Occupational Asthma in Singapore

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ABSTRACT

Aim: Since the first notified case of occupational asthma in 1983, a total of 90 cases were confirmed as on 31st December 1999. In this study, demographic data, causative agents and impact on the workers were described.

Methods: The data was derived from notifications to the Ministry of Manpower and referrals to the Occupational Lung Disease Clinic jointly run with Department of Respiratory Medicine, Tan Tock Seng Hospital.

Results: Of the 90 cases, 19 (21%) were females and 71 (79%) were males. There were 48 (53%) Chinese, 22 (24%) Malays, 14 (16%) Indians and 6 (7%) of other ethnic origins. The mean age at diagnosis was 35.8 ± 9.3 yrs. The mean duration of exposure prior to onset of symptoms was 34.9 ± 57.3 months. The most common causative agent was isocyanates (28 cases, 31%) followed by solder flux (12 cases, 13%) and welding fumes (8 cases, 9%) respectively. Thirteen (14.4%) workers were assessed to have permanent disability under the Workmen's Compensation Act.

Conclusions: Since 1990, occupational asthma has overtaken silicosis and asbestosis as the most common occupational lung disease in Singapore. The most common causative agent is isocyanates. Occupational asthma is a condition associated with disability in the workplace and may still be largely under-reported.

Keywords: occupational asthma, isocyanates

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INTRODUCTION

Worldwide, previous studies have shown that asthma affects 5-10% of the population and 3-7% of new onset adult asthma may be caused by the workplace[1,2]. Occupational asthma has become the most common form of occupational lung disease in industrialised nations, surpassing silicosis, asbestosis, and occupational lung cancer[3-7]. In Singapore, the last review was a decade ago with 35 cases reported between the period 1983 to 1990, during which it was postulated that occupational asthma may be largely under-reported[8].

With technological advances and change in industries, we decided to re-examine our cases of occupational asthma. The aim of our study was to describe the demographic data, causative agents and impact of occupational asthma on the workers in Singapore in the period between 1.1.1983 and 31.12.1999.

METHODS

Occupational asthma is defined as a disease characterised by variable airflow limitation and/or airway hyperresponsiveness due to causes and conditions attributable to a particular occupational environment and not to stimuli encountered outside the workplace. Two types of occupational asthma are distinguished by whether they appear after a latency period: immunological and nonimmunological[9].

The data was derived from notifications to the Ministry of Manpower and referrals to the Occupational Lung Disease Clinic jointly run with Department of Respiratory Medicine, Tan Tock Seng Hospital in the period between 1.1.1983 and 31.12.1999. A retrospective analysis of the data was then carried out. The cases were diagnosed from history taking, factory visits, peak expiratory flow rate (PEFR), nonspecific bronchoprovocation and/or specific inhalation challenge.

Occupational asthma was made a compensable occupational disease under the Workmen's Compensation Act in 1985. Criteria for evaluating permanent impairment were developed based on pulmonary function tests and the need for maintenance medication for asthma (use of bronchodilators, inhaled and/or oral steroids)[10].

Statistical analysis using SPSS V7.5 statistical package was used. Student's t-test was used to compare means if the data was normally distributed while Mann-Whitney U-test was used for non-parametric testing. Chi-square test was used to compare proportions. The ethnic composition in our study was compared with

that of the Singapore population[11] using chi-square analysis. p values of <0.05 were considered statistically significant.

RESULTS

Since the first notified case of occupational asthma in 1983, a total of 90 cases were confirmed as on 31st December 1999 (Fig. 1). Since 1990, occupational asthma has overtaken silicosis and asbestosis as the most common occupational lung disease in Singapore (Fig. 2).

Of the 90 cases of occupational asthma, 19 (21%) were females and 71 (79%) were males. There were 48 (53%) Chinese, 22 (24%) Malays, 14 (16%) Indians and 6 (7%) of other ethnic origins. As compared to the Singapore population[11], there were significantly more Malays and Indians. The mean age at diagnosis was 35.8 ± 9.3 yrs.

The most common causative agent was isocyanates (28 cases, 31%) followed by solder flux (12 cases, 13%) and welding fumes (8 cases, 9%) respectively (Table I).

The median duration of exposure prior to onset of symptoms was 12.0 months (range 1- 300 months) (Table I). The causative agent with the shortest median duration of exposure prior to onset of symptoms was isocyanates (2.5 months). Overall, the median duration of exposure prior to onset of symptoms was not statistically different between the patients with and without past history of asthma (9.5 versus 13.0 months, p = 0.216) (Fig. 3). The median duration of exposure was also not statistically different when the causative

Fig. 1 Number of occupational asthma cases in Singapore.

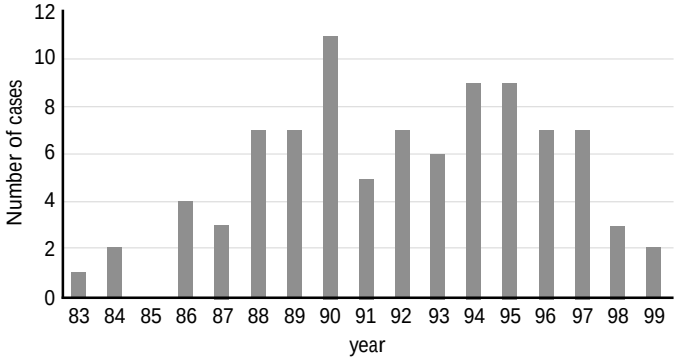


Fig. 2 Occupational lung diseases in Singapore, 1980-1999.

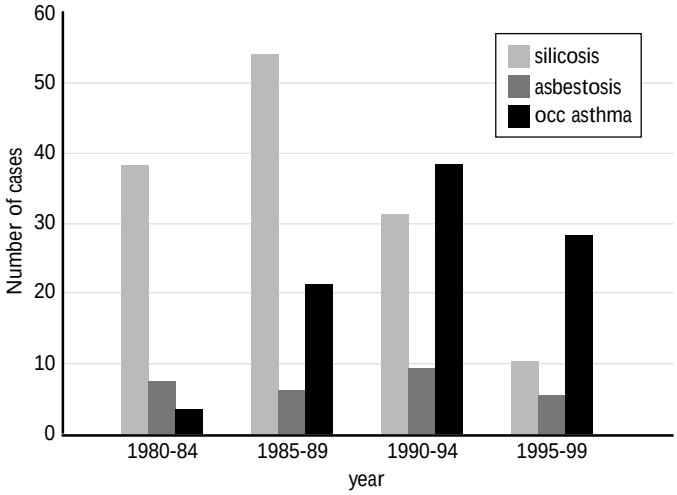


Table I. Causative agents in occupational asthma.

Causative agent	Median exposure (mths)	Range of exposure (mths)	Number of cases (%)
Isocyanates	2.5	1 – 264	28 (31.1)
Solder flux	12.0	1 – 60	12 (13.3)
Welding fumes	13.5	4 – 24	8 (8.9)
Wood dust	56.5	36 – 300	4 (4.4)
Solvents	36.0	24 – 56	3 (3.3)
Amines	3.0	1 – 12	3 (3.3)
Cement	21.0	12 – 30	2 (2.2)
Coolant mist	70.0	4 – 136	2 (2.2)
Ammonia	45.5	5 – 86	2 (2.2)
Anhydride	9.0	1 – 17	2 (2.2)
Flour	78.5	25 – 132	2 (2.2)
Smoke	78.5	4 – 153	2 (2.2)
Others*	15.5	1 – 191	20 (22.2)
Total	12.0	1 – 300	90 (100)

* ampicillin, ranitidine, heat, tylosine, PVC, ozone, cyanoacrylate, acid, synthetic rubber, barley, chlorophyll, sewage, dander, metal fumes, plastics, bird, acetaldehyde, dust, fragrances, formaldehyde.

Fig. 3 Duration of exposure prior to onset of symptoms in patients with and without past history of asthma.

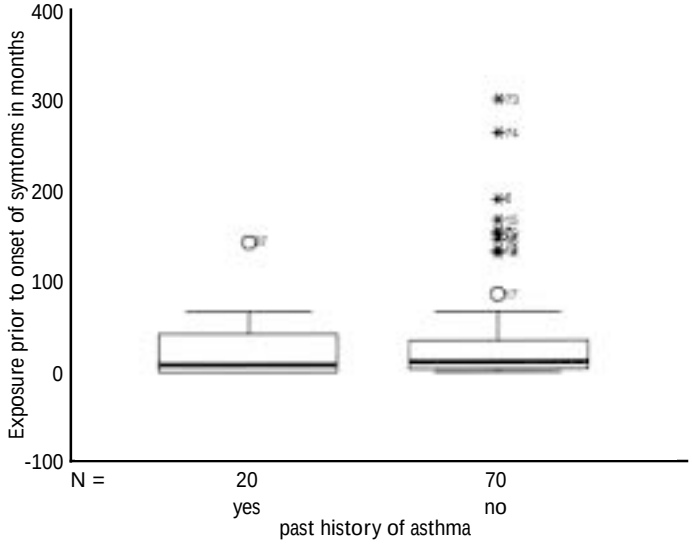
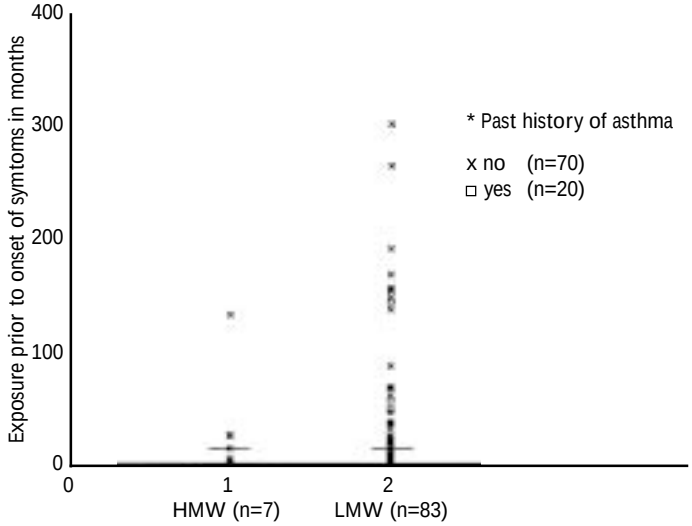


Fig. 4 Duration of exposure (months) prior to onset of symptoms according to HMW/LMW agents.



– Median exposure not statistically different between HMW (high molecular weight agents) and LMW (low molecular weight agents) (13.0 mths vs 12.0 mths) (p = 0.85).

* No statistically significant relationship between types of occupational asthma and past history of asthma.

agents were classified into HMW (high molecular weight) or LMW (low molecular weight) agents (13.0 versus 12.0 months, p = 0.85). When the causative agents were classified into nonimmunological and immunological categories, the median duration of exposure prior to onset of symptoms was also not statistically different (18.0 versus 12.0 months, p = 0.4). There was also no statistically significant relationship between the types of occupational asthma according to causative agents and past history of asthma (Fig. 4 and 5).

Thirty (33.3%) workers were assessed to have temporary disability while 13 (14.4%) workers were assessed to have permanent disability under the Workmen's Compensation Act.

It is known that factors which can increase the risk of occupational asthma include past history of asthma and history of smoking and hence we looked into the prevalence of these in our study. Twenty (22.2%) gave a past history of asthma. 8 (8.9%) of our patients were ex-smokers while 9 (10%) of our patients were current smokers.

A comparison between our current study period and that published earlier[8] showed that there was no statistical difference in gender, age distribution, mean duration of exposure prior to onset of symptoms and types of causative agents. There were, however, more Indians in the second study perod (p = 0.008) (Table II).

DISCUSSION

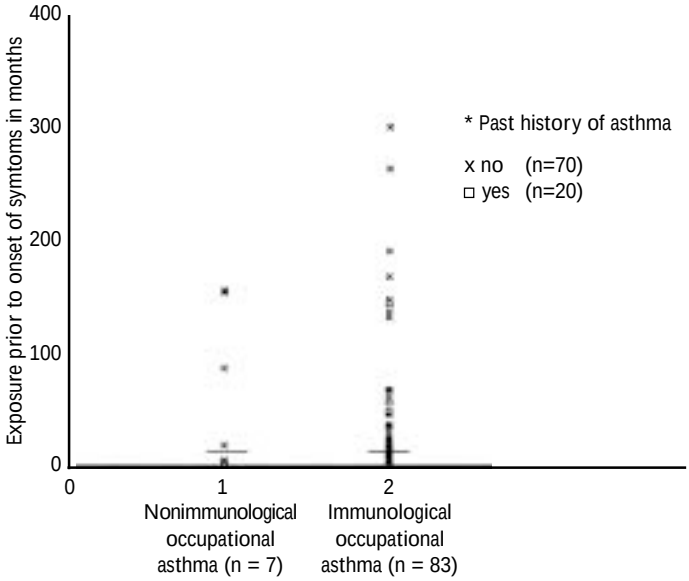
The first review of occupational asthma in Singapore was published in 1991 when it was described as one of the newer occupational diseases in Singapore[8]. As in other industralised nations[3-7], occupational asthma has now overtaken silicosis and asbestosis as the most common occupational lung disease in Singapore. This trend probably arose with the advancement in technology and change in industries.

With a greater number of cases reviewed as compared to the earlier one, this review is a better reflection of our cases of occupational asthma. Isocyanates still form the most common causative agent and present with the shortest median duration of exposure prior to onset of symptoms leading to earlier detection of occupational asthma. In our series, occupations at risk of exposure to isocyanates include polyurethane foam workers, painters, varnishers and insulators. Tarlo et al showed that occupational asthma due to isocyanates had shorter latent period before onset, shorter duration of symptoms before diagnosis and had a better outcome compared to occupational asthma induced by other work agents[12].

Exposure to diisocyanates is now recognised as a leading cause of occupational asthma. They are used worldwide in many important industries. For instance, they are used extensively in the automobile industry for production of foam rubber cushions, dashboards, body parts, and for finish coatings. Almost all mold and core processes in modern steel foundries require methylene diphenyldiisocyanate (MDI). They are also used as insulating materials in the building industry. Prepolymers of hexamethylene diisocyanate (HDI), MDI and their respective polyisocyanate oligomers are found in spray paint. Prepolymerized toluene diisocyanate (TDI) and

vapors of TDI are also used in spray lacquering[9]. A detailed past and present occupational history is hence paramount to the approach to someone with suspected occupational asthma. Clinical examination may reveal wheezing on chest auscultation if the worker has continuous symptoms. Investigations which should be performed include pulmonary function testing (pre- and post-bronchodilator) to document reversible airway obstruction and peak expiratory flow rate (PEFR) monitoring during working periods and off duties to show work-related airway obstruction. An increase in nonspecific airway hyperresponsiveness (NSBH) within 24 hours after a controlled laboratory challenge or a known workplace challenge may have diagnostic significance, especially if the increase in NSBH subsides in the ensuing weeks[9]. Although specific bronchial inhalation challenge is the "gold standard" for confirming a diagnosis of isocyanate asthma, such testing is not without risk to the patient and should only be performed in specialised centers with all safety measures recommended by international guidelines[1]. In addition, although challenges with isocyanates are highly specific if the test is positive, they may not be sensitive enough especially if they are performed with subthreshold concentrations.

Fig. 5 Duration of exposure (months) prior to onset of symptoms according to types of occupational asthma (nonimmunological/immunological).



– Median exposure not statistically different between nonimmunological and immunological types of occupational asthma (18.0 mths vs 12.0 mths) (p = 0.40).

* No statistically significant relationship between types of occupational asthma and past history of asthma.

Table II. Comparison between current study and previous study[8] in terms of gender, ethnic groups, age, mean exposure prior to onset of symptoms and causative agents.

	1.1.1983 – 31.12.1990[8]	1.1.1991 – 31.12.1999	P value
Gender (Males/Females)	28/7	43/12	0.837
Chinese	22	26	0.149
Malays	10	12	0.467
Indians	1	13	0.008
Other ethnic groups	2	4	1.000
Age (mean ± s.d.)	33.8 ± 10.1	37 ± 8.7	0.106
Mean duration of exposure prior to onset of symptoms	36.7 ± 54.1	33.8 ± 59.7	0.814
Isocyanates	13	15	0.324
Solder flux (no. of cases)	3	9	0.289
Welding fumes (no. of cases)	3	5	1.000
Wood dust (no. of cases)	1	3	1.000
Solvents (no. of cases)	1	2	1.000
Amines (no. of cases)	1	2	1.000
Cement (no. of cases)	1	1	1.000
Coolant mist (no. of cases)	1	1	1.000
Ammonia (no. of cases)	1	1	1.000
Anhydride (no. of cases)	2	0	0.149
Flour (no. of cases)	1	1	1.000
Smoke (no. of cases)	0	2	0.519
Others (no. of cases)	7	13	0.686

Hence, a negative specific inhalation challenge does not necessarily rule out the diagnosis of occupational asthma. The American Conference of Governmental Industrial Hygienists[13] has set the threshold limit value (TLV) of toluene diisocyanate (TDI) and methylene diphenyldiisocyanate (MDI) as 0.0005 ppm. Exposure of workers should be kept within these limits through engineering control measures (such as enclosures and local exhaust ventilation) and the use of appropriate respirators. Removal of affected workers from the exposed agent is the mainstay of treatment. $Beta_2$ agonists may be used to prevent or reverse asthmatic reactions but more severe asthma induced by diisocyanates should be treated like any asthma occurrence, with inhaled $beta_2$ agonists and high-dose steroids. A regular surveillance program conducted in industries such as the isocyanate surveillance program in Ontario[12] may be beneficial in earlier diagnosis and prevention of occupational asthma.

Overall, the current data correlated well with that of the previous study published locally[8]. However, compared to studies overseas[1,2,14], our incidence of occupational asthma appears lower. This could either reflect the fact that occupational asthma remains largely under-recognised and under-reported, or the incidence is indeed lower as compared to that in other countries. Factors associated with a worse outcome in occupational asthma include long exposure to agents before asthma onset as well as continuing exposure[15]. With its medico-legal implications and its effect on health and economy, a greater awareness by the workers as well as physicians-in charge will be paramount to the management of occupational asthma. Preventive measures by the employers should also be reinforced.

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