Problem-Solving Techniques in Occupational Medicine

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The diagnosis of occupational illnesses may be considerably more difficult than is the case with occupational injuries because of a variety of factors: an intervening latency period, uncertainty in identifying the most significant chemical or physical exposures, determination of exposure levels retrospectively, and coordination of the physician with regulatory and workers' compensation bureaucracies. Such problem-solving techniques as retrospective industrial hygiene and attention to in-situ chemistry can act as means of reducing the uncertainty in making the diagnosis of occupational illness. Advance familiarity with workers' compensation and state or federal regulatory agencies can further facilitate diagnosis and patient advocacy.

Few areas of medicine so thoroughly challenge the family physician's technical and organizational skills as the realm of occupational injuries and illnesses. Based upon survey data, the Bureau of Labor Statistics of the US Department of Labor estimates that some five million occupational cases occur each year nationally, of which 2 to 3 percent qualify as "occupational illnesses," ie, responses to noxious physical, chemical, or biological agents in the workplace.[1] Facility in obtaining a detailed occupational history, access to information resources regarding toxicology, and finally, the interface with industrial hygiene personnel, regulatory agencies, and state workers' compensation systems all present barriers to successful diagnosis and patient advocacy in the field of occupational medicine. A number of excellent articles have appeared in the primary care literature emphasizing occupational history-taking and information resources.[2-7] The aim of this article is to highlight problem-solving techniques in the context of brief case histories.

Illustrative Cases

Case 1

A 40-year-old, female, electronics worker was evaluated after having been examined and observed overnight in an emergency room for the effects of an acute overexposure to phosphine gas (PH_3), used in semiconductor fabrication. While

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this agent is capable of producing pneumonitis and noncardiogenic pulmonary edema in sufficient concentrations,[8] in this case the symptoms of lightheadedness and dyspnea were not accompanied by any radiographic or electrocardiographic changes, and arterial blood gases showed only a mild respiratory alkalosis without hypoxemia. The patient was a nonsmoker with no identified chronic health problems, and abnormalities on physical examination in the emergency room were confined to an apparent chemical conjunctivitis.

On reevaluation four months later, the patient reported having had mild exertional dyspnea and wheezing away from the workplace, which had largely resolved. More significantly, she complained of symptoms consistent with hyperventilation when she smelled the characteristic garlic-like odor of phosphine that occurred transiently on the job, an odor she had previously tolerated before her overexposure. Use of an aerosol bronchodilator in the interval since her emergency room visit helped with her apparent transient bronchial hyperreactivity, but did not control her work-related symptoms. In her own words, she felt "paranoid" about her work environment and desired reassignment or job retraining.

Physical examination revealed a relaxed woman with a resting respiratory rate of 20 per minute, chest clear to auscultation, no peripheral cyanosis or clubbing, and no murmur or gallop on cardiac examination. Pulmonary function testing showed only minimal reversible airflow obstruction (forced expiratory volume in one second was 2.0 L, or 83 percent of predicted, rising to 2.58 L, or 107 percent, after bronchodilators), with normal lung volumes, arterial blood gases, and carbon monoxide diffusion capacity. No bronchial provocation testing was done, as phosphine acts as an irritant rather than an allergen and is capable of producing tracheitis and bronchospasm in essentially any individual.[9]

While industrial hygiene measurements of the patient's work environment were not available, information on the odor threshold for phosphine, variously reported as 0.02 to 3.0 ppm,[9,10] raised the possibility that both the existing Occupational Safety and Health Administration (OSHA) standard for an average exposure of 0.2 ppm and a recommended short-term exposure limit of 1 ppm[11] were exceeded on more than one occasion. More importantly, since the previously tolerated odor of phosphine was now associated by the patient with an episode of intense respiratory irritation, it was fair to state that she was psychologically sensitized to her work environment. Barring industrial hygiene measures sufficient to control phosphine concentrations below the odor threshold, she could not remain in the work environment without experiencing episodes of hypervenilation. Reassignment was obtained with no recurrence of symptoms.

Comment

Attempts to reconstruct workplace conditions based on historical factors (such as the perception of an odor) or on biological monitoring (such as blood levels of a toxin) might be termed *retrospective industrial hygiene*. While not identical to workplace air sampling, these techniques do have specific reference to the actual exposure conditions that pertained during a particular time frame. (For some exposures, such as lead, both industrial hygiene and biological monioring may be required by standards.) A workplace inspection, by contrast, may or may not document representative conditions, depending on a variety of factors, including the specific circumstances of an acute overexposure and the degree to which the work environment has been modified in anticipation of the inspection. The following case illustrates this factor.

Case 2

A 32-year-old automobile mechanic presented to his family physician with the complaint of headaches that were global in location, daily in occurrence, and not associated with visual auras, nausea, localized weakness, or sensory disturbance. He was a nonsmoker and denied any history of sinusitis or allergies. He did feel that this problem was associated with the institution one month earlier of several heat-conserving measures in his workplace at the beginning of the cold weather season. These measures included keeping the garage doors partially or completely closed (even

when cars were running), placing plastic trash bags over the roof vents, and using an unvented, catalytic kerosene heater indoors. He had been off work for several days at the time of the first visit, and while there had been some improvement in his symptoms, he still complained of occipital headaches.

The patient had borderline hypertension (140/90 mmHg), and there were normal findings on cardiopulmonary, neurological, and fundoscopic examination. Musculoskeletal examination revealed moderate occipital and trapezius muscle group tenderness. A baseline carboxyhemoglobin level was obtained and was reported as "undetectable—less than 5 percent." The patient was prescribed a muscle relaxant and mild analgesic, and he was instructed to return to work and to report for another carboxyhemoglobin determination at the close of his second day back at work. On follow-up, the patient reported a good therapeutic response only until he had been back in the workplace for a few hours, after which time his headache recurred. The diagnosis was made of headaches secondary to carbon monoxide intoxication when the second carboxyhemoglobin level was reported to be 17 percent. The presumed mechanism of temporal generalization of these headaches was via occipital muscle spasm.

The patient was advised of his options under the California Occupational Health and Safety Administration of filing an anonymous complaint, or of informing his employer, suggesting that the employer request a voluntary inspection without threat of penalty. He chose the latter course. The employer, however, contended that the workplace could not be responsible for the intoxication, but proceeded to modify the workplace (ie, remove bags from the vents and maintain garage doors wide open) while simultaneously maintaining that an inspection would "prove that the workplace was not at fault." An industrial hygiene inspection, not surprisingly, found the workplace air within legal standards for carbon monoxide.

After eliminating any possible outside sources of carbon monoxide exposure for this worker, the following calculation was performed: assuming a four-hour half-life for carbon monoxide elimination in an active individual,[12] for the patient to have a 17 percent carboxyhemoglobin level at the end of an eight-hour workday as the sole consequence of an exposure outside of the workplace, the patient would have had to come to work with a carboxyhemoglobin level four times that, or 68 percent. With the range for production of a state of coma being 38 to 60 percent,[9] this was unlikely to have been the sequence of events. The workers' compensation insurance carrier apparently agreed, and temporary disability was granted without adjudication.

Comment

Workers' compensation, as it is variously implemented by individual states, is a no-fault insurance system that generally compensates for work-related health problems without regard to culpability, relieving workers from the compensatory exclusion of "contributory negligence" as well as protecting the employer from lawsuit in most cases ("exclusive remedy"). The principal built-in incentive is for the employer to maintain a favorable claims record in order to keep premiums down. To this end, most workers' compensation insurance carriers employ their own industrial hygienists and safety personnel who can consult in their clients' workplaces.

The vast majority of employers comprehend the indirect nature of the system and are able to learn from a claims experience, even if the lesson is only to insist on more stringent adherence to safety procedures by employees. Unfortunately, however, some employers may act as though workers' compensation benefits come out of their own pockets, and may subject an employee who files such a claim to harrassment. In the case under discussion, the patient-worker was, indeed, discharged without cause. He was subsequently awarded back pay under provisions of Section 132(a) of the California Labor Code, which prohibits discrimination on the basis of having filed a workers' compensation claim. Antidiscrimination remedies also exist for workers who request OSHA inspections.

The work situation described was a particularly flagrant example of disregard for worker safety in its failure to exhibit even commonsense protective measures. The goals of maintaining a reasonably warm work environment while properly venting exhaust gases could be achieved by practicing

direct venting of cars' tailpipes to suction hoses. While this solution is ideal, it is not required by statute, and in the present case, after initial intervention, the expedient of sacrificing worker comfort by employing "wide-open" ventilation was taken. The patient-worker himself reported complete resolution of headaches when he was removed from the work situation, and no recurrence in his subsequent place of employment, another garage with more adequate ventilation.

Case 3

A 37-year-old man was seen in follow-up after having been examined in the emergency room for transient upper respiratory tract irritation, followed by headaches, vertigo, abdominal pain, and diarrhea. He also reported several episodes of vomiting of bilious material. Onset of symptoms had occurred acutely at work, where he was involved in recycling barrels from various sources for use in local canneries. His job had involved removing the tops from used barrels, pouring out the contents, "flaming" the barrels in a furnace (with no exhaust stack), and using a torch to cut barrels down. The current batch of barrels was marked as having contained TOK, which was later learned to be the herbicide nitrofen (2,4 dichlorophenyl-p-nitrophenyl ether), currently under suspension because of imputed teratogenicity and carcinogenicity.

Original examination in the emergency room revealed a mildly distressed man whose heart rate went from 84 to 100 beats per minute going from supine to standing position, with blood pressure steady at 130/80 mmHg, temperature 98.0° F, respirations 16 per minute. Abdominal examination showed mild diffuse tenderness to direct palpation, with no guarding or rebound. The chest was clear to ausculation, and no murmur, gallop, or rub was heard on cardiac examination. Ear, nose, and throat examination was unremarkable; no nystagmus was observed, and pupils were noted to be equal, but the pupillary diameters were not noted. A white cell count showed $13 \times 10^3 / \mu L$, with no band forms, 66 percent segmented neutrophils, 26 percent lymphocytes, and 4 percent each monocytes and eosinophils.

On reexamination, three days after the onset of symptoms, the patient continued to complain of decreased appetite, occasional nausea, and intermittent diarrhea. Vital signs were normal, pupils were noted to be 3 mm in diameter and reactive to light, and abnormalities on physical examination were confined to mild diffuse abdominal tenderness. A repeat blood count, as well as electrolytes, glucose, blood urea nitrogen, creatinine, total bilirubin, and aspartate amino transferase, was normal, as were red blood cell and plasma cholinesterase. The additional history was obtained of a fellow worker similarly exposed and similarly affected. (This worker was subsequently examined, with similar physical and biochemical findings as the index case.) Symptoms resolved gradually over the following week.

Comment

Symptoms consistent with a viral illness may confound the diagnosis of such occupational syndromes as metal fume fever[13] and polymer fume fever[14] (respiratory symptoms), or organophosphate insecticide poisoning[15] (gastrointestinal symptoms). In this case, the strong temporal association between exposure and symptoms, as well as the suggestive history of having a similarly stricken co-worker, lends support to the possible etiologic role of nitrofen vapor inhalation, skin absorption, or inhalation of nitrofen pyrolysis products in producing the clinical syndrome described. No organophosphate pesticide residues were found on subsequent industrial hygiene inspection; however, the limited acute human toxicity data available on nitrofen were consistent with its role as causative agent.

The possible role of nitrofen pyrolysis (thermal decomposition) products would be an example of what might be termed *in situ chemistry*, a class of phenomena active in the above-mentioned syndromes of metal fume fever (due to the zinc oxide fumes produced when welding on galvanized iron) and polymer fume fever (due to polytetrafluoroethylene pyrolysis products produced when cigarettes, contaminated with Teflon because of a lack of handwashing on the job, are smoked). In situ chemistry also plays a role in household toxic exposures involving the mixing of bleach (sodium

hypochlorite) with ammonia, producing chloramine gas, or with acidic toilet bowl cleaners, producing chlorine gas. Finally, much of the toxicity from smoke inhalation is currently postulated to derive from toxic pyrolysis products of natural and synthetic materials.[16]

In the foregoing case, because of a worker history of questionable disposal methods for the contents of the barrels being recycled, as well as in compliance with reporting requirements in California for suspected pesticide-related illnesses, the local health department was alerted. This move eventuated in the involvement of state and federal environmental authorities, identification of the site as a toxic waste dump, and the issuance of a cleanup order. Additionally, California OSHA was contacted, resulting in the issuance of multiple citations against the employer for unsafe working conditions. Neither worker reported a repetition of symptoms after industrial hygiene intervention took place, although both workers have since found other employment.

Discussion

Occupational diseases present particular challenges to the primary care physician. Causation may be considerably more obscure than is the case with an acute occupational injury. Physician ambivalence may be aroused by a perception of patient motivation toward "secondary gain," and the physician may feel reluctant to interfere with the employer's business. Together with the considerable difficulties of mastering the technical data base in occupational medicine, these factors conspire to foster an atmosphere of benign neglect toward the diagnosis of occupational disease.

On the other hand, alert primary care practitioners have played a role in the discovery of new occupational syndromes: aseptic necrosis of the jaw (and later aplastic anemia and osteogenic sarcoma) in radium dial painters,[17] angiosarcoma of the liver in vinyl chloride monomer exposure,[18] and oat cell carcinoma of the lung in bischloromethyl ether exposure.[19] Attention to such analytical techniques as retrospective industrial hygiene and the potential for in situ chemistry can provide tools in the analysis of otherwise confusing clinical pictures. Furthermore, familiarity with workers' compensation and federal or state rules can facilitate a physician's interaction with these bureaucracies. Such preparation and analytical approaches can significantly aid in the recognition of and intervention in occupational diseases, essential roles for the primary care physician.

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