"OCCUPATIONAL ASTHMA" FROM THE USE OF COAL AS FUEL

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SYNOPSIS

The author, who is an occupational medicine physician at the time of occuprence, suffered two acute attacks of wheezing broncho-constriction, each occurring seven months apart, upon inhalation of combustion products of coal burning. He was aged 34 years then and has a history of syn-pharyngitic Glomerulo-Nephritis with Hypertension. He is a non-smoker and has no childhood asthma. There is no family history of asthma. He is non-atopic. Individual susceptibility, possible sensitization and acclimatization are discussed.

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INTRODUCTION

Coal burning has never been documented as a cause of occupational asthma(1-4). This paper deals with the inhalation of combustion products of coal burning or coal gas from incomplete combustion presumably and discusses on the possible agents arising therefrom, that may cause asthma.

Occupational asthma is difficult to define. A practical and accurate definition of occupational asthma is variable air flow obstruction caused by specific exposure in the work place(1). Whilst the author was not strictly working with coal at the material times, he was otherwise so engaged as to constitute working.

Coal is fossilized vegetable substances which, having been subjected to pressure and temperature over geological eras, have been converted to carbon rich compounds(3). China holds twelve per cent of the proven recoverable resources of bituminous coal of the world. Hence, this type of coal is commonly burned for fuel in China households.

Coal in general is a complex substance containing some hundreds of hydrocarbons. Bituminous coal contains up to seven per cent moisture, thirty per cent ash and a mixture of organic compounds of a predominantly aromatic character(3), for example benzene, toluene and aromatic amines like p-phenylenediamine. If coal gas is presumably produced as a result of incomplete combustion of coal, the composition of that gas varies considerably. Approximate composition includes nitrogen (and its oxides, particularly nitrogen di-oxide), saturated and unsaturated hydrocarbons and tarry vapours of cyclic hydrocarbons like benzene and toluene, polycyclic hydro-carbons like naphthalene, anthraces(4). In addition, there are components of ammonia, hydrogen sulphide and possibly varous cyanides like toluene diisocyanate and naphthalene diisocyanate. Taken as a whole, the various composite agents exert their effects on the uppoer respiratory tract and lung parenchyma by way of irritation, as well as physical and chemical asphyxiation.

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EPISODE ONE

The author lodged with his brother-in-law in Xiamen, China, from sixth to tenth September 1986. Whilst there, he had to enter the kitchen daily to get to the only source of tap water. The kitchen is also where the coal burner is situated .Coal is burnt as a cooking fuel. No coal gas is used as a fuel.

The kitchen is about two metres wide, four metres deep and three metres high. The door is one metres wide and two and one half metres high. The solitary window is one metre high and one and one half metres wide. General ventilation is inadequate. There is no local exhaust ventilation.

On the second day of arrival, the author put the kettle onto the coal burner. He stood and waited for the boil for about fifteen minutes, thereupon he was seized by sudden tightness of chest, cough and dizziness. As he left the kitchen and moved to the living room about ten metres away, severe wheezing and use of accessory muscles of respiration set in. Copious amounts of sputum were expectorated.

Having realized the hazardous effects of combustion products of coal burning and presumably also coal gas from incomplete combustion, on the respiratory system, he asked for bronchodiators and was given ephedrine tablets, which was on standby in the house, because his twelve year old nephew has a history of Bronchial asthma. The nephew is not a blood relative but is related by marriage.

Wheezing subsided after about an hour didn't recur the same day or any day of his remaining stay, for on subsequent entries to the kitchen, he flung the window wide open and kept the door ajar.

EPISODE TWO

This occurred on 13 April 1987 at the same household, again as a result of trying to boil water over the coal burner. Although the author knew the possibility of recurrenece of asthma, the window of the kitchen had to be kept shut because of torrential pain. Because of the resulting poor ventilation, the burning coal did not have adequate oxygen supply and began to smoulder and produced a haze of smoke.

Wheezing set in about five minutes after standing next to the burning coal. It was worse than in the first episade, there being no relief after an hour with taking of an Ephedrine tablet (麻黃). One puff of Isoprenaline inhaler provided relief from the wheezing after about twenty minutes.

The author is non-atopic based on a negative history of eczema and rhinitis.

DISCUSSION

Was it occupational asthma or was it not. What shone through were the causality and specificity of the boiling work exposure. The two episodes described illustrated the hazards of the combustion products of coal burning and presumably coal gas from incomplete combustion as well.

As stated in the introduction, combustion releases water vapours from its moisture content and incomplete combustion presumably releases coal gas. The latter consists of ammonia, benzene, toluene, naphthalene, anthracenes and varous cyanides.

Postulated as follows are the most likely asthmogenic agents in the circumstances. This is done by exclusion. Water vapour is not asthmogenic, neither is ammonia, which is a known upper respiratory tract excoriant and an asphyxiant. Nitrogen is also a physical asphyxiant, Its oxides, in particular, dioxide, is well known to be able to cause bronchiolitis fibrosa obliterans and then delayed pulmonary oedema, not asthma(5). Benzene toxicity, acute and chronic, is well documented. In particular, chronic toxcity can cause Diguigliamo's Erythroleukaemia(6). Acute toxicity, like narcosis, is common to all hydrocarbons but no asthmatic reaction to it has been recorded to date. In the acute stage, toluene behaves similarly to benzene. Chronic toluene toxicity causes a condition similar to Goodpasture's disease(7). Hydrogen sulphide also does not cause asthma. Neither will the naphthalenes, which have been documented to cause haemolytic anemia and hepatorenal vesical congestion(8). The anthracenes, especially benzanthracene, has been implicated as a cause of lung cancer, not asthma(9). Three candidates stand as likely asthmogenic agents. All three may have been released from incomplete combustion of the coal and they share a common characteristic, that is the ability to sensitize. Of the aromatic amines postulated to be present in the tarry vapour mentioned in the text. p-Phenylenediamine is a notorious sensitizing asthmogenic agent(10). While the first episode of wheezing may or may not have been due to individual susceptibility in a non atopic and non asthmatic individual (the author), the second episode of "worsened" asthmatic reaction was more than likely to be due to sensitization possibly by p-Phenylenediamine, toluene diisocyanate or naphthalene diisocyanate. Having narrowed down the postulated asthmogenic candidates to p-Phenylenediamine, toluene diisocyanate and naphthalene diisocyanate, one can quite easily dismiss ash as the culprit.

CONCLUSION

Ventilation of the kitchen appeared to be the single most important mitigating factor. Wheezing in a non atopic and non asthmatic individual points to the possibility of idiosyncrasy and sentization by the three postulated agents. Acclimatization of regular users of coal burners is evident in the fact that, other than the nephew who is asthmatic, none of the two others household members ever experienced asthmatic reactions.

The author's personal idiosyncratic or possibly sensitized response to inhalation of combustion products of coal burning and coal gas from presumed incomplete combustion, has been described as one of occupational asthma. Peak expiratory flow (PEF) before and after the two wheezing attacks were within normal and almost similar (Height 185 cm, Age 34 years. PEF: 660 L/Min. Predicted value is 650 L/Min.)

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