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Occupational asthma among hospital health care personnel: a cause for concern?

In recent years there has been increasing recognition of the importance of exposure to agents in industry as a cause of asthma. Occupational asthma has now emerged as the most prevalent occupational lung disease in many western countries. There have been over 250 compounds reported as possible causes of occupational asthma, and with the ever increasing number of new substances being introduced this number is likely to increase.[1] Although concerns have recently been expressed about the effects of exposure to chemicals in some areas of the hospital environment,[2,3] there is little information on the overall prevalence of occupational asthma among different groups of health care workers. There has, however, been an increase in the number of individual case reports of health care workers developing asthma as a result of substances encountered in the work place. These include laboratory technicians, nurses, doctors, and, most recently, radiographers. With the implementation of more rigorous health and safety legislation in many industrialised countries,[4] it is likely that the number of health care professionals seeking advice from medical colleagues about respiratory symptoms in the hospital work setting will increase.

Asthma in health care workers

Health care workers may be exposed to a wide range of substances, some of which have already been clearly implicated as causative agents of occupational asthma in industry. The first case of occupational asthma in a health care worker was probably caused by exposure to ipecacuanha powder in a hospital worker of the 19th century.[5] However, it is only in recent years that the problem has been given some limited recognition as reflected by attempts to determine prevalence data in a number of at risk health care groups.

A recent study of respiratory therapists in the United States reported that the overall prevalence of asthma in this group of professionals was greater (18·7%) than that of a control group derived from other health care groups (5·8%; odds ratio 3·2).[6] The authors were unable to explain this difference on the basis of selection or information bias, nor were they able to implicate any single putative factor such as glutaraldehyde or contaminants of bronchodilator solutions (such as sulphites) to which they had been exposed. In a study which included measuring immunological status, airway responsiveness, and specific bronchial challenges, Malo *et al* reported a prevalence of 4% for occupational asthma to psyllium (ipsaghula) in 193 of 248 nurses from four institutions.[7] Occupational exposure to glutaraldehyde among hospital staff has been a cause of recent concern. Exposure may be associated with nasal symptoms, eye symptoms, and skin rashes. Respiratory symptoms have been reported in personnel at levels of exposure less than those recommended.[8] Although there have been individual reports of occupational asthma, symptoms are generally considered to result from a direct irritant effect of the biocide rather than an acquired hypersensitivity.[9] No data on changes in airway function on exposure were given in these cross sectional studies. Although allergy to latex has been known for some time, it has only recently been shown to be a cause of asthma among health care workers. Sensitisation, as shown by a positive skin test, has been reported in 10% of theatre nurses.[10] Recently 29 health care workers from several disciplines have been reported with asthmatic symptoms and positive skin tests to latex.[11] No data are offered on changes in airway calibre associated with exposure to latex, but it is noteworthy that a number of patients had a previous history of asthma.

Further information is clearly required on the prevalence of asthma in these and other categories of potentially at risk health workers such as those working in renal dialysis units, operating theatres, and diagnostic imaging departments who are regularly exposed to a range of biocides. Similarly, other groups exposed to protein compounds, pharmaceutical agents, volatile gases, and glues may require investigation. Compounds reported to have caused occupational asthma in the health care environment are listed in the table, which also outlines the considerable range of hospital departments where hazards may be encountered.

High molecular weight chemicals

Allergens of high molecular weight (greater than 5000 daltons) are usually proteins of animal, plant, bacterial, or fungal origin. The diagnosis of occupational asthma resulting from this group of compounds is often considered straightforward. There is often, therefore, a clear relation between chest symptoms and occupational exposure. In many cases patients complain of allergic symptoms affecting the eyes, nose, or skin. In virtually all the reported cases of occupational asthma in health care workers related to high molecular weight compounds the onset of asthma symptoms was preceded or accompanied by other allergic symptoms such as rhinitis, conjunctivitis, or even contact dermatitis and urticaria. Although it is generally accepted that atopic patients are at greater risk of developing asthmatic symptoms following exposure to these compounds, there has been one report of four non-atopic laboratory technicians developing specific IgE antibodies and asthmatic symptoms following exposure to the proteolytic enzyme bromelain while working in a blood bank.[12] It is also generally considered that sensitisation and the development

Substances described as causing occupational asthma among health care personnel

Substance	*Occupation*	*Department*
High molecular weight		
Animal proteins[35]	Laboratory workers	Research laboratory
Psyllium (ipsaghula)[13]	Nurse	Geriatrics
Pancreatic extracts[14]	Nurse	Paediatrics
Bromelain[12]	Laboratory technician	Blood bank
Bovine serum albumin[36]	Laboratory technician	Pathology
Latex[15]	Nurse	Surgery
"Allergen exposure"[37]	Technician	Pulmonary medicine
Corn starch (rubber glove)[38]	Obstetrician	Obstetrics
Low molecular weight		
Formaldehyde[16]	Nurse	Renal dialysis unit
Glutaraldehyde[24,27]	Nurse, radiographer	Endoscopy, radiology
Hexachlorophene[17]	Nurse	Paediatrics
Chloramine[18]	Laboratory technician	Pathology
Methyl methacrylate[20]	Theatre nurse	Orthopaedics
Methyl blue[19,39]	Nurse, ECG technician	Cardiology
Enflurane[23]	Anaesthetist	Anaesthésia
Isonicotinic acid hydrazide[26]	Hospital pharmacist	Pharmacy
Chlorhexidine[21]	Auxiliary nurse, midwife	Obstetrics
Sulphathiazoles[40]	Nurse	General surgery
Ethylene oxide[25]	Nurse	Renal dialysis unit
Terpene (rubber glove)[22]	Laboratory technician	Pathology
Radiograph fixative/ developer[27,28]	Radiographer	Diagnostic imaging

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of symptoms following exposure to such compounds occurs within the first or second year. However, in five nurses with psyllium (ipsaghula) induced asthma,[13] the latent or asymptomatic period of exposure was approximately 10 years. Similar prolonged latent periods have been reported in other cases of asthma resulting from exposure to high molecular weight compounds, including reports of occupational asthma in nurses caused by pancrex powder[14] and latex rubber.[15] In all cases sensitisation was confirmed by specific immunological tests such as a radioallergosorbent or skin prick test for the presence of specific IgE antibodies.

Low molecular weight chemicals

The diagnosis of occupational asthma resulting from exposure to low molecular weight chemicals is invariably more difficult. While differing diagnostic criteria have been used in reports of such chemicals inducing asthma in health care workers, virtually all showed confirmatory objective evidence of changes in airway calibre following exposure to the sensitising agent. The demonstration of late asthmatic responses following specific inhalation tests remains the gold standard for diagnosing asthma caused by exposure to low molecular weight chemicals, and these have been reported with formaldehyde,[16] hexachlorophene,[17] chloramine,[18] methyl blue,[19] and methyl methacrylate.[20] By contrast, early asthmatic responses have been reported in two patients with "normal" airway reactivity challenged with chlorhexidene,[21] and in a laboratory technician challenged with rubber gloves in which a terpene was implicated as the causative agent.[22] Changes in self-recorded air flow measurements at work following exposure to enflurane in an anaesthetist,[23] and glutaraldehyde in an endoscopy nurse,[24] formed the basis for the diagnosis of occupational asthma in these two case reports. An increase in airway reactivity occurred in a nurse with work related asthmatic symptoms after challenge with ethylene oxide.[25] In only one report – that of asthma due to isonicotinic acid[26] – was the presence of a specific IgE response to a protein conjugate to the sensitising agent shown. Recently both glutaraldehyde (used as a hardening agent) and fixative solution (containing ammonium thiosulphate) have been implicated as causes of asthma among radiographers.[27,28]

Diagnosing occupational asthma

In all cases of occupational asthma a careful history is a prerequisite for accurate diagnosis. The history must provide complete details of all symptoms and work activities. The duration, location, and pattern of symptoms must be related to type, nature, quantity, and intensity of exposure to suspected agents. As outlined above, many patients with occupational asthma resulting from exposure to high molecular weight chemicals develop symptoms of allergic rhinitis, conjunctivitis, urticaria, or contact dermatitis before developing asthmatic symptoms. Asthmatic symptoms may develop immediately or soon after exposure to the sensitising agent, and the relation between exposure and symptoms may be clear. Sensitisation to high molecular weight chemicals can be confirmed by skin prick test or a radioallergosorbent test. The presence of work related asthma of recent onset, with confirmation of sensitisation to the suspected allergen, is considered sufficient by many physicians to make a diagnosis of occupational asthma caused by high molecular weight chemicals.

The diagnosis of asthma as a result of exposure to low molecular weight chemicals is often less clear. Many of the low molecular weight chemicals (in particular the biocides) irritate mucosal surfaces and thus symptoms may be dismissed by the health care worker as resulting from this irritant effect. Similarly, an association between symptoms and exposure to a sensitising agent may not be apparent because asthma caused by low molecular weight chemicals may induce atypical non-specific symptoms such as cough or chest discomfort. More classic symptoms such as wheeze and chest tightness may not occur until late in the evening or during the night after exposure. Hospital staff within many departments may work a very varied shift and the type of professional activity may also vary from day to day. In endoscopy units and operating theatres, therefore, levels of exposure to compounds might vary from individual to individual and from day to day. Similarly, many hospital staff work on periodic night or weekend shifts, often followed by variable periods away from work and away from further exposure to a sensitising agent. Such common variations in work practice in the hospital milieu will sometimes result in intermittent and unpredictable exposure to the sensitising agent. As a consequence, any relation between symptoms and work may be difficult to establish, thus delaying diagnosis.

Although clinical diagnosis of occupational asthma caused by exposure to a low molecular weight chemical is quite sensitive, it is not very specific[29] and objective measurements of asthma and any relation between symptoms and work should be made. Two-hourly self-recorded peak flow measurements for two weeks at work and two weeks away from work have been found to be a useful method of assessing work related respiratory symptoms.[30] Care must be taken to ensure that the time of starting and finishing work, and the type of work and substances to which the health care worker has been exposed, must be included in order to interpret the significance of the airflow indices being assessed. Measurements of non-specific airway reactivity to histamine or methacholine following a period at work, and after a period away from work, may also give valuable additional information. Specific bronchial provocation tests may be required in some cases where the diagnosis remains uncertain, or where the worker has been exposed to a number of potential sensitising agents, or when further uncontrolled exposure is considered dangerous.

Treatment

The only effective treatment for occupational asthma is avoidance of further exposure to the sensitising agent. This usually requires relocation and retraining. Unfortunately, in industry experience has shown that workers find themselves significantly worse off after been diagnosed as having occupational asthma.[31] In the health care setting most individuals have undertaken several years of specialised professional training and relocation or retraining of these personnel would be costly, both for the worker and the health authority. This further emphasises the need for care and precision in making the diagnosis.

Prevention strategies

The justification for the continued use of sensitising substances in the hospital environment needs to evaluated. Thus, simple substitution of psyllium (ipsaghula) by other equally effective laxatives would solve any further potential problems with this substance. In cases such as pancreatic enzyme-induced asthma the increased usage of granules or capsules instead of powder has already substantially reduced any potential risk to nursing staff. Exposure to latex allergens is greatest in areas of multiple glove changes,[32] and increased use of plastic and non-powdered rubber gloves would result in reduced exposure levels. However, simple replacement strategies may not be practicable with other compounds such as glutaraldehyde

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used as a sterilising agent in endoscopy units and as a hardening agent in radiograph developer material in radiology departments. It is likely, too, that the chemical property which makes glutaraldehyde a useful agent may also be precisely that quality which causes it to act as an irritant and potential sensitising agent. Simple replacement of one agent with another may therefore only substitute one sensitising or irritant agent for another. This is clearly suggested by the number of biocidal agents which have been described as causing occupational asthma. It would seem sensible, therefore, to reduce exposure to as low a level as possible if substitution is not feasible. Such reduction in exposure may require changes in work practice and improvement in facilities, particularly in areas such as isolation, ventilation, and waste disposal. Most importantly, personnel require education and supervision in the proper handling of substances.[33] While monitoring atmospheric levels of sensitising agents may provide useful information on overall levels of exposure, it is often difficult to relate to individual exposures and individual symptom profiles.

Ultimately, early detection and removal from further exposure may be the most useful method of avoiding the development of symptoms of chronic asthma in patients with occupational asthma.[34] Although there is no agreement on the best method of detecting occupational asthma in a work place, surveillance by questionnaire with subsequent removal from the work place during the course of further investigations should be considered. Finally, it is important to emphasise that it is likely that some compounds in long established use may go undetected as a cause of asthma; for example, glutaraldehyde and latex rubber were used for many years before hypersensitivity responses were reported. Greater access to information about the proper handling and use of substances may minimise the risk of sensitisation and development of symptoms.

Conclusion

In the course of their professional activities in hospital doctors, nurses, and other paramedical personnel may be exposed to many high and low molecular weight compounds capable of inducing occupational asthma. A rising number of individual case reports have documented hospital acquired occupational asthma in endoscopy suites, operating theatres, laboratories, and diagnostic imaging departments. Some prevalence studies suggest appreciable degrees of risk in groups such as respiratory therapists and those exposed to ipsaghula (psyllium), glutaraldehyde, and latex. Although there is increasing awareness of this problem among health care professionals, there are few well conducted controlled studies of potentially at risk groups such as endoscopy nurses or radiographers. If the experience of quantifying occupational asthma in the industrial environment is replicated in the hospital setting, it is likely that carefully conducted studies will reveal a greater prevalence of work related asthma among health professionals than is currently realised.

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