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Case Presentation

Acute onset of a vesiculopustular rash in an ICU patient.

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Abstract

A 63 year-old woman with hyperthyroidism was admitted to the Medical Intensive Care Unit for ARDS following damage to her lungs from propylthiouracil. She was placed on 250 mg SSKI PO TID as an alternative therapy until thyroidectomy could be performed. Four days after admission, she abruptly developed an acneiform rash on her face, shown to be iododerma. The eruption rapidly resolved after discontinuation of the SSKI.

Key words: iododerma, acneiform rash, wolff-chaikoff effect, vesicuopustule

Case synopsis

A 63 year-old woman with hyperthyroidism was admitted to the Medical Intensive Care Unit for ARDS following damage to her lungs from propylthiouracil. She was placed on 250 mg saturated solution of potassium iodide (SSKI) PO TID as an alternative therapy until thyroidectomy could be performed. Four days after admission, she abruptly developed an acneiform rash on her face. Within 2 days, she developed pustules, vesiculopustules and bullae on her neck, trunk and extremities. (FIGURE 1A) All investigations, including direct fluorescence antibody, viral culture, fungal culture and bacterial culture showed no evidence of infection. Biopsy showed intraepidermal and dermal neutrophilic infiltration consistent with iododerma. SSKI was discontinued and thyroidectomy was performed. Within 4 days, new lesions stopped erupting and older lesions began resolving.



Figure 1A. (Lesion-Abdomen.jpg): Iododerma. Day 4, Abdomen with multiple grouped papulovesicles showing first biopsy site.

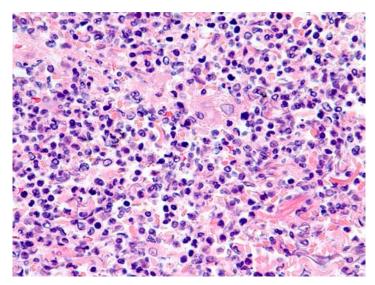


Figure 1B. Iododerma (H&E 40x) Biopsy of Left inner thigh was performed, revealing intraepidermal neutrophils with dermal abscess composed primarily of neutrophils, consistent with a neutrophilic dermatosis.

Discussion

The Wolff-Chaikoff effect is an autoregulatory phenomenon in which elevated levels of iodide inhibit the formation and release of thyroid hormone. Administration of iodide results in rapid and dramatic reduction in basal metabolic rate, but this effect is temporary. Iodide is generally used as an emergency measure in the setting of severe thyrotoxicosis or in preparation for thyroidectomy.

The most common cutaneous manifestation resulting from intake of iodide, the anionic form of iodine, is an acneiform eruption consisting of pustules on the face and trunk. Bullous and vesicular lesions are not uncommon and in advanced cases, lesions can become vegetative and fungating. A similar eruption can occur following intake of other halogenated compounds containing fluorine and bromine. The differential diagnosis includes Sweet syndrome, deep mycosis, pemphigus vegetans, mycosis fungoides, bacterial folliculitis, and disseminated herpes simplex and herpes zoster. Biopsy will clarify the diagnosis among these entities.

In acute iododermas, histopathologic changes can be non-specific, but the predominant findings include a diffuse dermal inflammatory infiltrate composed mostly of neutrophils with few plasma cells, mast cells, and eosinophils [2] (FIGURE 1B). Although there was no significant follicular involvement in our case, the presence of this finding assists in diagnosis. In chronic lesions, pseudoepitheliomatous hyperplasia and a mixed cellular infiltrate may be seen [3]. Resolution occurs after elimination of iodide. Lesions resolve in 4 to 6 weeks, leaving only mild pigmentary changes and occasionally dermal atrophy [6]. The pathophysiologic mechanism by which iodide induces skin changes is not entirely clear; in some cases iodide may act as a hapten that combines with serum proteins [4, 5].

Exposure to iodide can occur through multiple sources, including medications such as amiodarone and potassium iodide (sometimes found in expectorants) [7], wound products such as povidone iodine and iodoform gauze [8, 9], and radiology contrast dyes [10].

Iododermas are rare in patients with normal kidney function and those reported generally occurred in patients with renal insufficiency [1]. Delayed clearance and prolonged circulation of iodide may be one cause of iododerma. Importantly, exposure can be occult and considerable probing into the patient's medical history may be necessary to elicit a source of exposure to iodides. In any generalized pustular or vesicobullous eruption, particularly one associated with vegetating lesions, iododerma should be included in the differential diagnosis.

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