

Herbal tea asthma

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Some of the better recognised occupational asthmas associated with exposure to materials of plant origin include asthma from wheat and other grain flours, western red cedar, castor beans, and coffee beans.^{1 2} "Tea worker's asthma" is less well known although it has been described in both case reports and group studies.³⁻⁸ As with many occupational syndromes, it was first reported some time ago,⁸ followed by a lengthy hiatus of research activity, and finally became the subject of renewed interest in recent years. Tea associated reactive airway disease has been reported in cultivators of common tea (*Camellia sinensis*),⁶ in processors of this tea,^{3 4} and in processors of various different "herbal" teas. These herbal teas have included sage, camomile, dog rose, and mint.^{5 7} We present a case of reactive airway disease associated with occupational exposure to a mixture of herbal teas and documented by a positive inhalation challenge test.

Case report

The patient is a Hispanic man who was 33 at the time of his initial visit. He was referred for an assessment to determine whether his bronchial asthma might be occupationally related. The patient had been in good health until about three years previously when he began to experience shortness of breath, wheezing, and cough productive of white sputum. These symptoms resulted in multiple visits to the emergency room. Although the patient described a sense of fever and reported chills, he was never noted to be febrile on any of these visits. Emergency room records, however, documented tachypnoea, diffuse rhonchi, and wheezes by physical examination. Bronchodilators gave good symptomatic relief. X ray examinations were carried out on several of these visits without any evidence of parenchymal infiltrates. Peripheral eosinophilia ranged from 8 to 14% (absolute count from 570-1230/mm³) during these exacerbations. Outpatient pulmonary function testing two years before his initial assessment at our clinic (one year after the onset of symptoms) yielded

the following values: FVC 5.50 (exp 5.20); FEV₁ 3.09 (exp 4.01); FEF_{2.5-7.5} 2.37 (exp 4.36). FEV₁ increased to 3.86 and FEF_{2.5-7.5} to 2.78 after administration of bronchodilators. (All values in litres.) Arterial blood gasses were essentially unremarkable.

About six months before his first respiratory complaints the patient had begun employment processing herbal teas. His job responsibilities includes packing bulk tea in both leaf and finely ground form. The herbal tea consisted of a mixture of materials not all of which the patient could identify but did include chaparral, red clover, and mint. He described the work environment as being quite dusty. In the first seven months of the patient's employment no respiratory protection was provided. Later he was supplied with a filter type, disposable face mask. By this time symptoms of dyspnoea had developed.

Respiratory symptoms were worse by the end of the work week and would improve over the weekend. There was no complaint of delayed symptoms some hours after work. Eventually, the symptoms became severe enough so that he voluntarily ceased employment in the herbal tea facility. Nevertheless, his respiratory complaints continued. The total duration of employment was 18 months.

There was no history of childhood asthma or atopy or of serious respiratory infection. The patient had been in a motor vehicle accident five years before presentation and had been admitted to hospital for a left sided rib fracture and minimal pneumothorax that resolved without complication. He is a non-smoker. Past occupational history was non-contributory. There was no family history of asthma or lung disease.

On initial physical examination the patient was noted to be mildly tachypnoeic with scattered expiratory rhonchi and wheezes by pulmonary auscultation. The rest of the physical examination was unremarkable. There were no nasal polyps, lymphadenopathy, cardiac abnormalities, or peripheral cyanosis, clubbing, or oedema.

Analysis of quantitative immunoglobulins showed an IgE of 720 IU/ml (normal range less than 100

IU/ml); IgG 1170 mg/100 ml (normal range 759–1228 mg/100 ml), IgA 320 mg/100 ml (normal range 104–228 mg/100 ml); and IgM 104 mg/100 ml (normal range 64–174 mg/100 ml). Radioallergosorbent testing (RAST) yielded a positive response to common ragweed, English plantain, wheat, oats, peanuts, soy beans, house dust, and perennial rye grass. RAST was negative for mixed moulds including aspergillus and to dog and cat epithelium. The patient underwent pulmonary challenge testing by placing a herbal tea sample from his former place of employment in and out of a plastic bag and inhaling the generated dust. The measured baseline FEV₁ was 3.1 l, decreasing to 1.6 l five minutes after initial inhalation. Coughing, wheezing, and conjunctival infection were noted by physical examination. Thirty minutes after treatment with metaproterenol inhalent, the FEV₁ returned to the baseline value.

Discussion

This patient's history exhibits many of the features of classic occupational asthma: symptoms beginning insidiously after an initial quiescent "honeymoon" exposure period; symptoms improving after several days away from exposure and worsening by the end of the work week; increasing severity of symptoms with continued exposure; and, as is seen not uncommonly, symptoms persisting even after cessation of the original exposure. The patient's eosinophilia and raised IgE are consistent with allergic asthma.

The negative RAST assay for mixed moulds in this case is as interesting as the positive response to various plant substances. Since "tea factory cough" was described by Castellani and Chalmers some 70 years ago,⁸ it has been theorised that tea associated reactive airway disease may be caused by fungal contaminants. This supposition has been reiterated by Uragoda³ and Gu Xue-Qi.⁹ The negative RAST in this case would argue against such a fungal aetiology. This patient showed a continuing pattern of hyperreactivity that may include sensitivity to a variety of allergens. It cannot be determined if this hyperreactivity was present subclinically before his initial workplace sensitisation or if, alternatively, the patient's primary workplace sensitisation may have

been aetiologically concerned in triggering a wider pattern of non-specific hyperreactivity.

The precise allergen in this case was not identified. Herbal tea, unlike common tea, is in many cases a mixture of plant species, some of which may be relatively more immunogenic than others. Zuskin and Skuric, in a study that was able to compare several different teas, found sage to have the highest allergenicity by skin testing although it did not seem to be more potent in terms of symptoms or spirometric changes.⁵ In a study of workers exposed to mixed herbal tea dusts, Castellan *et al* could find no correlation between dust exposure and preshift and postshift changes in FEV₁, although self reported symptoms were significantly higher in workers exposed more heavily.⁷ Unfortunately, FEF₂₅₋₇₅ was not measured in that study. Nevertheless, there has been at least one case report of anaphylaxis associated with ingestion of herb tea.¹⁰ The herbal tea worker, such as this patient, may be more at risk of immune mediated asthma simply because of the variety of potentially immunogenic exposures in the workplace.

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