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CASE REPORT

Iododerma after Lugol use for pre-operative preparation of thyroidectomy

Iododerma após uso de lugol no pré-operatório de tireoidectomia

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Abstract

A 35-year-old man with hyperthyroidism reported the sudden occurrence of ulcerated and painful oral lesions, painful ulcerative-necrotic lesions on the face, trunk, upper and lower limbs and purpuric circinate lesions on the scalp, 6 days after starting the treatment with oral iodine. The clinical history, cutaneous-mucosal lesions, and histopathological findings support the diagnosis of iododerma secondary to oral iodine treatment. Iododerma is a rare disease caused by iodine exposure that can develop after oral or intravenous use, rarely after topical use of iodine and is often associated with systemic manifestations related to vasculitis, which can be fatal, as in the present case.

Keywords: Contrast media/adverse effects. Drug eruptions/diagnosis. Drug eruptions/etiology. Iodine/adverse effects.

Resumo

Um homem de 35 anos, com hipertireoidismo, relatou a ocorrência repentina de lesões orais ulceradas e dolorosas, lesões ulcerativas-necróticas dolorosas na face, tronco, membros superiores e inferiores e lesões circinadas purpúricas no couro cabeludo, seis dias após início do tratamento com iodo oral. A história clínica, as lesões cutâneo-mucosas e os achados histopatológicos apoiam o diagnóstico de iododerma secundária ao tratamento oral com iodo.

O iododerma é uma doença rara causada pela exposição ao iodo, desenvolvida após o uso oral ou intravenoso ou, raramente, após o uso tópico de iodo e muitas vezes está associada a manifestações sistêmicas relacionadas à vasculite, que podem ser fatais, como no presente caso.

Palavras-chave: Erupção por fármaco/diagnóstico. Erupção por fármaco/etiologia. Iodo/efeitos adversos. Meios de contraste/efeitos adversos.

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Introduction

Iododerma is a rare disease caused by systemic iodine exposure and rarely after its topical use[1,2]. Systemic exposure can occur when potassium iodine is used for the treatment of some diseases such as asthma, hyperthyroidism, chronic bronchitis and sporotrichosis, or when iodinated radiocontrast media is used[1,2]. This disease mainly affects patients with kidney disease, due to the difficulty in excreting iodine and, consequently, accumulation in the bloodstream[3,4].

The pathogenesis of iododerma is not clear. Some authors consider it as a late hypersensitivity reaction in individuals with repeated antigenic stimulation[1].

Case report

A 35-year-old male patient with hypertension and hyperthyroidism due to Graves' disease, submitted to subtotal thyroidectomy 15 years ago, was admitted to undergo total thyroidectomy due to refractory hyperthyroidism despite the use of propylthiouracil and methimazole. As a pre-operative treatment and according to his body weight (98 kg) he was given 90 mg/day of 5% lugol solution, a concentrated solution of potassium iodine (6 mg/drop, i.e., 15 drops). During hospitalization, on day 6 after onset of lugol solution, he developed a painful aphthous ulcer and necrotic lesions in the dorsal region of the tongue and in the vestibule of the lower lip (Fig. 1A). In 2 days, new purpuric lesions with a circinate pattern appeared on the scalp (Fig. 1B) associated with vesicopustular lesions (Fig. 1C) that evolved into round, painful ulcerative-necrotic lesions, on the face, trunk, upper and lower limbs (Fig. 1D). Laboratory tests revealed elevated transaminases (AST 161 U/L; ALT 619 U/L) and total bilirubin (6.91 mg/dL) due to elevation of both direct and indirect bilirubin (respectively, 3.45 and 3.46 mg/dL), and deterioration of renal function (creatinine 2.12 mg/dL, urea 72 mg/dL). Serologies for hepatitis B and C, HIV and syphilis were negative.

Histopathology of a vesicopustular lesion on the patient's thigh revealed an area of epidermal ulceration with an underlying dermal abscess containing mononuclear inflammatory cells, eosinophils and some neutrophils (Figs. 2A and B). Histopathology of a scalp lesion showed hyperkeratosis with epidermal hyperplasia, edema of the upper dermis, a dermal inflammatory reaction mainly around hair follicles and blood vessels, composed by mononuclear cells with an peripheral empty perinunclear halo, resembling fungal bodies in cryptococosis, eosinophils and neutrophils (Fig. 2C), and abscesses with mononuclear cells, eosinophils, some neutrophils and basophilic debris (Fig. 2D). Histopathological findings were compatible with iododerma.

Lugol solution was withdrawn at day 8 and methylprednisolone 1 mg/kg/day was started. The patient recovered significantly from skin and oral mucosa lesions. After 5 days, he developed upper digestive tract hemorrhage with melena which led to a progressive drop in hemoglobin level, soon followed by a severe hemorrhage, which led to refractory hypovolemic shock and death.

Discussion

The diagnosis of iododerma is undertaken based on the patient's history and clinical signs. Histopathology shows, in early lesions, superficial microabscesses and macroabscesses within the epidermis and dermis. Later, in the course of the eruption, ulceration, pseudoepitheliomatous hyperplasia, formation of horn pearls, interstitial superficial dermis infiltrate of eosinophils, and features of allergic vasculitis can develop[2]. Elevated blood or urine iodine levels and histologic findings are supportive of the diagnosis; however, they are not pathognomonic[1,5,6].

Clinically, iododerma presents with polymorphic lesions, such as pustules, papulopustules, vesicles, ulcers, vegetations, nodules, hemorrhagic, and urticarial lesions. The disease has been described on the face, trunk, and extremities[2,7]. Systemic reactions such as fever, abdominal pain, and mucous membranes hemorrhages have been described, namely in gastrointestinal tract hemorrhages as observed in our patient, and have been attributed to an underlying vasculitis, simulating periarteritis nodosa[7].

Histopathological findings may include mononuclear inflammatory infiltrate, especially neutrophils and acellular structures with an empty halo, similar to cryptococci, which lead to diagnostic difficulties in the context of suspected infectious disease[8].

Differential diagnosis includes folliculitis, blastomycosis, pyoderma gangrenosum, pemphigus vegetans, mycosis fungoides, tertiary syphilis, cutaneous metastasis, and sweet syndrome[1,9].

Treatment of iododerma involves removal of the causative agent, showing spontaneous resolution in approximately 4-6 weeks. Systemic corticosteroids, cyclosporine or even hemodialysis may be necessary in more severe or refractory cases[1,4,10,11].

In this article, we present the case of a young patient with a fatal outcome after a severe reaction to the use

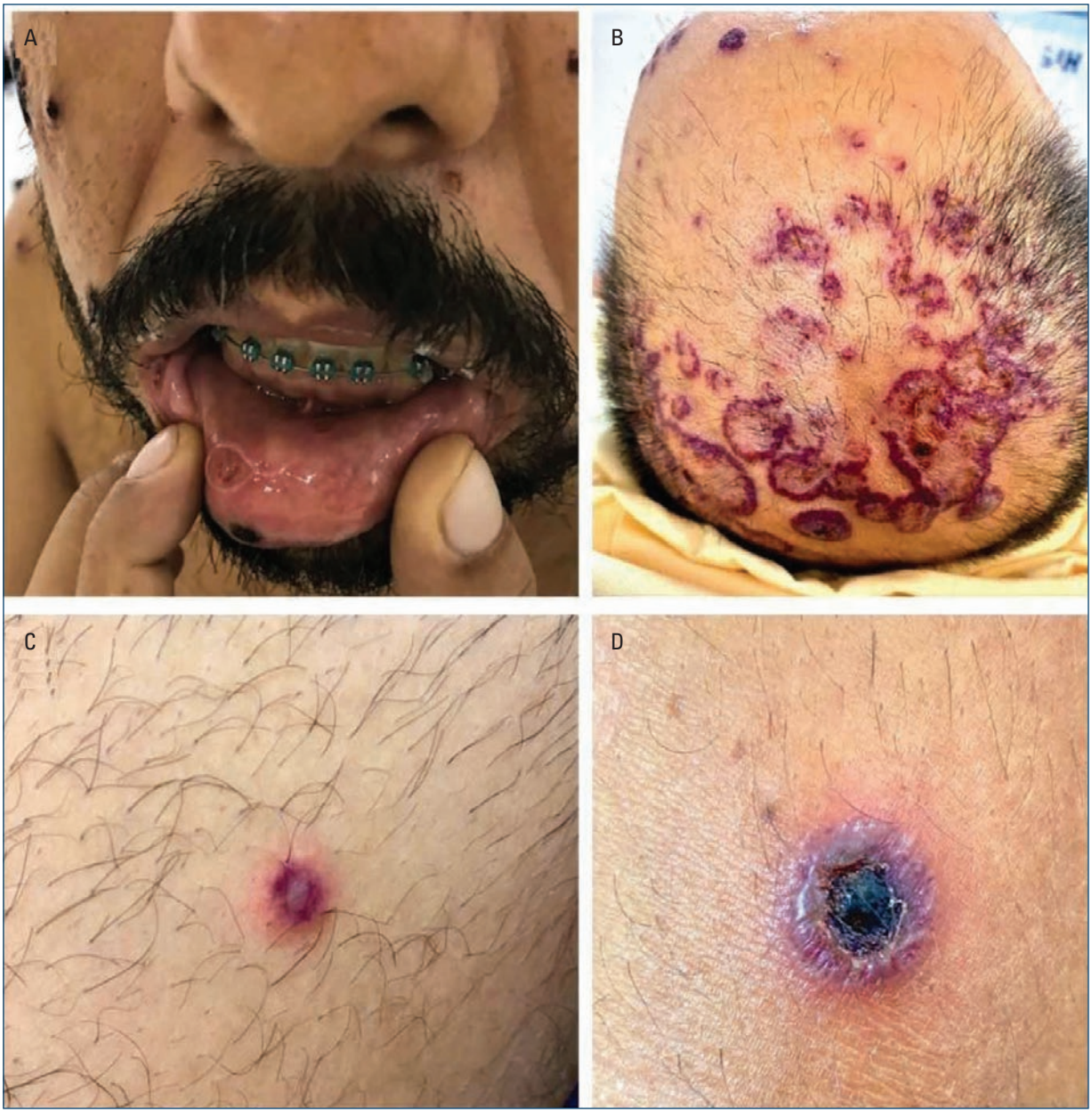

Figure 1. Lesions on the 3rd day after admission (8 days after beginning the Lugol solution treatment). **A:** ulcerated and painful lesions in the vestibule of the lower lip. **B:** purpuric lesions, with a circular pattern, on the scalp (biopsied lesion). **C:** vesiculopustular lesion with erythematous borders suggesting early necrosis on the lower limbs. (Biopsied lesion). **D:** rounded, painful ulcerative-necrotic lesion on the trunk.

of iodine in the preoperative preparation for thyroidectomy due to Graves' disease. Before the admission, the patient had a normal renal function, with no history of any kidney disease, which could be a high risk factor for iododerma.

The dermatologist must be aware of the possibility of skin and systemic reactions in patients exposed to iodine. In addition, every medical professional must be aware of the severity signs for early intervention when necessary.

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Conflicts of interest

The authors have no conflicts of interest to declare.

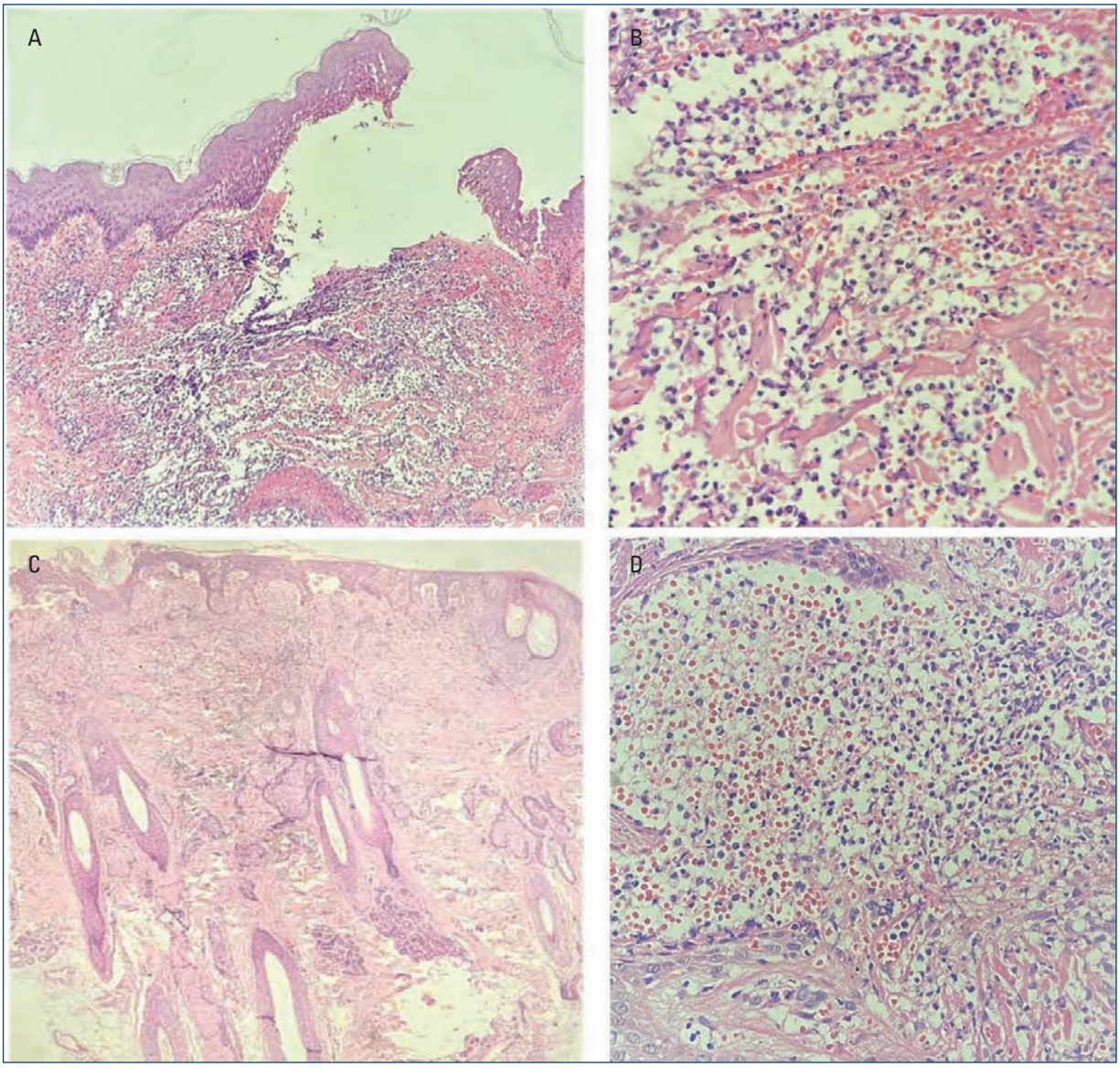

Figure 2. Histopathological examination–Thigh lesion. **A:** epidermal ulceration with the formation of an underlying abscess (H&E- ×20). **B:** mononuclear inflammatory cells, eosinophils and some neutrophils. (H&E- ×40). Scalp lesion. **C:** hyperkeratosis with hyperplasia of the epithelium, edema of the upper dermis and an inflammatory reaction with mononuclear inflammatory cells, eosinophils and some neutrophils occupying the upper and middle dermis portion between follicles (H&E- ×20). **D:** abscess formation in the middle dermis composed of mononuclear cells, eosinophils, some neutrophils and basophilic debris (H&E- ×40).

Ethical disclosures

Protection of people and animals. The authors declare that for this investigation no experiments were carried out on humans and/or animals.

Data confidentiality. The authors declare that they have followed the protocols of their work center regarding the publication of patient data.

Right to privacy and written consent. The authors declare that they have received written consent from the patients and/or subjects mentioned in the article. The corresponding author must be in possession of this document.

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