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Case Report

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Diffuse Large B Cell Lymphoma of Hard Palate-A Rare Case Entity with Literature Review

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ABSTRACT

Introduction: The most common Non-Hodgkin's Lymphoma in the oral cavity is Diffuse Large B Cell Lymphoma (DLBCL), followed by mantle cell lymphoma, extranodal marginal zone lymphoma of Mucosa-Associated Lymphoid Tissue (MALT lymphoma) and Burkitt lymphoma. The literature shows a very few case reports of primary MALT lymphoma. Primary involvement of the oral cavity by NHL is very rare. Extranodal DLBCL of the palate are extremely rare, with isolated case reports in the literature.

Case Report: Here we are discussing about a case of an 84-year-old male known case of Hepatitis B infection (since 2014) and a chronic tobacco chewer for the last 9 years presented with a complaint of toothache (bilateral lower molar) of 06 months duration and a non-healing ulcer on the hard palate of 04 months duration. Patient underwent an incisional biopsy and was diagnosed with extra-nodal diffuse large B-cell lymphoma, GCB type.

Conclusion: DLBCL have several subtypes with different clinical courses. Identification of these subtypes has prognostic and therapeutic significance. They are aggressive tumours that can be rapidly fatal if left untreated. So early diagnosis plays an important role. Complete remission can be achieved with early diagnosis and intensive chemotherapy.

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Introduction

The lymphomas can be broadly grouped into Hodgkin's and non-Hodgkin's based on the morphology of tumour cells. Non-Hodgkin Lymphomas (NHL) are more common than Hodgkin lymphoma in the head and neck region [1]. The most common NHL in the oral cavity is Diffuse Large B Cell Lymphoma (DLBCL), followed by mantle cell lymphoma, extranodal marginal zone lymphoma of Mucosa-Associated Lymphoid Tissue (MALT lymphoma) and Burkitt lymphoma [2-4]. DLBCL includes several forms of lymphoma with certain features like B cell phenotype, diffuse growth pattern and aggressive clinical history. It commonly affects old age, the sixth to seventh decade of life and presents as a rapidly enlarging mass at a single nodal or extranodal site [5]. Primary involvement of the oral cavity by NHL is very rare and constitutes approximately 3–5% of all NHL [2-3]. The common extranodal sites of NHLs in the head and neck region are Waldeyer's ring, palatine tonsil, salivary glands, base of tongue and oropharynx [2]. Extranodal DLBCL of the palate are extremely rare, with isolated case reports in the literature.

The extranodal MALT lymphoma is a low-grade B-cell NHL that arises from the B lymphoid cells located in the marginal zone of secondary lymphoid follicles. The literature shows a very few case reports of primary MALT lymphoma [6]. As oral lymphomas in early stages can mimic more common inflammatory or reactive disease processes so these can easily be misdiagnosed.

Case Report

Our patient 84 years old male known case of Hepatitis B infection (since 2014) and chronic tobacco chewer for last 9 years presented with complaint of toothache (bilateral lower molar) for 06 months and a non-healing ulcer on hard palate for 04 months which was insidious in onset, associated with pain and had gradually progressed from few millimeters to wide visible defect for 01week duration before presenting at our center [Fig 1].

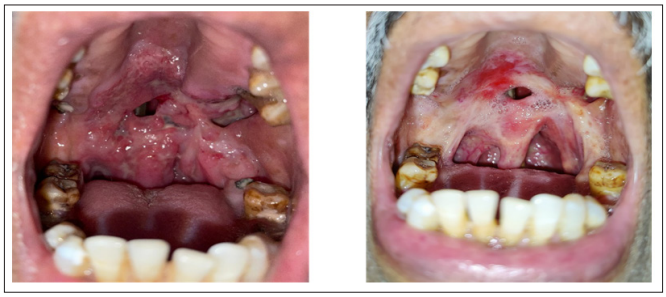

Figure 1: Pre and Post Chemotherapy

He also complained of nasal regurgitation and dysphagia for 01 week. Patient did not complain of any dyspnea, neck swelling or voice change. There was no history of hemoptysis, hematemesis, hematochezia or epistaxis.

On examination, the Patient was partially edentulous with poor dental hygiene. A single defect was present in the hard palate and soft palate, crossing midline, extending to (L) RMT, (L) Anterior tonsillar pillar & uvula. No trismus or ankyloglossia. On fibre-optic laryngoscopy - B/L TVC were mobile & Hypopharynx & endolarynx were normal. There was no visible lesion in the nose or nasopharynx.

MRI of face & neck revealed ill-defined heterogeneously enhancing lesion in post 1/3rd of hard palate left side and adjacent soft palate, involving gingivobuccal sulcus extending into left retromolar trigone and pterygomandibular raphe, extending posterolaterally into masticator space infiltrating pterygoids and medially extending into adjacent pharyngeal wall, also involving torus tubarius and fossa of rosenmuller on left, posteriorly involving prevertebral muscles on left and inferomedially involving left glossoepiglottic fold palatoglossal fold & left tonsillar fossa.

Biopsies from the hard palate was done at another hospital before presenting to our center - 1st biopsy showed granulation tissue with no evidence of malignancy & 2nd biopsy report was suggestive of a granulomatous lesion.

Based on the presentation and clinical examination, the following were considered as differential diagnoses: 1. Necrotising sialometaplasia, 2. Infections (tuberculosis, histoplasmosis), 3. Salivary gland malignancies (mucoepidermoid carcinoma, adenoid cystic carcinoma), 4. Squamous cell carcinoma, 5. Connective tissue malignancies.

As biopsy reports were not satisfactory with clinical presentation and progression of disease, an incisional biopsy was again carried out from both the hard and soft palate.

On histopathological examination, [Figure 2] there was dense infiltration of the ulcer bed by lymphoid cells set against a background of cytoplasmic debris. The lymphoid cells were predominantly intermediate-sized, had irregularly cleaved nuclear margins, coarse chromatin & scant cytoplasm. There was associated sparse accompanying mature lymphocytes, histiocytes, a few plasma cells and prominent vascular proliferation.

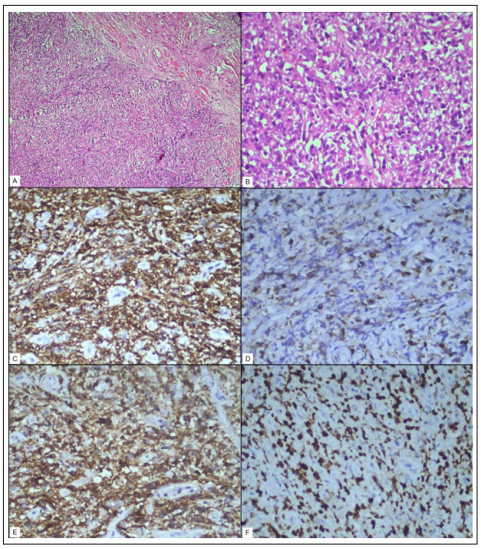

Figure 2: HPE Images

- A: Hematoxylin and Eosin stain (Original magnification 40x) Representative image from the hard palate lesion biopsy shows diffuse infiltration by lymphoid cells
- B: Hematoxylin and Eosin stain (Original magnification 400x) The lymphoid cells are composed of intermediate to large-sized cells having open chromatin, conspicuous nucleoli and scant cytoplasm.
- C: Immunohistochemistry with CD20 (Clone L26, RTU, Pathnsitu) (Original magnification 400x) highlight the predominant lymphoid cell population
- D: Immunohistochemistry with CD3 (Clone EP41, RTU, Pathnsitu) (Original magnification 400x) highlights few scattered background mature lymphocytes
- E: Immunohistochemistry with CD10 (Clone MMEP, RTU, Pathnsitu) (Original magnification 400x) also highlight the predominant lymphoid cell population
- F: Immunohistochemistry with Ki67 (Clone MIB1, RTU, Pathnsitu) (Original magnification 400x) highlights the brisk proliferative index of the cells

Morphological features were suggestive of NHL with possibilities of either Extranodal NK/T cell lymphoma or MALT. Points in favour of Extranodal NK/T cell lymphoma were – immunopositivity for CD3, CD7, and CD8 with CD5 negativity. Points against this diagnosis and favouring MALT were – Diffuse CD20, CD79a and CD43 positivity. But the point against MALT was the Ki67 index of 40-50%. Further IHC workup was carried out, which showed the predominant lymphoid cell population of intermediate to large cells to be CD20(membranous -85%) positive B lymphocytes, which were also seen to express CD10(membranous) and CD34(aberrant, dim and membranous) while being negative for MUM1. Hence, a diagnosis of Extra-nodal diffuse large B-cell lymphoma, GCB type was made.

Bone marrow aspirate and biopsy were negative for bone marrow involvement by hematolymphoid malignancy.

A tumour board was held, and the patient was further planned for chemotherapy. Patient received chemotherapy (CHOP regimen) Inj Cyclophosphamide 250 mg for 3 days with Doxorubicin, vincristine & prednisolone. The patient was again evaluated after 3 months post-chemo and showed regression of disease with drastic improvement in clinical symptoms.

Discussion

DLBCL is a malignant proliferation of large B cells with very heterogeneous morphology, phenotype and molecular and clinical findings. They are aggressive tumours that can be rapidly fatal if left untreated. So early diagnosis plays an important role. Complete remission can be achieved with early diagnosis and intensive chemotherapy.

Extra-nodal B cell lymphomas of the palate are exceptionally rare presentations with isolated case reports and are known to transform from MALT. A few of the reported cases are mentioned in the table below [Table 1].

References	Age/Gender	Clinical features	Imaging	HPE	IHC	Diagnosis	Treatment	Follow- Up
Janardan M et al [8]	32/F	Chronic Peridontal abscess, forming ulcer over 2 weeks	CBCT - Hyperdense mass in left maxillary sinus extending into oral cavity perforating the palatal floor	Sheets of large to medium sized atypical round cells in deeper connective tissue	Positive for CD45, CD20. MUM1 positive	DLBCL of maxillary sinus	Chemotherapy	Regression in size of tumor over period of 3 months
Sahoo et al. [9]	76/M	Single well circumscribed Swelling in anterior hard palate	CT maxilla showed a space occupying hypodense lesion in anterior maxilla with perforation and extension into maxillary antrum	Diffuse proliferation -large atypical lymphoid cells with high N/C ratio, coarse chromatin & prominent nucleoli	CD20 and CD45 positive	Primary DLBCL of Anterior palate	Chemotherapy	Swelling resolved completely without evidence of recurrence for 18 months
Kaur et al. [7]	57/M	Swelling in hard palate for 2 years	MRI : 3.8x3.6x3.3 cm soft tissue submucosal mass in left maxilla involving pterygoid fossa extending into infratemporal fossa	Monocytic lymphoid cells admixed with scattered large cells	Positive for CD20, Bcl2, MNDA (focal), CD21 highlighted residual germinal centre	Extranodal MALT lymphoma of hard palate	EBRT to left infratemporal fossa - Dose 36 Gy over 1 month	Complete remission without recurrence for 1 year
Abeles et al [10]	49/M	Nasal congestion, purulent rhinorrhea, progressive difficulty breathing for 4 months	CT : hyperdense left nasal sidewall mass extending into maxillary alveolus & right nasal floor lytic lesion	Diffuse atypical lymphoid infiltrate with predominantly small and medium sized lymphocytes	Positive for CD20, Bcl-2, Bcl-6 and negative for CD5, CD10 & MUM1	DLBCL of nasal bone, maxilla and hard palate	Chemotherapy (R-CHOP) following surgical resection	No recurrence in 18 months follow up
Souto et al [11]	85/F	Ulcer on palate of 2 months duration		Proliferation of large lymphoid cells with high nucleus: cytoplasm ratio, coarse chromatin and inconspicous nucleoli with abnormal mitotic figures	Positive for anti-CD20, CD79a, CD10, MUM-1, Bcl-6, Bcl-2. Negative for anti-CD3, CD5 and CD138	DLBCL-NOS of hard palate	Chemotherapy (R-CHOP) followed by RT	Complete resolution of disease with disease free period in 31 months follow up
Lancet Oncol 2018 [12]	69/F	Bleeding & fetid tumor on left side of palate for 6 months	CT : Bone destruction involving palate		CD20+ , CD30+ , EBV+, MUM1+ , CD10-, Bcl6-, CD15-, Ki67 45%	Ebstein-Barr virus positive B-cell lymphoma, Burkitt lymphoma	Radiotherapy & Chemotherapy (CHOP) - 8 cycles in 3 weeks	Remmission over period of 10 months follow up
J Exp Ther Oncol. 2017 [13]	40/F	Swelling on right side of face since 45 days and gradually increased in size. Intra-orally an ulcero-proliferative growth on the right side of hard palate		Lymphoproliferative disorder	Positive for CD45, PAX5, Bcl2, Ki67, CD138 and negative for CD3, CD10, CD20 and CD30	DLBCL of hard palate		

Microscopic diagnosis of such lea lesion is also challenging, as inflammation is a common occurrence in oral biopsies, and it might be difficult to differentiate reactive lymphocytic proliferation from neoplastic proliferation.

DLBCL have several subtypes with different clinical courses. Identification of these subtypes has prognostic and therapeutic significance. The importance of classifying DLBCL based on cell of origin as activated B cell (ABC) or germinal centre B cell (GCB) is widely accepted based on differences in clinical outcome and gene expression.

Conclusion

DLBCLs should always be considered as a differential diagnosis for rapidly progressing solitary ulcers of the palate. Immunomarkers might be of great help in such situations, and stratification of DLBCL by IHC is an effective method of delineating the subtypes [7-13].

Declarations

- The authors have no relevant financial or non-financial interests to disclose.
- The authors have no competing interests to declare that are relevant to the content of this article.
- All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest or non-financial interest in the subject matter or materials discussed in this manuscript.
- The authors have no financial or proprietary interests in any material discussed in this article.
- No identification of any patients was done in the paper. Appropriate sections covered to hide identity.
- Consent from the patient was taken.
- Having done for an emergency, prior sanction not taken, and retrospective waiver taken in view of a life-saving procedure
- Data is available for the journal's perusal

Ethical Statement:

- All the authors mentioned have contributed to the paper
- No funding has been received for the above study
- This paper is in accordance with the 1964 Helsinki Declaration for compliance with ethical standards
- There is no conflict of interest for the authors' contributions.
- No identity of the patients has been revealed while writing the paper.

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