DOI: 10.7860/JCDR/2026/90523.24386

Case Report

Internal Medicine Section

Exophytic Major Recurrent Aphthous Stomatitis (Fuchs Syndrome) Triggered by Pregnancy and Lactation: A Case Report

MITTAL SINDHAV[1], BHARAT CHAUDHARY[2], RONAK SHAH[3], PARTH SHAH[4]

ABSTRACT

Fuchs syndrome, the most severe form of major Recurrent Aphthous Stomatitis (RAS), is characterised by large, deeply penetrating oral ulcers that cause significant morbidity. While its pathogenesis involves immune dysregulation, nutritional deficiency, and hormonal influences, pregnancy and lactation-associated exacerbations remain poorly documented, and atypical exophytic presentations are exceptionally rare. Hereby, the authors report the case of a 32-year-old lifelong lacto-vegetarian female, actively breastfeeding, who presented with a third episode of severe oral ulceration over 18 months each episode temporally associated with late pregnancy, early postpartum or active lactation. On this occasion, a 6 mm ulcer progressed to a 6 cm exuberant flower-like exophytic inflammatory mass covering the entire lower lip despite seven days of oral acyclovir therapy, initiated on the basis of incidental Herpes Simplex Virus Type 1-Immunoglobulin G (HSV-1 IgG) seropositivity. Negative Herpes Simplex Virus-Immunoglobulin M (HSV-IgM) and absence of vesicular lesions excluded active herpetic infection. Borderline vitamin B12 at the lower end of the reference range (234 pg/mL) and low-normal ferritin (30 ng/mL) were identified as contributory nutritional factors in a vegetarian patient with increased lactational demands. Systemic prednisolone (20 mg daily tapered over 13 days) produced dramatic clinical improvement within 72 hours and complete epithelialisation by day 21, with no recurrence at three months. The present case illustrates the diagnostic pitfall of equating isolated HSV-1 IgG positivity with active infection, highlights the role of hormonal immunomodulation in precipitating major RAS, and reinforces the primacy of systemic corticosteroids in managing severe immune-mediated oral ulceration.

Keywords: Corticosteroids, Hormonal immunomodulation, Oral ulceration, Pregnancy complications, Vitamin B12 deficiency

CASE REPORT

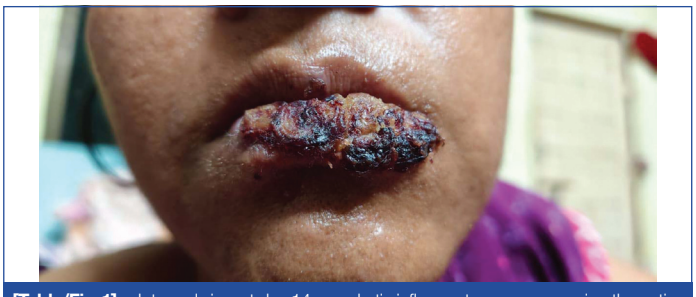
A 32-year-old female, a homemaker and lifelong lacto-vegetarian presented to the Emergency Department with severe, painful oral ulceration that had prevented oral intake for five days. She was actively breastfeeding her six-month-old second child and was in lactational amenorrhoea.

There was no history of dental trauma, recent dental procedures, tobacco or alcohol use, or long-term medication, and dental hygiene was satisfactory. Family history was negative for recurrent aphthous ulcers, Behçet's disease, inflammatory bowel disease, coeliac disease, or other autoimmune conditions.

This was the patient's third episode of severe oral ulceration in 18 months, each linked to a distinct hormonal state. The first occurred in the seventh month of her second pregnancy: a single 4-5 mm ulcer that resolved within five days with a multivitamin injection, omeprazole 20 mg daily, folic acid 5 mg daily, and topical antiseptic gel. The second developed six months postpartum during active breastfeeding: an 8 mm ulcer persisting seven days, treated similarly over one month with complete resolution.

The current episode began with a single, painful 6 mm ulcer on the lower lip (pain 7/10 on the Visual Analogue Scale), with no identifiable trigger such as trauma, diet, stress, or new medication. On day six, oral acyclovir 400 mg three times daily was started at an outside facility on the basis of an incidental HSV-1 IgG titre of 27 IU/mL, without confirmatory IgM testing or clinical correlation. Despite seven days of antiviral therapy, the lesion progressed dramatically: by day 13 it had transformed into a flower-like exophytic inflammatory mass approximately 6 cm in maximum dimension, covering the entire mucosal surface of the lower lip. No regional lymphadenopathy was detected at any stage. A 5 mm round, flat ulcer developed concurrently on the palm, following a parallel course.

On examination at day 14 the patient was conscious, alert, and haemodynamically stable (blood pressure 118/76 mmHg, pulse 78/min, temperature 37.0°C), with no pallor, icterus, pedal oedema, or lymphadenopathy. Oral examination revealed the ulcerative lesion described above, with irregular raised borders and a yellow-white pseudomembranous covering overlying granulation tissue [Table/Fig-1]; it was extremely tender, soft, and non indurated, with no bleeding, vesicles, crusting, or satellite lesions. The surrounding mucosa was otherwise normal, and no sharp teeth or dental appliances were identified. Cutaneous examination confirmed the 5 mm round, flat, non exophytic palmar ulcer with no vesicles, bullae, or target lesions. Systemic examination of the cardiovascular, respiratory, abdominal, and neurological systems was unremarkable, and the patient denied fever, weight loss, night sweats, arthralgia, ocular symptoms, genital ulcers, or gastrointestinal complaints.



[Table/Fig-1]: Intraoral view at day 14, exophytic inflammatory mass covering the entire lower lip, approximately 6 cm, with irregular borders and pseudomembranous covering.

Investigations

Haematological, biochemical, microbiological, and serological investigations performed on day 14 are summarised in [Table/Fig-2]. Serum vitamin B12 (234 pg/mL) was borderline and ferritin (30 ng/mL) low-normal, consistent with the nutritional demands of lactation in a vegetarian patient; all other parameters were within normal limits.

Biopsy was deferred given the rapid response to corticosteroids within 72 hours, complete resolution by day 21, young age,

Investigation	Result	Reference range
Haemoglobin	13.0 g/dL	12.0-15.5 g/dL
Total leucocyte count	8980/µL	4000-11000/µL
Platelet count	347,000/µL	150,000-400,000/µL
Erythrocyte Sedimentation Rate (ESR)	9 mm/hour	<20 mm/hour
C-Reactive Protein (CRP)	2.0 mg/L	<5 mg/L
AST / ALT	24 / 32 IU/L	<40 IU/L
Serum creatinine	0.8 mg/dL	0.5-1.1 mg/dL
Vitamin B12	234 pg/mL	200-900 pg/mL
Serum ferritin	30 ng/mL	15-150 ng/mL
Serum Iron	70 µg/dL	60-170 µg/dL
Total iron-binding capacity	300 µg/dL	250-450 µg/dL
HIV I & II antibodies	Non-reactive	Non-reactive
Anti-tTG IgA	Negative	Negative
HSV-1 IgG	27 IU/mL	>22 IU/mL = positive
HSV IgM	<0.5 IU/mL	<0.9 IU/mL = negative

[Table/Fig-2]: Laboratory investigations performed at presentation (Day 14). All other values were within normal limits.
AST: Aspartate aminotransferase; ALT: Alanine transaminase; Anti-tTG IgA: Anti-tissue transglutaminase immunoglobulin A; HIV: Human immunodeficiency virus

absence of malignant features, and two prior similar self-resolving episodes. HSV PCR and Tzanck smear were not performed; active HSV infection was excluded on negative IgM, absent vesicular morphology, and clinical failure of acyclovir. Autoimmune markers were not assessed given the characteristic trajectory and complete steroid-responsive resolution. Borderline B12 and low-normal ferritin were interpreted as contributory rather than causative.

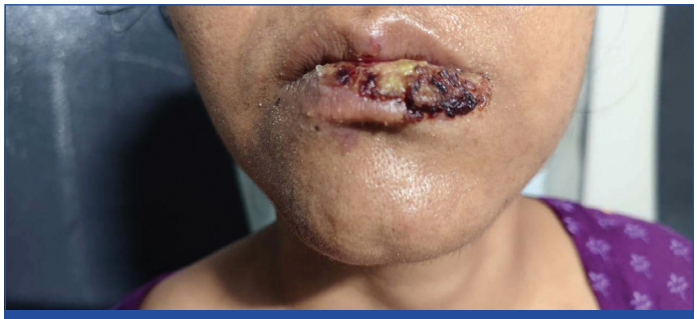
Recurrent herpes simplex virus infection was excluded by negative HSV IgM, absence of vesicular lesions, involvement of non-keratinised mucosa, and clinical deterioration despite seven days of acyclovir, to which genuine herpetic infection typically responds within 48-72 hours. Behçet's disease was excluded given the absence of genital, ocular, or articular involvement, normal inflammatory markers (ESR 9 mm/hour, CRP 2.0 mg/L), and clustering with hormonal states rather than a chronic relapsing course. Nutritional deficiency alone could not explain the severity or exophytic morphology, particularly as prior episodes recurred despite supplementation. Inflammatory bowel disease was excluded by the absence of gastrointestinal symptoms and negative coeliac serology, and autoimmune mucocutaneous disorders by the absence of vesiculobullous lesions and complete corticosteroid response. Oral squamous cell carcinoma was excluded given young age, absence of risk factors, induration, fixation, or lymphadenopathy, and complete healing without recurrence at three months.

Treatment

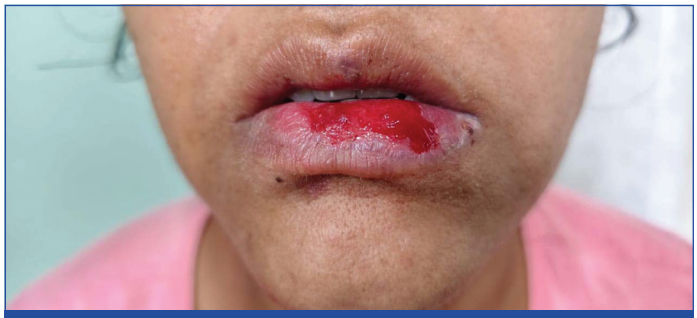
Initial oral acyclovir 400 mg three times daily for seven days worsened the lesion, confirming a non-herpetic aetiology. Definitive treatment began on day 14 with tapering systemic prednisolone (20 mg daily for five days, 10 mg daily for five days, 5 mg daily for three days; 13-day course). Oral cefixime 200 mg twice daily for five days and topical mupirocin 2% twice daily for five days addressed suspected secondary bacterial colonisation. Long-term prophylaxis included a daily multivitamin with zinc, folic acid 5 mg daily, dietary counselling, and a switch to sodium lauryl sulphate-free toothpaste.

Outcome and Follow-up

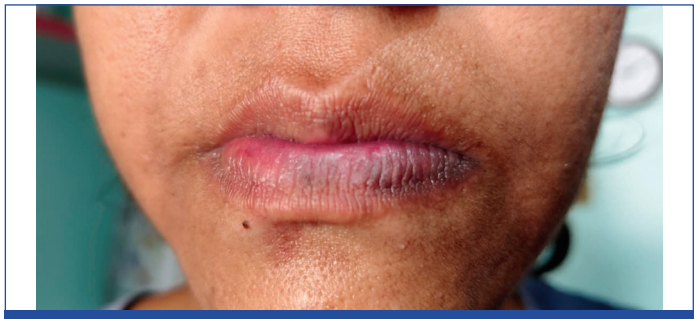
The response to corticosteroids was rapid. Within 72 hours (day 17), pain fell from 7/10 to 3/10, the patient tolerated a soft diet, and approximately 50% of the exophytic tissue had regressed [Table/Fig-3]. By day 21, complete epithelialisation had occurred, the palmar lesion had resolved, pain was 0/10, and a normal diet was tolerated [Table/Fig-4]. Breastfeeding continued uninterrupted throughout. At three months, the patient remained asymptomatic with no recurrence or scarring, and continued nutritional supplementation [Table/Fig-5].



[Table/Fig-3]: Seventy-two-hour response of steroid treatment - Clinical photograph at day 17 (3 days, after initiating corticosteroid therapy) showing marked improvement. Significant reduction in lesion size and regression of exophytic tissue. Patient able to close mouth and tolerate normal diet.



[Table/Fig-4]: Healing Ulcer after slough separation revealing red granulation tissue, Clinical photograph at day 21 showing marked improvement. The pseudomembranous slough has separated, revealing healthy red granulation tissue and active re-epithelialisation. Dark blue staining from topical antiseptic treatment visible.



[Table/Fig-5]: 3-month follow-up showing complete resolution: Clinical photograph at 3-month follow-up demonstrating complete resolution. Normal mucosal appearance with no residual scarring. No evidence of recurrence.

DISCUSSION

The present case documents a morphologically exceptional variant of major RAS, in which a small aphthous ulcer underwent progressive exophytic transformation over thirteen days into a six-centimetre, flower-like lesion spanning the entire lower lip. Standard major-type (Sutton) ulcers typically measure 1-3 cm, are deeply indurated, and persist for ten days to six weeks, with roughly two-thirds healing with scarring [1]. The present lesion exceeded this threshold more than twofold and assumed a morphology rarely reported; its rapid, complete resolution with systemic corticosteroids alone, without biopsy or recurrence, supports an immune-mediated rather than neoplastic process.

A clinically instructive feature is the temporal link between all three episodes and distinct hormonal states across a single reproductive continuum - late pregnancy, early lactation, and active breastfeeding. RAS is driven by T-lymphocyte-mediated immune dysregulation, with Th1 cytokines including Interleukin-2 (IL-2), Interleukin-6 (IL-6) and Tumour Necrosis Factor-alpha (TNF-α) implicated in cytotoxic ulceration [2], and gene-expression studies confirm a predominant Th1 signature without Th2 overexpression [3]. Pregnancy is characterised by a physiological Th1-to-Th2 shift protecting

the semi-allogeneic fetus, a protection that dissipates abruptly after delivery, when abrupt immune reconstitution can provoke exaggerated inflammatory responses [4]. Hormonal influences on aphthous severity and a specific association with breastfeeding have both been previously documented [5,6]. As per authors' knowledge, three serially documented episodes spanning a single pregnancy-to-lactation continuum, culminating in an exophytic presentation, have not been previously reported.

Nutritional deficiency was an important contributing factor. The patient's vitamin B12 (234 pg/mL), though within the reference range, was suboptimal for mucosal health given the increased demands of lactation in a vegetarian. A randomised controlled trial found B12 supplementation reduced RAS recurrence regardless of baseline level [7], while dietary B12/folate intake is reduced in RAS patients compared with controls [8]. Concurrent deficiencies of haemoglobin, iron, B12, and folate correlate with RAS severity [9], and ferritin deficiency is significantly more common in young and middle-aged women with RAS [10]. The patient's ferritin (30 ng/mL) likely reflected insufficient iron stores for mucosal regeneration during lactation, compounded by a vegetarian diet limiting both iron and B12 intake.

The uncritical interpretation of HSV-1 IgG seropositivity caused diagnostic harm, delaying appropriate treatment and contributing to the lesion's dramatic progression. Acyclovir was started solely on an incidental IgG titre, without IgM testing or clinical correlation, resulting in a seven-day delay during which the lesion grew tenfold. Active herpetic disease requires IgM positivity, rising titres, or direct viral detection; elevated IgG with absent IgM confirms prior exposure, not reactivation, and the gold standard for diagnosing active HSV disease remains clinical recognition with direct viral confirmation rather than serology alone [11,12]. This case illustrates a pattern of serological overinterpretation that clinicians must guard against when evaluating progressive, atypical oral ulceration.

The dramatic corticosteroid response is consistent with evidence identifying systemic corticosteroids as the most effective intervention for severe major RAS, with improvement typically evident within 48-72 hours [13]. Regression of a six-centimetre exophytic lesion within 72 hours of starting prednisolone confirmed its inflammatory pathogenesis and served as therapeutic proof-of-diagnosis where biopsy was appropriately deferred. Long-term prophylaxis focused on nutritional optimisation with zinc, folic acid, and vitamin B12, alongside trigger avoidance including Sodium Lauryl Sulphate (SLS)-free toothpaste, since SLS is known to disrupt the oral mucosal barrier and precipitate aphthous episodes in susceptible individuals.

CONCLUSION(S)

The present case underscores that isolated HSV-1 IgG seropositivity must never be equated with active herpetic infection, since acting on serology alone delayed appropriate treatment and allowed a small aphthous ulcer to progress into an exophytic six-centimetre mass. It further demonstrates that major RAS can be precipitated or exacerbated by the hormonal transitions of late pregnancy, early postpartum, and active lactation, compounded by borderline vitamin B12 and low-normal ferritin in a vegetarian mother with increased lactational demands. The dramatic regression achieved within 72 hours of systemic corticosteroid therapy, with complete epithelialisation by day 21 and no recurrence at three months, confirms the immune-mediated nature of the lesion and affirms systemic corticosteroids as the treatment of choice for severe major RAS. Clinicians managing atypical, progressive oral ulceration in peripartum women should correlate serology carefully with clinical findings, screen for correctable nutritional deficiencies, and consider hormonally driven immune dysregulation before pursuing invasive investigation. Early recognition of this pattern can shorten time to effective therapy and avoid unnecessary antiviral treatment and biopsy.

REFERENCES

[1] Neville BW, Damm DD, Allen CM, Chi AC. Oral and Maxillofacial Pathology. 4th ed. St Louis: Elsevier; 2016.
[2] Slebioda Z, Szponar E, Kowalska A. Etiopathogenesis of recurrent aphthous stomatitis and the role of immunologic aspects: Literature review. Arch Immunol Ther Exp (Warsz). 2014;62(3):205-15.
[3] Lewkowicz N, Kur B, Kurnatowska A, Tchorzewski H, Lewkowicz P. Expression of Th1/Th2/Th3/Th17-related genes in recurrent aphthous ulcers. Arch Immunol Ther Exp (Warsz). 2011;59(5):399-406.
[4] Mor G, Cardenas I. The immune system in pregnancy: A unique complexity. Am J Reprod Immunol. 2010;63(6):425-33.
[5] Sunardi SU, Rahardjo TBW, Baziad A, Auerkari EI. The role of estrogen receptor beta on severity of recurrent aphthous stomatitis (RAS). J Int Dent Med Res. 2017;10:711-14.
[6] McCullough MJ, Abdel-Hafeth S, Scully C. Recurrent aphthous stomatitis revisited; clinical features, associations, and new association with infant feeding practices? J Oral Pathol Med. 2007;36(10):615-20.
[7] Volkov I, Rudoy I, Freud T, Sardal G, Naimer S, Peleg R, et al. Effectiveness of vitamin B12 in treating recurrent aphthous stomatitis: A randomized, double-blind, placebo-controlled trial. J Am Board Fam Med. 2009;22(1):9-16.
[8] Kozlak ST, Walsh SJ, Lalla RV. Reduced dietary intake of vitamin B12 and folate in patients with recurrent aphthous stomatitis. J Oral Pathol Med. 2010;39(5):420-23.
[9] Sun A, Chen HM, Cheng SJ, Wang YP, Chang JYF, Wu YC, et al. Significant association of deficiencies of haemoglobin, iron, vitamin B12, and folic acid and high homocysteine level with recurrent aphthous stomatitis. J Oral Pathol Med. 2015;44(4):300-05.
[10] Bao ZX, Shi J, Yang XW, Liu LX. Hematinic deficiencies in patients with recurrent aphthous stomatitis: Variations by gender and age. Med Oral Patol Oral Cir Bucal. 2018;23(2):e161-e167.
[11] Arduino PG, Porter SR. Herpes simplex virus type 1 infection: Overview of relevant clinico-pathological features. J Oral Pathol Med. 2008;37(2):107-21.
[12] Fatahzadeh M, Schwartz RA. Human herpes simplex virus infections: Epidemiology, pathogenesis, symptomatology, diagnosis, and management. J Am Acad Dermatol. 2007;57(5):737-63.
[13] Warrier A, Sruthi M, Anbarasi K. Comprehensive management of major aphthous ulcer. BMJ Case Rep. 2021;14(5):e241010.

PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of Emergency Medicine, Smt. B. K. Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth Deemed to be University, Vadodara, Gujarat, India.
2. Consultant Dermatologist, Department of Dermatology, Aastha Hospital, Patan, Gujarat, India.
3. Associate Professor, Department of Critical Care Medicine, Smt. B. K. Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth Deemed to be University, Vadodara, Gujarat, India.
4. Assistant Professor, Department of Anaesthesiology, Ananya College of Medicine and Research, Vadodara, Gujarat, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Mittal Rohan Sindhav,
1388, Akashgagana, Behind Akashvani Radio Station, Vadodara-390001, Gujarat, India.
E-mail: mittalmeghraj7@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Jun 03, 2026
- Manual Googling: Jul 23, 2026
- iThenticate Software: Jul 25, 2026 (1%)

ETYMOLOGY: Author Origin

EMENDATIONS: 8

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **May 15, 2026**
Date of Peer Review: **Jun 15, 2026**
Date of Acceptance: **Jul 28, 2026**
Date of Publishing: **Sep 01, 2026**