

Silk-Induced Asthma

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ABSTRACT

The existence of professional allergens in the home environment is important because their avoidance usually resolves the problem completely. We report on a case of an asthmatic woman who, for five years, was helping her husband create large artistic screens with the cocoons of the silkworms. Extensive laboratory evaluation revealed that she was allergic to silk and her symptoms resolved in three months following discontinuation of her exposure to silk allergens. (Allergy and Asthma Proc 20:107–108, 1999)

Silk¹ is made from the unwound cocoons of a particular group of moths. The finer grades are harvested before the moth emerges (i.e., the moth is killed) because, when it emerges, it eats through one end of the cocoon, cutting most of the fibers into much shorter lengths. Most silk is cultivated, and produces a very fine white product. Other silk is wild and produces a shades-of-beige product. Silk can be made from certain varieties of spider cocoons, but they are much more difficult to farm than the silkworm moth cocoon. In recent years, improvements have been made in sericulture (silkworm raising), and thus, silk is much less expensive than it used to be. The oldest silks found are from China, dated to 1500 BC, although the word for silk in Chinese is known to be older than that. In China during the Han Dynasty (200 BC–AD 200), silk was part of a soldier's standard wages. The establishment of the Silk Road sometime between 160 and 140 BC marks the beginning of silk use in the West. It was still rather rare, but when the Crusaders began returning from the mideast with silk, demand developed and better trade routes to Europe were established. Venice, Lucca, and Florence became renowned centers of silk weaving starting in the 14th century. These cities specialized in heavy brocades and patterned velvets, which were exported to the whole of northern Europe.

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Silk and silkworm proteins have been clearly incriminated for provoking occupational asthma.¹ The mechanism through which specific allergens trigger asthma is immunologic and is attributed to the production of specific IgE antibodies against certain silk proteins.^{1,2} Silk sensitization is more common in atopic individuals, that is, subjects with congenital predisposition to produce specific IgE antibodies to common environmental allergens.³ Although the main trigger for thinking of silk hypersensitivity type I is occupational exposure to this allergen,^{3,4} in some instances, like in the case presented here, silk treatment can be made in the home environment as well. Looking for professional allergens in a home environment is important because their avoidance usually leads to complete cure of the disease.

CASE REPORT

A 28-year-old woman, high school teacher, was referred for allergy evaluation because of chronic rhinitis and asthma starting three years previous. She was receiving pharmacotherapy with albuterol, sodium cromoglycate, and theophylline. In a spirometry performed before the initial examination, the forced expiratory volume in one second (FEV₁) was 1.90 L (normal value: 3.10 L) and the forced vital capacity was 3.20 L (normal value: 3.60 L). These parameters were improved following albuterol inhalation. Lung specialists diagnosed nonallergic asthma and replaced sodium cromoglycate with beclomethasone dipropionate. The disease started with watery rhinorrhea, sneezing, nasal itching, severe nasal congestion mostly at night, and dyspnea mostly after midnight. Her symptomatology was not environmental-specific, although there was a mild worsening of her symptoms during spring and autumn.

For the last five years, her husband was occupied at home with a form of local art creating large screens with the cocoons of the silkworms. Cocoons were painted by her husband and were stuck together with an ordinary glue used to glue paper to form a picture on a canvas. The patient helped him in her free time without worsening of her symptoms. She was only cutting cocoons into different shapes (e.g., butterflies, flowers, etc.) and had no access to paints or glues that were used exclusively by her husband.

Her personal history revealed no coexisting diseases. Her mother suffered from severe perennial bronchial asthma.

The patient submitted to skin-prick testing with the most common aeroallergens and the serum total IgE levels were evaluated along with testing for the presence of specific IgE for common allergens with the RAST method. To exclude a possible involvement of silk sensitivity, a specific identification of specific IgE for silk with the RAST method (Pharmacia) was performed. The patient underwent skin tests and both nasal and bronchial challenge for silk sensitivity. Skin testing, because of a lack of silk extract, was performed with a modification of the scratch method. In the inner surface of the forearm, a microscopic laceration of the skin (approximately 1 cm²) was performed with a regular needle followed by scratching of the wound with the outer surface of the silk worm's cocoon. The same procedure was performed with both the silkworm nymph and the inner surface of the cocoon. Three atopic patients served as the control group. Tests were evaluated after 15 minutes, and the appearance of a wheal bigger than that of histamine (1 mg/mL) was considered as positive. Both nasal and bronchial provocation were performed with the classic method of reproducing the working conditions in a hospital room.^{5,6} The patient brought cocoons with her and was asked to do what she was doing at home while helping her husband. Spirometry was performed before and every 5 minutes during the work. Albuterol and cromolyn were stopped 24 hours before the provocation.⁶

RESULTS

Skin tests and RASTs to common allergens were negative. Serum total IgE level was elevated (334 KU/L). Specific IgE levels to silk waste (K73: quilt stuffing containing silk waste from *Antheraea* sp. living on oak leaves) were 31.2 peripheral resistance units (PRU)/mL (grade 4) and silk (K74: cocoons from *Bombyx mori* living on mulberry leaves) were 12 PRU/mL (grade 3). Skin tests to the outer surface of the cocoon were strongly positive (4+) and, to a lesser degree, to the silk-worm's nymph (3+). In the control group, skin tests were all negative. Both nasal and bronchial provocation were positive. Seven minutes after the beginning of the usual work, she developed nasal symptoms (sneezing, catarrh, nasal itching, and nasal congestion). Nasal symptomatology was more severe as provocation testing was in progress. Spirometry performed every five minutes was still normal. Forty-three minutes after the beginning of the provocation, the patient experienced mild dyspnea. On-site spirometry showed significant decrease of FEV₁ (from 2.7 L to 1.7 L: 39% reduction). The challenge was discontinued and an inhaled bronchodilator was instituted (albuterol). A second spirometry after three minutes showed that FEV₁ was back to normal (2.90 L). Provocation challenge was considered positive and the patient remained under close observation for six hours with spirometry performed every hour for detection of a late-phase reaction. FEV₁ remained steady during the observation

period. The patient avoided the allergen for the next few months (her husband moved the whole setup to her mother-in-law's house), and she was able to discontinue albuterol and cromoglycate within three months. In a new spirometry performed four months after the challenge, the recorded values were normal for her age and height.

DISCUSSION

It is well known that sensitization to allergens does not necessarily imply an etiologic relationship between the allergen and the disease.^{11,12} In this specific case, the positive nasal and bronchial provocation to silk, in combination with the improvement of the disease in such a short period (3 months) after the complete avoidance of the responsible allergen, confirms the etiologic relationship of asthma caused by sensitization to silk. Avoidance of occupational allergens does not always lead to complete cure of asthma. The duration of asthma after the discontinuation of exposure to a specific allergen depends on the origin of the occupational allergen and the exposure time.^{12,13} For example, Red Cedar dust that causes asthma can last even for five years after change of occupation.¹⁴ As for silk allergy, there are not many papers published. In the present case, asthma symptoms completely resolved in three months despite the fact that the patient has been exposed to silk for five years and had symptoms for three years.

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