

Case report

Occupational pharyngitis associated with allergic patch test reactions from acrylics

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A female dentist specialized in orthodontics repeatedly developed symptoms of pharyngitis at work. A chamber provocation test indicated that her symptoms were caused by acrylics. Prick tests with acrylics were negative, while patch tests were strongly positive although the patient had no skin symptoms. The relationship between the symptoms and the patch test results is discussed. It is suggested that type IV allergic reactions may be involved in symptoms of the upper respiratory tract.

Occupational pharyngitis is usually caused by chronic exposure to dry, dusty, or hot air, or by tobacco smoke, or sometimes by industrial fumes (3, 5). Occupational irritant or allergic pharyngitis caused by specific chemicals is verified only occasionally. Here we present the case of a dentist who was diagnosed as having occupational pharyngitis caused by dental acrylics. She had no skin symptoms, but strong patch test reactions to several acrylics.

Case report and methods

A non-smoking (never smoked) 45-year-old female dentist specialized in orthodontics started getting symptoms of irritation and soreness of the throat at her workplace in the spring of 1990. The symptoms disappeared during weekends and holidays and reappeared 2–3 days after restarting work. The dentist had been working as an orthodontist since 1979, being increasingly exposed to acrylics because of the growing use of dental composite resins in her practice. She did not have tightness of breath, asthmatic symptoms, rhinitis or conjunctivitis. Twenty-five years earlier she had had tonsillectomy. Her own and family history of atopy were negative, and she had never had any skin problems.

Several dental personnel at her workplace had become allergic to acrylics (11) and therefore she consulted the university dermatology clinic, where positive patch test reactions to acrylics were revealed. As she had no skin symptoms, she was referred to our clinic to clarify whether the respiratory symptoms were occupational. Skin tests were per-

formed, scored and evaluated as previously described (1, 7). On patch testing, the standard series, as well as the plastics and glues series (Chemotechnique Diagnostics AB, Malmö, Sweden) were negative, but 18 of 30 acrylics (Chemotechnique) provoked mild to strong allergic reactions (Table 1). Three acrylics from the dentist's workplace also induced strong positive patch test reactions. Both components of the Scotchbond-2-Dental Adhesive System (3M Corporation, St. Paul, MN, USA), namely Scotchprep Dentin Primer (1% wt/wt in petrolatum), containing 30–65% of 2-hydroxyethyl-methacrylate (2-HEMA; (11)), and Scotchbond-2-Light Cure Dental Adhesive (containing 50–60% BIS-GMA and 40–50% 2-HEMA; (11)) caused 3 + allergic test reactions. Also Servibond Primer (manufactured by Ortho Enterprises Ltd, Sheffield, UK and distributed by Servident, Turku, Finland) gave a positive patch test reaction (1%, 3 +; 0.1% 2 +; 0.01% negative; all test substances in petrolatum). According to the manufacturer, Servibond Primer contains triethylene dimethacrylate and isobutyl methacrylate, but the manufacturer did not provide more detailed information on the components or their proportions. The standard prick test series of environmental allergens (10) were negative, as were prick tests with latex, methyl methacrylate (MMA) conjugated human serum albumin (HSA; (9)) and polyethyleneglycol methacrylate, and MMA in water (1%). The patch test substances (in petrolatum), 2-HEMA (0.5%) and TREGDMA (0.1%) also gave negative prick test results.

Table 1. Patch test results with the acrylics (6-day reading)

(Meth)acrylate series (1–30)		% (w/w)	
Ethyl acrylate	(EA)	0.1	3+
Butyl acrylate	(BA)	0.1	2+
2-Ethylhexyl acrylate	(2-EHA)	0.1	–
2-Hydroxyethyl acrylate	(2-HEA)	0.1	3+
2-Hydroxypropyl acrylate	(2-HPA)	0.1	3+
Methyl methacrylate	(MMA)	2	3+
Ethyl methacrylate	(EMA)	2	3+
n-Butyl methacrylate	(BMA)	2	2+
2-Hydroxyethyl methacrylate	(2-HEMA)	2	3+
2-Hydroxypropyl methacrylate	(2-HPMA)	2	3+
Ethylene glycol dimethacrylate	(EGDMA)	2	3+
Triethylene glycol dimethacrylate	(TREGDMA)	2	2+
1,4-Butanediol dimethacrylate	(BUDMA)	2	1+
Urethane dimethacrylate	(UEDMA)	2	–
2,2-bis[4-(2-methacryloxyethoxy)-phenyl]propane	(BIS-EMA)	1	–
2,2-bis[4-(methacryloxy)phenyl]propane	(BIS-MA)	2	–
2,2-bis[4-(2-hydroxy-3-methacryloxypropoxy)phenyl]propane	(BIS-GMA)	2	–
1,4-Butanediol diacrylate	(BUDA)	0.1	3+
1,6-Hexanediol diacrylate	(HDDA)	0.1	2+
Diethylene glycol diacrylate	(DEGDA)	0.1	3+
Tripropylene glycol diacrylate	(TPGDA)	0.1	–
Trimethylolpropane triacrylate	(TMPTA)	0.1	–
Pentaerythritol triacrylate	(PETA)	0.1	?+
Oligotriacrylate 480	(OTA 480)	0.1	–
Epoxy diacrylate	(BIS-GA)	0.5	–
Urethane diacrylate (aliphatic)	(al-UDA)	0.1	–
Urethane diacrylate (aromatic)	(ar-UDA)	0.05	–
Triethylene glycol diacrylate	(TREGDA)	0.1	3+
N,N-Methylenediacrylamid	(MBAA)	1	–
Tetrahydrofurfuryl methacrylate	(THFMA)	2	3+

Chamber provocation test. The patient was exposed in a chamber to a placebo chemical (a polyol), Servibond Primer and Scotchbond Dentin Primer. The method was the same as used for lung provocation tests (9, 14). Servibond exposure was performed with 2 different concentrations. One (Servibond and Scotchbond) or 3 drops (Servibond) of the dental acrylics were smeared on an objective glass and left in the chamber with the patient for 30 min. The patient's otorhinolaryngeal (ORL) status was estimated immediately before, 30 min after, and 10 h after the beginning of the exposure. The signs of redness and swelling were rated with a 4-stage scale: 0 = no; 1 = mild; 2 = moderate, and 3 = abundant. The test was interpreted as positive if both signs (redness and swelling) were increased by at least one point. A cell smear of the throat was taken 2 h after the exposure. Respiratory function (FEV) was followed for 12 h after the exposure.

The placebo and Scotchbond Dentin Primer had no effect. Servibond Primer (in 2 concentrations, see above) caused significant swelling and redness of the throat, which was considered diagnostic of occupa-

tional pharyngitis. None of the exposures affected pulmonary function. Pharynx cell cytology was not influenced by the exposures; the amount of neutrophils, non-ciliated and epithelial cells remained unchanged, and mast cells, eosinophils and lymphocytes were absent. Servibond (3 drops) had no effect on the ORL status of 5 healthy controls.

Other examinations. The blood and urine pictures were within normal range. The leukocyte differential count, the number of eosinophils and IgE (40 kU/l) were normal. Thorax and sinus X-ray, spirometry with the bronchodilatation test, and histamine provocation (9, 10) were normal.

Discussion

Acrylics are volatile liquids at room temperature and may irritate the respiratory tract. Rats exposed to 75 ppm of acrylic acid revealed damage to the nasal mucosa within 14 days, and mice showed similar damage with a much smaller dosage (5 ppm) (13). Increasing amounts of various types of acrylics are used in dentistry (17) and dental personnel exposed to acrylics often complain of respiratory symptoms. The newer acrylic products may even be more allergenic than the older ones (8, 11) and possibly also stronger irritants.

The symptoms of pharyngitis are discomfort in the throat, pain on swallowing and occasionally earache. The mucous membranes of the throat are inflamed, i.e., hyperemic and swollen. In addition, in hypertrophic pharyngitis there are islands of lymphoid tissue in the posterior pharyngeal wall. Pharyngitis is usually associated with upper respiratory infection, but also irritants, and probably allergens can cause an inflammatory condition. The most common irritants causing pharyngitis are industrial fumes and tobacco smoke (3, 5). Occupational pharyngitis, either irritant or allergic, caused by specific chemicals is very rarely diagnosed in Finland, according to the statistics on new cases of occupational diseases (Table 2). In 1990 2 cases were diagnosed (Table 2): the present case, and one caused by styrene.

The chamber provocation test combined with the anamnestic data was considered sufficient to diagnose the case as occupational pharyngitis caused by

Table 2. Incidence of occupational pharyngitis and allergic rhinitis in Finland during 1986–1990

	1986	1987	1988	1989	1990
Occupational pharyngitis	1	2	2	1	2
Occupational allergic rhinitis	127	128	150	228	298

acrylics, and the patient has consequently been compensated for an occupational disease. The prick test reactions were negative, however, and the allergic nature of the pharyngitis could not be proved. In rare cases, acrylics have caused type I allergy, asthma, and/or positive immediate skin tests (4, 12, 15, 16). Evidently, the immediate pharyngitis that developed after the chamber provocation test with acrylics was irritant in nature, although type I allergy could not be completely excluded because the prick test reactions may have been "false negative". HSA-conjugated prick test substances (9) are largely manufactured on a trial and error basis (18) and we cannot be certain that our test substances are sufficient to detect type I allergy, as we have no proven cases of IgE-mediated acrylic allergy to compare our results with.

Interestingly, the patient had strong allergic patch test reactions to several acrylics, although she had never had skin symptoms, i.e. she had "latent" type IV contact allergy. It seems feasible that the patch test reactions had some significance to the clinical signs of pharyngitis. The anamnestic data indicated that the patient had delayed symptoms of pharyngitis. After vacations the symptoms did not start immediately, but after 2–3 days of work. All the necessary cellular elements for a delayed type IV allergic reaction are present in the mucosa of the pharynx (6), and acrylics are volatile at room temperature. The great number of positive allergic patch test reactions indicates both cross and concomitant sensitization (8, 11). Accordingly, the sensitized patient may get symptoms from many acrylics.

The patient had probably become sensitized from skin contact with acrylics, although the significance of airborne contact cannot totally be neglected. As the patient was aware of the sensitizing capacity of the acrylics, she probably succeeded in avoiding direct skin contact, and accordingly had no skin symptoms (latent allergy).

We have earlier reported on patients with occupational allergic rhinitis and dermatitis from fur dyes (2). Nasal challenge with paraphenylenediamine (1% w/w in petrolatum) or scrapings from the leather side of suspected furs caused a delayed rhinitis reaction 8–12 h after the challenge. The patients also had positive patch test reactions to the same allergens. The present and the above previous cases indicate that type IV allergic reactions may be involved in allergy of the upper respiratory tract.

In conclusion, we have presented a subject who got immediate reactions from acrylics when exposed in a provocation chamber. This was possibly caused by irritant mechanisms. Further, our patient had allergic patch test reactions to acrylics. It is hypothesized that these type IV allergic reactions were involved in the pathomechanism of pharyngitis.

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