Case Report

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Squamous Cell Carcinoma of Tongue in a Patient with Immune Thrombocytopenia: A Case Report

Oncology Section

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ABSTRACT

Oral Tongue Squamous Cell Carcinoma (OTSCC) is among the most aggressive forms of oral cancer, particularly prevalent in South Asian populations. Immune Thrombocytopenia (ITP), an autoimmune haematological condition that destroys peripheral platelets via antibody, rarely occurs with solid epithelial tumours. The simultaneous occurrence of OTSCC and ITP creates diagnostic and therapeutic challenges. Hereby, the authors present a case report of a 45-year-old Indian male with a six-month-old chronic, non healing ulcer on the left lateral border of the tongue. A 4.7×1.7×3.2 cm ulcer proliferative lesion with nodal spread was seen on Magnetic Resonance Imaging (MRI), while histological investigation revealed a well-differentiated squamous cell carcinoma. Results showed severe thrombocytopenia (38,000/mm³) and significant megakaryocytic activity in the bone marrow, supporting immune-mediated platelet degradation. A combination of high dose corticosteroids, rituximab, and Intravenous Immunoglobulin (IVIg) led to haematological restitution with platelet counts reaching 300,000/mm³, allowing for safe chemoradiotherapy. After follow-up, the patient showed partial tumour shrinkage and good tolerance without haemorrhagic sequelae. This remarkable concurrence highlights the importance of early detection, careful exclusion of mimic reasons, and a precisely coordinated multidisciplinary approach that relies on haematologic stabilisation for curative oncological therapy.

Keywords: Autoimmune haematological disorder, Corticosteroids ,Oncological therapy

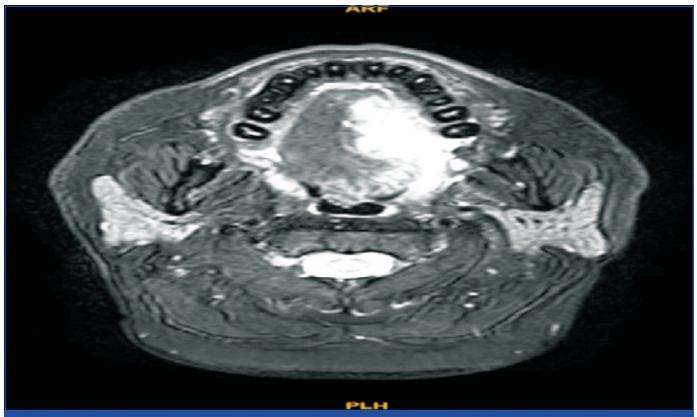
CASE REPORT

A 45-year-old Indian male, a chronic tobacco chewer with no other co-morbidities, arrived with a six-month history of a non healing ulcer on the left lateral edge of the tongue. Patient is a chronic tobacco chewer since 18 years. He used to take 5-6 gutkha packets per day. An intraoral examination revealed an indurated ulcer of 2×1 cm in size, accompanied by restricted tongue mobility; however, no spontaneous bleeding or petechiae were observed. During Bilateral neck examination no cervical lymph nodes were palpabale. The systemic examination showed no signs of hepatosplenomegaly, lymphadenopathy, or cutaneous ecchymoses.

A contrast-enchanced MRI of the head and neck was subsequently performed using gadopentetate dimeglumine (10 mL Magnavist, intravenous), with axial and sagittal acquisitions; axial sections were primarily utilised for assessing tumour depth of invasion and local spread. The MRI protocol incorporated standard head and neck sequences, including:

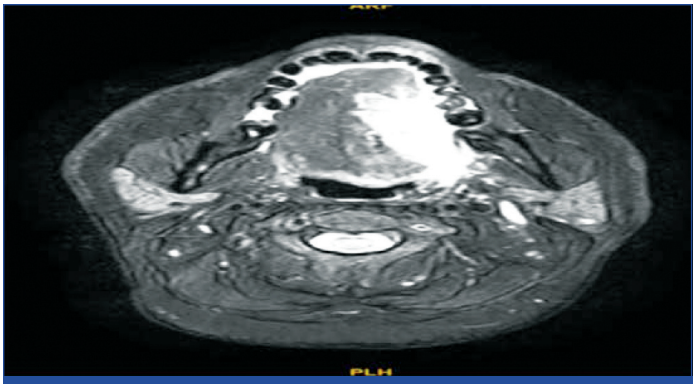
- T1-weighted Imaging (T1WI) for detailed anatomical depiction, with bright fat and dark Cerebrospinal Fluild (CSF);
- T2-weighted Imaging (T2WI) for fluid-sensitive contrast, highlighting oedema and lesion- CSF interfaces;
- Diffusion-Weighted Imaging (DWI) to identify areas of restricted diffusion typical of high-cellularity tumours;
- Short-Tau Inversion Recovery (STIR) for fat suppression and clearer visualisation of oedema; and
- Contrast-enhanced T1-weighted imaging (CE-T1WI) to delineate enhancing components of the lesion following contrast administration.

The MRI demonstrated a heterogeneously enhancing ulcer-proliferative lesion measuring 4.7×1.7×3.2 cm, involving the anterior two-thirds and posterior one-third of the left-side of the tongue, with infiltration into the intrinsic tongue musculature, adjacent salivary gland tissue, and surrounding cartilaginous fibers [Table/Fig-1]. The lesion showed posterior extension toward the oropharynx as well as lateral involvement of the mandibular alveolar region [Table/Fig-2]. Multiple necrotic lymph nodes were noted at levels IIB and III, along with several sub-centimetric bilateral cervical lymph nodes. High-resolution Computed Tomography (CT) of the thorax showed no evidence of pulmonary metastasis.



[Table/Fig-1]: Axial CE-MRI image showing a heterogeneously enhancing ulcero-proliferative lesion involving the left lateral border of the tongue, with invasion of adjacent intrinsic tongue muscles. The lesion appears hyperintense on T2-weighted/STIR imaging.

In the evaluation of a persistent non healing ulcerative lesion of the tongue, a broad differential diagnosis was systematically considered to ensure thorough clinical assessment. Common aetiologies included traumatic ulceration secondary to sharp dental margins or ill-fitting prostheses, aphthous ulceration presenting as recurrent painful mucosal breaks, and oral squamous cell carcinoma, the most prevalent malignant neoplasm of the tongue, typically manifesting as a slowly enlarging lesion that may initially be asymptomatic before progressing to pain. Additional routinely encountered conditions such as oral lichen planus, a chronic immune-mediated inflammatory disorder, and primary or recurrent herpes simplex infection were also evaluated. Less frequent but clinically relevant considerations comprised syphilitic chancre of primary syphilis, tuberculous involvement of the oral cavity, opportunistic fungal



[Table/Fig-2]: Axial CE-MRI image at a slightly lower section showing posterior extension of the left-sided tongue lesion toward the oropharynx and adjacent musculature. Mild asymmetry and involvement of deep tongue structures were evident.

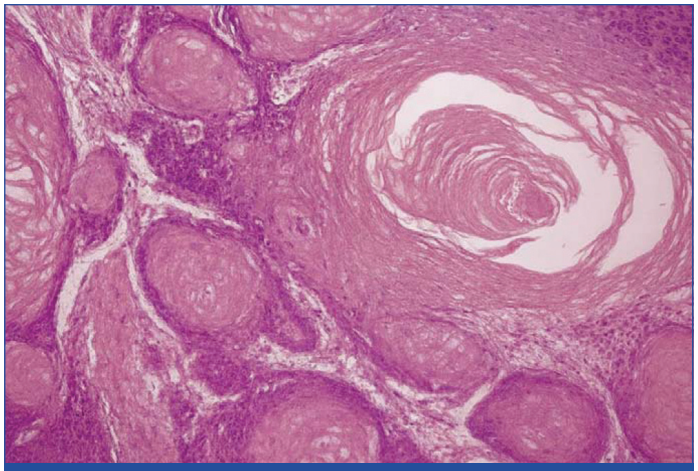
infections including candidiasis, autoimmune blistering disorders such as pemphigus vulgaris, and nutritional deficiencies particularly of vitamin B12 and iron which are known to predispose to chronic mucosal ulceration. Based on the lesion's clinical progression and supportive radiological findings, a provisional diagnosis of malignancy of the oral tongue was established.

Cytology was not undertaken, as exfoliative cytology provides limited diagnostic accuracy in non healing tongue ulcers due to inadequate sampling and inability to assess depth of invasion. In accordance with National Comprehensive Cancer Network (NCCN) guidelines [1], an incisional biopsy was performed to obtain sufficient tissue for definitive histopathological diagnosis. The clinical photograph of a non healing ulcerative lesion on the lateral aspect of the tongue with surrounding erythema and induration. Sutures from the incisional biopsy are visible at the center of the lesion [Table/Fig-3].

[Table/Fig-3]: Non healing ulcerative lesion on the lateral aspect of the tongue with surrounding erythema and induration.

Histopathological analysis confirmed well-differentiated squamous cell carcinoma of the tongue [Table/Fig-4]. As per clinical 8th American Joint Committee on Cancer (AJCC) Tumor, Node, Metastasis (TNM) staging of head and neck cancer, patient classifies as cT3N2cM0, Group Stage IVA [2,3].

The above photomicrograph demonstrates sections showing tumour arranged in islands and nests, a pattern characteristic of epithelial neoplasms. The tumour cells exhibit abundant eosinophilic cytoplasm, vesicular nuclei, and prominent nucleoli, features indicative of active cellular proliferation. The presence of keratin pearls confirms squamous differentiation, which is a hallmark of well-differentiated squamous cell carcinoma. These histopathological {Haematoxylin and Eosin (H&E)} findings collectively support the diagnosis by showing both architectural (islands/nests) and cytological (keratinisation, nuclear features) evidence of squamous malignancy.



[Table/Fig-4]: Tumour arranged in islands and nests. Tumour cells have abundant eosinophilic cytoplasm, vesicular nuclei and prominent nucleoli. Keratin pearls are seen. (H&E, 40x).

Routine haematological evaluation, including complete blood counts, liver and renal function tests, and coagulation profile, was performed and found to be within normal limits except for marked thrombocytopenia, with platelet counts of 38,000/mm^3 confirmed on serial assessments [Table/Fig-5]. Peripheral smear demonstrated normocytic, normochromic red cells with significantly reduced platelets and no blasts or schistocytes, while bone marrow examination showed intact megakaryocytes, supporting a diagnosis of secondary ITP.

The patient was administered intravenous methylprednisolone (250 mg daily for 3 days), followed by oral prednisolone (1 mg/kg/day), leading to a small increase in platelet count to 65,000/mm^3. Due to ongoing instability, rituximab (375 mg/m^2 weekly for 4 doses) was commenced but resulted in a mediocre response (60,000/mm^3). The subsequent injection of IVIG (1 g/kg for 2 days) resulted in a significant increase in platelet counts to 300,000/mm^3, enabling oncological intervention [Table/Fig-5].

The multidisciplinary tumour board was done post recovery of platelets. Patient was advised concurrent chemoradiotherapy as the ultimate therapeutic approach considering the local disease load and stable haematologic condition.

Oncological Treatment Summary:

The patient received concurrent chemoradiotherapy consisting of:

- **Chemotherapy agent:** I njection Cisplatin
- **Dose:** 40 mg/m^2
- **Schedule:** Administered intravenously once weekly during the course of radiotherapy
- **Radiotherapy dose:** 70 Gy in 35 fractions over seven weeks using Intensity Modulated Rasiotherpay (IMRT) technique.

At weekly follow-up during chemoradiotherapy, patients' blood routine investigations were monitored including complete blood counts (Haemoglobin, total leucocyte counts with differential and platelet counts). They were found to be within normal limits during treatment.

The patient survived treatment without haemorrhagic complications. Nutritional rehabilitation and speech therapy commenced. Patient was being called for monthly follow-up for three months post commencement of chemoradiotherapy as per NCCN Guidelines. At one month of his follow-up, his blood counts showed thrombocytopenia and rest all parameters were normal. He was kept on oral steroids to maintain his platelet counts. During the most recent follow-up at three months, the patient continues to be clinically stable andresponse evaluation imaging revealed partial reduction of the tongue lesion and nodal condition. He is under integrated oncological and haematological monitoring.

Parameters	At diagnosis	Post- IVIG treatment	1-month Post-chemoradiation	Reference range
Haemoglobin (g/dL)	10.2	11.4	12.1	12-16
Total leucocyte count (×10^9/L)	6.8	7.2	6.5	4-11
Neutrophils (%)	68	65	62	40-75
Lymphocytes (%)	26	28	30	20-40
Platelet count (×10^9/L)	38	30	88	150-400
Peripheral smear	Thrombocytopenia; no atypia	Improved platelet count	Thrombocytopenia	—
AST (U/L)	28	30	27	5-40
ALT (U/L)	32	34	30	5-45
ALP (U/L)	88	92	85	40-120
Total bilirubin (mg/dL)	0.9	0.8	0.7	0.2-1.2
Serum creatinine (mg/dL)	0.8	0.9	0.8	0.6-1.3
BUN (mg/dL)	14	16	15	7-20
PT (seconds)	13.1	13.4	13.0	11-15
INR	1.02	1.05	1.01	0.9-1.2
LDH (U/L)	180	170	165	140-280
HIV, HBV, HCV	Negative	—	—	—

[Table/Fig-5]: Laboratory parameters.
AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline phosphatase; BUN: Blood urea nitrogen; PT:Prothrombin time; INR: International normalised ratio; HIV: Human Immunodeficiency virus; HBV: Hepatitis B virus; HCV: Hepatitis C virus; LDH: Lactate dehydrogenase

The trend of platelet counts over time in the patient diagnosed with OTSCC and concurrent ITP. For most of the recorded period, the platelet counts remain persistently low, consistent with chronic thrombocytopenia due to ITP has been depicted in [Table/Fig-5]. A sharp transient spike is observed around April 2025, which likely corresponds to therapeutic intervention most commonly steroid therapy, IVIG, or platelet transfusion administered to optimise the platelet count before a diagnostic or therapeutic procedure. Following this temporary rise, the platelet levels gradually decline back to patient's baseline thrombocytopenic range. This pattern reflects the typical clinical course of ITP, where platelet counts shows fluctuating levels and may exhibit short-lived responses to treatment. The graph demonstrates the challenges in maintaining stable platelet counts in such patients, highlighting the importance of close monitoring throughout cancer evaluation and treatment.

DISCUSSION

Oral cancers remain a major oncological challenge in South Asia, with OTSCC identified as one of the most aggressive subtypes. Its biological behaviour is driven by early regional metastasis, extensive local invasion, and a high potential for functional morbidity involving speech and deglutition [4,5]. Epidemiological analyses from India have highlighted a sustained rise in oral cancer burden over recent decades, attributed primarily to persistent exposure to carcinogenic risk factors such as tobacco chewing, alcohol use, and poor oral hygiene practices [4,5].

The coexistence of OTSCC with ITP represents an exceedingly rare clinical scenario. ITP, as defined by the American Society of Haematology (ASH), is an autoimmune condition characterised by accelerated platelet destruction and impaired megakaryocyte maturation, often confirmed through bone marrow examination and exclusion of secondary causes [6]. In the present case, the diagnosis of immune-mediated thrombocytopenia was established following the demonstration of preserved megakaryopoiesis and absence of marrow infiltration- criteria aligning precisely with the ASH 2019 guidelines [6].

The interplay between ITP and solid tumours has been scarcely documented in the literature. Ekstrand C et al., first described this association in a series of patients with breast carcinoma, suggesting immune dysregulation secondary to neoplastic antigenic stimulation as a plausible mechanism [7]. Subsequent literature by Martin DN et al., expanded on this association, systematically reviewing cases of ITP linked to various solid tumours and emphasising its rarity and diagnostic complexity [8]. However, reports linking ITP with squamous malignancies of the oral cavity remain virtually absent.

In the context of head and neck neoplasms, the co-existence of ITP has only recently been brought to clinical attention. Enrique B et al., presented a case of paraneoplastic thrombocytopenic purpura secondary to pulmonary squamous cell carcinoma, underscoring the potential for immune-mediated cytopenias to emerge as paraneoplastic manifestations [9]. Similarly, Ghosh A et al., reported one of the first documented instances of ITP secondary to carcinoma of the tongue, in which profound thrombocytopenia complicated oncological management and required multimodal immunotherapy [10]. The findings of the present case closely parallel these recent reports, particularly in the diagnostic and therapeutic challenges encountered.

From a therapeutic perspective, corticosteroids remain the cornerstone of initial ITP management, as reinforced by the ASH guidelines [6]. However, the incomplete haematologic response in steroid-refractory cases necessitates adjunctive immunomodulatory therapy. Rituximab, a monoclonal antibody targeting CD20-positive B cells, has emerged as an effective option in this subset, with Dong W et al., and Cooper N and Newland AC documenting durable platelet recovery in otherwise resistant ITP cases [11,12]. In concordance with these findings, partial response to corticosteroids in the present case mandated escalation to rituximab, followed by IVIG, which elicited a robust haematologic rebound.

The observed clinical response to IVIG is consistent with the mechanistic understanding proposed by Provan D and Newland AC, who noted that IVIG achieves rapid platelet increment through Fc receptor saturation and inhibition of autoantibody-mediated platelet clearance [13]. The resulting rise in platelet counts to 300,000/mm^3 provided a safe therapeutic window for initiating definitive oncologic therapy. Once haematologic stability was achieved, concurrent chemoradiotherapy was undertaken without haemorrhagic complications, in line with current recommendations that stress timely oncologic intervention following haematologic stabilisation [6,12].

The present case thereby reinforces several pivotal principles: 1) the necessity of meticulous diagnostic exclusion to confirm immune-mediated thrombocytopenia; 2) the value of sequential, multimodal immunotherapy tailored to the severity and refractoriness of thrombocytopenia; and 3) the critical importance of multidisciplinary

collaboration between haematology and oncology teams to ensure both haematologic and oncologic objectives are achieved. The rarity of reported cases- particularly in the context of OTSCC- underscores the need for further documentation to refine management algorithms and enhance understanding of this complex clinical interplay.

CONCLUSION(S)

The present case represents an extremely unusual occurrence of OTSCC with immunological thrombocytopenia, a dual disease that complicates diagnosis and treatment. Rapid detection of immune-mediated thrombocytopenia, along with careful haematologic stabilisation, established the necessary conditions for starting comprehensive oncologic treatment. The present paper highlights the necessity of a multidisciplinary approach, where the integration of haematologic and oncologic methods is crucial for achieving favourable clinical results in difficult cases.

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