces, like HIV Tat and specific variants of HIV p17, which have oncogenic functions.[9] The study by Asadi et al. (2023) provided evidence of an increased likelihood of developing NHL or multiple myeloma in individuals with lead exposure.[10]

The most common extranodal site for NHL in Western countries is the gastrointestinal tract (4-20% of all NHL cases), whereas in India, the most frequent site is the head and neck region, accounting for 50.9% of cases.[8] The male-to-female ratio ranges between 2.31:1.[11] T-cell NHLs constitute an uncommon subset, accounting for ~12% of all lymphomas and often presenting as extranodal disease.[12] These may present a tumour or an ulcerated lesion in the mouth, frequently involving the palate, gingiva, and tongue. The lesions grow rapidly and may lead to destruction of the jawbones.

NHLs are present in two histologic patterns: nodular and diffuse. In a nodular pattern, large clusters of neoplastic cells are present, typically of B-cell origin and generally associated with a favourable prognosis. The diffuse pattern shows a monotonous distribution of cells with no evidence of nodularity or germinal centre formation, which is similar to our case and can be of B-cell origin or T-cell origin.[13] On H&E sections, T-cell NHL often presents as diffuse sheets of atypical lymphoid cells that can mimic other "round cell tumours" such as diffuse large B-cell lymphoma, anaplastic large cell lymphoma, and small round blue cell tumours (such as Ewing sarcoma or rhabdomyosarcoma).[14] The differential diagnosis hinges on careful assessment of the size of the round cell and its growth pattern, but ultimately requires IHC to rule out mimics.

To determine the origin of a round cell tumour, a panel of IHC markers is employed. CD45 (leukocyte common antigen) is pivotal in confirming hematopoietic lineage, while cytokeratin (AE1/AE3) indicates epithelial origin, and vimentin supports mesenchymal differentiation. In our case, strong cell membrane positivity with CD45 confirmed hematopoietic origin, effectively excluding round cell tumours of epithelial and mesenchymal origin. T-cell lineage is confirmed by positivity for CD3, CD5, etc, though aberrant loss of some markers is common. Negative staining for B-cell markers (CD19, CD20), myeloid markers (MPO), or muscle markers (desmin, myogenin) helps exclude other round cell neoplasms. Thus, the combination of H&E morphology with a tailored IHC panel is essential for distinguishing T-cell NHL from its morphologic mimics and confirming a definitive diagnosis.[15]

The management of NHL varies depending on the histology, but it frequently involves chemotherapeutic agents such as CHOP. In

addition, Rituximab can also be given if it is a B-cell NHL. In addition to chemotherapy, other treatment modalities include radiotherapy, biologic therapy, and targeted therapy.[16] FDG-PET/CT is integral to lymphoma management, offering superior accuracy in staging, guiding therapy decisions, and refining radiation planning. It enables early response assessment, confirms remission or residual disease at treatment completion, and facilitates relapse detection and biopsy guidance, making it an important imaging modality in lymphomas.[17]

The prognosis of NHL varies based on several factors, including disease extent, clinical staging, histopathological subtype, and comorbid conditions such as HIV infection. In individuals with HIV, NHL generally carries an unfavourable prognosis due to its aggressive course and increased susceptibility to opportunistic infection.[18] Histologically, indolent NHLs typically display nodular/follicular morphology, presenting as chronic relapsing diseases, and, though associated with long survival, remain largely incurable at advanced stages (III–IV). In contrast, aggressive NHLs exhibit diffuse morphology, progress rapidly, and, despite their shorter natural history, can often be managed with intensive chemotherapy regimens administered with curative intent.[13]

Early diagnosis of NHL is crucial because it directly influences prognosis and survival rates. Detecting the disease at an early stage allows for timely intervention, often with less intensive therapy, and improves the likelihood of long-term remission or cure in aggressive subtypes.

CONCLUSION

T-cell NHL involving the oral cavity is uncommon but should be carefully distinguished from other intraoral malignancies during diagnostic evaluation. IHC is the cornerstone of early, accurate diagnosis, providing the precision needed to classify lineage, subtype, and biological behaviour for appropriate therapy. Dentists are often the first to detect suspicious oral lesions, and their vigilance in screening, with timely oncology referral, is crucial for improving patient outcomes.

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