

Iododerma Following Exposure to Iodine: A Case of Explosive Acneform Eruption Overnight

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PRACTICE POINTS

- Iododerma should be considered for patients who develop rapidly progressive, vegetative lesions, especially in those with renal failure. A thorough history should be obtained in these cases, focusing on medications and recent studies involving iodinated contrast.
- The most important first step in treating iododerma is to remove the iodine-containing agent to avoid continued exposure.
- Therapies for iododerma include supportive care, cyclosporine, systemic corticosteroids, and hemodialysis in severe cases.

To the Editor:

Iododerma is a rare dermatologic condition caused by exposure to iodinated contrast media, oral iodine suspensions, or topical povidone-iodine that can manifest as eruptive acneform lesions.¹⁻³

A 27-year-old woman in septic shock presented for worsening facial lesions that showed no improvement on broad-spectrum antibiotics, antifungals, and antivirals. She initially presented to an outside hospital with abdominal pain and underwent computed tomography (CT) with intravenous (IV) iodinated contrast; 24 hours after this imaging study, the family reported the appearance of “explosive acne overnight.” The lesions first appeared as vegetative and acneform ulcerations on the face. A second abdominal CT scan with IV contrast was performed 4 days after the initial scan, given the concern for spontaneous bacterial peritonitis. Hours after the second study, the lesions progressed to involve the buccal mucosae, tongue,

mucosal airway, and distal arms and legs. She became progressively disoriented and developed an altered mentation over the course of the following week. Due to progressive facial edema, she required intubation 5 days after the second CT scan.

The patient had a medical history of end-stage renal disease secondary to crescentic glomerulonephritis on peritoneal dialysis. Physical examination revealed numerous beefy-red, heaped-up, weepy, crusted nodules clustered on the face (Figure 1) and a few newer bullous-appearing lesions on the hands and feet. She had similar lesions involving the buccal mucosae and tongue with substantial facial edema. Infectious workup was notable for a positive skin culture growing methicillin-susceptible *Staphylococcus aureus*. All blood and tissue cultures as well



FIGURE 1. Numerous beefy-red, heaped-up, weepy, crusted nodules clustered on the face. An endotracheal tube was placed shortly after admission due to rapidly declining mental status.

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The authors report no conflict of interest.

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doi:10.12788/cutis.0481

as serologies for fungal and viral etiologies were negative. A tissue biopsy revealed necrosis with a neutrophilic infiltrate with mixed cell inflammation (Figure 2), and direct immunofluorescence was negative.

The patient initially was thought to be septic due to viral or bacterial infection. She was transferred from an outside hospital 7 days after the initial appearance of the acneform lesions, having already received IV contrast on 2 occasions within the first 48 hours of illness. Infectious disease was consulted and initiated broad-spectrum antiviral, antimicrobial, and antifungal therapy with acyclovir, linezolid, meropenem, and later micafungin without improvement. The diagnosis of iododerma ultimately was established based on the patient's elevated urinary iodine levels with preceding iodine exposure in the context of renal failure. The preferential involvement of sebaceous areas and pathology findings were supportive of this diagnosis. Aggressive supportive measures including respiratory support, IV fluids, and dialysis were initiated. Topical iodine solutions, iodine-containing medications, and additional contrast subsequently were avoided. Despite these supportive measures, the patient died within 48 hours of admission from acute respiratory failure. Her autopsy attributed "septic complications of multifocal ulcerative cutaneous disease" as the anatomic cause of death.

Iododerma is an extremely rare neutrophilic dermatosis. The proposed mechanism of action involves a cell-mediated hypersensitivity reaction to iodine with induction of neutrophil degranulation.² There have been documented cases with exposure to oral potassium iodide supplements, amiodarone, topical povidone-iodine, and IV iodinated contrast material.¹⁻³ Iododerma typically presents 1 to 3 days after exposure to iodine. The most common source is IV radiocontrast. Diagnosis is based on the clinical presentation including acneform to vegetative nodular or bullous eruptions involving sebaceous areas in the context of recent iodine exposure. Elevated urinary iodine levels and histologic findings of neutrophilic infiltrate of the dermis support the diagnosis.^{3,4}

Although there have been reported cases of iododerma in patients with normal renal function, patients with renal failure are much more susceptible due to the decreased clearance of iodine.⁵ The plasma half-life of radiocontrast is 23 hours in patients with end-stage renal disease vs 2 hours in patients with normal kidney function.³ Dosage adjustments for renal impairment have not been well studied, and no specific guidelines exist for the prevention of iododerma in patients with renal failure.

The first step in treating iododerma is to remove the offending iodine-containing agent. In most cases, cutaneous lesions resolve in 4 to 6 weeks after discontinuation of the source of iodine; however, there have been reported fatalities in the literature secondary to pulmonary edema in patients with iododerma.^{6,7} Despite the rarity and

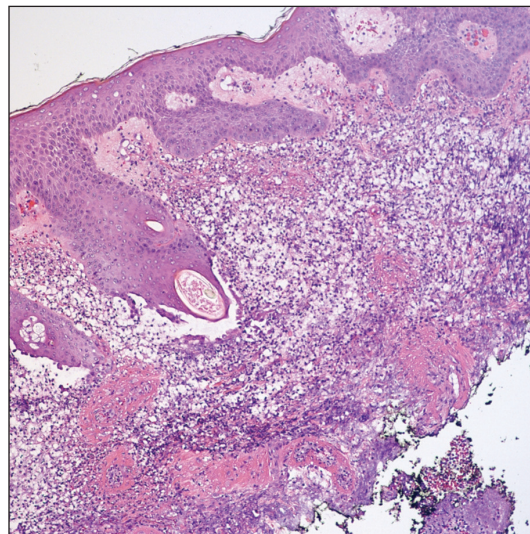


FIGURE 2. A biopsy from the right cheek showed a dense, dermal, mixed cell infiltrate with a predominance of neutrophils and edema (H&E, original magnification $\times 10$).

diagnostically challenging nature of iododerma, early recognition of this disease is crucial. Although our patient showed symptoms of iododerma after 1 dose of radiocontrast, she was not diagnosed at that time and received a second imaging study with contrast less than 48 hours later. These 2 consecutive exposures to iodine as well as the delayed diagnosis unfortunately resulted in rapid clinical deterioration.

The mainstay of therapy for iododerma includes avoidance of iodine-containing materials as soon as the diagnosis is suspected as well as supportive care. Patients have been successfully treated with systemic corticosteroids, with the addition of cyclosporine and hemodialysis in severe cases.³ Patients with a history of iododerma are advised to avoid iodine in their diet, in topical preparations, and in future imaging studies.⁸

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