

Two cases of airborne allergic contact dermatitis caused by methylisothiazolinone in paint

Michael D. Lundov¹, Holger Mosbech², Jacob P. Thyssen³, Torkil Menné³ and Claus Zachariae³

¹Department of Dermato-Allergology, National Allergy Research Centre, Gentofte University Hospital, Copenhagen University, DK-2820 Gentofte, Denmark,

²Department of Dermato-Allergology, Allergy Clinic, Gentofte University Hospital, DK-2820 Gentofte, Denmark, and ³Department of Dermato-Allergology, Gentofte University Hospital, DK-2820 Gentofte, Denmark

doi:10.1111/j.1600-0536.2011.01924.x

Key words: airborne allergic contact dermatitis; methylisothiazolinone; paint.

Correspondence: Michael Dyrsgaard Lundov, Department of Dermato-Allergology, National Allergy Research Centre, Gentofte University Hospital, Ledreborg Alle 40, 1, DK-2820 Gentofte, Denmark. Tel: +4539777309; Fax: +4539777118. E-mail: midylu01@geh.regionh.dk

Conflicts of interest: The authors have declared no conflicts.

Methylisothiazolinone (MI) in combination with methylchloroisothiazolinone (MCI) is one of the most frequent causes of preservative contact allergy (1). Since the early 2000s, MI has been used without MCI in industrial products, and since 2005, MI has been permitted in cosmetic products sold within the EU. The first cases of isolated MI contact allergy were occupational, and in 2010,

the first cases of cosmetic-related MI contact allergy were published (2–5). Previously, MCI/MI was frequently used to preserve paints, and in some cases this chemical caused airborne allergic contact dermatitis (6–8). In a study by García-Gavín (4), two patients experienced airborne contact dermatitis probably caused by either MCI/MI or MI in a carpet glue or wall paint. A recent study showed that painters constitute nearly half of occupational dermatitis cases caused by MI, a finding that may indicate that MI is used frequently in paints (5). In this case report, we present the first cases of airborne allergic contact dermatitis caused by paint preserved with MI.

Case 1

A 36-year-old non-atopic man was referred in 2009 for a second opinion because of suspected occupational contact dermatitis. The patient worked as an assistant manager in a casino. In November 2007, he developed dermatitis on the legs that spread to the face, shoulders, back, abdomen, and arms. He complained about periods with intense headache. The symptoms worsened at work, but improved on days off. During 4 weeks of holiday, dermatitis and headache almost disappeared. Patch testing showed a positive reaction (++) to formaldehyde (2% aqua) and doubtful (+?) reactions to fragrance and MI (0.2% aqua). Formaldehyde and fragrance contact allergy were considered not to be of clinical relevance following screening of personal and occupational products, whereas MCI/MI was found in a liquid soap at work, and MI alone was found in wall paint that had been used on several occasions in the casino. The liquid soap was not considered to be of clinical relevance, as his hands were not affected. The patient returned to work full time in the casino from April to October 2010 after 2.5 months of sick leave, during which the dermatitis had disappeared. In October 2010 he resigned, and shortly after this he moved into a newly refurbished apartment. Two days later, symptoms reoccurred: headache, dermatitis on the abdomen and left eyelid, and a burning sensation in the face. An investigation of the different products used in the refurbishment showed that the apartment had been painted with wall paint containing MI. The symptoms continued for about 1 month when the patient lived in the apartment. For a period of 3 weeks, he was only in the apartment over the weekends. Dermatitis and the burning sensation improved in this period, but he experienced flare-ups during the weekends, when he stayed in the apartment. Three months after the patient moved into the apartment, the burning sensation in the face and dermatitis on the abdomen disappeared. However, eyelid dermatitis and occasional headache remained.

Case 2

A 57-year-old non-atopic man, a heavy smoker with no previous chest symptoms but a long history of hand eczema and contact allergy (positive patch test reactions to formaldehyde, quaternium-15, DMDM hydantoin, *p*-phenylenediamine, melamine formaldehyde, urea formaldehyde, MCI/MI, and MI), participated in a clinical investigation on the dose–response relationship of MI in MI-allergic patients (9). During the study period, he and his wife began to paint the walls in their house. The patient used an isolation paint that contained benzisothiazolinone (BIT), MI, and MCI/MI. The concentrations of BIT, MI and MCI/MI in this product were 8.5, 1.2 and 0.3 ppm, respectively. All three isothiazolinones were used to preserve the ingredients in the paint, and they were not added to the finished products as preservatives; hence the low concentrations. The patient's wife painted with two other types of wall paint, also containing BIT, MI, and MCI/MI. The concentrations of BIT, MI and MCI/MI in one of the paints were 172, 113 and 0.91 ppm, respectively. BIT and MI were added as preservatives in the finished product, whereas MCI/MI was a residue from one of the ingredients in the products. In the final product, the concentrations of BIT, MI and MCI/MI were 187, 187 and 10 ppm, respectively. All three isothiazolinones were added as preservatives to the finished product. After a few days, the patient developed facial erythema, cough, and difficulty in breathing. He was hospitalized because of the skin and respiratory problems. Oral prednisolone (25 mg/day) and cetirizine (10 mg/day), as well as low-dose inhaled corticosteroid, were initiated. The patient recovered quickly, and was discharged after only one night. He stayed in his house over the weekend, but did not feel well. For these reasons, he moved out of the house for the following 3 weeks, and, during this period, the dermatitis and respiratory problems improved. After agreement with his general practitioner, he stopped prednisolone therapy and returned to his home. Three days later, all symptoms reoccurred, including dermatitis reactions at the sites where he had been exposed to MI in the dose–response patch test study 2 months earlier and around the eyes (Fig. 1). He took prednisolone 25 mg, and was now seen at the allergy ward, presenting with severely reduced lung function, with both obstructive and restrictive patterns, even after inhaled β_2 agonist: forced expiratory volume in 1 s (FEV₁), 1.4 l (39% of expected); forced vital capacity (FVC), 2.2 l (47% of expected), an elevated concentration of NO in exhaled air (85 ppb), indicating eosinophilic bronchial inflammation (normal value, <25 ppb), but normal diffusion capacity. He had almost total nasal obstruction. The dose of prednisolone was increased,

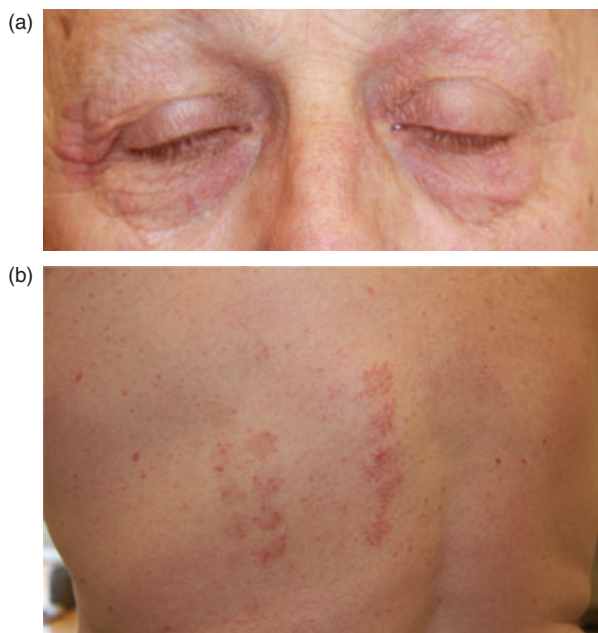


Fig. 1. Reactions to airborne exposure to methylisothiazolinone (MI) in case 2. (a) Reactions around the eyes. (b) Flare-up of MI dose-response patch test reactions 2 months after application.

he was put on high-dose inhaled and nasal corticosteroid, and he returned to his home, where ventilation in the painted rooms was increased. After 2 weeks, his lung function had doubled (FEV_1 , 3.0 l; FVC , 4.0 l), the concentration of NO in exhaled air was normal, he had no respiratory symptoms, and he showed clear nasal improvement. He again stopped taking prednisolone, and this time had no relapse of asthma and rhinitis. Symptoms and lung function at follow-up after 2 weeks were unchanged.

Discussion

We present two severe cases of airborne allergic contact dermatitis caused by exposure to wall paint preserved with, among other substances, MI. MCI/MI has long been recognized as a source of airborne contact dermatitis,

owing to the use of this chemical in wall paints (6–8). One patient also had severe asthma and rhinitis, most likely caused by the same substances. Induction of asthma has also previously been described in an isothiazolinone-manufacturing plant (10). In case 2, the patient was exposed to three different isothiazolinones, all at various concentrations. In two of the paints, BIT and MI were present at high concentrations, whereas the highest concentration of MCI/MI was only 10 ppm. The patient was also MCI/MI-allergic, and this could also have triggered the reactions. However, because of his distinct flare-up reactions on the areas (back and forearm) where he had previously reacted to MI, and the low concentration of MCI/MI in the products, we are convinced that the airborne contact dermatitis was caused by MI. Concomitant reactions between MCI/MI and MI in patch tests are seen in 40% of cases, and were seen in both of our cases (5).

MI is an upcoming contact allergen that has received increased attention over the last few years. In 2005, the EU allowed MI as a preservative in cosmetics, with a maximum permitted concentration of 100 ppm. In industrial products, there is currently no maximum permitted concentration of MI, and there are no labelling requirements for MI in industrial products. This makes it difficult for painters, as well as consumers, to avoid exposure to concentrations of MI that are higher than what is considered safe in cosmetics. We showed in a previous study that painters constitute the majority of allergic occupational MI contact dermatitis cases (5). During the past year, we have had several severe cases of allergic contact dermatitis caused by MI on the hands in painters. Furthermore, a clinical study showed that 7 (64%) of 11 MI-allergic patients reacted to an exposure of only 50 ppm MI in a repeated open application test (9).

Recently published results on MI allergy show that the prevalence of MI contact allergy is high, and that even small concentrations of MI are sufficient to elicit allergic reactions (5, 9, 11). These two cases of airborne MI contact dermatitis constitute yet another reason to reconsider the current legislation for both industrial and consumer products concerning exposure to MI.

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