

Case report

Dentist's occupational asthma, rhinoconjunctivitis, and allergic contact dermatitis from methacrylates

Background: Allergic contact dermatitis (ACD) caused by (meth)acrylates (MA) is common in dental personnel. MAs have also caused asthma and rhinoconjunctivitis, but asthma, rhinoconjunctivitis and ACD caused by MAs in the same patient appears to be very rare.

Methods: Occupational asthma and rhinoconjunctivitis were diagnosed in a dentist according to patient history, PEF monitoring, and a work-simulated bronchial provocation test. ACD was diagnosed by skin-patch testing with MAs with the occlusive Finn Chamber-technique.

Results: The patient's skin-prick test reactions to common environmental allergens and MAs were negative. The total IgE was not elevated. Occupational asthma was diagnosed by a specific inhalation challenge test in which the patient handled liquid dental MAs for 30 min causing a delayed 23% reduction in FEV1. The provocation test also resulted in rhinoconjunctivitis. On patch testing, positive reactions were provoked by several MAs including 2-hydroxyethyl methacrylate (2-HEMA) to which the patient was occupationally exposed. The patient has not been able to continue her work with dental MAs.

Conclusions: A case of occupational asthma, rhinoconjunctivitis and ACD caused by dental acrylate compounds is presented. Patients with respiratory hypersensitivity from MAs have to stop working with MAs, whereas patients with ACD from MAs need to avoid direct contact with MAs, but can often continue in their present job if they use no-touch techniques.

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Acrylates, methacrylates and epoxy acrylates, here called (meth)acrylates (MA) are widely used in dentistry. Allergic contact dermatitis (ACD) caused by MAs is well known in dental personnel. The clinical picture of dermatitis is usually pulpitis of the fingertips or dermatitis of the face, caused by airborne MAs (1). MAs are also an important occupational hazard to the respiratory system of dental workers (2, 3). Both ACD and respiratory hypersensitivity caused by MAs have increased greatly over the past decade in Finland (3, 4). So far more than 50 cases of ACD from MAs and close to 20 cases of occupational respiratory disease from MAs have been diagnosed at the Finnish Institute of Occupational Health. Here we report the first detailed description with both occupational skin and respiratory hypersensitivity from MAs.

Methods

Spirometry was performed with a rolling-seal spirometer (Mijnhardt, Vicatest 3, Bunnik, The Netherlands) connected to a microcomputer (Medicro MR-3, Kuopio, Finland), and Viljanen's reference values were used (5). The histamine challenge test was performed according

to Sovijärvi's method (6), following FEV1 (forced expiratory volume in one second) values with a Vitalograph S bellow spirometer (Vitalograph, Buckingham, UK). A 15% reduction in FEV1 was considered significant, and the provocative dose of histamine diphosphate causing a 15% reduction in FEV1 (PD15) was measured. The hyper-responsiveness was graded as strong (PD15 < 0.10 mg), moderate (0.11–0.40 mg) or slight (0.41–1.6 mg).

Routine skin prick tests (SPTs) to common environmental allergens were performed as described earlier (7). Prick tests were also performed with natural rubber latex (Stallergenes S.A., Fresnes Cedex, France), chloramine T (1% and human serum albumin (HSA) conjugate), and acrylates (Chemotechnique Diagnostics AB, Malmö, Sweden); 2-hydroxyethyl methacrylate (2-HEMA), methyl methacrylate, BIS-GMA, ethyleneglycol dimethacrylate, triethyleneglycol dimethacrylate (2%) and triethyleneglycol diacrylate (0.1%). 2-HEMA and BIS-GMA were also tested as HSA conjugates, which were prepared as previously described (8). Patch tests were performed according to the recommendations of the International Contact Dermatitis Research Group (ICDRG) with the occlusive Finn Chamber® (Epitest, Tuusula, Finland) technique. The patch test concentrations are given in Table 1.

Inhalation challenge tests with a placebo (Coca solution) and dental liquid MAs were performed in a 6-m³ challenge chamber according to the international guidelines as described previously (3). The products used by the patient in her work were used in the work simulating challenge tests (Scotchbond® primer containing 40% of HEMA and adhesive containing 62% of BisGMA and HEMA 37%).

Table 1. The patient's positive patch test reactions with a (meth)acrylate series including 35 test substances

(Meth)acrylate	Source	Abbre-viation	Patch test concentration (%) (w/w) (all allergens in petrolatum)	Patch test result
Glycidyl methacrylate	O	GMA	0.1	1+
Ethyl acrylate	C	EA	0.1	1+
Ethyl methacrylate	C	EMA	2	1+
2-Hydroxyethyl methacrylate	T	2-HEMA	1	2+
2-Hydroxypropyl methacrylate	C	2-HPMA	2	2+
Ethyleneglycol dimethacrylate	T	EGDMA	2	2+
Methyl methacrylate	C	MMA	2	1+

C = Chemotechnique (Malmö, Sweden); T = Trolab (Reinbek, Germany), O = prepared by ourselves.

No concentration measurements were carried out. The FEV1 and PEF values during the challenge test procedure were measured by a portable pocket-size spirometer (OneFlow tester ATS 94, Fuchs Medical, Saint Romans, France). The clinical symptoms and lung auscultation were recorded as well. The ocular reaction following the skin-patch tests to MAs was evaluated by an optometrist, as delayed conjunctivitis from MAs has earlier been reported (9).

Case report

A 47-year-old nonsmoking female dentist had been working in general dentistry for 22 years performing dental fillings, orthodontics, prosthetics and dental surgery. She had not had allergic symptoms as a child; however, her sister expressed an atopic constitution. When investigated at our institute she had had symptoms of rhinoconjunctivitis and sneezing for 12 years, cough attacks for 10 years, and shortness of breath for 2 years. Furthermore, she had had hand and face dermatitis for 3 years. The symptoms were work-related and disappeared during weekends and holidays. The patient associated the eye and respiratory symptoms to making dental fillings with photo-bonded resins and to working with dental prostheses. The dermatitis also got worse from disinfectants and natural rubber latex gloves. The patient had occasionally used inhaled epinephrine or salbutamol for nearly 10 years. During the past few years, she needed bronchodilating medication almost every day at work. Asthma medication with inhaled steroids was begun more than 1 year ago. During this medication PEF-flow monitoring at work and at home (3 weeks) showed values between 540 and 500 l/min during days off, whereas the lowest values during working days were 420 l/min. The results pointed towards occupational asthma. The patient was remitted to us mainly because of the work-related respiratory symptoms but also because her hand dermatitis had worsened. When seen by us, a fissured, purulent pulpitis was observed on her left thumb, and milder dermatitis was present on the sides of the left thumb and the left forefinger. No asthmatic rales were heard from her lungs. She had been on sick leave for 2 weeks.

Results

The patient's spirometry was normal and there was no significant response in the bronchodilation test (FVC 3.88 l, 108% of predicted, FEV1 3.12 l, 106% of predicted, and FEV1/FVC 80.55%, 98% of predicted). The histamine challenge test showed moderate bronchial hyper-reactivity with PD15 0.255 mg. The patient had been on sick leave for 2 weeks before the histamine challenge. There were no positive reactions in SPTs with common environmental allergens, natural rubber latex,

chloramine-T, or acrylates. The total serum IgE was normal, 35 kU/l. The eosinophils in the peripheral blood were normal (256). The placebo challenge test was negative. In the first inhalation challenge test with metacrylates, the adhesive (20 drops altogether during 30 min) induced cough, rhinoconjunctivitis and a 10% decrease in FEV1 after 45 min. In the second test, with both the adhesive and the primer (40 drops during 30 min), an early late 23% FEV1 reduction was recorded, at a maximum at 3 h, as well as increased symptoms with dyspnea. Before the tests the inhaled steroids had been stopped for 8 days. Patch testing with a MA series showed allergic reactions to several MAs, including 2-hydroxyethyl methacrylate (2-HEMA), present in Scotchbond (Table 1). In addition, patch testing induced itching, swelling and soreness of the eyelids, maximal during the 3-day patch test reading. An optometrist's consultation indicated that the symptoms were in accordance to delayed allergic conjunctivitis. She was ordered sick leave. On a control visit 10 months later, the patient did not have any symptoms of dermatitis or rhinoconjunctivitis. She still used inhaled steroids, and occasionally bronchodilators, e.g., when exposed to cigarette smoke. She did not use antihistamines, nasal steroids or eye drops. Two months before the control visit she had performed a work trial but developed strong respiratory reactions. It was concluded that she could not continue in her present work and needed retraining.

Discussion

ACD from MAs is a well known occupational hazard in dental personnel (1). MAs may also cause respiratory hypersensitivity (2, 3) but the mechanism is not known, and IgE-mediated reactions from MAs have not been reported although immediate skin reactions of an unknown mechanism occur occasionally (10, 11). This reaction pattern was also evident in our patient: ACD could be established by patch testing, whereas skin prick testing to MAs was negative. A bronchial provocation test needs to be performed in order to diagnose respiratory hypersensitivity (2, 3). As is usual,

the bronchial provocation tests were stopped when one positive test had been recorded although the patient had been exposed to many other MAs at work. Accordingly, she may have had respiratory hypersensitivity to several MAs. The positive patch-test reactions to many MAs (Table 1) may represent concomitant sensitization to several MAs, although cross-reactivity probably explained part of the positive reactions.

We have not earlier come across patients with asthma, rhinoconjunctivitis and ACD caused by MAs in the same patient, but we have had an earlier, closely related case. Another dentist had asthma and conjunctivitis from MAs and ACD from other acrylates (3).

Our patient developed eye symptoms during patch testing which can be considered as one type of systemic ACD (12). In most cases, systemic ACD refers to systemic reactions from ingested or inhaled allergens in patients previously sensitized by percutaneous absorption of the allergen (12). However, flare-ups during patch testing can also be manifestations of systemic ACD. They can be flare-ups of either earlier patch-test reactions, or, as in our patient, of contact dermatitis reactions elsewhere. These reactions may be of clinical relevance. Accordingly, inhaled MAs may exacerbate previous sites of dermatitis, and exposure to MAs via the skin, e.g., hands, may result in systemic dermatitis elsewhere, for example on the eyelids.

The patient had been exposed to MAs for nearly 20 years, and she developed respiratory symptoms after about 10 years of exposure. This long latency period is in accordance with our earlier findings on methacrylate induced asthma in dental personnel (3). ACD from MAs may necessitate change of career. However, our advice to patients with ACD has been to avoid direct contact with MAs using no-touch techniques (1). We have not recommended the patients to change their occupation. If patients with ACD from MAs would be at an increased risk of occupational asthma from MAs, it would be necessary to discontinue exposure to MAs. Even though the present patient developed both ACD and respiratory hypersensitivity to MAs, there is no firm evidence that patients with possibly lymphocyte-mediated allergy to MAs are at greater risk to develop occupational asthma than workers without such allergy to MAs. Accordingly, we continue to inform our patients with ACD from MAs to avoid skin contact with MAs but to continue in their previous occupation. This same principle also applies to, e.g., epoxy resin allergy. Epoxy resins are common causes of occupational ACD, and may cause asthma (13), but patients with ACD from epoxy resin are usually advised to continue in their jobs, but using good protection. Patients with occupational asthma from MAs have to stop working with MAs, as our patient did.

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