NOTES & COMMENTS

Ichthyosiform eruption caused by paradichlorobenzene toxicity from toilet freshener inhalation

To the Editor: We read with great interest the article in this issue by our colleagues Lee and Stavert reporting an ichthyosiform eruption caused by paradichlorobenzene (PDB) toxicity after chronic mothball ingestion. We would like to present an additional case of ichthyosiform eruption secondary to paradichlorobenzene toxicity by different mechanism.

We present a case of a previously healthy 19-year-old woman with a medical history of bipolar disorder and anemia who presented to the emergency room with altered mental status, unusual body odor, and ichthyosiform hyperpigmented plaques on her trunk, neck, axillae, and extremities (Fig 1). The patient had a cesarean section complicated by postpartum hemorrhage 1 month before presentation and a multimonth history of slowly progressive neurologic deterioration and skin plaque development. At the time of admission, computed tomography of the head, electroencephalogram, and spinal fluid analysis were unremarkable, with magnetic resonance imaging of the brain showing leukoencephalopathy. Skin punch biopsy found papillomatosis, an intact granular layer, and sparse perivascular lymphocytic infiltrate. With the diagnosis still unclear, additional history obtained from the patient revealed a several-month history of intentional inhalation of toilet fresheners. At this time, a urine test for 2,5-dichlorophenol (a metabolite of PDB, the primary chemical in toilet fresheners) was performed, and was found to be 620 mg/L, which is 3 times the upper limit of normal for an occupational exposure. This finding confirmed our diagnosis of

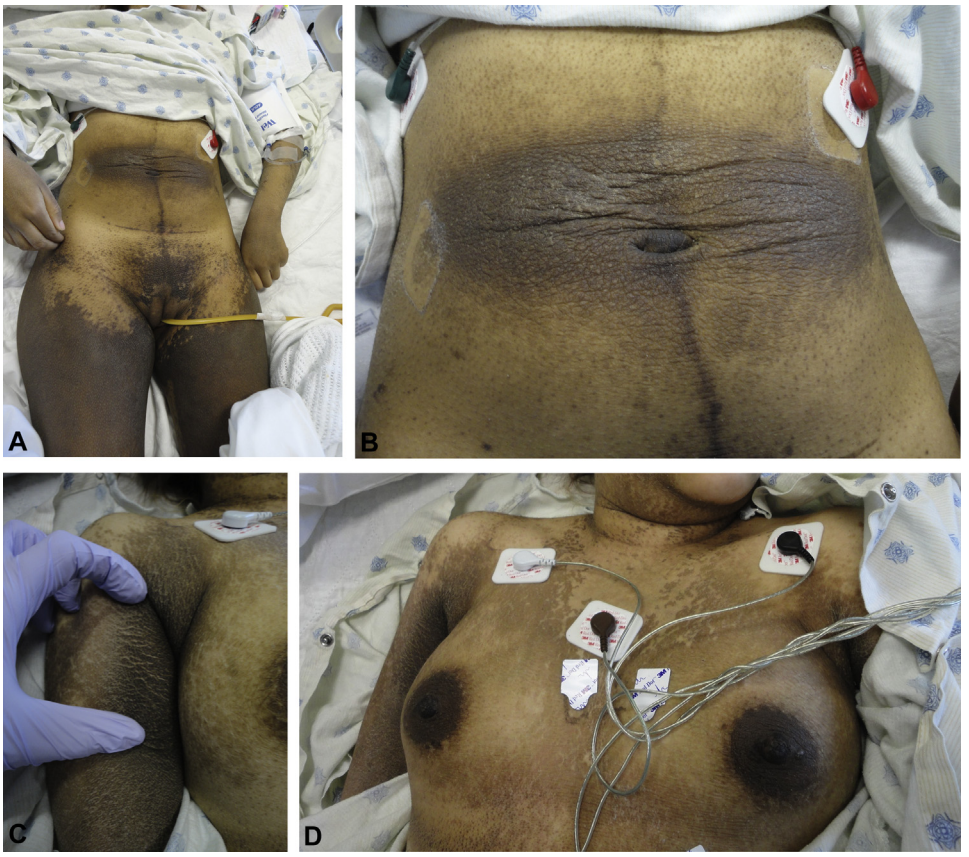

Fig 1. PDB toxicity leading to ichthyosiform hyperpigmented plaques of the lower extremities (**A**) and abdomen (**B**), and right arm/chest (**C** and **D**).

chemical encephalopathy and ichthyosiform dermatitis secondary to PDB toxicity.

This brings to light the fact that toilet fresheners and mothballs contain PDB and can be intentionally inhaled for a high and can cause a similar clinical presentation.[1] We would also like to draw attention to the possible implication of anemia in PDB intoxication. Although our colleagues presented a patient with sickle cell trait, our patient had a known history of anemia and a recent pregnancy complicated by postpartum hemorrhage. The association between pregnancy, pica, and paradichlorobenzene ingestion has been reported in a postpartum woman and pregnant woman who ingested mothballs.[2,3] Our case, as well as those of our colleagues, perhaps suggests that paradichlorobenzene inhalation or ingestion is not driven purely by a psychologic entity, but also a misplaced physiologic drive like pica.

Our patient initially experienced only minor improvement in her dermatologic and neurologic symptoms but eventually recovered her ability to speak and walk after several months of supportive care and discontinuation of the offending agent, as has been shown previously.[1] For severe cases, it has been proposed that paradichlorobenzene should be administered in a taper to prevent withdrawal symptoms, which mimic those of PBD intoxication.[4] Conversely, if PBD exposure is not discontinued in a timely fashion, permanent organ damage or death can occur.[5]

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