Chemical-induced sclerodermoid disease triggered by pressure washing bleach solution



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INTRODUCTION

Chemical-induced sclerodermoid disease (CISD) is a scleroderma-like disorder that most commonly affects those working industrial jobs with chemical exposure. CISD has been well-reported, secondary to aromatic hydrocarbon, vinyl chloride, epoxy resin, and pesticide exposure.^{1,2} Cutaneous signs of CISD include acrosclerosis and morpheaform plaques. We report a novel case of CISD attributed to a pressure washing product containing sodium hypochlorite and sodium hydroxide, which are the ingredients found in household bleach.

CASE REPORT

A man in his 60s presented with complaints of rash and skin tightness after pressure washing his house with Mold Armor EZ House and Siding Wash. He had not worn protective clothing. The chemical came in contact with his forearms and hands, especially the right, which held the spray wand and "drenched" his socks. He showered several hours later after experiencing itching and redness of contacted sites. Symptoms persisted over the ensuing weeks with the development of "tightness" in his right arm and difficulty moving his hands, especially the right one. His past medical history included Grave disease; family history included rheumatoid arthritis. Review of systems was otherwise negative.

Physical examination demonstrated acrosclerotic changes in both hands, with erythema and induration extending up the forearms. Induration was more notable on the right side. Joint mobility of the right hand was decreased. Erythematous to violaceous plaques covered both the legs in a sock distribution (Fig 1, *A* and *B*). Histopathology of a punch biopsy from the right forearm demonstrated sclerosis of

Abbreviation used:

CISD: chemical-induced sclerodermoid disease

the dermis, increased fibroblasts with diminished collagen fenestrae, and perivascular lymphocytic infiltrate with loss of adnexal fat, typical of morphea (Fig 2, *A* and *B*).

Given the distribution of involvement and history of chemical exposure, a diagnosis of CISD was made. Treatment included a prednisone taper and class I topical corticosteroid, followed by methotrexate 10 mg weekly and home broad-band ultraviolet A light therapy. Improvement in redness, induration, and mobility was noted at the 1-month follow-up, with subsequent steady improvement. At the 6month follow-up, the redness and induration of the legs had resolved. The induration of the forearms was mild, as was the stiffness of the right hand joints.

DISCUSSION

CISD frequently presents with acrosclerosis and morpheaform plaques.^{1,2} Histological examination shows lymphocytic infiltration around the dermal vessels and changes in the collagen matrix, closely resembling the findings in scleroderma.^{2,3} Commonly implicated chemicals include silica and organic compounds; pesticides have also been reported.² Exposure to Mold Armor, a registered pesticide containing sodium hypochlorite (5.25%) and sodium hydroxide (<1%) or similar bleaching agents has not been previously reported to cause CISD. Notably, household bleach contains sodium hypochlorite and sodium hydroxide in similar concentrations (1%-6% and 0.01%-1%, respectively).⁴ In this case, CISD was diagnosed based on the congruent history,

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Fig 1. A, Erythema and induration of forearms extending onto the dorsal aspect of the hands and digits. **B**, Erythematous to violaceous morpheaform plaques on the lower extremity in a sock distribution.

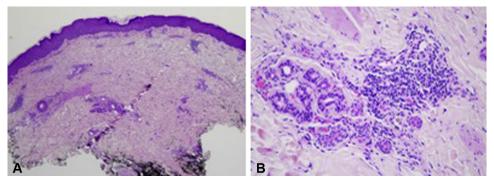


Fig 2. A, Sclerosis of the dermis with hyalinized collagen. **B**, Perivascular lymphocyte infiltration and loss of adnexal fat. (**A** and **B**, Hematoxylin-eosin stain; original magnifications: **A**, \times 4; **B**, \times 20.)

characteristic distribution, and clinicopathologic findings supporting a sclerodermoid/morpheaform process. A diagnosis of primary morphea was considered, but the distribution of erythematous plaques confined to chemical-exposed areas is unusual among morphea subtypes.⁵ The pathophysiology of CISD is unclear but is thought to be related to chemicals inducing autoantibody formation in genetically susceptible patients, such as our patient with a personal and family history of autoimmune disease.^{2,6} CISD may improve or resolve with the avoidance of the chemical. Severe disease can be treated with a combination of prednisone and methotrexate.^{2,5} Our patient improved with chemical avoidance and a combination of prednisone, methotrexate, and ultraviolet A therapies.

To our knowledge, CISD associated with a bleach product has not been previously reported. Because bleach is a commonly used household chemical, this occurrence is relevant to the general population. Consumers should be reminded to wear proper protective attire and promptly wash skin that comes in contact with pesticides and household chemicals. Clinicians should be aware that bleach-containing products may cause CISD. A detailed history including chemical exposures should be obtained when evaluating patients with morpheaform skin lesions.

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