Tea asthma: response to specific and non-specific challenges

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We recently were asked to see a patient with a history of “tea asthma.” Although she had been off work for months without any occupational exposure, and in addition had neither symptoms nor signs when examined, a specific inhalation challenge with tea was positive. A histamine test carried out before the challenge with tea had been negative but when repeated 24 hours after specific challenge it had become positive.

Case history

The patient was a 43 year old woman who had worked for the same company for 25 years. During the eight years before being seen she had cleaned the machines that initially fill tea bags and then later seal them. A fine, yellowish brown dust composed of microscopic tea particles was generated during the bagging. At the end of each day, the machines and immediate surroundings were covered with a fine layer of tea dust. Her symptoms had started 11 months before being seen and consisted of shortness of breath along with intermittent wheezing. She had noted that during a ten day holiday she had been much better and her symptoms had completely disappeared, only to recur when she went back to work. On close questioning, she also stated that she felt better at weekends and worse while at work and had missed several days due to shortness of breath and wheezing. Before the onset of symptoms she had missed almost no time from work at all. She gave no history of allergies or asthma and there was no history of atopy. She had smoked about ten cigarettes a day for around 25 years but gave this up at the onset of her symptoms.

On examination, she appeared well. The chest, and indeed the whole examination, was completely normal. Her chest radiograph likewise showed no abnormalities. Routine spirometry showed a FVC of 3-05 l (90% of the predicted) and a FEV₁ of 2-17 l (76% of the predicted). This yields a FEV₁/FVC of 71% which is slightly below the predicted normal for her age.

A histamine challenge was negative, showing only a slight fall in the FEV₁ (12%) at 8 mg/ml. Immediately thereafter she was given nebulised salbutamol and her FEV₁ returned to 2-41 l. Two hours later she was challenged with finely powdered dextrose which produced no response. The challenge was then repeated with tea dust. Both the dextrose and tea challenges were effected by having her sit in a small room and dropping the dextrose and tea dust from one tray to another. Measurements of her FEV₁ were carried out at five minute intervals for the first 15 minutes and then at quarter of an hour intervals for the next several hours. Before the tea challenge her FEV₁ was 2-45 l. Three hours after the challenge, her FEV₁ fell to 2 l (an 18% decrement). This was associated with tightness in her chest, but wheezes were not heard. After nebulised salbutamol, her FEV₁ returned within minutes to 2-45 l. She noted mild chest tightness over the night and had to use her salbutamol inhaler. The next day the histamine challenge was repeated. Her initial FEV₁ was 2-01 l (74% of the predicted) and fell to 1-67 l with the 2 mg/ml dose of histamine, thus indicating that she had an increase in her non-specific bronchial reactivity.

Discussion

Occupational asthma is best defined as variable airways narrowing causally related to exposure to a specific antigen in the workplace, be the latter dust, gas, vapour, or fume. The symptoms may occur shortly after exposure—that is, in the first 30 minutes—or may be delayed several hours, with the delay ranging from three to 16 to 24 hours. Once symptoms develop, they may take several weeks or longer to resolve. Difficulty may arise in distinguishing occupationally induced asthma from occupationally provoked asthma. For diagnostic, social, and medicolegal purposes it is frequently important to confirm that the asthmatic attacks are related to exposure to a specific agent. Although skin testing may yield positive results, the reactions are frequently non-
specific. Similarly, serial measurements of peak flow while at work, although helpful in providing circum-
estantial evidence, suffer from the drawback that there is substantial variability in peak flow and it is impos-
sible to ensure that the patient does not deliberately
"adjust" the results to match his history. The most
reliable diagnostic test, and indeed the gold standard
for the diagnosis of occupational asthma, is the
demonstration of a change in airway calibre produced
by the inhalation of the specific agent suspected of
causing the symptoms.3

Differences of opinion arise as to the most useful
criteria to establish a diagnosis of occupational
asthma. Airway hyperreactivity is usually measured
by the method described by Cockcroft et al,6 and
involves the inhalation of increasing doses of his-
tamine or methacholine. Non-specific airway reac-
tivity is frequently present in patients with all forms
of asthma, including occupational asthma.7 Cartier et al
have shown that the severity of the late asthmatic
response is related to the magnitude and duration of
histamine bronchial responsiveness and have sug-
gested that specific and non-specific bronchial reactivity
result from the same mechanism—that is, inflamma-
tion and smooth muscle contraction.6 Non-specific
hyperresponsiveness, however, persists long after
airflow obstruction has returned to normal.7 Alter-
native explanations must therefore be considered.
Moreover, Burge and Hargreave et al have shown that
occupational asthma may be present in the absence of
increased sensitivity to histamine or methacholine,
suggesting that different mechanisms account for the
bronchial hyperreactivity in the two conditions.5,9

When a subject is challenged with the suspected
agent and this induces changes in non-specific
hyperreactivity in the absence of a change in airway
calibre this presents a diagnostic problem. Some
investigators have suggested that an increase in non-
specific airway reactivity after specific antigen
challenge may be more sensitive and just as specific as
changes in airway calibre.10 It has been suggested that
despite the lack of change in airway calibre on specific
challenge, increases in non-specific hyperreactivity
after challenge with the suspected agent are significant
and of diagnostic import. The basis for this assump-
tion is somewhat tenuous. Our patient, who has a rare
form of asthma and who had been separated for some
considerable time from exposure to the antigen, had
normal bronchial hyperreactivity before specific
challenge. She had, however, retained her sensitivity to
tea dust. The following day when the histamine
challenge test was repeated a pronounced increase in
her non-specific reactivity had developed.

It is clear from Chan-Yeung's studies that the
severity and persistence of asthma are related to the
persistence and severity of non-specific bronchial
hyperreactivity.2 It is doubtful, however, whether
affected subjects ever lose their specific sensitivity to
the antigen. Thus there are reports of people who have
had no exposure for many years and when they
subsequently undergo a specific challenge show a
definite decrease in FEV1, or similar index of airflow
obstruction.3 For the present it is suggested that the
gold standard for the diagnosis of occupational
asthma must remain inhalational challenge with the
suspected agent. A non-specific increase in bronchial
hyperreactivity in the absence of changes in airway
calibre may occur after non-specific challenge with
various irritants ranging from ozone to aerosolised
citric acid.10,11

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