

CASE LETTERS

Allergic contact dermatitis caused by a blood pressure cuff

To the Editor: N-isopropyl-N-phenyl-p-phenylenediamine (IPPD) is used primarily in the manufacture of black rubber products,¹ and its cross-reactivity with p-phenylenediamine (PPD), a common hair dye, is possible.

A 78-year-old nonatopic woman with a history of hypertension and coronary artery disease underwent a dipyridamole stress echocardiography. During this procedure, her blood pressure was monitored for 45 minutes. One day later, a pruritic, sharply demarcated, erythematous vesicular eruption developed at the site of contact with the blood pressure cuff. Slight improvement was obtained with topical corticosteroids (Fig 1) and the dermatitis completely cleared after 7 days of treatment with oral corticosteroids. A clinical diagnosis of contact dermatitis due to the blood pressure cuff was made. One month later, the woman was patch tested with the standard Italian Society of Allergological, Occupational and Environmental Dermatology (SIDAPA) series. At day 2 and day 4, a positive reaction (++) to IPPD (0.1% in petrolatum) was recorded. PPD (1% in petrolatum), included in the standard series, did not show a positive reaction. Strong response (++++) was elicited when a fragment of the blood pressure cuff was patch tested as is. A skin prick test to latex protein was negative. In addition, the patient gave a positive history of hand eczema to rubber gloves.

The inner part of the sphygmomanometer was composed of black rubber and lined by a nylon cuff. According to the manufacturer, there is no information available concerning the composition of the rubber. Chemical analysis was conducted to verify the presence of IPPD in the nylon cuff. In previous studies, the determination of IPPD was carried out by gas chromatography–mass spectrometry and high-performance liquid chromatography (HPLC).² In this study, after incubation with dichloromethane (CH₂Cl₂) for 24 hours, a piece of tissue extracted from the nylon section of the sphygmomanometer was submitted to HPLC interfaced with an API3000 mass spectrometer that was triple quadrupole equipped with a turbo ion spray. Spectroscopy was comparatively performed on the standard IPPD allergen. The quasimolecular ion m/z 227 [M + H]⁺ and the fragment m/z 184 confirmed the presence of IPPD in the tissue,³ which had a sample content of 38.5 ppm.



Fig 1. Allergic contact dermatitis caused by a blood pressure cuff.

To our knowledge, this is the first report describing allergic contact dermatitis due to IPPD in a blood pressure cuff. Orthopedic textile knee bandage and orthopedic hip brace have been reported to cause sensitivity to IPPD.^{1,4} Contact dermatitis after ambulatory blood pressure monitoring was reported in 2012⁵; however, those authors did not provide information regarding the responsible allergens.

The lack of interface between the nylon and rubber components and the repeated use of the sphygmomanometer could have caused the passage of the allergen by way of direct contact with the skin. The duration of sweating and occlusion (45 minutes) may also have favored the development of dermatitis. We emphasize the importance of chemical analysis and the possible risk of allergic contact dermatitis even during a medical test that is generally considered to be a safe procedure.

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Fixed drug eruption occurring in vitiliginous skin

To the Editor: In October 2012, a 50-year-old man presented with sudden-onset pruritic eruptions on the extremities. Physical examination showed depigmented macules of vitiligo, which had been present since childhood, scattered on the trunk and extremities. The halo phenomenon for a congenital melanocytic nevus was noted on the left thigh. In total, 12 isolated, irregularly shaped erythematous macules were recognized on some of the depigmented macules on the extremities. The erythematous macules were mainly present on the margins of vitiligo lesions. Laboratory tests were unremarkable. L-carbocisteine, clarithromycin, and dextromethorphan hydrobromide hydrate had been administered over the preceding 3 days for acute bronchitis. After discontinuing the medication at our recommendation, all the erythematous macules subsided within 3 days. Furthermore, according to the patient and his medication diary, in December 2011 he experienced a pruritic eruption on some of his depigmented macules that resolved within a week after taking a cold medication that contained L-carbocisteine.

After written informed consent was obtained, oral challenge tests were conducted; after 2 consecutive days of L-carbocisteine use (1500 mg/day), pruritic erythematous macules were induced at the previously involved sites (Fig 1). The histopathology of a biopsy specimen showed epidermal immigration of lymphocytes and basal vacuolated and apoptotic keratinocytes, which was consistent with fixed drug eruption (FDE). Oral provocations of the other 2 drugs (clarithromycin and dextromethorphan hydrobromide hydrate) yielded negative results. L-carbocisteine-induced FDE was diagnosed and the patient was advised against future L-carbocisteine use.

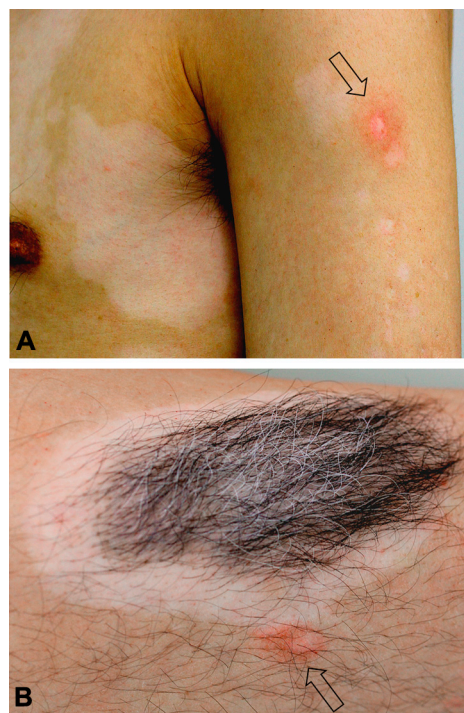


Fig 1. Fixed drug eruption. Multiple irregular erythematous macules of various sizes occurring after an oral provocation test with L-carbocisteine. **A**, Erythematous macule of fixed drug eruption surrounding a vitiligo lesion on the upper arm. **B**, A lesion of fixed drug eruption is also seen at the margin of a halo nevus on the thigh.

The oral challenge test is the gold standard for establishing the causative agent in FDE. It should be performed carefully, because it may involve a risk of the occurrence of new lesions and discomfort for the patient. In FDE, the adverse reaction is usually observed within 30 minutes to 8 hours after administration of the agent; however, as in our case, it may require more than 2 consecutive days to elicit the eruption because of hypersensitivity not to the agent itself but to its metabolites.¹

To our knowledge, there are no previously published reports of FDE occurring on vitiligo lesions. In addition, the eruptions were mainly present at the margins of the vitiligo lesions. Some cases of FDE have been reported to have appeared in particular areas of predilection, including location of physical trauma.² The pathogenesis of FDE is believed to involve activated T cells residing in the affected epidermis. A homogeneous population of T cells persists after clinical resolution of FDE and becomes reactivated after reexposure to the causative drug.³ The leading theory for the cause of systemic vitiligo implicates an autoimmune pathomechanism involving activated T cells that are recognized as infiltrating the perilesional margin.⁴