DEPARTMENT OF HEALTH & HUMAN SERVICES

National Institute for
Occupational Safety and Health (NIOSH)

Memorandum

Date: August 26, 2013

From: Research Epidemiologist, FSB, DRDS
Thru: Chief, FSB
Team Leader, FSB

Subject: Reprint of Publication

To: Deputy Director, DRDS

Attached is a reprint of a book chapter I co-authored that recently appeared in In: Malo J-L, Chan-Yeung M, Bernstein D, editors. Asthma in the Workplace, 4th Edition. The DRDS clearance number is 2013-002B. It is provided for your retention.

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Thank you,

Paul Henneberger

Paul Henneberger, MPH SCD

Attachment:

cc:
Director, DRDS
Director, EID
Chief, FSB, DRDS
Chief, SB, DRDS

10 Management of the worker

André Cartier, Paul K. Henneberger, and Stuart M. Brooks

CASE HISTORY

A 25-year-old worker has been employed for 5 years in a small family-owned bakery that employs three other bakers and two helpers.

1. *During his apprenticeship course, the worker received information on possible allergies to cereals, enzymes, and other products in flour.*
2. *He started sneezing at work 1 year before being seen at the clinic, and his wife noticed that he had some wheezy breathing at home in the evening for the past few months*
3. *The local health department had started a surveillance program in bakeries in the area. After some hesitation, the employer allowed an industrial hygienist from that department to visit the workplace and a nurse to meet with the workers.*
4. *An informative session was offered and short self-administered questionnaires were completed by all attending workers.*
5. *Because the worker had positive responses on the self-administered questionnaires, he met with the occupational nurse, who inquired about the details of his symptoms with more detailed medical questionnaires. The results of these questionnaires indicated that the worker had symptoms consistent with work-related rhinitis and asthma.*
6. *The worker was seen by the occupational physician of the local health department. Skin prick tests to various cereals and enzymes showed positive immediate skin reactions to wheat and soya flours allergens. He was given inhaled salbutamol on demand.*
7. *The nurse and physician suggested that the worker make an appointment at a specialized center for further testing.*
8. *The worker, a recent immigrant with two children, feared losing his job but followed the advice and scheduled an appointment.*
9. *Monitoring of peak expiratory flows (PEFs) and nonspecific bronchial responsiveness (NSBR), at work and off work, suggested the diagnosis of OA to flour.*
10. *A claim was filed to the Workers' Compensation Board and the worker was removed from work.*
11. *Following the diagnosis of OA, he was given the opportunity to follow a course on truck driving and was able to find a new job.*
12. *Two years later, the worker was asymptomatic requiring no treatment for asthma. He was reassessed by the Workers' Compensation Board to determine if he had permanent disability.*

INTRODUCTION

Asthma is the most frequent respiratory chronic disorder in working adults. Although often unrecognized, occupational factors may account for one of six cases of adult asthma (1). As reviewed in the chapter 1, work-related asthma refers to either occupational asthma (OA) (either sensitizer- or irritant-induced) or work-exacerbated asthma (WEA). The purpose of this chapter is to review the management of workers with work-related asthma, using an illustrative case similar to the one used to illustrate the various steps in the assessment of work-related asthma in chapter 6. All items of this case history (followed by numbering) illustrate specific items that will be covered in the chapter. However, reactive airways dysfunction syndrome and irritant-induced asthma will not be reviewed here as the subject is covered in chapter 21.

Several guidelines (1–6) on the management of OA have been published or updated in the recent years, which have outlined various aspects of the management of workers with work-related asthma and will be covered in the form of questions. The management of work-related asthma includes prevention, assessment, treatment, and compensation. This chapter focuses mainly on OA treatment, whereas other topics are covered in other chapters.

WHEN SUSPECTING OA, HOW SHOULD THE ASTHMA BE TREATED, SHOULD THE WORKER CONTINUE TO WORK, AND WHEN SHOULD HE/SHE FILE A CLAIM FOR COMPENSATION? (SEE CASE HISTORY 6–10)

As reviewed in chapter 6, the diagnosis of asthma should not rely only on history, as this is too often the case, but on objective evidence of either reversible airways obstruction or increased bronchial responsiveness. Once the diagnosis of asthma is confirmed, the physician has to determine if symptoms at work are due to asthma and if the worker has WEA or OA.

During the investigation, asthma should be treated according to current guidelines on asthma treatment, initially with inhaled beta-2 agonist taken on demand. If this is not enough to control symptoms, inhaled corticosteroids

may be added. Ideally, during the investigation, long-acting beta-2 agonists, long-acting anti-cholinergic, and leukotriene receptor antagonists or inhibitors should be avoided in order to properly assess the relation of symptoms with work but this may not always be possible. In certain cases, theophylline may be added to control symptoms. Controller medication (inhaled corticosteroids ± theophylline) should be kept at a minimum dosage and the dose should not be modified during monitoring.

In order to reduce the socio-economic burden related to a wrong diagnosis, it is highly recommended to avoid taking a subject off work before confirming work-related asthma. However, if asthma symptoms are too severe and uncontrolled, work withdrawal may be necessary, but this should be kept at a minimum to avoid loss of income and stress for the worker. Once asthma is controlled on a minimum dose of medication, the worker should be asked to return to work with continued monitoring while keeping the dose of controlled medication constant and not reduced. Indeed, too often, when taken off work, the patient is treated aggressively for asthma with rapid control and then returned to work while controller medication is reduced or even stopped—this per se may be enough to induce a flare-up of asthma, which may be misdiagnosed as due to work. Monitoring of PEF and nonspecific bronchial responsiveness (NSBR) assessed by the provocative concentration of methacholine causing a 20% fall (PC20-M) in the forced expiratory volume in 1 second (FEV1) may allow confirming of OA, or specific inhalation challenges may be required to confirm or exclude the diagnosis, as reviewed in chapter 9. Although the investigation of work-related asthma may be performed by any physician, referring the worker to a specialized center may facilitate the investigation and is usually recommended (1).

Deciding when it is appropriate to file a claim to either a Workers' Compensation Board or the worker's insurances may be delicate and needs open discussion with the worker. Indeed, in some instances, it may be better to file a claim only when there are sufficient data either confirming the diagnosis of OA or making it likely as some employers may refuse to collaborate and may lay-off a worker simply on presentation of a claim. Although in most countries, the worker is in theory protected by law against such dismissal, it should be kept in mind that this may occur and put the worker in a precarious psychosocial situation. Ideally, the worker should be kept at work until he/she is assessed by a compensation board and a final decision is made.

With salbutamol, the worker was able to continue to work, although he remained symptomatic and was awakened occasionally at night and was symptom free when he was off work for more than a day. He was referred to a specialized center in OA, where he had serial measurements of PC20-M and monitoring of PEF while he was still at work. He had significant and recurrent falls in PEF at night and during the day while working with improvement over weekends and during 2 weeks off for his holidays. His PC20-M improved from 2 mg/mL at the end of a working week, confirming the diagnosis of asthma, to 9.0 mg/mL at the end of his holidays. With these findings combined with positive skin tests to wheat to which he was regularly exposed, the diagnosis of OA to wheat was confirmed. A claim was filed to the Workers' Compensation Board that, upon reviewing the investigation, accepted the diagnosis of OA to flour.

ONCE THE DIAGNOSIS OF OA IS CONFIRMED, WHAT IS THE BEST WAY TO IMPROVE THE OUTCOME? (SEE CASE HISTORY 10–12)

Many studies have confirmed that the prognosis of OA is influenced by host and workplace factors, as reviewed extensively by Maestrelli et al. (7). The worst outcomes are associated with lower airway caliber, lower PC20-M, and stronger asthmatic responses to specific inhalation challenges at the time of diagnosis, as well as a longer duration of exposure while symptomatic. This stresses the importance of early diagnosis of OA and early removal from exposure. Older age at diagnosis seems to be associated with poorer prognosis, while atopy and smoking are not related to asthma outcome. However, as smoking is associated with poorer asthma control (8,9), workers who smoke should be encouraged to quit smoking.

Reducing Exposure Versus No Exposure—Does it Make a Difference in OA?

The main aspect of management of a case of OA is the reduction, either complete or partial, of the causative exposure. This issue has been addressed in three very comprehensive reviews recently published (6,10,11). The following paragraphs summarize four recommendations from the European Respiratory Society (ERS) Report (6) that address using reduction or cessation of exposure in the management of OA. These recommendations were reassessed in a review article by the same professional group in 2012 (11).

- *Patients, physicians, and employers should be informed that persistence of exposure to the causal agent is likely to result in a deterioration of asthma symptoms and airway obstruction* (strong recommendation, moderate quality of evidence).

The Task Force identified nine studies that compared continued exposure to causative agents versus complete cessation of such exposure in the same types of workers (12–20). While asthma symptoms continued in nearly all patients for whom exposure persisted, about one-third of the patients who ceased all exposure actually recovered. A few studies examined objective measurements of respiratory function associated with persistence of exposure. Three studies documented a decline in FEV1 (12,14,19) and two of them an increase in NSBR (12,19), with these findings statistically significant in only one of the studies (19).

- *Patients and their attending physicians should be aware that complete avoidance of exposure is associated with the highest probability of improvement, but may not lead to a complete recovery from asthma* (strong recommendation, moderate quality of evidence).

The ERS Report cited a review that included literature to 2004 (21), which provided average estimates of 32% (95% CI 26–38) for recovery from symptoms and 73% (95% CI 66–79) for persistence of increased NSBR among OA cases that completely avoided further exposure. The authors of the ERS Report updated the review through 2009 and identified similar trends for the same outcomes: 15.5% (95% CI 8.3–27.1) for symptomatic recovery and 67.2% (95 CI 45.7–83.2) for persistence of increased NSBR.

- *Reduction of exposure to the causal agent can be considered as an alternative to complete avoidance in order to minimize adverse socioeconomic consequences; however, there is insufficient available evidence to recommend this option as a first-choice therapeutic strategy. This approach requires careful medical monitoring in order to ensure an early identification of asthma worsening* (weak recommendation, low quality of evidence).

The ERS Task Force members reviewed 10 articles that addressed whether reduction of exposure was as effective as complete cessation of exposure. A meta-analysis of the pooled data had the following findings about reducing exposure in comparison to ceasing exposure entirely: lower likelihood of improvement and recovery of asthma-related symptoms, and higher risk of worsening of symptoms and NSBR. An important caveat about the studies was that the reductions in exposure were not measured, so there was no way to determine if the downward adjustments in levels were truly "substantial" (10). While a couple of articles (22,23) suggest that reducing exposure was associated with less unemployment than cessation of exposure, the Task Force could not conclude that the socio-economic consequences were less for reduction versus cessation of exposure.

- *The use of respiratory protective equipment should not be regarded as a safe approach, especially in the long-term and in patients with severe asthma* (strong recommendation, low quality of evidence).

The ERS Report noted that several controlled studies tested the short-term impact of using respiratory personal protective equipment (PPE) in OA cases to limit exposure (24–28). While these studies did show some positive impact on respiratory symptoms and function (e.g., level and variability of peak flows), the protection was not complete and not all subjects realized benefits. One study showed that subjects with more severe asthma realized no protective effect from using respiratory PPE (28). At least one retrospective investigation of workers with red cedar OA found evidence, suggesting a long-term benefit of using respiratory PPE (29). Among the workers who remained exposed to red cedar after onset of OA, those with stable asthma were more likely to use a twin-cartridge respirator at work than those with worsening disease.

Medical Management

This topic has been well covered in recent reviews and guidelines (1,3,6,30).

The pharmacologic management of workers with OA who have and have not stopped exposure does not differ from the management of nonwork-related asthma. There is insufficient evidence to support systematic treatment with high-dose inhaled corticosteroids in addition to cessation of exposure to the causal agent (6). Asthma should be controlled with the minimum amount of medication, and the worker should be well educated on the means to achieve this control.

In workers who continue exposure to their offending agent, even if reduced, the need of medication is usually greater but asthma can usually be controlled—this may not preclude deterioration of asthma and there is currently no means to predict who will have a worse outcome if exposure is not completely stopped. Recent anecdotal reports have suggested that omalizumab treatment may improve asthma control in bakers with OA with continuous exposure (31,32), but further studies are needed before this approach can be recommended. Similarly, subcutaneous immunotherapy with some high-molecular weight (HMW) allergens (33) may be useful in certain cases, although this is limited as standardized extracts are lacking for most agents and there is potential for serious adverse effects. There is some suggestion that sublingual immunotherapy may offer some protection in workers with latex allergy. At present, however, there is a lack of evidence-based information on the effectiveness and adverse effects of specific immunotherapy with HMW allergens.

Rehabilitation/Work Reintegration

Once the diagnosis of OA is confirmed and the decision is made for the worker to stop the exposure completely, vocational rehabilitation should be offered to the worker to reduce the psychosocial burden of the disease (34). There is a need for active communication between the treating physician and the workplace and the compensation board/insurance company to ensure the best chances for the worker to return to full employment in safe conditions. The relevant sensitizer and any cross-reacting sensitizers must be completely avoided. Furthermore, if the worker has persistent asthma, he may need protection from environmental exposure to irritant triggers such as cold air, fumes, and dusts, and he may also have some exercise limitation. Follow-up of the worker in his new environment with appropriate adjustment in exposure or medication is necessary.

In workers with OA who continue to work with reduced exposure, or in the same workplace with supposedly no exposure to the relevant sensitizer, close monitoring of symptoms, airway caliber, and NSBR is essential to insure that the asthma does not get worse.

Upon review and acceptance of the diagnosis of OA by the Workers' Compensation Board, the baker was referred to the workers' reintegration program. As the company was a small family business with no possibilities to find him a proper job where he would not be exposed to flour, he was given the opportunity to retrain in a new trade. He followed a truck driving course and was able to find a job with adequate income.

Compensating the Worker for Disability–Impairment

Once OA has developed, there is good evidence that the specific sensitization to the offending agent is long lasting even following cessation of exposure and normalization of NSBR (35–37). Therefore, workers with OA should be considered as being permanently disabled as they cannot be re-exposed to their offending sensitizer and this may be associated with loss of income. As most subjects with OA have persistence of increased NSBR, with or without symptoms requiring medication, there is a need to assess if there is permanent impairment and disability after cessation of exposure. Most subjects reach a plateau of improved FEV1 by 1 year and a plateau of improved NSBR by 2 years while other individuals do not. Several schemes for compensation (38–40) have been proposed and this is discussed in chapter 13.

When reassessed 2 years after cessation of working in the bakery, the worker was asymptomatic, requiring no asthma medication. He had a normal spirometry and PC20-methacholine >32 mg/mL, thus normal. He had reached maximal medical improvement (MMI), showed no objective findings for permanent pulmonary impairment, and received no further disability compensation.

Surveillance, Prevention

Any case of OA should be considered a sentinel event. This topic is covered in chapter 12, but should be considered as part of the management of OA.

ONCE WORK-EXACERBATED ASTHMA (WEA) IS CONFIRMED, WHAT IS THE BEST WAY TO IMPROVE OUTCOME?

The management of WEA was addressed in the consensus statement on work-related asthma that the American College of Chest Physicians (ACCP) published in 2008 (30). One of the consensus statements from that document was: "In individuals with irritant-induced asthma or WEA, the panel advises optimizing asthma treatment and reducing the exposure to relevant workplace triggers. If not successful, change to a workplace with fewer triggers is suggested in order to control asthma." This advice was provided in the context of limited data on the natural history and management of WEA. This is an area where further studies are needed to provide evidence to refine the current recommendations.

Indeed, in these circumstances, the role of the physician is to insure that there is sufficient patient education on the role of trigger factors in the workplace and home environment, avoidance of allergens, explanation of the significance of environmental controls, and the importance of compliance with prescribed medications usage, especially the continuation of regular use of maintenance-inhaled corticosteroids. Reduction of exposure to irritants should be recommended. Use of respiratory protection may be offered to workers but these devices are often difficult to wear. In certain jurisdictions, claims for WEA are accepted by the compensation boards, facilitating the rehabilitation of the worker.

DISCLAIMER

The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

REFERENCES

1. Fishwick D, Barber CM, Bradshaw LM, et al. Standards of care for occupational asthma: an update. Thorax 2012; 67: 278–80.
2. Beach J, Rowe BH, Blitz S, et al. Summary, Evidence Report/ Technology Assessment No. 129. Diagnosis and management of work-related asthma. Rockville, MD: Agency for Healthcare Research and Quality, 2005.
3. 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.
4. Tarlo SM, Liss GM. Evidence based guidelines for the prevention, identification, and management of occupational asthma. Occup Environ Med 2005; 62: 288–9.
5. Henneberger PK, Redlich CA, Callahan DB, et al. An official American Thoracic Society statement: work-exacerbated asthma. Am J Respir Crit Care Med 2011; 184: 368–78.
6. Baur X, Sigsgaard T, Aasen TB, et al. Guidelines for the management of work-related asthma. Eur Respir J 2012; 39: 529–45.
7. Maestrelli P, Schlunssen V, Mason P, Sigsgaard T. Contribution of host factors and workplace exposure to the outcome of occupational asthma. Eur Respir Rev 2012; 21: 88–96.
8. Livingston E, Thomson NC, Chalmers GW. Impact of smoking on asthma therapy: a critical review of clinical evidence. Drugs 2005; 65: 1521–36.
9. Thomson NC, Shepherd M, Spears M, Chaudhuri R. Corticosteroid insensitivity in smokers with asthma: clinical evidence, mechanisms, and management. Treat Respir Med 2006; 5: 467–81.
10. Vandenplas O, Dressel H, Wilken D, et al. Management of occupational asthma: cessation or reduction of exposure? A systematic review of available evidence. Eur Respir J 2011; 38: 804–11.
11. Vandenplas O, Dressel H, Nowak D, Jamart J. What is the optimal management option for occupational asthma? Eur Respir Rev 2012; 21: 97–104.

12. Chan-Yeung M, Maclean L, Paggiaro PL. Follow-up study of 232 patients with occupational asthma caused by western red cedar (Thuja plicata). J Allergy Clin Immunol 1987; 79: 792–6.
13. Rosenberg N, Garnier R, Rousselin X, Mertz R, Gervais P. Clinical and socio-professional fate of isocyanate-induced asthma. Clin Allergy 1987; 17: 55–61.
14. Gannon PF, Weir DC, Robertson AS, Burge PS. Health, employment, and financial outcomes in workers with occupational asthma. Br J Ind Med 1993; 50: 491–6.
15. Moscato G, Bertoletti R, Biscaldi G, et al. Occupational asthma: fate and management after the diagnosis. G Ital Med Lav 1993; 15: 27–31.
16. Tarlo SM, Banks D, Liss G, Broder I. Outcome determinants for isocyanate induced occupational asthma among compensation claimants. Occup Environ Med 1997; 54: 756–61.
17. Merget R, Schulte A, Gebler A, et al. Outcome of occupational asthma due to platinum salts after transferral to low-exposure areas. Int Arch Occup Environ Health 1999; 72: 33–9.
18. Orriols R, Drobnic ME, Munoz X, Rodrigo MJ, Morell F. Occupational asthma due to isocyanates: study of 21 patients. Med Clin (Barc) 1999; 113: 659–62.
19. Valentino M, Rapisarda V. Course of isocyanate-induced asthma in relation to exposure cessation: longitudinal study of 50 subjects. G Ital Med Lav Ergon 2002; 24: 26–31.
20. Padoan M, Pozzato V, Simoni M, et al. Long-term follow-up of toluene diisocyanate-induced asthma. Eur Respir J 2003; 21: 637–40.
21. Rachiotis G, Savani R, Brant A, et al. Outcome of occupational asthma after cessation of exposure: a systematic review. Thorax 2007; 62: 147–52.
22. Burge PS. Occupational asthma in electronics workers caused by colophony fumes: follow-up of affected workers. Thorax 1982; 37: 348–53.
23. Vandenplas O, Jamart J, Delwiche JP, Evrard G, Larbanois A. Occupational asthma caused by natural rubber latex: outcome according to cessation or reduction of exposure. J Allergy Clin Immunol 2002; 109: 125–30.
24. Slovak AJ, Orr RG, Teasdale EL. Efficacy of the helmet respirator in occupational asthma due to laboratory animal allergy (LAA). Am Ind Hyg Assoc J 1985; 46: 411–15.
25. Kongerud J, Rambjor O. The influence of the helmet respirator on peak flow rate in aluminum potroom. Am Ind Hyg Assoc J 1991; 52: 243–8.
26. Laoprasert N, Swanson MC, Jones RT, Schroeder DR, Yunginger JW. Inhalation challenge testing of latex-sensitive health care workers and the effectiveness of laminar flow HEPA-filtered helmets in reducing rhinoconjunctival and asthmatic reactions. J Allergy Clin Immunol 1998; 102: 998–1004.
27. Muller-Wening D, Neuhauss M. Protective effect of respiratory devices in farmers with occupational asthma. Eur Respir J 1998; 12: 569–72.
28. Taivainen AI, Tukiainen HO, Terho EO, Husman KR. Powered dust respirator helmets in the prevention of occupational asthma among farmers. Scand J Work Environ Health 1998; 24: 503–7.
29. Côté J, Kennedy S, Chan-Yeung M. Outcome of patients with cedar asthma with continuous exposure. Am Rev Respir Dis 1990; 141: 373–6.
30. Tarlo SM, Balmes J, Balkissoon R, et al. Diagnosis and management of work-related asthma: American College of Chest Physicians consensus statement. Chest 2008; 134: 1S–41S.
31. Olivieri M, Biscardo CA, Turri S, Perbellini L. Omalizumab in persistent severe bakers' asthma. Allergy 2008; 63: 790–1.
32. Perez PA, Bueso FA, Garcia LJ, et al. Effect of omalizumab treatment in a baker with occupational asthma. J Investig Allergol Clin Immunol 2008; 18: 490–1.
33. Sastre J, Quirce S. Immunotherapy: an option in the management of occupational asthma? Curr Opin Allergy Clin Immunol 2006; 6: 96–100.
34. Tarlo SM, Boulet LP, Cartier A, et al. Canadian Thoracic Society. Guidelines for occupational asthma. Can Respir J 1998; 5: 289–300.
35. Lemière C, Cartier A, Dolovich J, et al. Outcome of specific bronchial responsiveness to occupational agents after removal from exposure. Am J Respir Crit Care Med 1996; 154: 329–33.
36. Lemière C, Cartier A, Malo JL, Lehrer SB. Persistent specific bronchial reactivity to occupational agents in workers with normal nonspecific bronchial reactivity. Am J Respir Crit Care Med 2000; 162: 976–80.
37. Lemière C. Persistence of bronchial reactivity to occupational agents after removal from exposure and identification of associated factors. Ann Allergy Asthma Immunol 2003; 90: 52–5.
38. Harber P. Assessing disability from occupational asthma. A perspective on the AMA Guides. Chest 1990; 98: 232S–5S.
39. Malo JL. Compensation for occupational asthma in Quebec. Chest 1990; 98: 236S–9S.
40. ATS. Guidelines for the evaluation of impairment/disability in patients with asthma. American thoracic society. Medical section of the American lung association. Am Rev Respir Dis 1993; 147: 1056–61.