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Case Report

Dentistry Section

Chronic Inflammatory Tongue Lesion in an Elderly Immunocompromised Female with Familial Predisposition: A Case Report

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ABSTRACT

Tongue lesions are often quite difficult to diagnose. This is especially true in older adults and those with weakened immune systems because such lesions can be even more difficult and can even seem to be premalignant and/or infectious lesions. They can be caused by immune and/or nutritional dysregulation, as well as by more systemic illnesses. This report describes a 71-year-old female with Chronic Lymphocytic Leukaemia (CLL) and autoimmune arthritis who presented with a persistent, painful ulcerative lesion on the mid-dorsal surface of the tongue. The lesion had been present for six months and was associated with occasional spontaneous bleeding. Comprehensive clinical, haematologic, and radiologic evaluation ruled out fungal infection and malignancy. Histopathology revealed epithelial atrophy with subepithelial inflammatory infiltrate, consistent with a benign chronic inflammatory process. Also, remarkable was the family background of this patient, as her mother suffered an undiagnosed condition for many years and her brother had a fissured tongue, or a tongue with deep grooves, but had no symptoms of any disease, which could point to a possible hereditary condition in that family. The patient was diagnosed and managed conservatively with a mouthwash with benzydamine, mucopain, and vitamin A and multivitamin supplements, which improved the ulcer by 90% in the eight weeks and complete symptom resolution on follow-up at 12 weeks. This case underscores the importance of a systematic, multidisciplinary approach in evaluating chronic tongue lesions in immunocompromised individuals. Early recognition of benign inflammatory patterns can prevent unnecessary invasive investigations and patient anxiety. The co-existence of autoimmune and haematologic disorders, along with familial occurrence, further highlights the multifactorial nature of such lesions and their relevance in oral medicine practice.

Keywords: Autoimmune diseases, Chronic disease, Oral mucosa, Oral ulcer, Vitamin A

CASE REPORT

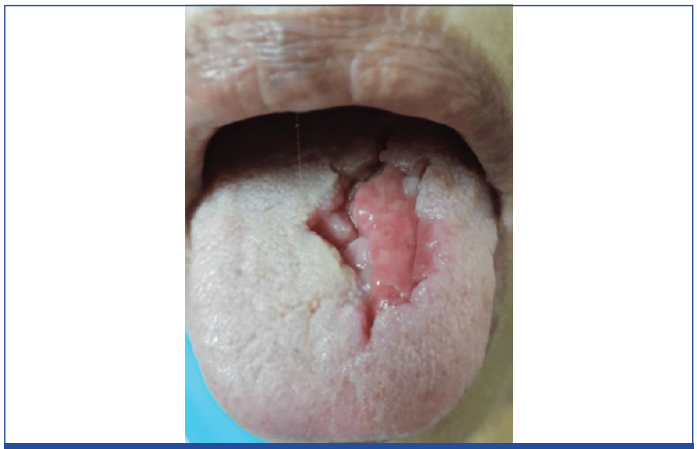
A 71-year-old woman consulted the oral medicine department for a painful ulcer-like lesion of the mid-dorsal surface of the tongue that had been present for approximately six months. The lesion had started as a shallow, midline fissure; however, it went deeper and widened and had burning sensations and periodic bleeding on its own without being triggered by eating or brushing. The ulcer was painful, especially when consuming spicy foods. She denied fever, dysphagia, weight loss, or general malaise.

Medical history: The patient had Chronic Lymphocytic Leukaemia (CLL) for seven years and autoimmune arthritis for 10 years. Both conditions were being followed, but she was not taking any medications. She also reported no tobacco, alcohol, or betel-nut use.

Family and dental history: Patient's mother had a similar undiagnosed condition of the tongue and her brother had an asymptomatic fissured tongue, indicating possible genetic involvement. No dental history was noted.

Systemic history: There was no history of diabetes mellitus, gastroesophageal reflux disease, Crohn's disease, or burning mouth syndrome. The patient denies any gastrointestinal symptoms or symptoms of nutritional malabsorption.

Intraoral examination: A single ulcerative lesion measuring 1.5×1 cm was noted on the mid-dorsal tongue with an erythematous base and irregular white pseudomembranous margins [Table/Fig-1]. The lesion was mildly tender, non-indurated, and not raised. Lateral borders of the tongue and other mucosal sites appeared normal. Salivary flow was slightly reduced with viscous saliva but without candidal plaques. No cervical lymphadenopathy was detected.



[Table/Fig-1]: Clinical photograph showing ulcerative lesion with erythematous base and irregular white margins on the dorsum of the tongue.

Investigations: Routine haematologic analysis revealed marked lymphocytosis consistent with CLL {White Blood Cell (WBC)=72.8×10³/µL; lymphocyte=68.4×10³/µL; smudge cells present}. Random blood glucose was 112 mg/dL. Thyroid profile showed subclinical hypothyroidism {Thyroid Stimulating Hormone (TSH)=5.06 µIU/mL; Free T4=0.91 ng/dL}. Vitamin D and B12 were normal, and the coagulation profile was within reference limits.

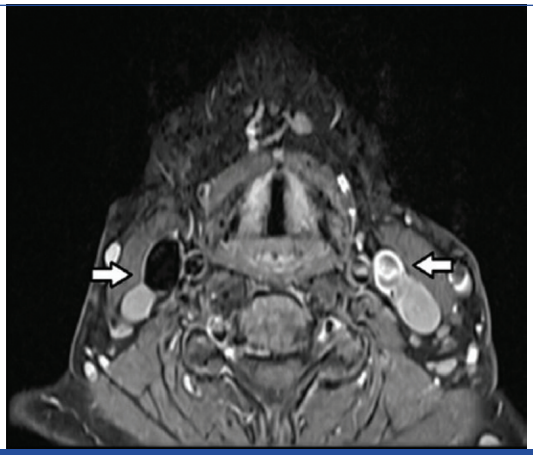
Immunological evaluation indicated CD3 T-cell count=780 cells/µL, confirming mild T-cell suppression. Fungal smear and KOH mount were negative.

Since atypical clinical and haematologic features were present, an incisional biopsy was performed from the deepest margin of the ulcer on the mid-dorsal surface of the tongue, including both the ulcer edge and adjacent clinically normal mucosa. It was done under local anaesthesia. Histopathological examination showed epithelial atrophy, chronic inflammatory infiltrate with a greater cell area being subepithelial, as well as an aggregation of plasma cells and lymphocytes. Nevertheless, there were no signs of malignancy, dysplasia, or fungal elements [Table/Fig-2].

Feature examined	Findings
Type of biopsy	Incisional biopsy from the deepest margin of the ulcer (mid-dorsal tongue)
Epithelial findings	Epithelial atrophy; surface desquamation
Subepithelial tissue	Dense chronic inflammatory infiltrate
Predominant inflammatory cells	Lymphocytes and plasma cells
Dysplasia	Absent
Malignant cells	Absent
Granuloma formation	Absent
Fungal hyphae (KOH/Histology)	Absent
Overall impression	Chronic benign inflammatory lesion

[Table/Fig-2]: Summary of histopathological findings.

After the biopsy, an MRI of the oral cavity and neck was done to look for possible deeper soft-tissue involvement or nodal disease. MRI and associated radiographs [Table/Fig-3] showed no soft-tissue mass, no tumour invasion, and no tumour infiltrate, but there was bilateral reactive cervical lymphadenopathy. Routine microbiological fungal investigations of the KOH mount were negative. [Table/Fig-4] shows the results of routine blood tests to be increased lymphocytosis consistent with CLL and blood glucose levels of 112 mg/dL. Based on the above combined results, the condition was diagnosed as a chronic inflammatory lesion of the tongue rather than an infective or neoplastic process.



[Table/Fig-3]: Axial T2-weighted MRI of the neck showing bilateral reactive cervical lymphadenopathy without soft-tissue mass or tumour infiltration.

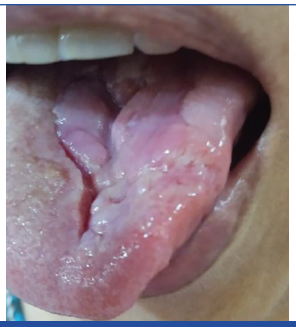
Test/investigation	Result	Interpretation	Reference range
White blood cell (WBC) count	72.81×10³/µL	Marked leukocytosis consistent with CLL	4-11×10³/µL
Absolute lymphocyte count	68.44×10³/µL	High- consistent with CLL	1-3×10³/µL
Smudge cells	Present	Suggestive of CLL	—
Random blood glucose	112 mg/dL	Within the non-diabetic range	<140 mg/dL (random)
TSH	5.06 µIU/mL	Subclinical hypothyroidism	0.4-4.5 µIU/mL
Free T4	0.91 ng/dL	Subclinical hypothyroidism	0.8-1.8 ng/dL
Vitamin D	39.8 ng/mL	Sufficient	30-100 ng/mL
Vitamin B12	428 pg/mL	Normal	200-900 pg/mL
Serum LDH	255 U/L	Mildly elevated (possible cell turnover)	140-280 U/L
Coagulation profile (APTT/BT/CT)	Normal	Normal coagulation	APTT: 25-35 s; BT: 2-7 min; CT: 8-15 min
CD3 T-cell count	780 cells/µL	Mild T-cell suppression	900-2,500 cells/µL
KOH/fungal smear	Negative	Fungal infection unlikely	—
Incisional biopsy (histopathology)	Epithelial atrophy with surface desquamation; subepithelial chronic inflammatory infiltrate; no dysplasia, no fungal elements	Malignancy and fungal infection excluded	—

[Table/Fig-4]: Summarises laboratory and microbiological findings used to exclude systemic metabolic causes and infection. Abbreviations: LDH: Lactate dehydrogenase; APTT: Activated partial thromboplastin time; BT: Bleeding time; CT: Clotting time

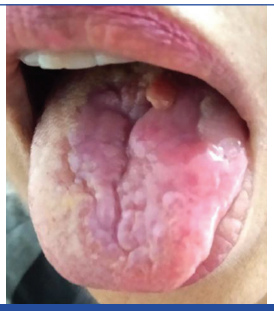
Diagnosis and Management

Findings confirmed a chronic benign inflammatory tongue lesion. As reports were pending, an antifungal therapy (clotrimazole oral paint) was trialed prior to biopsy, as oral candidiasis is an easily treatable and common cause of tongue lesion symptoms in immunocompromised patients. After a fungal infection and dysplasia were excluded, a more conservative approach was taken in symptom-focused management, incorporating benzydamine hydrochloride (0.15%) mouthwash, Mucopain gel, vitamin A and multivitamin oral medications to assist in epithelium repair. The patient was instructed to refrain from consuming irritants and spicy foods and to maintain proper oral hygiene.

Follow-up and outcome: At four weeks, pain and burning sensation had markedly reduced and bleeding had stopped. By eight weeks, the lesion healed completely, leaving mild surface fissuring without tenderness [Table/Fig-5]. During a 12-month follow-up, the patient remained asymptomatic with no recurrence, malignant transformation, or new mucosal lesions [Table/Fig-6]. Her systemic conditions (CLL and arthritis) remained clinically stable without medications.



[Table/Fig-5]: Post-treatment photograph after eight weeks showing healed tongue surface with mild residual fissuring and no ulceration.



[Table/Fig-6]: Twelve-month follow-up image demonstrating normal mucosal appearance and absence of recurrence.

DISCUSSION

Chronic inflammatory lesions of the tongue can present a diagnostic dilemma, especially in elderly and immunocompromised individuals [1]. Such lesions often resemble premalignant or infectious conditions, leading to unnecessary concern and overtreatment if not accurately recognised [1]. The present case is distinctive because it occurred in an elderly woman with two systemic diseases- CLL and autoimmune arthritis- both known to impair immune regulation and delay mucosal healing.

Remarkable about this case is the chronological pairing of a woman with both systemic autoimmune diseases: CLL and chronic autoimmune arthritis, both of which are expected to affect some parts of the immune system and slow the rate of normal healing of the oral mucosa [2]. The involvements of the tongue in the form of fissured and migratory patterns are known to be self-resolving and to carry no significance; however, patients with diseases that modulate the immune system may display lesions that cause burning and residual inflammation, which complicate proper diagnosis [3].

There are reports in the literature that describe chronic ulcerative or deep fissure-like lesions of the tongue in various immunocompromised states, including CLL, Human Immunodeficiency Virus (HIV) infection, post-transplant immunosuppression, and autoimmune disorders [4,5]. Many of these cases demonstrate delayed mucosal healing, exaggerated inflammatory response, and atypical ulcer morphology, not dissimilar from the present case [4,5]. Immunocompromised patients may also demonstrate secondary bacterial or fungal colonisation, which further alters the clinical appearance and may mimic more serious pathology [6]. Chronic inflammatory lesions of the dorsal tongue are known to often present with persistent erythema, ulceration, or fissuring in such patients with immunologic dysfunction and often require histopathological evaluation to rule out high-risk pathology [7].

A key consideration when evaluating tongue protrusion ulcers is the obligatory consideration of malignancy risk [8]. Oral squamous cell carcinoma may present in the dorsal tongue in the form of a non-healing ulcer, a deep fissure, or a slow-healing fissure-like ulcer, all of which should be considered in older individuals. The dorsal and lateral borders of the tongue are some of the highest-risk intraoral sites. Therefore, in aged or systemically compromised individuals, biopsy is a necessity [9].

From an epidemiological standpoint, it has been documented that chronic inflammatory or ulcerative lesions of the tongue, especially when associated with atrophy and chronic irritation, will present a slight risk of malignant transformation, and even more so when dysplastic changes are present [10]. The risk of malignant transformation has been reviewed in chronic inflammatory lesions of the tongue or lichenoid-type atrophic lesions and the rates observed within the range of 0.4% to 5% depending on the additional risk factors that are present in the individual, which may be of older age, tobacco or alcohol use, and the degree of epithelial dysplasia [11,12]. Despite the current patient's biopsy reflecting no dysplasia, it was the combination of her age and immunological status that warranted considerable investigation, especially in the areas of biopsy and MRI, to below whether a neoplastic pathology was present. The pathology was non-malignant and the tissue displayed chronic inflammatory degeneration. The benign histology and absence of invasive changes on imaging reassured that the lesion represented a chronic inflammatory.

In this patient, chronic inflammation coupled with occasional spontaneous bleeding likely resulted from superficial vascular fragility within the inflamed mucosa. Similar presentations have been described by Assimakopoulos D et al., and Jainkittivong A and Langlais R, who reported that benign inflammatory tongue lesions can easily be mistaken for fungal or neoplastic pathology [13,14].

The combination of autoimmune arthritis and CLL points to the potential presence of more than one immune-related problem within the lesion. With autoimmune arthritis, there are higher levels of certain pro-inflammatory cytokines, especially Tumour Necrosis Factor (TNF)-α, Interleukin (IL)-1β, and IL-6. These are cytokines that can alter epithelial cellular tight junctions and make the interstitial spaces within a mucosa more vulnerable to micro injuries and there can also be a slowdown in the normal rate of cellular renewal by the keratinocytes [15]. When there is also a CLL, the effect is even more pronounced due to a lower quality immune response, with particular attention to B and T lymphocytes, a drop in antibodies, and the weakening of any monitoring immune response within a tissue milieu [16]. Because of these immune defects, the inflammation within a tissue is much less prone to proper healing and there is a higher chance for a chronic inflammation to arise due to the presence of even small breaks within the tissue. In total, the autoimmune arthritis will increase the permeability of epithelial surfaces due to the cytokines and the CLL will contribute to a weakening of the immune response. These make the formation of long-standing inflammatory lesions on the oral mucosa much more likely [17]. Furthermore, the positive family history- with the patient's mother having a similar undiagnosed tongue condition and her brother showing asymptomatic fissured tongue- supports a possible genetic predisposition, consistent with reports by Picciani BL et al., describing familial clustering of benign inflammatory glossal lesions [18].

Differential diagnoses in this case included oral candidiasis, lichen planus, and early squamous cell carcinoma, all of which were excluded by fungal studies, histopathology, and MRI imaging. Assimakopoulos D et al., emphasised that such atypical inflammatory lesions, if misinterpreted, often lead to repeated biopsies or unwarranted antifungal therapy [13].

The patient's excellent response to Mucopain gel, benzydamine mouthwash, and vitamin supplementation reinforces the effectiveness of conservative, symptom-focused management once infection and malignancy are excluded. vitamins, particularly vitamin A and zinc, aid epithelial regeneration and modulate immune response, thereby supporting mucosal healing [1,19].

This case highlights the importance of a systematic, multidisciplinary diagnostic approach combining clinical, histological, and imaging evaluation. In elderly and immunocompromised patients, awareness of such benign inflammatory lesions can prevent unnecessary anxiety and invasive procedures. The familial tendency observed in this patient also points toward the need for further genetic and immunologic studies to better understand hereditary influences on oral mucosal diseases.

CONCLUSION(S)

Management of chronic ulcerative conditions involving tongue lesions in elderly and systemically immunocompromised patients is difficult and time-consuming due to the possibility of these conditions being malignancies or chronic infections. We, therefore, systematically and stepwise combined the clinical and the histopathology, and the multidisciplinary syndromes to achieve the correct diagnoses. After the malignancy and infectious conditions were ruled out, the conservative approach of managing with topical analgesia and mucosal protectants and oral vitamins had good results.

This case also suggests a possible genetic or immunologic predisposition due to the familial occurrence, which also implies the need to further study the chronic mucosal inflammation and the possible hereditary factors. In patients with systemic immunological disorders, close and longitudinal follow-up is absolutely necessary to study recurrences and the risk of malignancy. This report reinforces the importance of acknowledging the fact that chronic and ulcerative lesions of the tongue in the elderly are not all potentially malignant. This careful evaluation and awareness of the inflammatory pattern is vital in avoiding invasive procedures that cause anxiety and, hence, improve the diagnostic and overall care.

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