



Painful ulcers on gingiva, tongue, and buccal mucosa

We had one clue that helped explain the ulcers in the patient's mouth: He had an accompanying low-grade fever.

A 29-YEAR-OLD MAN with no prior history of mouth sores abruptly developed many 1- to 1.5-mm blisters on the gingiva (FIGURE 1A), tongue (FIGURE 1B), and buccal mucosa (FIGURE 1C), which evolved into small erosions accompanied by a low-grade fever 5 days prior to presentation. The patient had no history of

any dermatologic conditions or systemic illnesses and was taking no medication.

- WHAT IS YOUR DIAGNOSIS?
- HOW WOULD YOU TREAT THIS PATIENT?

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Dr. Brodell has received consultant fees from Intraderm Pharmaceuticals and has performed clinical trials for Genentech, Novartis, Glaxo-Smith-Kline, Corrona Registry, and Janssen Biotech, Inc. Dr. Fitzpatrick reported no potential conflict of interest relevant to this article.

FIGURE 1

Painful mouth ulcers



Our patient had painful, irregular ulcerations with a yellowish membrane and erythematous halo on the gingiva (A), lesions on the ventral surface of the tongue (B), and multiple grouped ulcerations on the buccal mucosa (C).



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➤ Utilize PCR for the diagnosis of herpetic gingivostomatitis because of its sensitivity, specificity, and rapid turnaround time.

Diagnosis: Acute primary herpetic gingivostomatitis

Herpes simplex virus (HSV) is the causative agent for acute primary herpetic gingivostomatitis.¹ HSV-1 is primarily responsible for oral mucosal infections, while HSV-2 is implicated in most genital and cutaneous lower body lesions.¹ Herpetic gingivostomatitis often presents as a sudden vesiculoulcerative eruption anywhere in the mouth, including the perioral skin, vermilion border, gingiva, tongue, or buccal mucosa.² Associated symptoms include malaise, headache, fever, and cervical lymphadenopathy; however, most occurrences are subclinical or asymptomatic.²

■ **A diagnosis that's more common in children.** Primary HSV occurs in people who have not previously been exposed to the virus. While it is an infection that classically presents in childhood, it is not limited to this group. Manifestations often are more severe in adults.¹

Following an incubation period of a few days to 3 weeks, the primary infection typically lasts 10 to 14 days.^{1,2} Recurrence is highly variable and generally less severe than primary infection, with grouped vesicles often recurring in the same spot with each recurrence on the vermilion border of the lip. Triggers for reactivation include immunosuppression, pregnancy, fever, UV radiation, or trauma.^{1,2}

Differential includes other conditions with mucosal lesions

Acute herpetic gingivostomatitis must be distinguished from other disease processes that cause ulcerative mucosal lesions.

■ **Aphthous stomatitis** (canker sores) is the most common ulcerative disease of the oral mucosa.³ It presents as painful, punched-out, shallow ulcers with a yellowish gray pseudomembranous center and surrounding erythema.³ No definitive etiology has been established; however, aphthae often occur after trauma.

■ **Herpangina** is caused by coxsackie A virus and primarily is seen in infants and children younger than 5.⁴ The papulovesicular lesions primarily affect the posterior oral cavity, including the soft palate, anterior tonsillar pillars, and uvula.⁴

■ **Allergic contact dermatitis** is precipitated by contact with an allergen and

presents with pain or pruritus. Lesions are erythematous with vesicles, erosions, ulcers, or hyperkeratosis that gradually resolve after withdrawal of the causative allergen.⁵

■ **Pemphigus vulgaris.** Oral ulcerations of the buccal mucosa and gingiva are the first manifestation of pemphigus vulgaris in the majority of patients, with skin blisters occurring months to years later over areas exposed to frictional stress.⁶ Skin sloughs may be seen in response to frictional stress (Nikolsky sign).⁶

The new Dx gold standard is PCR

Acute herpetic gingivostomatitis usually is diagnosed by history and hallmark clinical signs and symptoms.¹ In this case, our patient presented with a sudden eruption of painful blisters on multiple areas of the oral mucosa associated with fever. The diagnosis can be confirmed by viral culture, serology with anti-HSV IgM and IgG, Tzanck preparation, immunofluorescence, and polymerase chain reaction (PCR).¹ Viral culture has been the gold standard for mucosal HSV diagnosis; however, PCR is emerging as the new gold standard because of its unrivaled sensitivity, specificity, and rapid turnaround time.^{7,8} Specimens for PCR are submitted using a swab of infected cells placed in the same viral transport medium used for HSV cultures.

Our patient's culture was positive for HSV-1.

Prompt use of antivirals is key

Treatment of acute HSV gingivostomatitis involves symptomatic management with topical anesthetics, oral analgesics, and normal saline rinses.¹ Acyclovir is an established therapy; however, it has poor bioavailability and gastrointestinal absorption.¹ Valacyclovir has improved bioavailability and is well tolerated.¹ For primary herpes gingivostomatitis, we favor 1 g twice daily for 7 days.¹ Our patient responded well to this valacyclovir regimen and healed completely in 1 week. **JFP**

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CASE REPORT

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LETTERS

Dismantling the opioid crisis

Dr. John Hickner's editorial, "Doing our part to dismantle the opioid crisis" (*J Fam Pract* 2019;68:308) had important inaccuracies.

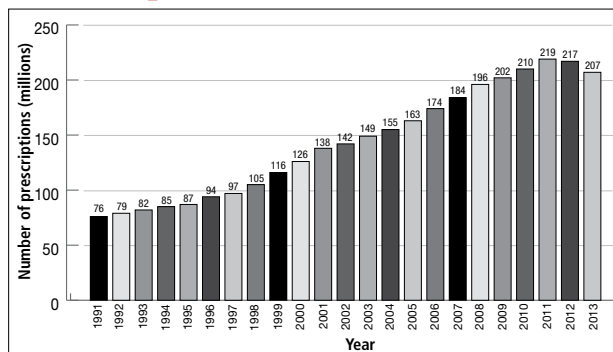
The Joint Commission, for which I serve as an executive vice president, did not "dub pain assessment the 'fifth vital sign.'" The concept of the fifth vital sign was developed by the American Pain Society in the 1990s.¹ It gained national attention through a Veterans Health Administration initiative in 1999.² And in 2001, the Joint Commission (then the Joint Commission on Accreditation of Healthcare Organizations or JCAHO) issued its Pain Standards.

Dr. Hickner wrote that the push to assess for pain as the fifth vital sign was a central cause of the opioid epidemic; however, this is contrary to published data on the epidemic. Total opioid prescriptions had been steadily increasing in the United States for at least a decade before the Pain Standards went into effect in 2001 (FIGURE).³ Between 1991 and 1997, the number of prescriptions increased from 76 million to 97 million. The rate of increase from 1997 to 2011 appears to have been more rapid, which is likely due to the 1995 approval of the new sustained-release opioid OxyContin and the associated aggressive marketing campaigns to physicians.

Your readers should know that we, at the Joint Commission, are also "doing our part to dismantle the opioid crisis." In 2016, we completely revised our Pain Standards, adding new criteria to help address the epidemic. Some

FIGURE

Opioid prescriptions dispensed by US retail pharmacies, 1991-2013³



adjustments include: requiring improved availability of nonpharmacologic therapy, encouraging engagement of patients in pain management plans, enhancing accessibility of Physician Drug Monitoring Program tools, and monitoring opioid prescribing.

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