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Spotlight on acute oral ulcers in children

Ioanina Parlatescu[1], Dragos Epistatu[2], Vlad Dima[3], Roxana Bohiltea[4,5], Valentin Varlas4,5

[1]Oral Medicine Department, Faculty of Dental Medicine, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[2]Department of Radiology, Faculty of Dental Medicine, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[3]Department of Neonatology, Filantropia Clinical Hospital, Bucharest, Romania
[4]Department of Obstetrics and Gynecology, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania
[5]Department of Obstetrics and Gynecology, Filantropia Clinical Hospital, Bucharest, Romania

ABSTRACT

Acute oral ulcers in children are painful lesions associated or not with other general symptoms. The etiology is variable. The anamnesis details regarding the history of the present lesion, accompanying general symptoms, and associated medical diseases are the basis for a correct diagnosis. Reactive lesions are the most frequently encountered in children. The treatment is usually topical and aims to reduce the symptoms and to heal the lesion.

Keywords: oral mucosa, ulcers, children

INTRODUCTION

Oral mucosa ulcerations in pediatric patients are commonly encountered in primary care of clinical practice of dental and general practitioners, pediatrics, otolaryngologists. The estimated prevalence of oral ulcers in children is 9% [1]. The primary clinical diagnosis has a vast variability, but the associated general and local symptoms and signs can guide and focus the diagnosis. Both presences of child-related pain and the refusal of food intake determine the parents to seek medical advice.

Pathologically, the oral ulcerations are breaks or disruptions of the mucosa continuity, affecting the epithelium's full-thickness through the basal membrane and into the underlying connective tissue. The surface has a whitish- yellow color, and it is covered by fibrin, a necrotic mass, or the roof of the blister or vesicle [1]. The ulcer is considered primary if it is not preceded by other lesions and secondary if the ulcer is preceded by vesicles or bullae [2]. There are differences regarding the causes of oral ulcers in adults and children. In children, the oral ulcers etiology comprises viral infections, autoimmune or allergic causes, local traumatisms, systemic disorders such as bowel diseases or vitamin deficiencies, so the differential diagnosis is broad.

As part of the diagnostic process, the anamnestic data may detect the presence of local trauma, a repetitive pattern of lesions, or systemic diseases. Depending on the length of the condition, oral ulcers are divided into two main types: acute ulcers that have a sudden onset and last up to 2 weeks and chronic ulcers that have been evolving for more than 2 weeks. This classification is unanimously accepted, although there was no international consensus to establish it [3]. Depending on the number of lesions, ulcerations can be single or multiple. Unique lesions can be traumatic or aphthae, while multiple lesions can be caused by infections or general conditions. This narrative article describes the most common acute oral ulcers based on their main clinical features.

Corresponding author:
Dragos Epistatu
E-mail: dragos.epistatu@umfcd.ro

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REACTIVE LESIONS

Most oral ulcers are caused by trauma [4]. In children, most frequent is physical trauma and rare chemicals. The local trauma is connected with sharp edges of the tooth (Figure 1), dental appliances, rough brushing, burns with hot food or drinks, or even unintentional injuries because of accidental falls. These ulcerations may be noticed after dental treatment until complete remission of anesthesia if the child is not supervised [5].

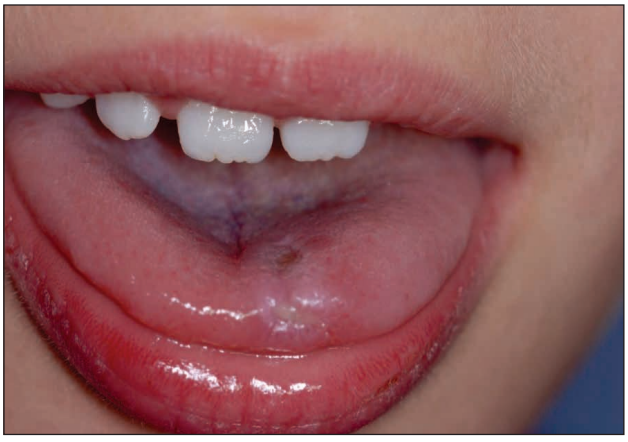


FIGURE 1. Ulcerative lesion of the tongue caused by repetitive rubbing motion with the tooth edge

Clinical it is a unique ulcer with slightly raised white, keratotic margins located on the buccal mucosa, tongue, or lower lip [6]. The discussion with the child and the parents, along with the location of the ulcer, is useful to detect the causative traumatic factor. The parafunctional habits could raise difficulties in diagnosis, mainly if the parents did not observe the habit previously. Chemical-induced oral ulcers are connected with the prolonged topical exposure of the substances such as acetylsalicylic acid or oral hygiene products [3]. The removal of the traumatic factor favors the remission of the ulcer in a maximum of 2 weeks [5]. Its persistence over this period calls for a more detailed evaluation such as general investigations and even a histopathological evaluation.

THE APHTHOUS ULCERS

The "canker sores" or aphthous ulcers are lesions with typical clinical and morphological features distinct from other oral ulcerations. They are painful ulcerations that appear on the mobile mucosa, not on the mucosa that covers the bone. The lesions can be solitary or multiple [6]. Most often, they have an erythematous area at the edge of the lesion. Vesicles or blisters do not precede the onset of ulceration.

In some cases, an erythematous area can precede the appearance of aphthae. Depending on the size, they are divided into minor aphthous ulcers with a diameter of less than 1 cm, major ulcers over 1 cm, and herpetiform ulcers of 1 mm in diameter (Figure 2). The most common are minor aphthae which have an erythematous halo and usually heal in 10 to 14 days. The herpetiform ulcers are multiple on the surface (over 10 lesions).

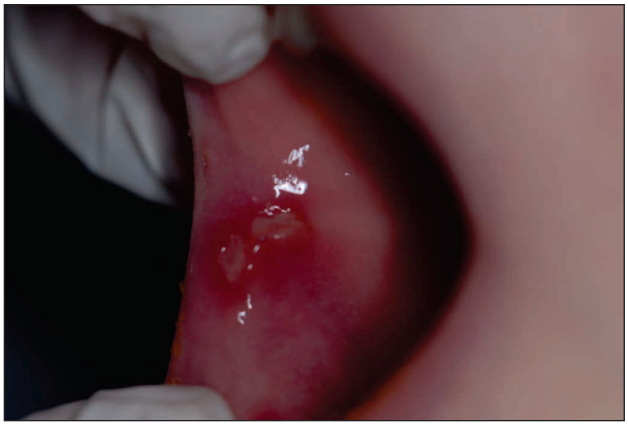
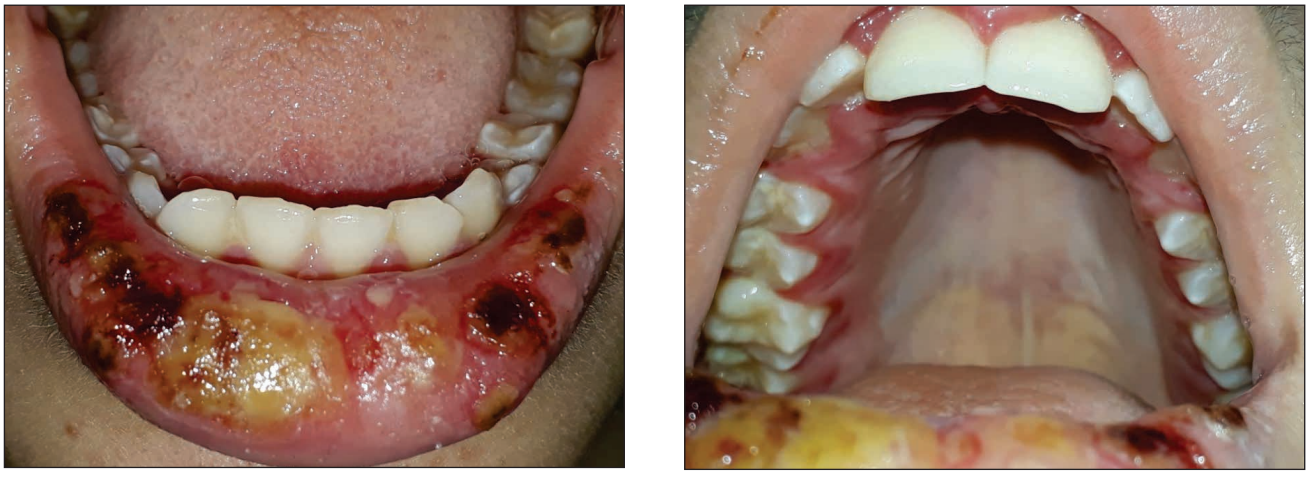


FIGURE 2. Aphthous ulcers of the right buccal mucosa. The margins are erythematous

The aphthae etiology is multifactorial, and the pathogenesis is immunologically mediated. The recurrent aphthous lesions produce functional disturbances and are associated with higher frequencies of deficiencies in hemoglobin level, serum iron, vitamin B12, folic acid, and vitamin D [7,8]. The treatment aims to reduce the symptoms, heal the lesions, and increase the periods between outbreaks for recurrent aphthous ulcers [9]. Topical corticosteroids, anesthetics, and antiseptics such as chlorhexidine are used in the therapy for aphthous lesions. Caution is recommended in dosing, and the usage frequency of local corticosteroids in an adherent vehicle is limited to the lesional area [3].

THE VIRAL ULCERS

Oral ulcers of viral cause are preceded by vesicles. The vesicles break quickly, and the secondary lesions are multiple painful ulcers. Herpes virus simplex type I and II produce the primary infection named primary herpetic gingivostomatitis and also secondary infection. The infection with herpetic virus type I is the most common cause of oral ulcers in children from 6 months to 5 years [1]. The primary herpetic gingivostomatitis has the onset with fever, malaise, adenopathy, and then a widespread vesicular and ulcerative eruption which involves any area of the oral mucosa, including the lips (Figure 3 A,B). The gingival involvement (herpetic gingivitis) presents in addition to vesicles and erythematous and swelling of the interdental papillae and fixed gingiva. Severe forms of herpetic infections are signs of immunosuppression and raise the need for further



A B

FIGURE 3. Primary herpetic gingivostomatitis in a 12 years old child. **A.** ulcerative lesions covered with crusts on the lower lip. **B.** Herpetic gingivitis

investigations such as a complete blood count and HIV test [4]. In most children, the eruption is self-limited in 5 to 7 days [6]. The therapy includes acyclovir used in local or systemic, or other antivirals.

The primary infection of Epstein-Barr virus (type III of herpes virus) causes infectious mononucleosis and superficial ulcers of the oral mucosa [4]. On the oral mucosa, the infection with cytomegalovirus can produce ulcers with long duration in immunodeficient individuals [4].

Herpangina is an acute respiratory tract infection caused by Coxsackie virus A and Enterovirus-A71 frequent in children under 2 years [6]. After a sudden onset of fever and dysphagia, the clinical specific signs are detectable. These are enanthem, and multiple oral ulcers preceded by vesicles located on the oro-pharynx and tonsils [10].

Hand, foot and mouth diseases are caused by the infection with Coxsackie virus. In North America, most hand, foot, and mouth disease cases affect children under 10 years old [11]. The oral ulcers are accompanied by cutaneous lesions like maculopapular or papulovesicular rash on hands and feet, asthenia, and low-grade fever. Oral, topical supportive measures favor the healing, which is usually in 10 days [11].

ALLERGIC ETIOLOGY

Erythema multiforme or polyforme is an immune-mediated mucocutaneous disease which has pathogenic mechanism types III and IV of hypersensibility [9]. Its onset can be precipitated by a viral, bacterial (*Mycoplasma pneumoniae)*, or a pharmacologic antigen [12]. A herpetic eruption can be followed by an episode of erythema multiforme. The oral mucosal lesions are present in the minor and the major form of erythema multiforme (minor form affects the skin and one mucosa, the major form affects the skin and other mucous membranes) [13]. Oral erythema, erosions, ulcers, and bullous lesions are present with a rapid onset. The skin lesions are typical targets with bullae in the central zone [14]. The disease has an acute, self-limited evolution. The diagnosis is based on the clinical features and detection and removal of the causative trigger. The treatment depends on the severity of the lesions, topical anti-inflammatories substances bring relief and help the healing.

ORAL ULCERS AND SYSTEMIC CONDITIONS

In children, inflammatory bowel diseases such as Crohn's disease, ulcerative colitis, and celiac disease associate oral ulcerative lesions, which can be the first clinical sign of the disease [9]. Their pathogenesis is in connection with haematinic deficiencies in celiac disease and Crohn's disease. There are different clinical aspects of ulcers in each disease. In celiac disease, oral ulcers have the same appearance as aphthous ulcers. In Crohn's disease, there are chronic linear ulcers with mucosal tags in the buccal vestibule. In ulcerative colitis, the lesions are termed pyostomatitis vegetans and present aphthous ulcers with multiple pustules [7].

The hematological conditions associated with oral ulcers are anemias, leukemias, lymphomas, multiple myeloma, and neutropenia of any cause. In these cases, the oral ulcers have the features of aphthous lesions [3,7].

OTHER CONDITIONS

Necrotizing sialometaplasia is the presence of a deep necrotic ulcer located on the hard or soft pal-

ate caused by ischemia. It is a rare benign disease favored by direct trauma, nervous bulimia, or smoking and alcohol use [4,6].

Oral mucositis characterized by erythema, swelling, and ulcers can be produced by radiotherapy or chemotherapy [3] at 5 to 7 days from the initiation of the treatment [6].

Acute necrotizing ulcerative gingivitis is a non-specific inflammatory, infectious disease encountered in immunocompromised individuals [8]. It starts in the interdental papillae and extends to the gingiva and neighboring mucosa. The lesions are remit after general antimicrobial treatment and local hygienisation.

CONCLUSIONS

Oral acute ulcers in children have a broad range of causes and pathogenesis. Traumatic, viral, immune, or allergic etiology can be determined. The basis for the diagnosis is the clinical features, the exhausted lesional history, and the general associated signs and symptoms.

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