

Occupational Asthma Caused by Sculptured Nails Containing Methacrylates

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Background In recent decades, the use of artificial nails including methacrylates (MAs) has increased. This study presents the first two clinical cases of occupational asthma (OA) caused by sculptured nails containing MAs.

Methods In both cases, OA was diagnosed on the basis of a work simulation test combined with the patient's history of occupational exposure and respiratory symptoms.

Results Both patients developed work-related respiratory symptoms 4–5 years before the current examinations. Previously, the first patient (a 30-year-old female) was diagnosed as having allergic contact dermatitis (ACD) to MAs; the second patient (a 27-year-old female) had no skin symptoms. In both cases, a dual asthmatic reaction was observed in the bronchial provocation test, in which the patients simulated their work using their own products including MAs.

Conclusions Sculptured nails containing MAs can induce OA in nail technicians. Products not containing MAs should be used to prevent OA and ACD. If MAs are used, they should be handled with appropriate safety measures. *Am. J. Ind. Med.*

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KEY WORDS: allergic contact dermatitis; artificial nails; methacrylates; nail technician; occupational asthma

INTRODUCTION

Acrylic compounds, such as methacrylates (MAs), are used in industry because of their good properties in polymerization processes. Therefore, they are used widely in paints, lacquers, adhesive glues, and coatings. In addition to industrial use, MAs are used in dentistry and orthopedics as fillings and prosthetic materials. Since the 1970s, they have also been used in the application of artificial fingernails [Spencer et al., 1997].

Of MAs, the adverse health effects of methyl methacrylate (MMA) are most studied. MMA is not only a skin irritant but it also has the potential to induce skin sensitization and allergic contact dermatitis (ACD) [CIDAD, 1998]. Based on case-reports and epidemiological studies, MMA is associated with occupational asthma (OA) [CIDAD, 1998]. In addition to skin and respiratory health effects, olfactory function [Schwartz et al., 1989], neurotoxicity [Seppäläinen and Rajaniemi, 1984], and adverse cardiac effects [NIOSH, 1976] of MMA have also been examined. The available epidemiological studies do not provide strong or consistent evidence of a carcinogenic effect of MMA on any target organ in humans [CIDAD, 1998; Tomenson et al., 2005]. MMA monomer as an ingredient in sculptured acrylic nails was banned in 1974 in the United States [Engasser et al., 2000]. Since then other MAs such as ethyl or butyl MA monomers have been used in this purpose, but they may be even stronger sensitizers than MMA [Kanerva et al., 1996].

The adverse health effects on skin are shared by other MAs. Related to artificial fingernails, the most frequent allergens triggering ACD have been reported to be

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2-hydroxyethyl methacrylate (2-HEMA) and 2-hydroxypropyl methacrylate (2-HPMA), followed by ethyleneglycol dimethacrylate (EGDMA) and ethylmethacrylate (EMA) [Lazarov, 2007].

Dental personnel has been reported to have OA as a result of exposure to MAs [Lindström et al., 2002; Piirilä et al., 2002]. The most important airborne exposures are 2-HEMA and triethyleneglycol dimethacrylate (TEGDMA) [Henriks-Eckerman et al., 2001]. Moreover, daily use of MAs has been related to a significantly increased risk of adult-onset asthma and nasal symptoms in dental assistants [Jaakkola et al., 2007]. A few studies also mention asthma in cosmetologists working with artificial fingernails [Spencer et al., 1997; Kreiss et al., 2006], but they do not, however, provide clinical data.

We report on two nail technicians who both developed OA from occupational exposure to sculptured nails containing MAs. One of the patients also had ACD from this exposure. The diagnosis of OA was confirmed by a specific work simulation test.

METHODS

One of the patients (Patient 1) was referred to Finnish Institute of Occupational Health (FIOH) for specialist examinations from a local central hospital and the other (Patient 2) from a local occupational health service unit. Informed consent was received from both of them to write this study.

Spirometry was performed with a rolling-seal spirometer (Mijnhardt, Vicatest 3, Bunnik, Netherlands) connected to a microcomputer (Medicro MR-3, Kuopio, Finland), and Viljanen's [1982] reference values were used. The histamine challenge test was performed according to the method of Sovijärvi et al. [1993], following the forced expiratory volume in 1 s (FEV₁) values with a Vitalograph S bellow spirometer (Vitalograph, Buckingham, UK). Measurements of exhaled nitric oxide (NO) were carried out using a chemiluminescence gas analyzer (NIOX, Aerocrine AB, Solna, Sweden) according to ATS FE_{NO} guidelines; values of >30 ppb were considered to be over normal values [Piipari et al., 2002; ATS/ERS, 2005]. Peak expiratory flow (PEF) measurements were performed at home and at the workplace according to the method of Burge [1982].

Skin prick tests (SPTs) to common environmental allergens were performed as described earlier [Kanerva et al., 1991]. SPTs were also performed to formaldehyde, ammonium persulfate, and MAs. The tested MAs with test concentrations were 2-HEMA (1% pet; Trolab, Hermal, Germany), 2,2-bis-[4-(2-hydroxy-3-methacryloxypropoxy)-phenyl]-propane (bis-GMA) (2% pet; Trolab), EDGMA (2% pet; Trolab), MMA (2% pet; Trolab), TEGDMA (2% pet; Trolab), and triethyleneglycol diacrylate (0.1%; Chemo-technique Diagnostics, Vellinge, Sweden). Additionally,

2-HEMA and bis-GMA were also tested as human serum albumin (HSA) conjugates, which were prepared as previously described [Tupasela and Kanerva, 1997].

Bronchial provocation tests were performed in an 8-m³ challenge chamber according to international guidelines [Allergy Practice Forum, 1992; Piirilä et al., 1998]. As a reference challenge test, Coca solution and lactose powder were used in Patients 1 and 2, respectively. In the active challenge test, the patients simulated their work in the challenge chamber using their own products including MAs, i.e., they attached the plastic nail with a glue, and then filed and sculptured the nails. During the active challenge test, which took 30 min, three sculptured nails were produced. The patients were monitored for 24 hr after each challenge test. A portable, pocketsize spirometer (One Flow, STI MEDICAL, Saint-Romans, France) recorded the lung function measurements (FEV₁, PEF); a drop of 20% in PEF or FEV₁ was regarded as significant. An asthmatic reaction was defined as follows: an immediate reaction causing a decrease of $\geq 20\%$ in the FEV₁ or PEF during the first post-challenge hour; a delayed reaction causing a similar decrease in FEV₁ or PEF after the first post-challenge hour; and a dual reaction as a combination of the afore-mentioned. Clinical symptoms and lung auscultation were recorded as well.

Acetone-soluble acrylates and MAs in gel nail materials and in gel nails were identified by gas chromatography-mass spectrometry (GC-MS) and quantified by liquid chromatography with ultraviolet (UV) detection at 210 nm [Henriks-Eckerman et al., 2004]. These were determined in the case of Patient 2; in the case of Patient 1, the products were not available for analysis.

RESULTS

Case Reports

Patient 1

The patient was a 30-year-old female who had worked for 6 years as a manicurist and a nail technician. Her main job was to apply sculptured nails and artificial tips to nails. The patient started the procedure by pushing the cuticles back, cutting the nails, and sanding the nail plate. She attached the plastic nail with a glue that contained both cyanoacrylate and MAs, and then filed and sculptured the nails. Each nail was treated three times with a gel that contained MAs. According to the Safety Data Sheets, the MAs and cyanoacrylate were the only known sensitizers in the products. Before the application of the next layer, each layer was hardened with UV light. The procedure was performed on a special downdraft table to minimize exposure. The patient did not use any respiratory personal protective equipment but she did use natural rubber latex gloves.

The patient had no atopic dermatitis in her childhood. She had smoked for 2 years in her 20's. The patient developed hand eczema 3 months after starting to apply artificial nails. She proved to be sensitized to 2-HEMA and EGDMA according to patch tests performed at a local central hospital 4 years later, after which the diagnosis of occupational ACD was issued. About 4 years prior to the examinations at the FIOH, she experienced rhinitis, wheezing, and dyspnoea during exercise. In pulmonary examinations at a local central hospital, spirometry was normal but the bronchodilatation test was positive. In the histamine challenge test, there was moderate bronchial hyperresponsiveness (PD_{15} 0.226 mg), and the patient had typical asthmatic symptoms (dyspnoea and wheezing). Asthma was diagnosed, and regular inhaled fluticasone medication with salbutamol on demand was started. Because she had respiratory symptoms when applying artificial nails, her asthma was also suspected to have an occupational origin. The patient was referred to FIOH for further respiratory investigations.

At FIOH, the patient had no respiratory symptoms, and lung auscultation was normal. SPTs to common environmental allergens, formaldehyde, and MAs were negative. X-Rays of the thorax and nasal sinuses were normal. Spirometry showed mild peripheral obstruction without a bronchodilatation effect. The exhaled NO was normal (17.1 ppb). In the histamine challenge test, mild bronchial hyperresponsiveness (PD_{15} 0.649 mg) occurred. A significant variation was noted in the PEF measurements at home and at the workplace: the PEF values varying from 360 to 580 L/min with a maximal diurnal variation of 26% and frequent bronchodilating effects up to 43%.

The reference bronchial challenge test was negative. In the active bronchial challenge test, a dual asthmatic reaction was noted: an immediate significant decrease of 25% in the FEV_1 , and 4 hr after the start, a delayed significant decrease of 37% in the FEV_1 (Fig. 1). After the delayed significant decrease, the patient received short-acting bronchodilating medication, after which the FEV_1 returned to normal.

On the basis on the work-related respiratory symptoms and findings in the workplace PEF follow-up, as well as the positive work simulation test, OA due to exposure to sculptured nails containing MAs was diagnosed. Minimizing the exposure to MAs was recommended, and asthma medication was continued with a combination of inhaled fluticasone and salmeterol. At the 6-month follow-up examination at FIOH, which is a normal procedure among patients in whom occupational disease is diagnosed at FIOH, the patient complained of nasal symptoms after long working days, but she had been able to continue her work as a nail technician. The diurnal PEF monitoring with regular asthma medication varied between 450 and 510 L/min, and her spirometry was normal without a bronchodilatation effect. On the clinical grounds, she was recommended to consider measurements of vocational rehabilitation, but she

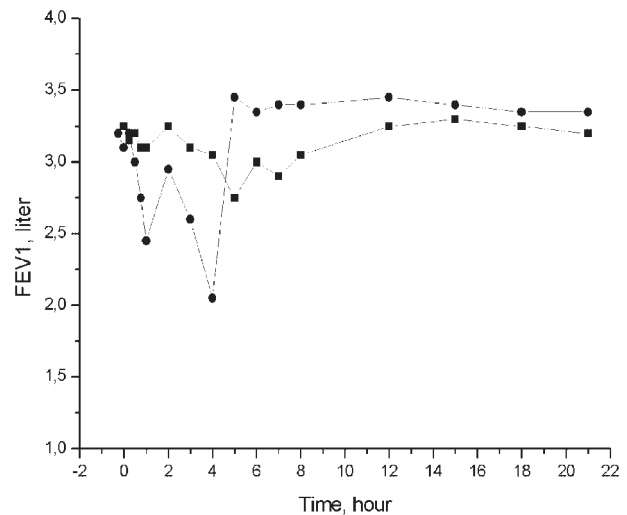


FIGURE 1. Forced expiratory volume in 1 s (FEV_1) follow-up during challenge tests of patient 1 with: (■) Coca solution; (●) methacrylates. Challenge occurred at 0 h.

was not willing to discontinue her career as a nail technician. She was advised to reconsider vocational rehabilitation later, if her asthma deteriorates under the post-FIOH surveillance maintained by her own doctor.

Patient 2

The patient was a 27-year-old woman who had worked for 5 years both as a hairdresser and as a nail technician preparing artificial gel nails. The process of preparing gel nails and the used products were similar to that described in Patient 1. This patient did not use any specific protective equipment.

The patient had had symptoms of allergic rhinitis with SPT positivity to cat and pollen allergens, and she did not smoke. About 5 years prior to examinations at FIOH, she had developed rhinitis, loss of voice, and recurrent sinusitis. The symptoms began during the first year after she started to apply gel nails. In pulmonary examinations at a local central hospital, the spirometry was normal, but there was significant diurnal variation in the PEF measurements and recurrent bronchodilating effects. In the histamine challenge test, there was mild bronchial hyperresponsiveness (PD_{15} 0.154 mg). On the basis of these examinations, bronchial asthma was diagnosed and asthma medication was started with inhaled corticosteroids. In spite of the asthma medication, she experienced dry cough, wheezing, and shortness of breath, especially when preparing gel nails. The patient was referred to FIOH for further examinations due to the clinical suspicion of OA. This patient had no skin symptoms.

At FIOH, the patient had no respiratory symptoms, and her lung auscultation was normal. Spirometry showed mild

peripheral obstruction. Moderate bronchial hyperresponsiveness (PD_{15} 0.29 mg) was noted in the histamine challenge test, and the exhaled NO value was increased (64.9 ppb). In the workplace PEF follow-up, there were no significant diurnal variations, but the patient did not prepare nails during the follow-up. SPTs to common environmental allergens showed allergy to animal epithelia (dog, cat) and to common pollens (alder, hay, mugwort) but no allergy to persulfates; MAs were not tested.

The reference bronchial challenge test was negative. In the active bronchial challenge test, a dual asthmatic reaction occurred. An immediate significant decrease of 20% in the PEF (and a drop of 16% in FEV_1) occurred 35 min after the start (Fig. 2). After 8 hr, a delayed significant drop of 27% in the PEF (19% in FEV_1) could be seen. The delayed drop fluctuated and was sustained until the patient received short-acting bronchodilating medication, after which the PEF and FEV_1 returned to normal.

On the basis of the work-related respiratory symptoms and findings in the pulmonary investigations, including a positive bronchial provocation test, OA due to exposure to sculptured nails containing MAs was diagnosed. Minimizing the exposure to MAs by using respiratory protective equipment was recommended, and asthma medication was continued with inhaled budesonide. At the 6-month follow-up at FIOH, the patient had been unable to continue her work as a nail technician because of respiratory symptoms. Diurnal PEF monitoring with regular asthma medication varied between 420 and 440 L/min, and her spirometry was normal, but the exhaled NO was high (59.5 ppb). Because work-related respiratory symptoms had continued, measurements of vocational rehabilitation were recommended, and the patient was willing to take part in them.

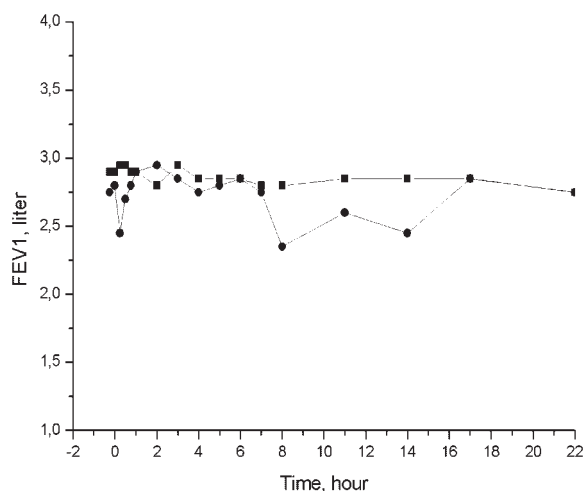


FIGURE 2. Forced expiratory volume in 1 s (FEV_1) follow-up during challenge tests of patient 2 with: (■) lactose powder; (●) methacrylates. Challenge occurred at 0 h.

Chemical Analysis of Gel Nail Materials

The concentrations of MAs in the gel nail materials and in the gel nails themselves were determined after the active challenge test of Patient 2 (Table I). The main MA was HEMA (8%) in the bonding agent and bis-GMA (42%) in the sculpture resin. The sculpture resin also contained 7% of volatile hydroxypropyl methacrylate (HPMA). The identification of the main MA in the sealing resin could not be confirmed. Hardened gel nails contained no detectable amounts of HEMA or aliphatic dimethacrylates.

DISCUSSION

There are at least two methods with which to construct photobonded sculptured nails [Constandt et al., 2005]. In the first method, a monomer liquid and polymer powder are mixed on the nail plate, after which the mixture is hardened by UV light ("acrylic nail"). In the second method, which was the method that both of our patients used, several layers of acrylic gel are applied on the nail plate, and every layer needs separate polymerization for some minutes by UV light ("gel nail"). Generally these processes involve acrylic or methacrylic acids and their esters, as well as cyanoacrylic acid and its esters [Kanerva et al., 1996; Constandt et al., 2005] to which substances the nail technicians are exposed.

ACD is a well-known adverse health effect of exposure to MAs, and it has been described both for dental personnel and for nail technicians [Alanko et al., 2004; Lazarov, 2007]. MAs can cause also respiratory hypersensitivity, including asthma, which has been found in dental personnel in clinical studies [Piiirilä et al., 1998; Lindström et al., 2002; Jaakkola et al., 2007] and which also has been suggested to occur in the application of artificial nails by two previous studies. The first of these latter studies focused on occupational hygiene without data on clinical characteristics or examinations [Spencer et al., 1997]. In the second study, a questionnaire-based survey conducted in the USA, the application of artificial nails was significantly associated with asthma arising in the course of employment as a cosmetologist (relative risk 2.83; 95% confidence interval 1.64–4.88) [Kreiss et al., 2006]. Our present study is the first clinical study including results of a work simulation test to report on OA in nail technicians. For both of our patients, OA from exposure to sculptured nails containing MAs was diagnosed on the basis of a work simulation test combined with the patient's history of occupational exposure and respiratory symptoms.

As the nail technicians polish and sculpture artificial nails, they are exposed to a powder that may still contain some unpolymersed acrylates and MAs. However, the main exposure comes from volatile chemicals, when the technician glues the artificial nails and applies the gel in layers. We analyzed the initial nail materials and the hardened gel nails

TABLE I. Concentrations of Methacrylates Identified in Gel Nail Materials and in Gel Nails

Product	Identified methacrylates	Concentration (% w/w)	Identification
Bonding agent	HEMA	7.5	GC-MS/LC
	EGDMA	0.8	GC-MS/LC
	TREGDMA	0.2	GC-MS/LC
	Ethylene glycol acrylate methacrylate	1	GC-MS
	Ethylene glycol-based dimethacrylates ^a	2	GC-MS
	Ethylene glycol monomethacrylate	0.5	GC-MS
	MMA	<0.05	GC-MS/LC
Sealing resin	HEMA	<0.01	GC-MS/LC
	MMA	<0.01	GC-MS/LC
	EGDMA	0.1	GC-MS/LC
	Ethylene glycol-based dimethacrylates ^a	20	GC-MS
Sculpture resin	HEMA	0.03	GC-MS/LC
	MMA	<0.01	GC-MS/LC
	HPMA	6.7	GC-MS/LC
	TREGDMA	≤0.1	GC-MS
	BUDMA	≤0.1	GC-MS
	Ethylene glycol-based dimethacrylates ^a	12	GC-MS
	Bis-GMA	42	GC-MS/LC
Gel nails	HEMA	<0.01	GC-MS
	Aliphatic dimethacrylates	<0.01	GC-MS

HEMA, hydroxyethyl methacrylate; EGDMA, ethylene glycol dimethacrylate; TREGDMA, triethylene glycol dimethacrylate; MMA, methylene methacrylate; HPMA, hydroxypropyl methacrylate; BUDMA, butandiol methacrylate; Bis-GMA, 2,2-bis-(4-(2-hydroxy-3-methacryloxypropoxy)phenyl)-propane; GC-MS, gas chromatography with mass selective detection; LC, liquid chromatography.

^aTentatively identified according to the presence of the ions 69 and 113 in the mass spectrum.

and found only traces of (small-molecular weight) acrylates in the gel nails but considerable amounts of acrylates in the materials. OA from exposure to MAs has been reported for dental personnel, even though the measured air concentrations are usually well below 100 µg/m³ [Henriks-Eckerman et al., 2001]. In an American study, in which MMA and ethyl methacrylate (EMA) concentrations were measured during the application of artificial fingernails, the former were non-detectable [Spencer et al., 1997], which, however, was an expected result, since there was only trace amounts of MMAs in the bulk products. In that study, although the EMA exposure was lowered significantly by the use of a ventilated table, the authors commented that such tables may not always be effective enough [Spencer et al., 1997].

The mechanism of OA induced by MAs is unclear. Both of our patients displayed a dual type of asthmatic reaction. In association with specific bronchial challenge tests, mainly late or dual asthmatic reactions has been reported to occur in dental personnel exposed to MAs [Piiirilä et al., 1998] or in other occupations exposed to other acrylates (e.g., cyanoacrylates) [Savonius et al., 1993]. These modes of asthmatic reactions refer usually, but not necessarily, to reactions other than hypersensitivity type I. Consistent with this, positive SPT reactions to (meth)acrylates have not been reported on, nor have immunoglobulin E (IgE) antibodies been found in

sera [Piiirilä et al., 1998; Lindström et al., 2002]. For our patients, the SPTs to MAs were negative for one and were, unfortunately, not performed for the other. Interestingly, our first patient not only developed OA, but also ACD from exposure to MAs. In line with this, her patch tests to MAs were positive but SPTs negative; therefore, the mechanism involved was a hypersensitivity type IV reaction, as the clinical picture of contact dermatitis indicated. This association between OA and ACD from exposure to MAs in the same person has been described earlier only once in the literature, in a dentist [Lindström et al., 2002]. Taken together, although our results do not rule out a possible IgE-mediated mechanism, the pathophysiology of OA in relation to MA exposure probably involves other immunological mechanisms. The situation is not dissimilar to other chemical induced OA, such as OA from exposure to diisocyanates, in which diisocyanate antigen specific IgE responses are not demonstrable in most workers with OA, and in which, therefore, immunologic as well as non-immunologic mechanisms have been accounted for [Bernstein and Jolly, 1999; Tarlo and Liss, 2002].

Useful recommendations to control chemical hazards during the application of artificial fingernails have been published, including ventilated tables, good work practices, and appropriate personal protective equipment [Spencer

et al., 1997; Anonymous, 2001]. Although our first patient used protective gloves and a ventilated table with a downward draft, she still developed ACD and OA due to skin and respiratory exposure to MAs, respectively. Obvious reason for development of ACD is that natural rubber gloves which she used do not provide good protection for MAs [Mäkelä et al., 1999]. Unfortunately, we had no information on the flow rates of the down draft table. It is also possible that the causative exposures occurred in other parts of the salon. Taken together, this case shows how difficult it is to control exposure to sensitizing chemicals, such as MAs.

In conclusion, we described the cases of two nail technicians who both developed OA from occupational exposure to sculptured nails containing MAs and for whom the diagnosis was confirmed by a work simulation test. Our findings confirm earlier indications of such an association and are important because the use of artificial nails is growing in popularity. Although we could not explicitly identify the causing agent(s) of OA, our results emphasize that adverse respiratory health effects of MAs are not restricted to MMA but also other MAs play a role. When handling substances with adverse health effects, the first priority is to eliminate the exposure completely by the substitution of non-toxic alternatives. Therefore, products that do not contain MAs should be used to prevent OA and ACD. If these are not available, MAs should be handled with appropriate safety measures including the use of a ventilated table with good work practices and appropriate personal protective equipment. If occupational symptoms develop, the worker should be transferred to other work tasks and referred to appropriate clinical medical investigations. If OA or ACD is diagnosed based on these investigations, the worker usually has to change her/his job. Finally, it is important to arrange the ongoing medical surveillance of the worker, and even more important it is, if a sensitized worker with asthma or ACD still remains in exposure.

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