J Oral Pathol Med (2004) 33: 243–5

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CASE REPORT

Facial *Candida albicans* cellulitis occurring in a patient with oral submucous fibrosis and unknown diabetes mellitus after local corticosteroid injection treatment

Hsin-Ming Chen[1,2], Chiang-Ching Shih[3], K. Lawrence Yen[4], Sheng-Ming Wang[5], Ying-Shiung Kuo[1,2], Mark Yen-Ping Kuo[1,2,*], Chun-Pin Chiang[1,2,*]

[1]*School of Dentistry, College of Medicine, National Taiwan University,* [2]*Department of Dentistry, National Taiwan University Hospital,* [3]*Department of Medicine,* [4]*Division of Otolaryngology, Head and Neck Surgery, and* [5]*Department of Dentistry, Koo Foundation Sun Yat-Sen Cancer Center, Taipei, Taiwan*

Facial cellulitis caused by odontogenic bacterial infection is frequently encountered; however, facial cellulitis caused by *Candida albicans* infection is rarely found. A patient with oral submucous fibrosis (OSF) and unknown diabetes mellitus (DM) was treated in our out-patient dental clinic by biweekly submucosal injection of 40 mg triamcinolone acetonide into bilateral buccal mucosae plus forced mouth opening performed by the two hands of the clinician. The interincisal distance of the patient improved from 28 to 48 mm after four times of steroid injection. The symptoms and signs of OSF also improved markedly. Unfortunately, facial candidal cellulitis occurred 2 months after the last time of steroid injection treatment. The infection was cured by incision and drainage, intravenous administration of amphotericin B (100 mg once a day for a week), and an appropriate medical control of DM. No recurrence of facial cellulitis was found during the follow-up period of 18 months. To prevent the occurrence of facial cellulitis after a high-dose steroid therapy, some prophylactic procedures should be taken before the initiation of the steroid treatment.

J Oral Pathol Med (2004) **33**: 243–5

Keywords: candidiasis; cellulitis; corticosteroid; oral submucous fibrosis

Case report

A 50-year-old male patient with oral submucous fibrosis (OSF) was treated in our out-patient dental clinic by biweekly submucosal injection of 40 mg triamcinolone acetonide in 2 ml of 2% xylocaine into bilateral buccal mucosae plus forced mouth opening performed by the two hands of the clinician. The interincisal distance (the linear distance between the incisal edges of upper and lower central incisors) of the patient improved from 28 to 48 mm after four times of steroid injection. In addition, the symptoms and signs of OSF also improved markedly. Unfortunately, a swelling at the right cheek of the patient was found 2 months after the last time of steroid injection treatment (Fig. 1). The patient visited our hospital for treatment. A needle aspiration yielded pus that was sent to the laboratory for culturing. *Candida albicans* infection was confirmed subsequently. At the same time, blood chemistry revealed fasting plasma glucose of 287 mg/dl and 12.2% of HbA1c (glycosylated hemoglobin, normal level 4–7%) in total hemoglobin. Diabetes mellitus (DM) was diagnosed, although the patient's initial fasting plasma glucose level was found to be 116 mg/dl before the start of steroid treatment. As no concomitant oral mucosal and odontogenic bacterial infection was noted, the patient was treated with 400 mg fluconazole per day without the combination of antibiotics. Two days later, the infection did not improve and even got worse. Therefore, the patient was admitted into our ward and magnetic resonance imaging (MRI) was arranged. The MRI of head showed an abscess in the right cheek extending from the lower eyelid to the lower mandibular border. Furthermore, bilateral maxillary sinusitis (more severe on the right side than on the left side) was also noted (Fig. 2). Incision and drainage was performed and 100 mg amphotericin B once a day was given to the patient by intravenous route. After culturing, *C. albicans* was still detected in the pus obtained from the incision and drainage procedure. Functional endoscopic sinus surgery was performed for the right maxillary sinusitis. Laboratory culture of the pus aspirated from the right maxillary sinus revealed the presence of *Klebsiella pneumonia*, but no fungal organism was observed. One week after intravenous amphotericin B treatment, the symptoms and signs improved and the patient was discharged. Maintenance dose of oral antifungal drug was given to the patient for an additional 1 week. The patient's DM was controlled by a physician in our hospital

Correspondence: Dr Chun-Pin Chiang, Department of Oral Pathology and Oral Diagnosis, National Taiwan University, 1 Chang-Te Street, Taipei 100, Taiwan. Tel.: +886 223123456/ext. 6855. Fax: +886 223893853. E-mail: cpchiang@ha.mc.ntu.edu.tw
*These two authors are equally contributed in this paper.
Accepted for publication May 21, 2003

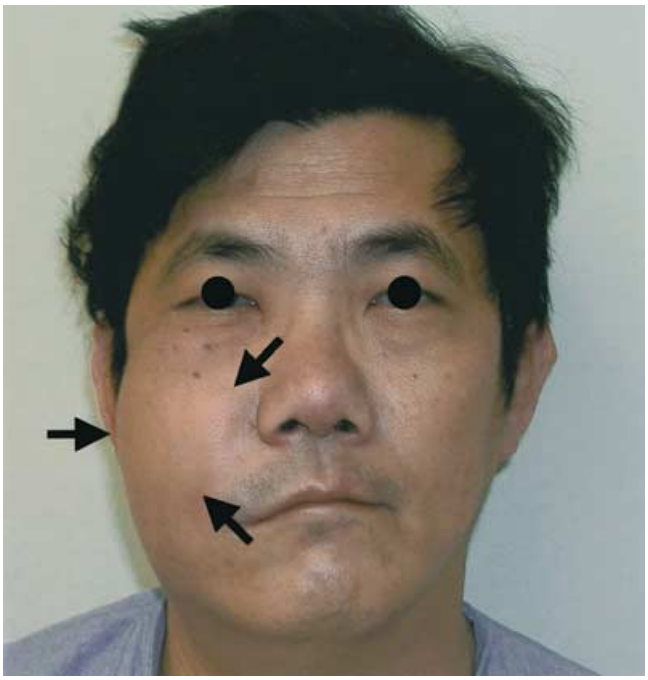

Figure 1 Clinical picture showed a swelling (arrows) at the right cheek of the patient.

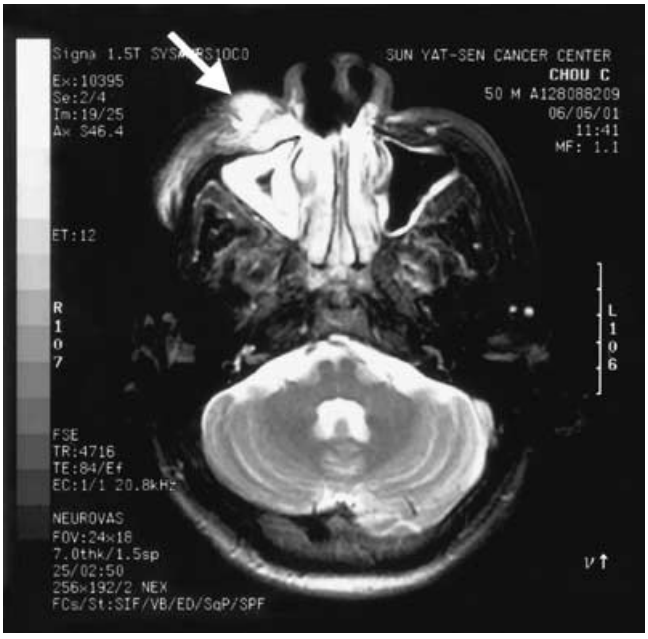

Figure 2 MRI of the head showed a nodule with increased signal intensity in the right cheek (arrow). Mucosal thickening with increased signal intensity was also noted at bilateral maxillary sinuses, and it was more severe on the right side than on the left side.

on a routine basis. No recurrence of the facial cellulitis was found during the follow-up period of 18 months.

Comments

In this case report, we presented a 50-year-old OSF patient with unknown DM who obtained a facial *C. albicans* cellulitis after four times of local steroid injection therapy. The infection was cured by incision and drainage, intravenous administration of amphotericin B, and an appropriate medical control of DM. Facial cellulitis caused by odontogenic bacterial infection is frequently encountered; however, facial cellulitis caused by *C. albicans* infection is rarely found. To the best of our knowledge, this was the first case reporting the complication of facial *C. albicans* cellulitis after local steroid injection in an OSF patient.

It was interesting to know why the patient got facial candidal cellulitis 2 months after the last time of steroid injection treatment. Candidiasis is caused predominantly by *C. albicans* and is the most common fungal infection in the oral cavity (1). As *C. albicans* is a weak pathogen, the occurrence of candidal infection needs local or systemic pre-disposing factors such as DM and systemic or local steroid therapy (2). Our OSF patient was treated by four times of biweekly submucosal injections of 40 mg triamcinolone acetonide (equivalent to 50 mg prednisolone) into bilateral buccal mucosae. This dosage of steroid treatment may cause local atrophic changes of buccal mucosa and may depress cell-mediated immunity of local tissue (3). The OSF oral mucosa originally has atrophic epithelium and decreased vascularity in the subepithelial connective tissue. These OSF mucosal changes plus local histologic and immunologic damages induced by steroids may predispose candidal infection. Actually, candidal infections of the tongue, pharynx and larynx have been reported after long-term use of steroid aerosols for the treatment of asthma (3). In addition, our patient was diagnosed having DM at the same time of facial candidal cellulitis. The local tissue factors and systemic factor like DM in this patient may predispose a subclinical candidal infection on the buccal mucosa surface in this patient during and after the steroid treatment. Microorganisms of *Candida* species are very common habitants in the oral cavity (1). Oral *Candida* species have been found in 70.8% of betel quid chewers and 69.2% of control subjects without betel quid chewing habits in a female Cambodian cohort (4). In addition, *C. albicans* is isolated from 27.1% of betel quid chewers (4). Thus, *C. albicans* may carry into the deep facial tissue by the needle or may invade into the deep facial tissue through the mucosal breaks caused by the needle or minor oral traumas. The latent microorganisms of *C. albicans* resided in the deep facial tissue may reactivate and proliferate under the conditions of immunosuppression and DM, finally resulting in abscess formation and facial candidal cellulitis in this patient.

The two other possible pathways for *C. albicans* infection were hematogenous dissemination from a systemic candidal infection and contiguous spread from a local candidal infection. In our patient, no symptoms and signs of a systemic candidal infection were found. In addition, facial, oral, radiographic, and laboratory examinations did not reveal any evidence of a local candidal infection from teeth, jawbones, maxillary sinuses, or skin. Therefore, it was of little possibility that the facial candidal cellulitis in this patient is caused by hematogenous dissemination from a systemic candidal infection or contiguous spread from a local candidal infection.

The use of glucocorticoid in nonphysiologic amounts over a long period will increase the risk of bacterial, viral, or

fungal infections. To prevent the steroid-induced complications, we suggest that any kinds of suspected oral candidiasis should be treated first before the start of a high-dose steroid therapy. Administration of 0.2% chlorhexidine gluconate as a mouthwash or of nystatin oral suspension in a dose of 0.6 million units four times a day for at least 1 week is strongly recommended. Nystatin should not be used in combination with chlorhexidine because it will form a nystatin–chlorhexidine salt that reduces the treatment individual effect of the two drugs each other (5). In those patients without any signs and symptoms of oral candidiasis, prophylactic procedures like thorough scaling and polishing of the teeth plus oral hygiene instruction were also recommended to eliminate the possible sources of infection prior to the institution of a high-dosage steroid therapy. In our dental clinic, these prophylactic procedures became routine works for the OSF patients who were planned to receive a high-dosage local steroid injection therapy. In addition, 0.2% of chlorhexidine gluconate solution for mouth rinse three times a day was given to the patient 7 days prior to the steroid injection procedure, and an additional oral rinse of the same solution just before the steroid injection procedure was used to minimize the microorganism load on the oral mucosal surface and to prevent the iatrogenic carriage of microorganisms into the deep facial tissues during the injection procedure.

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