CME Article

Lessons from an isocyanate tragedy

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ABSTRACT

This paper highlights the features of prevention of isocyanate-induced occupational asthma from various angles: the employer, the employee, as well as the occupational physician entrusted with the care of such occupationally-exposed group of workers. The difficulties posed by the variability of the presentation, not least because of the relatively long latency, as well as the sensitising nature of the hazard, are discussed.

Keywords: exposure monitoring, health surveillance, isocyanate, occupational asthma

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INTRODUCTION

"A 52-year-old manager of a furniture factory died of acute asthmatic attack when he was exposed to diisocyanate vapour at work. The vapour was emitted during the spray painting process carried outside the factory by his wife. The deceased had a previous history of acute asthmatic attack triggered by exposure to diisocyanate from spray painting." – Ministry of Manpower Singapore, OSH Alert September 2006.

The prevalence of occupational asthma among isocyanate-exposed workers has been variously reported to be between 5%–10% and 20%–30%.[1,2] Diisocyanate exposure has also been reported to be the most common cause of work-related new onset asthma.[3] Isocyanates are ubiquitous chemicals found in a wide range of industrial activities, including foam and paint manufacturing. Spray painters have often been singled out as being especially vulnerable to its harmful effects, largely because of their excessive exposure.[4,5] However, as this fatality clearly illustrates, even bystanders can be adversely affected.

Three out of every ten cases of fatal occupational asthma have been attributed to isocyanate exposure.[6] Deaths in an occupational setting, such as those seen in the isocyanate cases, do not occur *in vacuo*. Asthmatic attacks at the workplace is the pivotal event in the chain of events against which antecedent controls ought to have been in place, both as measures for its prevention, or failing which, once the asthma has been triggered, as measures for its mitigation.

CONTROLS

Every employer has a duty to provide safe working conditions for its employees. At its most fundamental, issues relating to the substitution of the hazard must be seriously considered.[7] In the case of isocyanates with its known possible harm, current technologies do not seem to offer any viable alternative. Employers must therefore be able to manage the risks accordingly.

Hazard and risk communication/training

For health hazards at the workplace, e.g. exposure to isocyanate, it is the responsibility of the employer to proactively educate its workers on the risks as well as to incorporate appropriate safety measures, instead of expecting the workers to read through the Material Safety Data Sheet for such information. Additionally, in any large group of workers, there will always be some who are more susceptible to isocyanate exposure, which has a variable period of latency of between ten months and 15 years after the initial exposure. [6]

Workplace risk reduction measures

Exposure monitoring and control traditionally hold the keys to prevention. In the case of isocyanates, this may be very difficult because of the very low concentration required to provoke bronchospasm once sensitisation has occurred. The most effective method for reducing workplace exposure then lies in the primary prevention of sensitisation in as many workers as possible. Is there a threshold level for sensitisation? Animal studies seem to suggest that there could be such a level, and observational studies seem to indicate an exposure level to range from ≤ 5 ppb to 5–10 ppb.[8]

Workplace monitoring, in order to be valid, has to be comprehensive. In one study of isocyanate exposure among auto repair workers, 380 samples were obtained from the various workstations in various locations, under different control situations. The polyisocyanate levels were found to range from 0.02 μg/m^3 in workplace background locations, to 108.1 μg/m^3 at near spray locations, to 3,119.6 μg/m^3 at spray locations.[4] The UK Health and Safety Executive's Occupational Exposure Limit (OEL) levels for the total reactive isocyanate group is 70 μg/m^3 for a ten-minute exposure, and 20 μg/m^3 for time-weighted exposure to any isocyanate.[9]

One shortcoming of isocyanate exposure monitoring at the workplace, like in all such endeavours, is that erroneously low readings may engender a misplaced sense of complacency. Workplace monitoring, if it is to

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be useful, has to be conducted with a forward plan for mitigation, including the use of spray booths, relocation of high-risk tasks away from other non-involved workers (e.g. administrative staff), strict adherence to the use of personal protective equipment, and an awareness that asthma can still occur despite preventive measures.

Emergency Response Measures

Worksite response to medical emergencies is also within the responsibility of the employers. This is particularly critical for businesses sited at remote locations, where help may not be expeditiously available.

HEALTH SURVEILLANCE[10]

Although a clinical history, such as that elicited by a questionnaire, is useful, it is inadequate on its own, and may suffer the risk of inaccuracy through concealment, and can potentially result in the underestimation of its severity. A history of atopy is particularly relevant for high molecular weight allergens, but less so for those with low molecular weight, like isocyanates. Atopy, being fairly prevalent in any given population, if considered as an exclusion criteria, would result in the wrongful exclusion of a significant number of workers who would have otherwise been safe for such jobs.[11] Demonstration of specific immunoglobin E (IgE) as an indicator of occupational asthma has high specificity, but low sensitivity.[10] Intuitively, a history of asthma would raise red flags, especially if it is current or recent, and may confuse the issue of causation. A previous history of asthma is, however, not significantly associated with occupational asthma.[12]

Serial Peak Expiratory Flow (PEF) measurement during working hours, immediately postshift, and off work hours (such as during vacation), is probably the most economical and practical measure to be undertaken in a medical surveillance programme. For large employers, this could be undertaken by an appointed health worker, with referrals to a physician for readings suspicious of work-related asthma.[7] Care has to be taken with the interpretation, particularly with the timing, magnitude of flow changes, exacerbation or abatement of symptoms, and the correlation to activities and exposure. The ideal, yielding highest sensitivity and specificity of test results, is PEF measured four times a day.[10]

Inhalation challenge testing

Specific bronchial provocation challenge is considered the gold standard for diagnosing occupational asthma from isocyanates. It may be mandatory in some countries as part of the investigation for statutory compensation. However, these tests are not without risks and need to be conducted in specialised centres.[13]

Frequency

The earliest recorded death after the appearance of signs and symptoms of occupational asthma is ten months.[6] This suggests that if workers showing the typical signs and symptoms are detected early enough and removed, irreversible damage can be prevented. Thus medical surveillance ought to start soon after commencement of work in an isocyanate environment, and be followed-up at regular intervals to detect early deterioration and to allow for remedial action, preferably removal and treatment. In view of the long latency period, occupational asthma surveillance must continue indefinitely, or for at least as long as there is exposure. In practice, loss of follow-up due to job changes is inevitable. Such job changes may contribute to the healthy worker bias, because those who had left may have done so voluntarily, precisely because of respiratory damage.

Early reporting by employees

Apart from routine medical surveillance, workers must be encouraged to report early for symptoms of breathlessness, chest tightness and discomfort. Closely related to this factor is the organisational climate and culture. A suspicious and autocratic management, wholly production- and goal-driven, is less likely to encourage dialogue and communication with workers over health problems. Needless to say, this can result in dire consequences.

Removal of the affected employee

Workers confirmed to have occupational asthma should be advised to avoid further exposure completely. Once sensitisation has occurred, the practical value of reducing exposure diminishes, as far as the susceptible individual is concerned. This often necessitates a job change, or if that is not possible, a severance from the employers. Early removal can potentially avert premature death, or at least irreversible lung damage.[14]

NOTIFICATION, COMPENSATION AND LIABILITY

Occupational asthma is a notifiable occupational disease. A statutory duty to notify arises under the Workplace Safety and Health Act. Although there is no need to seek approval from either the employees or the employers before notifying, it is good practice to have discussions with these relevant parties. Confirmation of occupational asthma entitles the worker to workman compensation, under a no-fault scheme, subject to disability assessment. If a worker is dissatisfied with, or fails to obtain workman compensation, he can take legal action and sue the company for negligence.[15] If a death has occurred, his

estate is entitled to remedy. It is also good practice to notify suspected cases where an occupational aetiology cannot be reasonably excluded. Novel substances suspected to be the cause can then also be investigated and confirmed by the relevant authorities.

CONCLUSION

Health incidents at the workplace deserve close scrutiny and investigation. There should be scope for early intervention and prevention. We should not be witness to another preventable death from occupational asthma due to exposure to isocyanates.

REFERENCES

1. Butcher BT, Jones RN, O'Neil CE, et al. Longitudinal study of workers employed in the manufacture of toluene diisocyanate. Am Rev Respir Dis 1977; 116:411-22.
2. Vandenplas O, Malo JL, Saetta M, Mapp CE, Fabbri LM. Occupational asthma and extrinsic alveolitis due to isocyanates: current status and perspectives. Am J Ind Med 1993; 50:213-28.
3. Goe SK, Henneberger PK, Reilly MJ, et al. A descriptive study of work aggravated asthma. Occup Environ Med 2004; 61:512-7.
4. Woskie SR, Sparer J, Gore ERJ, et al. Determinants of isocyanate exposures in auto body repair and refinishing shops. Ann Occup Hyg 2004; 48:393-403.
5. Talini D, Monteverdi A, Benvenuti A, et al. Asthma-like symptoms, atopy, and bronchial responsiveness in furniture workers. Occup Environ Med 1998; 55:786-91.
6. Ortega HG, Kreiss K, Schill DP, Weissman DN. Fatal asthma from powdering shark cartilage and review of fatal occupational asthma literature. Am J Ind Med 2002; 42:50-4.
7. Gannon PFG, Berg AS, Gayosso R, et al. Occupational asthma prevention and management in industry—an example of a global programme Occup Med (Lond) 2005; 55:600-5.
8. Baur X, Chen Z, Liebers V. Exposure response relationships of occupational inhalative allergens. Clin Exp Allergy 1998; 28:537-44.
9. Silk SJ, Hardy HL. Control limits for isocynates. Ann Occup Hyg 1983; 27:333-9
10. Nicholson PJ, Cullinan P, Taylor AJ, Burge PS, Boyle C. Evidence based guidelines for the prevention, identification, and management of occupational asthma. Occup Environ Med 2005; 62:290-9.
11. Gautrin D, Infante-Rivard C, Ghezzo H, Malo JL. Incidence and host determinants of probable occupational asthma in apprentices exposed to laboratory animals. Am J Respir Crit Care Med 2001; 163:899-904.
12. Mapp CE, Boschetto P, Dal Vecchio L, Maestrelli P, Fabbri LM. Occupational asthma due to isocyanates. Eur Respir J 1988; 1:273-9.
13. Banks DE, Tarlo SM, Masri F, Rando RJ, Weissman DN. Bronchoprovocation tests in the diagnosis of isocyanate-induced asthma. Chest 1996; 109:1370-9.
14. Tarlo SM, Liss G, Corey P, Broder I. A workers' compensation claim population for occupational asthma. Comparison of subgroups. Chest 1995; 107:634-41.
15. England and Wales Court of Appeal (Civil Division) Decisions. Parsons v Warren & Anor. In: British and Irish Legal Information Institute [online]. Available at: www.bailii.org/ew/cases/EWCA/Civ/2002/130.html. Accessed May 1, 2007.

SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME

Multiple Choice Questions (Code SMJ 200805A)

	True	False
Question 1. All workers suspected to have occupational asthma must have their diagnosis confirmed by:		
(a) Clinical history.	☐	☐
(b) History of work exposure.	☐	☐
(c) Serial peak expiratory flow.	☐	☐
(d) Specific bronchial provocation challenge.	☐	☐
Question 2. Workplace monitoring for occupational asthmatogens, like isocyanates, can achieve the following:		
(a) Identification of the effectiveness of control measures, such as ventilation controls, to reduce exposure.	☐	☐
(b) Improvement in the design and layout of the factory.	☐	☐
(c) Assessment of which activities carry the heaviest exposure.	☐	☐
(d) Establishment of safe exposure levels for workers to prevent occupational asthma.	☐	☐
Question 3. A worker confirmed to have occupational asthma should be:		
(a) Removed from the exposure.	☐	☐
(b) Observed to see if his/her asthma worsens.	☐	☐
(c) Subject to bronchial provocation challenge for legal determination of the cause.	☐	☐
(d) Investigated for atopy in order to guide treatment.	☐	☐
Question 4. A doctor treating a case of occupational asthma should:		
(a) Notify the Ministry of Manpower (MOM) under the Workplace Safety and Health Act.	☐	☐
(b) Seek permission of the patient before deciding to notify MOM.	☐	☐
(c) Seek permission of the workplace management before deciding to notify MOM.	☐	☐
(d) Never notify suspected cases.	☐	☐
Question 5. All workers have:		
(a) A right to be informed of the potential hazards that exist in the workplace.	☐	☐
(b) A right of access to the Material Safety Data Sheets of the chemicals used in the workplace.	☐	☐
(c) A right to workplace monitoring data information, to be provided by the employers.	☐	☐
(d) No rights as long as the employer pays him well.	☐	☐

Doctor's particulars:

Name in full: _____

MCR number: _____ Specialty: _____

Email address: _____

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(1)Answers will be published in the SMJ July 2008 issue. (2) The MCR numbers of successful candidates will be posted online at www.sma.org.sg/cme/smj by 15 July 2008. (3) All online submissions will receive an automatic email acknowledgment. (4) Passing mark is 60%. No mark will be deducted for incorrect answers. (5) The SMJ editorial office will submit the list of successful candidates to the Singapore Medical Council.

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