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1. Introduction

While odontogenic infections are daily encountered in dental and oral and maxillofacial surgery practices, some practitioners may be unfamiliar with the wide range of other infections of diverse etiology, some of them relatively uncommon, or even rare. Patients so affected come to their attention either through referrals from primary care providers or due to patients’ uncertainty about where to seek help for diseases manifesting themselves in the orofacial area. Also in hospital environment, where majority of oral and maxillofacial surgeons practice, one regularly receives requests for consultations about patients who need interdisciplinary cooperation despite the fact that their conditions primarily belong to the sphere of specializations like ENT surgery, ophthalmology, dermatology and others. The purpose of this chapter is to provide an update on such conditions and demonstrate the ways oral and maxillofacial surgeon can participate in their diagnosis and management.

2. Facial skin infections

2.1. Impetigo

Impetigo is a highly contagious infection of the superficial epidermis. It is usually caused by *Staphylococcus aureus* or Group A streptococci [1]. The form of impetigo that penetrates deeper into the dermis and may leave a scar is called ecthyma. Impetigo occurs most frequently among economically disadvantaged children aged 2–5 years, although older children and adults may also be afflicted under conditions of poor hygiene, high humidity and warm temperatures. Prospective studies of streptococcal impetigo have demonstrated that the responsible microorganisms initially colonize the unbroken skin. Inoculation of surface organisms into the skin happens after a mean interval of 10 days by abrasions, minor trauma, or insect bites [2].
2.1.1. Clinical presentation

The most frequent locations of impetigo are the face and extremities. Two clinical forms are recognized: non-bullous and bullous. The lesions of non-bullous impetigo begin as papules that rapidly evolve into vesicles surrounded by an area of erythema. Then they become pus-tules that gradually enlarge and break down over a period of 4–6 days to form characteristic golden yellow crusts [3]. (Figure 1)

![Figure 1. Non-bullous impetigo of paranasal skin with a central denuded area and peripheral crust.](image)

Bullous lesions appear initially as superficial vesicles that rapidly enlarge to form flaccid bullae filled with clear yellow fluid, which later becomes darker, more turbid, and sometimes purulent. The bullae may rupture, often leaving a thin brown crust resembling lacquer [4]. The lesions heal slowly and leave depigmented areas. Bullous impetigo is caused by strains of *S. aureus* that produce a toxin causing cleavage in the superficial skin layer. In the past, nonbullous lesions were usually caused by streptococci. Now, most cases are caused by staphylococci alone or in combination with streptococci [5].

2.1.2. Treatment

The therapeutic approach to impetigo depends on the number of lesions, their extent and location (in a face proximity to eyelids or mouth), and the need to limit spread of infection to other individuals. The best topical agent is mupirocin, although resistance has been described; other agents, such as bacitracin and neomycin, are considerably less effective. Topical therapy with mupirocin is equivalent to oral systemic antimicrobials and may be used when lesions are limited in number. Patients who have numerous lesions or who are not responding to topical agents should receive oral antibiotics effective against both *S. aureus* and *Streptococcus pyogenes*. The preferred antibiotics are penicillinase resistant penicillins or first-generation cephalosporins, because *S. aureus* currently accounts for most cases of bullous impetigo, as well as for a substantial portion of nonbullous infections [3].
2.2. Folliculitis

Folliculitis is defined as purulent infection of hair follicles limited to the epidermis. Predisposing factors are hot and humid conditions, obesity, diabetes mellitus, long-term antibiotic or corticosteroid use, and immunosuppression [1].

2.2.1. Clinical presentation and etiology

Folliculitis is characterized by clusters of small, erythematous papules or pustules, usually in body areas prone to friction and heavy perspiration. The face belongs to the most often involved areas. The most common form of folliculitis is sycosis barbae a staphylococcal infection related to shaving. (Figure 2)

![Figure 2. Sycosis barbae with peripheral cellulitis.](image)

The fungal counterpart of sycosis barbae is *tinea barbae* caused by various dermatophytes. Other possible etiological agents include *Enterobacteriaceae* (often associated with prolonged antibiotic therapy), *Pseudomonas aeruginosa* (associated with hot tubs and wet suits) [6], *Malassezia furfur*, herpes simplex virus, varicella-zoster virus and *Demodex* mites. Non-infectious folliculitis include eosinophilic folliculitis thought to be an autoimmune process directed against the sebocytes [7] and a papulopustular follicular eruption after treatment with epidermal growth factor receptor (EGF-R) inhibitors [8].

2.2.2. Treatment

Uncomplicated superficial folliculitis may respond to improved hygiene supported by use of antibacterial soap. If this simple measure is not sufficient, topical antibiotic cream can be used. Refractory or deep infections may require administration of systemic antibiotics. Selection of appropriate antibiotic is based on knowledge of the common microorganisms involved in the particular type of infection before results of microbiology examination and antibiotic sensitivity tests are available. Herpetic folliculitis responds to oral antivirals (e.g.,...
valaciclovir). Eosinophilic folliculitis may respond to isotretinoin, metronidazole, UV-B phototherapy, indometacin or itraconazole [1]. Infectious folliculitis may progress to involve deeper layer of the dermis and finally spread to subcutaneous tissue.

2.3. Furuncle and carbuncle

Furuncle is purulent infection involving the hair follicle and extending to surrounding subcutaneous tissue. (Figure 3)

![Figure 3](image1.png)

**Figure 3.** Furuncle of the upper lip. Note progression of infection to right paranasal area.

Furuncles can occur anywhere on hairy skin. A carbuncle is the coalescence of several furuncles with pus draining from multiple follicular orifices. Carbuncles frequently develop on the nape and are more likely to be seen in diabetic patients [3]. In immunocompetent individuals, furuncles and carbuncles are usually caused by *S. aureus*. (Figure 4)

![Figure 4](image2.png)

**Figure 4.** Carbuncle of right cheek in an immunocompetent patient.
2.3.1. Clinical presentation

In the face, furuncles are frequently seen on the chin, upper lip and paranasal area. Each lesion consists of an inflammatory nodule and an overlying pustule through which hair emerges. Furuncles of the nasal vestibule can be insidious and not obvious upon cursory examination and their symptoms, namely swelling of upper lip and infiltrate of upper oral vestibule, can lead to false impression of odontogenic infection. In patients affected by a facial furuncle, fever and malaise are common. Lesions are extremely painful and they are surrounded by area of cellulitis and collateral edema. (Figure 5)

![Figure 5. Furuncle of the nasal vestibule referred with suspicion of an odontogenic abscess.](image)

2.3.2. Treatment

Small furuncles may burst and heal spontaneously [1]. Application of moist hot dressing can promote drainage. Also gentle removal of overlying crust and necrotic central plug can be helpful; however attempts to express purulent content should be discouraged. Conservative management is preferable and only rarely cases of furuncles or carbuncles progressing into subcutaneous abscess require incision and drainage. In the face, whenever possible, this should be done through intraoral route to avoid facial scarring. Systemic antibiotics are necessary in instances of substantial collateral cellulitis, alteration of general condition and signs of developing facial thrombophlebitis. This initial empirical therapy should be aimed at supposed staphylococcal etiology. Until recently, staphylococcal infections acquired outside of the healthcare setting have been frequently methicillin-sensitive and responsive to a wide range of antibiotics. Since 1980, methicillin-resistant staphylococcus aureus (MRSA) infections have been reported in community outbreaks. These organisms have been called community-acquired or community-associated MRSA, as opposed to hospital acquired MRSA [9]. Hospital acquired MRSA is usually resistant to at least three β-lactam antibiotics and is usually susceptible only to vancomycin, sulfamethoxazole, and nitrofurantoin. Com-


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