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Chronic multifocal oral ulceration in a middle-aged man: diagnostic work-up and debridement-based supportive care

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Abstract

Chronic oral ulceration often signals a wide spectrum of disorders, so management must begin with a structured differential diagnosis after malignant and infectious causes have been excluded. A 50-year-old man presented with six months of painful multifocal ulcers involving the left lateral tongue, labial mucosa and bilateral buccal mucosa, accompanied by a 5 kg weight loss. Brush cytology showed chronic suppurative inflammation without dysplasia, complete blood count revealed mild normocytic anaemia, and dual syphilis serology (VDRL + TPHA) was non-reactive. Management comprised weekly debridement using 1% hydrogen-peroxide swabs followed by saline irrigation, twice-daily 0.2% chlorhexidine mouthrinse, and dietary counselling. Supra- and sub-gingival scaling was done once pain allowed. All lesions resolved by week 7; pain improved from 8/10 to 0. This case illustrates that after reasonable exclusion of malignancy and key infections, regular debridement combined with chlorhexidine and plaque control can accelerate healing of chronic multifocal ulcers. The protocol described may be readily applied in general dental practice, including settings with limited resources.

Introduction

Oral ulceration is a breach of both epithelium and underlying lamina propria which affects up to one-fifth of the population, most commonly as recurrent aphthous stomatitis (RAS) but also as manifestations of infection, neoplasia and systemic disease (Mortazavi *et al.*, 2016; Zhou *et al.*, 2022). Reported predisposing factors include genetic predisposition and immune factors, local trauma, drugs, stress and nutrient deficiencies (Yan *et al.*, 2022). Because many of these conditions share similar clinical features, a step-wise differential diagnosis that considers lesion duration, morphology, and location should precede any provisional diagnosis (Woo *et al.*, 2021).

We report a 50-year-old male with six-month multifocal ulcers that responded only after regular debridement and mechanical plaque control. The case illustrates a practical diagnostic pathway when the biopsy is not done and how weekly peroxide debridement and chlorhexidine mouthrinse can speed healing in a setting with limited resources.

Case report

Patient history

A 50-year-old male patient attended the Oral Medicine Clinic, Universitas Gadjah Mada Academic Hospital (Yogyakarta, Indonesia), complaining of persistent pain from multiple oral ulcers present for six months. Since his mid-thirties he had experienced minor oral ulcers that typically resolved fast. The current episode began abruptly six months earlier with larger ulcers appearing on several sites and never fully resolved. He denied issues with swallowing or moving tongue, lesions elsewhere, gastrointestinal symptoms, ocular complaints, smoking or regular medication. Oral intake was poor and he had lost 5 kg due to difficulty eating. He had been gargling with Betadine and salt water without improvement. The patient had difficulty speaking and communicated primarily through nodding or shaking his head.

Baseline examination (Day 0)

Extraoral examination revealed no palpable lymph nodes. Intraoral examination was limited because the patient could not open his mouth. It showed poor oral hygiene, with plaque and debris accumulating throughout the dentition and significant supragingival and subgingival calculus deposits on the lingual surfaces of the lower anterior teeth. Multiple ulcers with rolled, erythematous margins and yellow-white pseudomembrane were noted on the left lateral tongue, maxillary labial mucosa, and bilateral buccal mucosa. Informed consent was obtained, and the treatment was carried out according to hospital regulations.

At this first visit, lesion debridement (Table 1) was performed during the first visit using 1% hydrogen peroxide swabs, followed by saline irrigation and 0.12% chlorine dioxide (ClO_2) spray. The patient was prescribed 0.2% chlorhexidine (CHX) gluconate mouthrinse (10 mL, bid), B-complex vitamins, and instructed to brush twice daily with a soft brush while avoiding direct contact with ulcers. The supporting examinations ordered were complete blood count (CBC), brush cytology, gram stain for fungal elements and aerobic culture from ulcer bases.

Table 1. Debridement protocol

No.	Step & product used	Acceptable alternatives	Purpose / Rationale	Technique / Key points
1	Asepsis and isolation → 10% povidone-iodine swab	–	Reduce surface bioburden; protect field from saliva contamination	Swab perilesional mucosa for 30 s, gently blot excess solution and leave the surface moist. Place cotton rolls or gauze to keep area dry
2	Mechanical debridement → 1% hydrogen-peroxide swab (3-5 min)	Normal saline, chlorhexidine gluconate 0.1% – 0.2%, povidone iodine 10%	Break up pseudomembrane, expose viable tissue, lower microbial load	Use light, one-way strokes from the center of the ulcer outward. Continue until the pseudomembrane thins or pin-point bleeding appears. Change the swab as soon as it becomes coated. Work slowly to minimize pain
3	Irrigation → 0.9% normal saline	Aquadest, clean water	Neutralize residual peroxide, flush debris	Use gauze then blot the ulcer surface in one direction to remove residual debridement solution and loosened debris. Change the swab as soon as it becomes coated. Leave the surface moist.
4	Topical medication → Chlorine-dioxide spray 0.12 %	Triamcinolone acetonide 0.1% in ora base, Hyaluronic-acid (gel/rinse/spray) *Select the medication by lesion site, number, pain level.	Deliver antimicrobial or anti-inflammatory agent, form protective film	**For spray:** hold nozzle 1-2 cm from site, deliver 2-3 puffs, ask patient to avoid eating or drinking for 30 minutes to maximise contact time. **For paste/gel:** with a cotton pellet, micro-brush, or the blunt (back) end of a dental explorer, lay a thin film over the ulcer in one smooth stroke. Do not rub or scrub. Then ask patient to avoid eating or drinking for 30 minutes to maximise contact time.

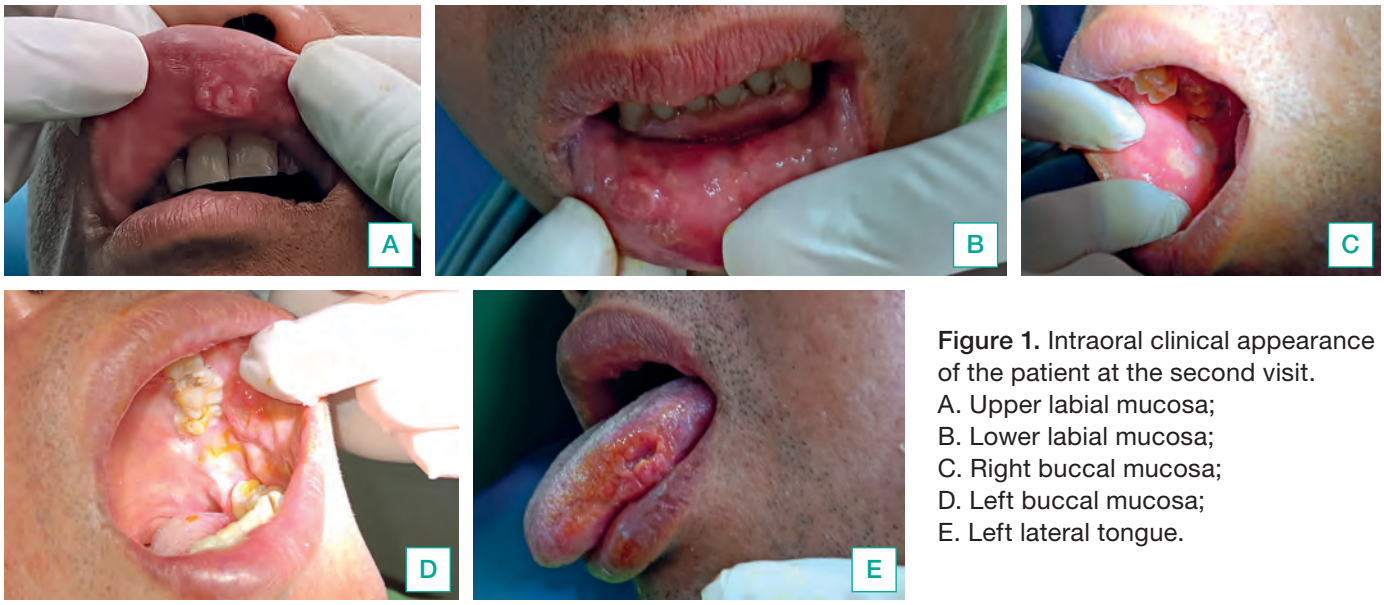

Figure 1. Intraoral clinical appearance of the patient at the second visit.
A. Upper labial mucosa;
B. Lower labial mucosa;
C. Right buccal mucosa;
D. Left buccal mucosa;
E. Left lateral tongue.

Week 1 review (Day 7)

The patient reported pain had lessened and speech improved. He admitted that he had previously refrained from brushing his teeth due to ulcer-related pain, opting only to gargle with salt water. Intraoral examination showed multiple ulcers measuring around 1-1.5 cm covered with a yellowish-white pseudomembrane with well-defined and regular edges were still present in several locations on the upper labial mucosa (Fig.1A); lower labial mucosa (Fig.1B); right and left buccal mucosa (Fig.1C, Fig.1D); and left lateral tongue (Fig.1E). Lesion debridement was repeated, and the same medication continued. CBC showed mild normocytic anaemia (Table 2). Brush cytology from the right buccal mucosa, upper labial mucosa and left lateral tongue revealed dense neutrophil–histiocyte infiltrates without epithelial atypia or malignant cells. The findings were interpreted as chronic suppurative inflammation, very likely caused by bacterial infection. Gram stain showed no yeast, pseudohyphae or true hyphae observed and the bacterial cultures on the tongue and labial mucosa yielded Gram-negative rod-shaped bacteria. On microbiology advice, syphilis serology (*Treponema Pallidum Hemagglutination*/TPHA and *Venereal Disease Research Laboratory*/VDRL) was requested.

Table 2. Blood test results

Parameter	Result	Unit	Reference range
Leukocytes	7.3	10^3/ul	3.8-10.6
Absolute Neutrophils	5.02	10^3/ul	1.65-4.97
Absolute Lymphocytes	1.30	10^3/ul	1.17-3.17
NLR (Neutrophil-lymphocyte ratio)	3.86		<3.13
Absolute Monocytes	0.73	10^3/ul	0.23-0.68
Absolute Basophils	0.03	10^3/ul	0.02-0.08
Absolute Eosinophils	0.22	10^3/ul	0.05-0.32
Erythrocytes	4.2	10^6/ul	4.4-5.9
Hemoglobin	12.4	g/dl	13.2-17.3
Hematocrit	36.9	%	40-52
Erythrocyte Sedimentation Rate	26	mm/hour	0-10
MCV	87.2	fl	80-100
MCH	29.3	Pg	26-34
MCHC	33.6	g/dl	32-36
Thrombocytes	188	10^3/ul	150-440
Neutrophils	69	%	50-70
Lymphocytes	18	%	25-40
Monocytes	10	%	2-8
Eosinophils	3	3%	2-4
Basophils	0	%	0-1
Band Neutrophils	0	%	1-5

Week 2 review (Day 14)

The patient reported significant improvement and no pain. Intraoral examination showed that ulcers were smaller with thinner pseudomembrane layer (Figure 2). TPHA and VDRL tests were non-reactive. Supra- and sub-gingival scaling plus lesion debridement were performed, and chlorhexidine mouthrinse was maintained.

Week 3 review (Day 21)

The patient no longer experienced pain, although a new ulcer had appeared on the anterior tongue four to five days prior. The patient denied any physical or psychological stressors but mentioned participating in late-night social activities. Intraoral examination revealed a 3-mm ulcer with a well-defined border and regular edges on the tongue tip, covered by a white-yellow pseudomembrane (Figure 3A). Other sites continued to heal (Figure 3B-E). Oral hygiene had markedly improved and the same regimen was continued.

Week 5 review (Day 35)

All ulcers had healed, with no new recurrences (Figure 4). A residual lesion measuring 2 mm on the lower right labial mucosa was in the healing phase. The patient was explained on the importance of maintaining oral hygiene, proper toothbrushing techniques, and adopting a balanced, nutritious diet while minimising activities that could cause physical or mental fatigue. A three-month recall with biopsy contingency (any ulcer > 3 weeks or recurrence < 6 months) was agreed.

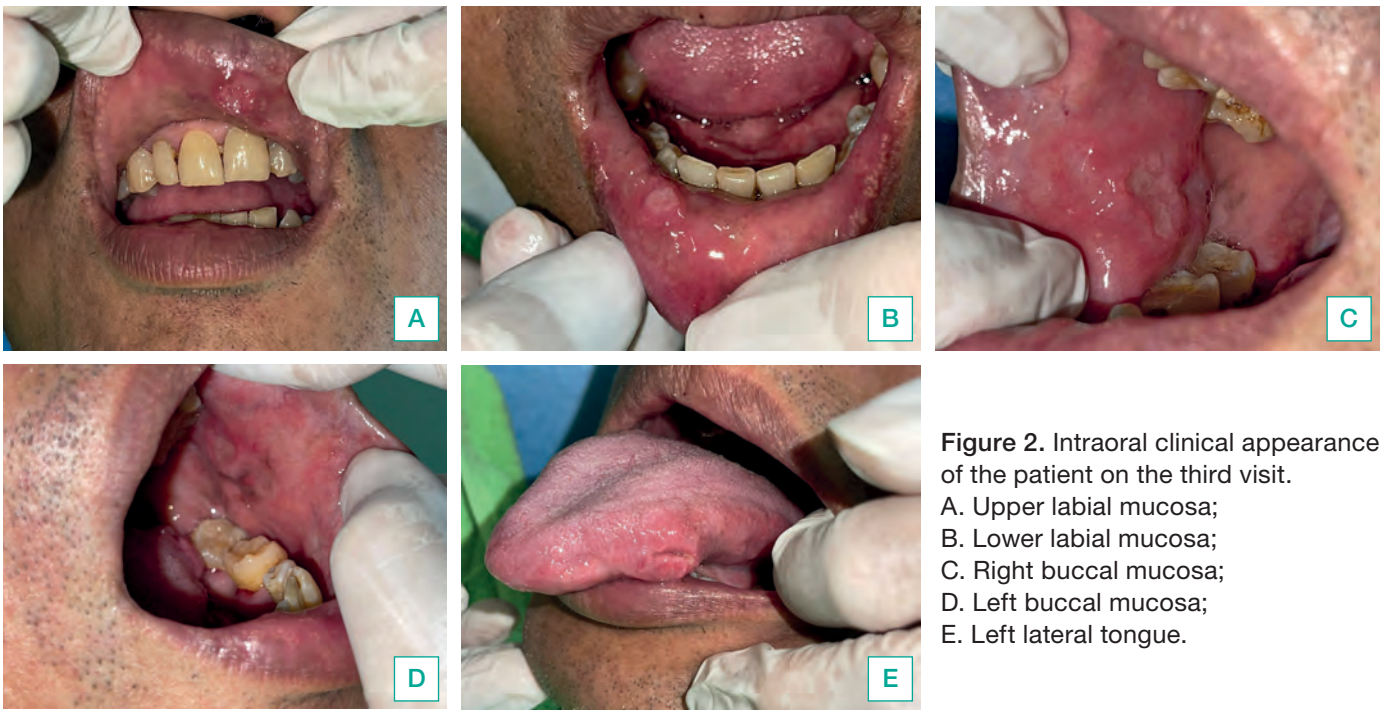

Figure 2. Intraoral clinical appearance of the patient on the third visit.
A. Upper labial mucosa;
B. Lower labial mucosa;
C. Right buccal mucosa;
D. Left buccal mucosa;
E. Left lateral tongue.

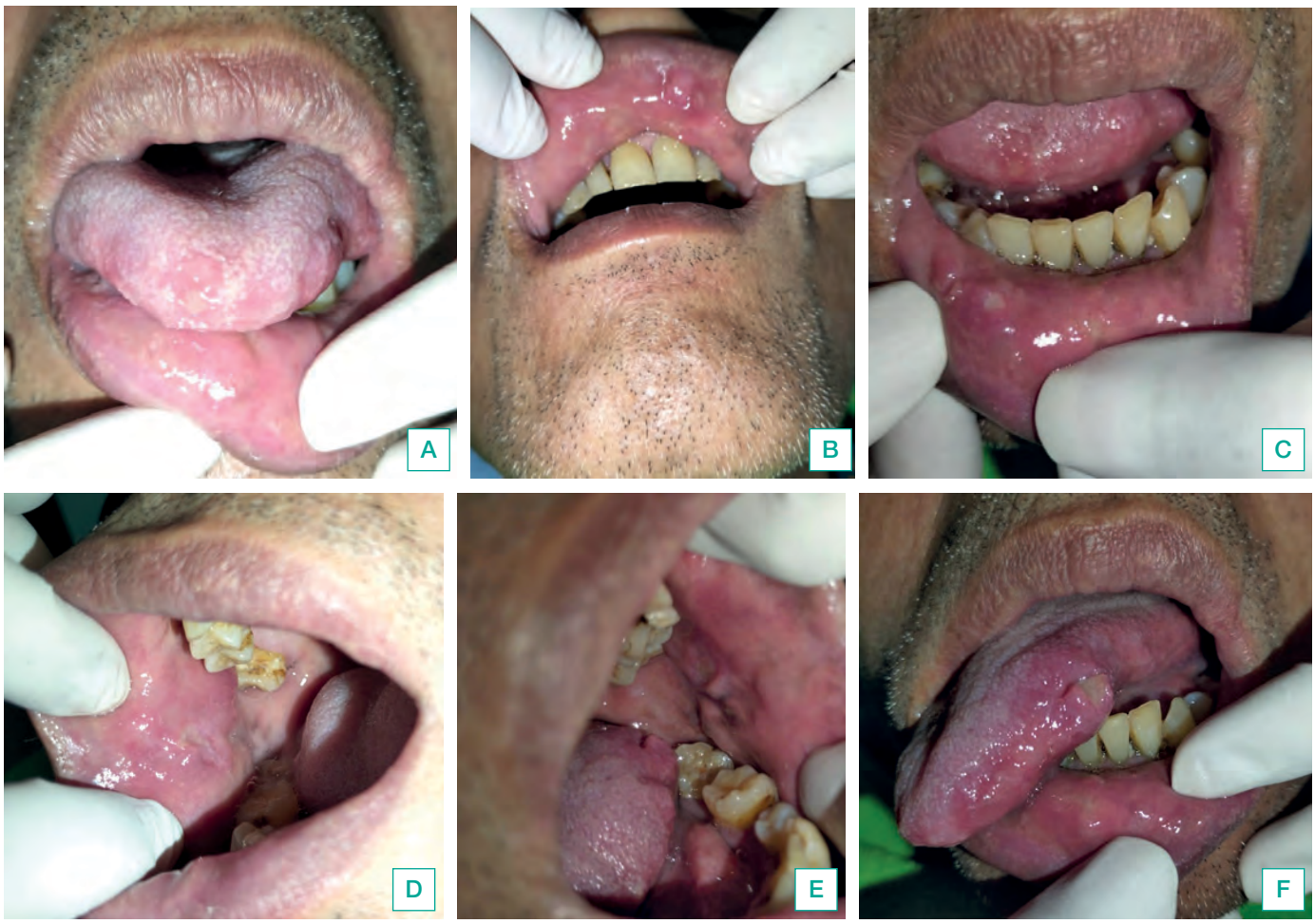

Figure 3. Intraoral clinical appearance of the patient on the fourth visit. A. New ulcer on the anterior tongue; B. Upper labial mucosa; C. Lower labial mucosa; D. Right buccal mucosa; E. Left buccal mucosa; F. Left lateral tongue.

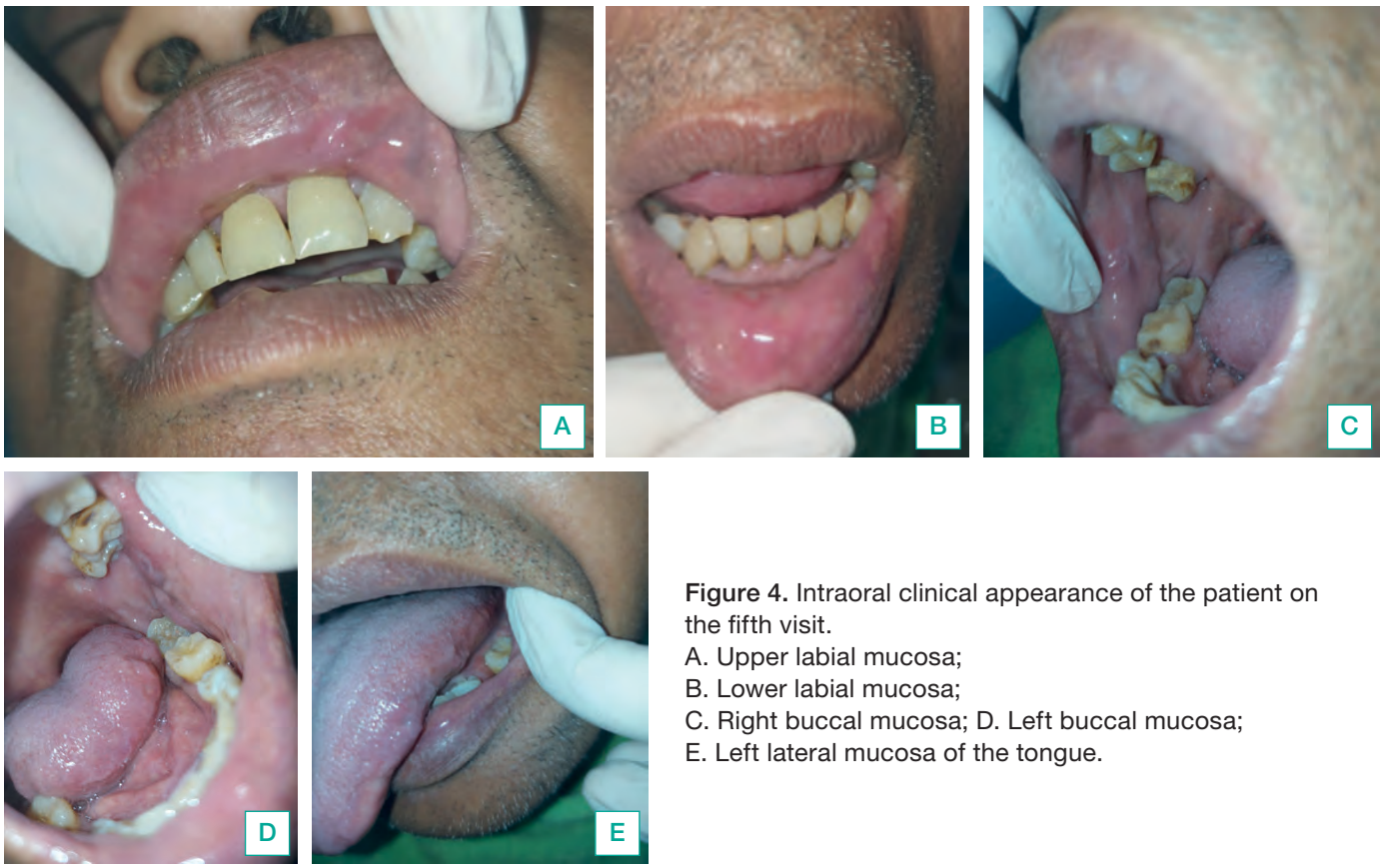

Figure 4. Intraoral clinical appearance of the patient on the fifth visit.
A. Upper labial mucosa;
B. Lower labial mucosa;
C. Right buccal mucosa; D. Left buccal mucosa;
E. Left lateral mucosa of the tongue.

Discussion

Ulcerative lesions share a common histology which is loss of epithelium and lamina propria but arise from diverse causes. Thus, diagnosis relies on careful history, morphology and site analysis. According to the duration- and number-based system of Mortazavi *et al.* (2016), the present lesions were chronic and multiple. Systemic conditions such as autoimmune disorders, nutritional deficiencies (particularly vitamin B12, iron, and folic acid), and gastrointestinal diseases were explored through laboratory tests and targeted history (Lodi *et al.*, 2019; Minhas *et al.*, 2019; Rodsaward *et al.*, 2017).

The patient's CBC revealed low erythrocyte, haemoglobin, and haematocrit levels with otherwise normal MCV, MCH and MCHC, indicating a mild normocytic-normochromic anaemia. Lymphocyte levels were below the normal range but not significantly, suggesting the possibility of physical or psychological stress (Herawati *et al.*, 2011). Elevated ESR and neutrophil-to-lymphocyte ratio (NLR) and monocyte levels indicated chronic low-grade inflammation (Forget *et al.*, 2017; Shi & Pamer, 2011). Iron studies and B-vitamin assays were advised but declined.

Given the ulcer's persistence (more than 2 weeks) and indurated edges resembling malignant features such as Oral Squamous Cell Carcinoma (OSCC), a biopsy was warranted as the gold standard to rule out malignancy. Brush biopsy or brush cytology was done in this case. Although brush cytology is reported to give about 95% sensitivity and 85% specificity for dysplasia, it cannot assess epithelial architecture; current guidelines therefore recommend incisional biopsy for any indurated or non-healing ulcer (Woo *et al.*, 2021). The brush cytology returned negative for malignancy, yet the residual false-negative risk was discussed with the patient. Because the patient showed good progression within two weeks of treatment, biopsy was deferred at that stage. A three-month recall was scheduled, with earlier biopsy if induration recurs or any ulcer persists beyond three weeks, implementing a safety-net strategy aligned with NICE guidance for suspected oral cancer, which advises urgent assessment for any oral ulcer persisting beyond three weeks.

In tropical countries such as Indonesia, a chronic oral ulcer lasting more than two weeks raises concern not only for malignancy but also for chronic granulomatous infections, most notably oral tuberculosis and deep fungal diseases such as histoplasmosis and blastomycosis. Indonesia is recognised by the World Health Organization as one of six high-tuberculosis-burden countries in the South-East Asia Region. The country also lies in the tropical belt where deep fungal infections such as histoplasmosis and blastomycosis can present as chronic oral ulcers that mimic malignancy (Chakrabarti, 2019; Kusmiati *et al.*, 2023). In our patient, Gram staining of ulcer exudate showed no yeast, pseudohyphae or true hyphae, ruling out superficial candidiasis. Deep mycoses can be definitively detected only through histopathological examination of biopsy sections stained with periodic-acid–Schiff (PAS) or Grocott–Gomori methenamine silver (GMS), which reliably expose the otherwise hidden fungal elements (Samaila & Abdullahi, 2011). Oral tuberculosis is likewise confirmed histologically, typically by identifying acid-fast bacilli on Ziehl–Neelsen stain (Mignogna *et al.*, 2000).

Chronic bacterial infections, particularly syphilis, can also cause non-healing ulcers. Primary syphilis infection often appears as a papule at the site of inoculation that quickly turns into an indurated, erythematous but typically painless chancre (Deng *et al.*, 2022). Although the patient experienced pain, the chronic nature of the ulcers and incomplete initial anamnesis required syphilis screening. Both the non-treponemal VDRL and the treponemal TPHA were non-reactive. VDRL detects about 78–86% of primary and about 100% of secondary cases, but its sensitivity declines in late infection (Seña *et al.*, 2010; Workowski *et al.*, 2021). Treponemal assays, by contrast, maintain ≥ 95% sensitivity throughout latent and tertiary stages and remain positive for life (Janier *et al.*, 2021; Workowski *et al.*, 2021). As the ulcers had persisted for six months, well beyond the usual 4- to 6-week seroconversion window, concordantly negative VDRL and TPHA results make active syphilis highly unlikely (Hook & Peeling, 2004).

Poor plaque control can delay oral-mucosal healing. Because the ulcers had been present for several weeks, the patient stopped brushing and switched to a soft diet, a mature biofilm therefore accumulated along the gingival margin and across the ulcer bases. Culture from the ulcer base in our patient yielded Gram-negative rod-shaped bacteria. Species-level identification was not available but this morphology is typical of anaerobes such as Fusobacterium nucleatum, Prevotella intermedia as well as the keystone periodontal pathogen Porphyromonas gingivalis, whose lipopolysaccharide and protease prolong local inflammation and slow re-epithelialization (Socransky & Haffajee, 2005). Recent 16S-rRNA profiling of recurrent oral-ulcer niches has also shown enrichment of Leptotrichia species, suggesting a broader polymicrobial role in impaired repair (Wang *et al.*, 2024). Because plaque biofilm was a plausible perpetuating factor, we prioritized supra- and sub-gingival scaling, which can remove up to 90% of bacterial load from periodontal pockets (Cobb, 2002). A course of chlorhexidine mouthrinse was added as an evidence-based adjunct to mechanical debridement to suppress recolonization and create a more conducive surface to healing (Brookes *et al.*, 2020).

The patient had already tried a Betadine gargle and saltwater rinses. Both are rational first-line measures because betadine gargle containing povidone-iodine is an antiseptic with broad-spectrum bactericidal properties, and salt solution moisturizes the oral cavity and promotes fibroblast migration, aiding wound healing (da Silveira Teixeira *et al.*, 2019; Huynh *et al.*, 2016). However, because the ulcers persisted, weekly debridement was introduced to remove necrotic tissue, debris, microbes and residual medications, thereby creating an optimal wound-healing surface. Irrigation was performed with the solution of choice; the tissue was cleaned and then irrigated again before the medication was applied (Wolcott & Fletcher, 2014). In this case, debridement employed 1% hydrogen peroxide, which kills pathogens through oxidative bursts and delivers transient oxygenation; its bio-effects are

dose-dependent, and concentrations above 3% risk mucosal cytotoxicity (Walsh, 2000; Zhu *et al.*, 2017). Residual peroxide was neutralized with saline rinse before the topical agent was applied. Given the patient's limited mouth opening, care was taken to avoid iatrogenic trauma. The combination of peroxide debridement, plaque control and topical antiseptics produced progressive healing at each visit.

Topical medication used for the patients were chlorine dioxide and chlorhexidine gluconate. Stabilised chlorine-dioxide (ClO_2) is an alcohol-free oxidising rinse that disrupts mature biofilm within 60 seconds. A 2020 systematic review and meta-analysis showed that ClO_2 mouthwashes reduce plaque and gingival indices as effectively as chlorhexidine, but without staining or taste alteration (Kerémi *et al.*, 2020). However, for daily use, 0.2% chlorhexidine gluconate (CHX) was selected. Chlorhexidine disrupts bacterial cell membrane permeability, causing cell lysis and death at concentrations above 0.1% (Brookes *et al.*, 2020). Also, because CHX is listed in the Indonesian National Formulary and reimbursed by BPJS-Kesehatan, whereas commercial ClO_2 rinses are not. B-complex vitamins were also prescribed to address possible micronutrient deficits, which can impair mucosal repair (Hanna *et al.*, 2022).

After excluding malignancy, syphilis, tuberculosis and deep mycoses, the working differential was reduced to aphthous-like ulcer (ALU) and atypical/complex recurrent aphthous stomatitis (RAS). ALU is a term reserved for ulcers that clinically mimic RAS but appear for the first time in adulthood, do not diminish with age and often show atypical size or distribution (Scully & Hodgson, 2008). While complex aphthosis is defined as the almost constant presence of three or more oral ulcers, with or without genital aphthosis, after Behçet disease has been excluded (Aquino & Jamora, 2020). Our patient fulfilled these latter criteria: multifocal ulcers were continuously present for six months, there were no ocular, genital, gastrointestinal or cutaneous manifestations, and serology/imaging showed no systemic disorder. We therefore assigned the provisional diagnosis idiopathic complex aphthosis (atypical RAS) while acknowledging that a definitive label awaits scalpel biopsy should lesions recur. The 5 kg weight loss was attributed to reduced oral intake from pain; no fever, night-sweats or gastrointestinal symptoms were reported. His mild normocytic anaemia is most plausibly nutritional, underscoring the role of adequate micronutrients in mucosal healing (Hanna *et al.*, 2022).

Conclusion

A chronic, multifocal oral ulcer demands a step-wise approach that first rules out malignancy and key infections before a benign label such as complex aphthosis is considered. In this patient, brush cytology, dual syphilis serology, culture, and targeted laboratory tests narrowed the diagnosis while a biopsy-contingent surveillance plan-maintained safety when the biopsy wasn't performed. Weekly 1% hydrogen-peroxide debridement, course 0.2% chlorhexidine, and supra-/sub-gingival scaling removed biofilm and necrotic tissue, allowing full re-epithelialization within seven weeks. The case shows that, in resource-limited settings, systematic assessment combined with sequential debridement and plaque control can expedite healing of chronic oral ulcers provided clear follow-up triggers are in place.

Author contributions

Conception or design of the work – AFA
Patient care – AAF, ARR, ADT, FN, VMK
Drafting the article – AAF, ARR, ADT
Critical revision of the article – FN, VMK, AFA
Final approval of the version to be published – all authors

Declaration of patient consent

The authors confirm that they have secured all necessary patient consent forms. The patient has given permission for their photographs and other clinical details to be published in the journal.

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Conflict of interest

The authors declare no conflicts of interest.

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