Emphysema due to smoke from a herbal asthma remedy

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Introduction
Smoke from herbal asthma remedies may cause the development of emphysema.

Case report
A 79-year-old man was referred to us for possible lung volume reduction surgery. He was a lifelong non-smoker but had had mild asthma since the age of 10. He had never required hospitalization nor used inhaled bronchodilators until a few years previously when he had been diagnosed with COPD. His MRC dyspnoea score was 4.

Spirometry revealed an FEV₁ of 0.88L (25% predicted), an FVC of 4.48 L (94%) predicted with an FEV₁/VC ratio of 24% and no additional improvement with bronchodilators. Lung volumes were increased; total lung capacity was 117% predicted and residual volume 156% predicted. Gas transfer values were reduced; TLco of 35% predicted, Kco of 63% predicted. Arterial PaCO₂ was 4.0 kPa and PaO₂ 7.2 kPa. CT thorax showed diffuse emphysema throughout both lungs, with bullous changes at both bases (Figure 1).

He reported no occupational exposure to any pulmonary irritants, nor any exposure to animals or birds. Further enquiry revealed that from the age of 12 years, for about 50 years, he had controlled his asthma by inhaling smoke from a preparation called Potter’s ‘Asthma Remedy’, the presumptive cause of his emphysema.

Discussion
This case highlights the need, especially in older patients, to consider historical medications and alternative therapies as well as current treatment. Asthma was described by early civilizations and treated with herbal smoke inhalation (Ebers Papyrus 1889). Traditional Chinese medicine advocated use of herbs, such as Ephedra sinica, containing ephedrine as a bronchodilator. Datura stramonium and Lobelia inflate, the key constituents of the Potters ‘Asthma Remedy’ (Saunders M. 2009 Inhalitorium Museum Collection. See http://www.inhalatorium.com/index.html) used by our patient, were commonly used in medicinal cigarettes in the United States,1 and mainland Europe until recently.2 Potter’s inhalation powder continued to be manufactured until 1988 when the UK’s Department of Health refused to renew the product licence.

Datura stramonium’s principle mechanism of action when the smoke is inhaled is anticholinergic bronchodilatation, with efficacy similar to salbutamol via metered dose inhaler,2 although the duration of action is shorter. Lobelia inflate was introduced as a medication in 1813 and is a plant alkaloid with mixed antagonism to nicotinic acetyl-choline receptors. Apart from its principle mechanism of bronchial vasodilatation, it has been used as a nicotine substitute, as it acts on central dopaminergic systems and for these reasons was often included in asthma cigarettes.1

While asthma and COPD are different diseases with differing clinical, pathological and immunological features, they share common processes of chronic airway remodelling and smooth muscle proliferation. In approximately 10% of patients, the specific features can overlap and are difficult to distinguish – especially in older people. The cornerstone for the development of COPD is mucosal irritation by smoke particles associated with cigarette smoke inhalation.1

DECLARATIONS

Competing interests
None declared

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with reactive oxygen species, inflammatory cytokines and proteolytic enzymes. It stands to reason that smoking asthma remedies, albeit for therapeutic purposes – would cause the same thermal injury and smoke particulate responses as other noxious inhalations. Even Herxheimer’s paper, extolling the acute beneficial effects of *D. Stramonium* inhalation on the airway, recalls the concerns of many authors that chronic airway irritation with these cigarettes could occur and others have highlighted the potential risks of contaminants, addiction, carcinogenic particles and chronic mucosal irritation. Asthmatics are more susceptible to oxidative stress from smoking damage than non-smokers and as such should avoid any form of smoke inhalation if possible.

We were unable to identify any studies of the chronic effects of either *Lobelia* or *D. Stramonium* smoke on the airways, nor longitudinal studies of the prevalence of chronic obstructive pulmonary diseases in patients who have used them. Presumably this is because they have generally been viewed (and advertised) as therapies not only for asthma but for COPD, and have not previously been thought of as a self-perpetuating cause. Active asthma has been associated with a 12-fold increased risk of developing COPD over 20 years follow-up. While chronic airway inflammation and remodelling might be causal, it is interesting to speculate that undocumented use of inhalational herbal therapy use might have contributed to this observation.

In summary, we present the case of a 79-year-old man who had never smoked tobacco, with severe emphysema, due to chronic inhalation of smoke from a herbal remedy for previously mild asthma. The probability of causation suggests that further epidemiological investigation into the currently unknown size of this problem is merited. A systematic approach to survey older asthma and COPD patients about their exposure may, therefore, be indicated in order to explore risk factors fully.

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