EDITORIAL

PREVENTION OF OCCUPATIONAL LUNG DISEASES

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SINGAPORE MED J 1996; Vol 37: 137-138

The diagnosis of an occupational lung disease is almost tantamount to an admission of failure in prevention. This is because in most situations, the factor(s) which cause an occupational lung disease are known and therefore worker exposure is preventable. But in real life, there continues to be occupational lung disease due to failure of control of known factors or the emergence of new occupational causes of lung diseases.

The profile of notifiable occupational lung diseases in Singapore is reviewed in a paper in this journal[1]. The list of notifiable occupational lung diseases in Singapore is limited to silicosis, asbestosis, mesothelioma, byssinosis and occupational asthma.

Silicosis was made legally notifiable under the Factories Act in 1970, while asbestosis, byssinosis and malignant mesothelioma were made notifiable in 1973 and occupational asthma in 1985. There have been no cases of byssinosis notified and as such in Singapore there are in practice only 3 types of notified lung diseases. Notification is an ongoing process occurring as and when cases are diagnosed. Its primary aim is to enable an investigation to be conducted to identify the problem so as to better control the occurrence of such diseases. The list of notifiable occupational lung diseases is very limited when considering the overall potential of such diseases. There is a need for a review to possibly enlarge the list of notifiable occupational lung diseases in order to identify and control them.

Silicosis was the most commonly notified occupational lung diseases in the 1970s with 240 cases on record and this number has dramatically fallen since, largely because of control measures which reduced the dust levels at workplaces to acceptable safe levels.

Similarly, asbestosis and malignant mesothelioma will decrease and disappear with better controls. For instance, since 1980, any factory using asbestos must inform the Ministry of Labour so as to ensure enforcement of control measures. Further, since October 1989, the import of all products containing crocidolite, amosite, amphiboles as well as building materials containing chrysotile have been banned by the Ministry of the Environment. From April 1995, all newly registered vehicles will be required to have asbestos-free brake and clutch linings.

With occupational asthma in Singapore, there has been an increasing trend of cases notified and today it is the most commonly notified occupational lung disease. This increasing trend is seen not only in Singapore but is a global phenomenon[2]. Further, studies show that asthma could persist in workers who have been removed from exposure to the incriminating agent[3]. For these reasons, it is appropriate that greater emphasis be focussed on the prevention of occupational asthma.

Prevention is traditionally classified as primary, secondary and tertiary. Tertiary prevention, which is largely the management of patients in order to prevent complications and deterioration, is not considered in this editorial. Primary prevention, to control exposure to agents which may cause occupational asthma, and secondary prevention for early detection of disease by clinical and biological means in order to abort or prevent the onset of occupational asthma are the foci of this editorial. This subject has been discussed in greater detail by Nordman[4] in a recent publication.

In primary prevention, the control of exposure to agents likely or known to cause asthma is of utmost importance. This could take the form of substitution of agents used or by a process of control of the work environment. A prerequisite for substitution is the need to disseminate information to industrialists to seek safer alternatives. For instance, isocyanates are still the most common cause of chemically induced asthma, while organic acid anhydrides are an increasing problem, so is also asthma caused by flour. Diisocyanates are still used in paints.

The use of enzymes, particularly alpha-amylase, has been implicated in the increasing trend of asthma induced by flour. The lesson from the use of enzymes in detergents and the problems has not been learnt.

Control of the work environment is an important area of primary prevention which requires the technical support and cooperation of medical, chemical, engineering and other experts.

Further, information of potential sensitisers should be available to the personnel responsible for ensuring the safety of the work environment. All too often, chemicals are only known by their trade names whereas the constituent chemicals should be identified so that users may become aware of potential sensitisers. There have been attempts at compiling a complete list of sensitisers in several countries[4] and such a list is useful for prevention purposes. Attempts have also been made to identify sensitisers according to their potency. Though information on exposure intensities needed for sensitisation is scarce and not reliable, it has helped in improving control measures. What is currently lacking are safe health limits based on the sensitising properties of various airborne allergens. But recently developed immunoassay measurements of protein allergens and a method directly measuring immunoglobulin binding of aeroallergens may make it possible to generate data for the setting of safe standards in the work environment.

The identification of susceptible persons is a much discussed issue in primary prevention. The consensus at present is that the identification of atopic individuals is not of much value in the prevention of occupational asthma. Further, it is a characteristic seen in about 30% of the working population making its identification of doubtful value for prevention purposes[4]. However, it is useful to identify persons with asthma or chronic bronchitis during pre-placement examinations as the condition of these subjects tend to deteriorate on exposure to non-specific irritants. Smoking appears to be a factor contributing to the development of specific IgE antibodies[5]. Though this has not

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been a confirmed observation, it is appropriate to advise workers against smoking, particularly in environments likely to have a risk of occupational asthma.

Early detection is the thrust of secondary prevention. This is particularly so as early detection followed by removal further improves prognosis. Early detection of susceptible individuals could be undertaken by questionnaires as well as routine skin prick testing of persons exposed to high molecular weight proteins. But it must be recognised that such early detection of susceptibles is only for more intense surveillance of these subjects and not any other action. The routine skin testing of persons exposed to low molecular weight chemicals is not indicated. The role and usefulness of bronchial hyperresponsiveness as a risk factor for occupational asthma is somewhat uncertain. The value of allergic rhinitis as a precursor to the development of occupational asthma is also not proven and requires further research.

These preventive measures together with improved diagnostic measures and epidemiological studies will be of value in the control of occupational asthma, the most important of occupational lung diseases today.

As stated at the beginning of this editorial, in effect in Singapore, only 3 occupational lung diseases are notifiable and notification is a primary pre-requisite in prevention. As such, it is appropriate to consider the widening of the scope of notifiable occupational lung diseases as a first stage in prevention.

References

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