

Ichthyosiform eruption caused by paradichlorobenzene toxicity from chronic mothball ingestion



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INTRODUCTION

Paradichlorobenzene (PDCB) is a common household deodorant that can produce neurotoxic effects in large exposure. Cutaneous manifestations of exposure are rarely reported. We present a case of PDCB intoxication caused by chronic mothball ingestion presenting with distinct cutaneous findings and associated histopathology.

CASE REPORT

A woman in her 50s with a history of substance use disorder, depression, sickle cell trait, and cirrhosis presented with a 3-week history of pruritic violaceous papules coalescing into plaques on the distal extensor arms and legs and the bilateral helices. The rash was spreading, nontender, and not associated with systemic symptoms and had not improved despite trials of 1% hydrocortisone cream, triamcinolone cream, and over-the-counter emollients. On physical examination, there were numerous 1 mm to 3 mm flat-topped violaceous papules coalescing into thin, confluent plaques, most evident on the bilateral helices, wrists and forearms, right hip, and distal lower extremities (Fig 1).

Initial clinical differential diagnosis included exanthematous lichen planus and a lichenoid drug reaction, although the patient denied any recent medication changes. A 4-mm punch biopsy obtained from the right thigh found mild acanthosis with alternating parakeratosis and perifollicular plugging, most suggestive of pityriasis rubra pilaris, and the patient was started on triamcinolone 0.1% cream twice a day and acitretin, 10 mg/d. Approximately 6 weeks later, she was hospitalized with suspected encephalopathy secondary to her underlying liver

Abbreviation used:

PDCB: paradichlorobenzene



Fig 1. PDCB toxicity. Patient initial presentation with flat-topped violaceous papules coalescing into plaques on the bilateral wrists and forearms.

disease, after presenting with generalized weakness, fatigue, weight loss and pain in the extremities sometimes localized to areas of her rash. The patient was discharged and seen in the dermatology clinic 7 weeks after initial presentation. On this visit, she presented with confluent acanthotic hyperpigmented plaques on the bilateral forearms and distal lower extremities and less well-defined plaques on the conchal bowls (Figs 2 and 3).

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Fig 2. PDCB toxicity 7 weeks after initial presentation. Right antecubital fossa with confluent acanthotic hyperpigmented plaques.

Given the evolution of her rash and systemic symptoms, the presentation was concerning for paraneoplastic acanthosis nigricans. A shave biopsy was obtained that demonstrated papillarity of the epidermis with thinning of the Malpighian layer and a basket-weave keratin layer of stratum corneum (Fig 4).

Slight acanthosis with basal layer hyperpigmentation and slight perivascular lymphocytic infiltrate was also visualized. One week later, before further workup was obtained, the patient was again hospitalized with altered mental status and failure to thrive, after presenting with weakness, fatigue, arm and leg pain, and blurry vision. During this episode, a collateral source revealed that since childhood the patient had occasionally ingested mothballs, and more recently she had increased to daily ingestion



Fig 3. PDCB toxicity 7 weeks after initial presentation. Bilateral knees with confluent acanthotic hyperpigmented plaques.

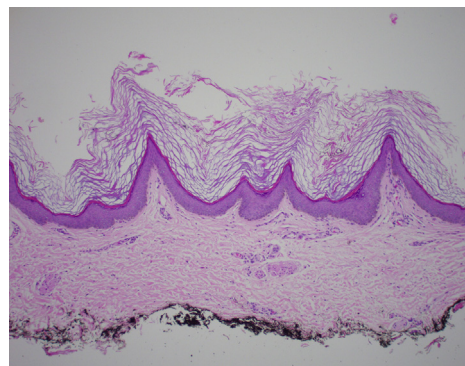


Fig 4. PDCB toxicity 7 weeks after initial presentation. Hematoxylin-eosin stain shave biopsy shows papillarity of epidermis with thinning of Malpighian layer and basket-weave keratin layer of stratum corneum.

because she “likes the smell.” Urine 2,5 dichlorophenol level, a metabolite of PDCB, was obtained and found to be greater than 50 mg/L (normal reference, undetectable).

Because there is no specific treatment for PDCB toxicity or PDCB-induced dermatosis, the patient was given supportive care and encouraged to discontinue ingestion. At follow-up 3 months later, she denied mothball ingestion, and her cutaneous and neurologic symptoms had resolved; however, her course has been complicated by occasional ingestion of mothballs in the months since.

DISCUSSION

We report clinical and histopathologic findings of PDCB toxicity in the setting of chronic mothball ingestion. Similar ichthyotic dermatoses have been reported in at least 3 other cases of PDCB ingestion,

including 2 twin women and a middle age woman.^{1,2} PDCB has also been reported to cause neurologic symptoms including encephalopathy, tremors, and muscle weakness in patients with exposure for more than a year.³⁻⁷ As with our patient, most of the other reported patients had a history of preceding psychiatric illness. Interestingly, PDCB toxicity from mothball ingestion was reported in the setting of pica,⁸ and our patient had a known history of sickle cell trait, a condition in which pica is frequently observed. Chronic PDCB use has also been associated with liver dysfunction.

Clinicians should have a heightened suspicion for PDCB intoxication in patients presenting with neurologic signs and hyperpigmented acanthotic or ichthyosiform plaques, particularly among patients with psychiatric conditions. A thorough history of chemical ingestions and exposures should be obtained in suspected cases, as this case shows that patients may be reluctant to divulge this information out of fear of stigmatization.

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