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LETTER TO THE EDITOR

ADVERSE REACTION TO IRRIGATION WITH POVIDONE-IODINE AFTER DEEP-IMPACTED, LOWER THIRD MOLAR EXTRACTION

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Povidone-iodine is most commonly used worldwide because of its germicidal activity, relatively low irritancy or toxicity and low cost. Frequently, povidone-iodine is used as a topical antiseptic for treating and preventing wound infection. In rare cases skin irritation or iododerma-like eruption could represent possible adverse effects due to the oxidative effects of iodine and allergic hypersensitivity reaction. In this report we describe a case of a massive adverse reaction to the irrigation of surgical wound dehiscence with 10% povidone-iodine solution after deep-impacted, lower third molar extraction. This reaction was related to a central neurotrophic reflex involving three trigeminal branches and probably due to a peripheral chemical insult of mandible nerve. This adverse reaction determined a severe edema and diffuse skin lesions, involving the whole left side of the face mimicking an iododerma-like eruption. These violent symptoms were solved after 60 days. Furthermore, we report a small permanent skin scar in the zygomatic area and transient alterations of facial sensitivity on the affected side which completely disappeared in 6 months.

Iodine compounds have been widely used as antiseptics and disinfectants for the skin before surgical procedures. Povidone iodine (PI) is a water-soluble combination of molecular iodine and the solubilizing agent polyvinyl-pyrrolidone (PVP-iodine). PVP-iodine is most commonly used worldwide because of its germicidal activity, relatively low irritancy or toxicity and low cost. Frequently, povidone-iodine is used as a topical antiseptic for treating and preventing wound infection (1). The iodine compounds are available in aqueous solution, tincture, aerosol, ointment or foam (2, 3). In rare cases skin irritation or iododerma-like eruption could represent possible adverse effects, even if less frequent, due to the oxidative effects of iodine and allergic hypersensitivity reaction (4, 5). Sometimes 10% povidone iodine solution can cause primary irritant dermatitis and allergic contact dermatitis (6-8). In literature the local irrigation before the tooth extractions by means of 10% PI solution of the gingival sulcus was described which is considered an important source of bacteremia following dental procedures (9, 10). The iodine compound is considered most effective in prevention of post-treatment bacteremia compared to chlorexidine or hydrogen peroxide. PI is also topically used during subgingival instrumentation in order to improve the outcome of non-surgical periodontal therapy (11). PVP-iodine has been used in a mouthwash to inhibit the development of gingivitis, for subgingival

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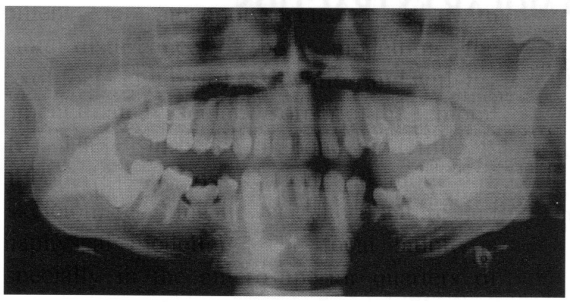

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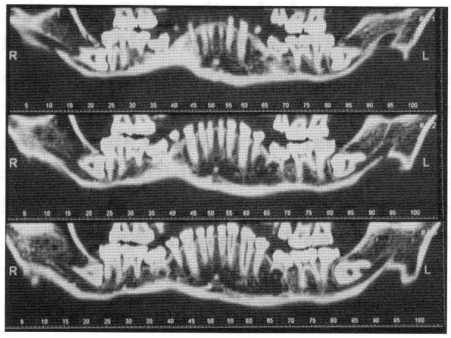

Fig. 1. a,b) *Orthopantomograpy and CT images for the correct evaluation before deep-impacted lower third molar extractions.*

irrigation in periodontal maintenance and in patients with recurrent periodontitis (12-14). In this report we describe a case of a massive adverse reaction to the irrigation of the dry alveolar socket with 10% povidone-iodine solution after impacted, lower third molar extraction.

Case report

At the Oral Surgery Division of the University of Naples "Federico II" we admitted a 31-year-old man without a past medical history of allergic events or other pathologies in order to perform an intervention for orthognatic surgery to correct a second skeletal class. Before the major surgery we planned the extraction of both lower third molars in deep impaction (Fig. 1). Following the lower, deep-impacted third molar extraction on the left side the patient had a surgical wound dehiscence (Fig. 2). After suture removal, irrigation with 10% povidone-iodine (PI) solution was used in order to clean and disinfect the post-extractive site and was repeated every week. After 2 weeks, during the irrigation with PI, unexpected and diffuse edema developed on the left side of the face involving the zygomatic, temporal, parietal and frontal area. The chin area was also involved in the edema. Severe alterations of neurological sensitivity on the affected facial side were immediately noted, related to the massive edema involving the facial nerve, associated to the transient damage on the lower and upper alveolar nerves with consequent hypoesthesia in the correspondent areas. The patient was admitted to hospital in order to begin a clinical follow-up and adequate medical therapy. A therapy with cortisone, antibiotics, enzymatic anti-inflammatories and B1, B6, B12 vitamins (Betametasone 4mg i.v./once in a day; Ceftriaxone 1g i.v./twice in a day; Escine 5mg i.v./twice in a day) was prescribed under gastric protection (Zantac i.v 50mg/day). Laboratory data showed elevated CK (1807 U/l) and AMS (1196 U/l) values, IgE value was modified (183 KU/l); the white blood cell count was within the normal limits. The ecography and CT images showed normal conditions of parotid gland and other facial structures; only a massive mandible lymphoadenopathy was noted. After 4 days the partial resolution of the edema began but multiple bullous lesions evolving in erosions and ulcerations were noted on the left side facial skin: these symptoms were compatible with an iododerma-like eruption. After 5 days, according to the consulting dermatologist, we prescribed the substitution of parenteral cortisone therapy with oral cortisone therapy (prednisone 25 mg, twice in a day) and the application of fucsid acid gel on the skin lesions (twice a day for 15 days). Work-up included lesion bacterial culture, which revealed normal skin flora, a Gram stain that showed no organisms and blood cultures that were negative. After consulting the neurologist we related this abnormal reaction to a central neurotrophic reflex of the trigeminal branches caused by a peripheral irritation of exposed mandibular nerve after the lower third molar extraction. After 15 days we also noted herpes-like lesions involving the hard and soft palate area. The ophthalmologic control pointed out left conjunctive hyperaemia, resolved after local therapy with nebicin

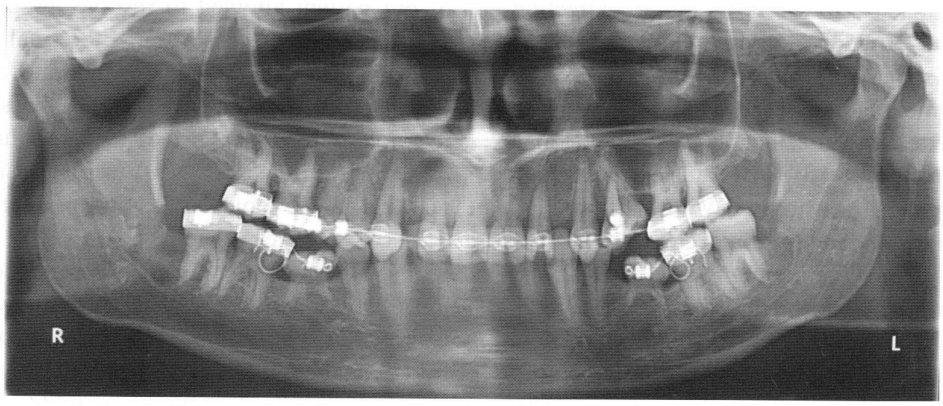

Fig. 2. *Radiographic control 20 days after the lower third molar extractions. On the left side we reported a dry alveolar pot-extractive socket.*

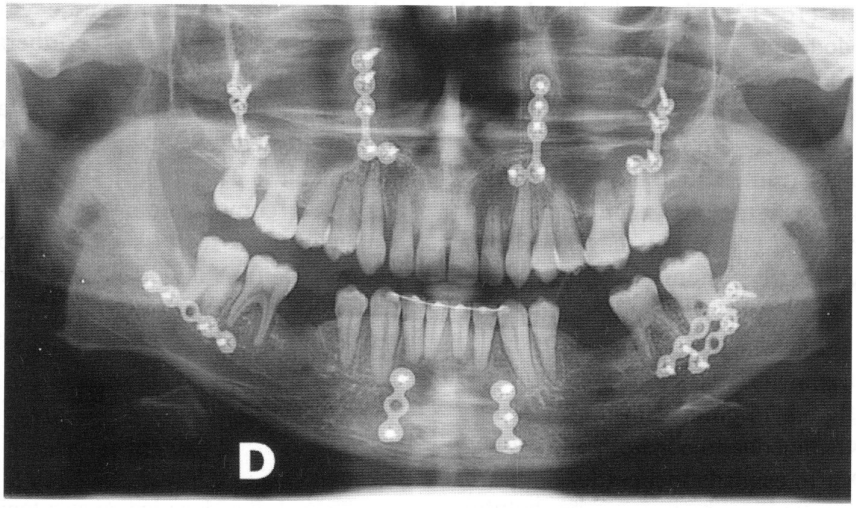

Fig. 3. *Radiographic image after the orthognatic surgery for a second skeletal class.*

for 2 weeks. After 30 days the altered laboratory data (CK, AMS and IgE) returned within normal limits. After 60 days the edema and skin lesions were completely resolved, only a small scar remained in the zygomatic area; while the alterations of facial sensitivity and the neurological damage to the lower and upper alveolar nerves completely disappeared after 6 months. After 1 year the same patient was submitted to the previously planned orthognatic surgery with excellent clinical and esthetic outcomes (Fig. 3).

DISCUSSION

The unexpected, unusual and massive reaction to the irrigation with 10% PI solution after the lower third molar extraction described in this report could be justified by the direct contact of the chemical agent with the exposed alveolar nerve after the deep-impacted lower third molar avulsion. We related this abnormal reaction to a central neurotrophic reflex of three trigeminal branches caused by an iodine peripheral irritation of exposed mandibular nerve

after deep-impacted lower third molar extraction. Following the povidone-iodine chemical insult to the mandibular nerve, a severe edema, several skin lesions, mimicking an iododerma-like eruption and herpes-like lesions at the hard and soft palate were reported in association of transient neurologic alterations of facial sensitivity. We related the high values of CK and AMS to the initial chemical necrosis of facial muscles; while the high value of IgE could be related to the afore-mentioned pseudo-allergic response. The diagnosis of iododerma rests on history and clinical findings, because no laboratory or histopathological finding is pathognomonic (15).

In literature patients have presented with polymorphous lesions that may mimic Sweet's syndrome, pemphigus vegetans, mycosis fungoides and blastomycosis (16, 17). The main histological features of iododerma are hyperkeratosis in epidermis, pseudoepitheliomatous hyperplasia and a dermal-epidermal separation with epidermal necrosis underlying abscess formation. A superficial and deep perivascular, interstitial and perifollicular infiltrate containing numerous neutrophils with abscess formations and eosinophils have also been noted. Histology of early lesions shows superficial microabscesses and macroabscesses within the epidermis and dermis. Upon discontinuation of iodide use, lesions resolve typically within 4-6 weeks, leaving only mild post-inflammatory pigment changes and possibly dermal atrophy. Treatment of iododerma has been attempted with systemic corticosteroids, which may be helpful. However, iododerma is generally a self-limited condition following cessation of iodide exposure (18). Iododerma is a rare skin eruption that is usually induced by the systemic use of iodide-containing radiographic contrast medium or treatment with oral potassium iodide therapy. Iododerma has also more rarely been reported to occur following topical application of iodine. Review of the literature yielded 18 cases of povidone-iodine-induced burn. This adverse effect of povidone-iodine application typically occurs when the povidone-iodine has not been allowed to dry or has been trapped under the body of a patient in a pooled-dependent position. All patients with a history of iododerma should avoid iodine in their diet, medications and radiographic studies. However, the adequate clinical management of our patient allowed to resolve the case without relevant consequences; in fact, the small permanent skin scar in the patient's zygomatic area is not serious, considering the violent and massive adverse reaction to the topical use of 10% PI solution.

Our case report shows a rare massive adverse reaction to the topical irrigation of surgical wound dehiscence after rinsing with 10% povidone-iodine solution after a left, deep-impacted, lower third molar extraction. This reaction was related to a central neurotrophic response involving three trigeminal branches and probably due to a peripheral chemical insult of exposed mandible nerve after the surgical dehiscence. This adverse reaction induced a severe edema and diffuse skin lesions, involving the whole left side of face, mimicking an iododerma-like eruption. These violent symptoms were resolved after 60 days. Furthermore, we report the small permanent skin scar in the zygomatic area, and transient alterations of facial sensitivity on the affected side which completely disappeared in 6 months. This report showed an unusual but aggressive complication which can occur when a chemical agent such as the 10% povidone-iodine solution comes into contact with the exposed mandible nerve after recent surgery. In this case the oral surgeon must avoid the use of this chemical agent in the oral cavity.

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