OCCUPATIONAL ASTHMA WITH A HIGH TITRE OF IgE (CASE REPORT)

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ABSTRACT

A case of occupational asthma due to exposure to epoxy resin smokes is described. The patient who was not atopic developed a high titre of IgA and IgE. When exposed to epoxy resins at the workplace he became dyspnoic and had decreased ventilatory volumes. After the bronchial provocation test reproducing conditions characteristic of his working environment, he developed typical symptoms of occupational asthma.

A case of occupational asthma in a 48 year-old worker who was mixing and decanting acids (HCl, H2SO4, CH3COOH, HF) for about ten years is described. During the last three months the worker has been exposed to the smoke of warm epoxy resins liquefied by pressing. His first troubles started about three years ago, with mild dyspnoea, when he was at work. Successively, and especially during the last three months, dyspnoea deteriorated and started to appear also 5-6 hours after the end of work. At admission to the clinic the patient had evident signs of expiratory rattles and hisses all over the lungs; X-ray showed mild accentuation of the vascular bronchial picture. FEV1 was reduced by 5% compared to the lowest theoretical value of C.E.C.A.; Tiffenau test showed a value of 51%; the residual volume was increased by 41%; ECG findings were normal. Patch test series using standard I.R.G.C.D. showed negative reactions. Prick tests (trees, grasses, weeds, flowers, cereals, flour, inhalants, epithelia, moulds) showed also negative reactions. The analysis of immunoglobulins showed the following results: IgA 234.5 mg/100 ml; IgG 932 mg/100 ml; IgM 121 mg/100 ml; IgE 3220 U/ml. Stool examination revealed no parasitic infestation. The patient after being removed from the work environment for 20 days did not complain of dyspnoea, which however reappeared three days after he resumed work again. The bronchial provocation test with acetylcholine (FEV1) did not change, compared to the baseline (or initial) values. The administration of salbutamol aerosol improved the ${\rm FEV_1}$ value by a rate of 30% compared the baseline value.

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The epoxy resin used in the working cycle served also for the bronchial provocation test. The exposure of the patient to the smoke of the heated resin provoked objective signs of dyspnoea; the measurements of FEV₁ were calculated by a closed circuit spirometer after 5, 30, 60, 180, 300 minutes and 24 hours after exposure had ceased. Immunoglobulins were determined at the beginning of the bronchial provocation test and again during dyspnoea. The following results were obtained:

- Before provocation: IgE 3220 U/ml (CRIST); IgG 1082 mg/100 ml (immunodiffusion according to Mancini); FEV₁ 2695 cm³;
- 60 minutes after provocation: FEV₁ 2255 cm³;
- 5 hours after provocation: FEV₁ 2475 cm³.

A dyspnoic attack with rare expiratory hisses in the middle of the chest occurred in the patient about 230 minutes after the provocation test. The relevant ventilatory tests and laboratory values were: FEV₁ 2145 cm³; IgE 3225 U/ml; IgG 1285 mg/100 ml. The patient needed medical help for dyspnoic troubles, and therefore the test was discontinued.

Back to work after discharge from the hospital, the patient took up a new job in the factory without contact with epoxy resins. He was followed-up monthly in the out-patient clinic as far as determination of immunoglobulins and control of respiratory troubles were concerned. Immunoglobulin values were in a normal range and no asthmatic dyspnoea was present IgE was 720 U/ml after the first and 470 U/ml after the second month (normal values 14–720 U/ml).

The data presented indicate that the subject who was not atopic developed occupational asthma by inhalation of epoxy resin smokes. Epoxy resins belong to the group of micromolecular substances having both irritating and sensitising properties. In our patient the diagnosis was proved by the bronchial provocation test which reproduced the conditions characteristic of the patient's usual working environment.