Brief Communication

Methylene Chloride Intoxication in a Furniture Refinisher

A Comparison of Exposure Estimates Utilizing Workplace Air Sampling and Blood Carboxyhemoglobin Measurements

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A 35-year-old furniture refinisher came to the occupational medicine clinic with complaints of upper respiratory irritation, fatigue, and lightheadedness occurring on a daily basis after using a methylene chloride-containing paint remover. Determinations of blood carboxyhemoglobin (COHb) on three occasions showed an apparently linear elevation of COHb as a function of hours worked on the day of sampling. COHb levels predicted from spot industrial hygiene measurements were in close concordance with those observed in the patient, indicating the potential usefulness of COHb monitoring in estimating airborne exposure levels.

Methylene chloride (or dichloromethane) is an organic solvent that has found wide use as a degreaser, paint remover, aerosol propellant, and a blowing agent for polyurethane foams, and as a solvent in food processing, photographic film production, and plastics manufacturing. Discovery of its unusual metabolic fate—conversion to carbon monoxide in vivo—has earned the compound a special place in the solvent toxicology literature. Demonstration of oncogenicity in experimental animals has occasioned a reconsideration of exposure limits, with emphasis upon stricter controls. In some workplaces, conditions prevail in which controls are inadequate to prevent even acute toxicity, much less long-term exposure risks.

Case Presentation

A 35-year-old male furniture refinisher was referred to the occupational medicine clinic for evaluation of intermittent eye and throat irritation, nasal discharge, nonproductive cough, fatigue, and lightheadedness that he had been suffering for 15 months. He denied headache or chest pain at the time of initial evaluation. He had worked full-time in his present job for approximately 2 1/2 years, applying an 80% methylene chloride, 20% methanol stripping solution to furniture with a brush and pump assembly. Other tasks included dipping furniture in tanks containing the stripper and water-blasting residual stripper and finish from furniture.

The patient wore rubber boots, a waterproof apron, neoprene gloves, goggles, and a half-mask organic vapor cartridge respirator. According to the patient, he could detect solvent smells within a few days of a respirator cartridge change, but described being limited to cartridge changes approximately once every 10 days because of the expense involved. While water-blasting furniture, the patient frequently removed his respirator.
because the mist increased the effort of breathing through it. A slot ventilation system had reportedly been connected around the periphery of the furniture-scrubbing tables in the past, but when these tables had been relocated about 1 1/2 years earlier, this system was left disconnected. Area ventilation was provided only by air movement through a large sliding door, in the front of the shop, that was normally kept open.

The patient noticed symptoms at work, with improvement on weekends and when on vacation. Symptoms were worse when using old respirator cartridges, on hot days, or with extensive use of the dip tanks. Symptoms of lightheadedness tended to peak in the afternoons and resolve in the evenings, whereas upper respiratory irritation frequently persisted through the work week. The patient was a nonsmoker, having quit 3 years earlier after smoking one quarter of a pack per day for 5 years. He consumed one or two six-packs of beer each weekend, but none during the work week. He took no regular medications and had no history of hemolytic anemia or other blood dyscrasias. There was no history of allergies or hay fever, and review of his nonoccupational exposures revealed no pets, passive smoking, excessive dust, or use of forced-air heating or woodstoves. His occasional hobby was oil painting (once or twice a month), at which times he kept windows open for ventilation.

Physical examination revealed slight injection of the tonsillar pillars and nasal mucosa, with no conjunctival injection. Results of cardiac and pulmonary examinations were normal. Examination of the abdomen revealed a nontender and non-nodular liver edge two fingerbreadths below the costal margin; the liver span was not clearly percussible. The patient behaved normally during a mental status examination, which included alertness, affect, orientation, short-term memory, and calculation. Results of a limited neurological examination (cranial nerves, deep tendon reflexes, sensation, Romberg, and gait) were within normal limits. Review of the results of a chemistry panel obtained 3 months before examination was remarkable only for an aspartate aminotransferase of 44 Karmen units/mL (normal, <40 Karmen units/mL), with normal alanine aminotransferase, alkaline phosphatase, and bilirubin levels.

A presumptive diagnosis was made of upper respiratory irritation and transient symptoms of central nervous system depression secondary to methylene chloride exposure. Note was also made of an isolated, marginally elevated liver function test and possible hepatomegaly.

A carboxyhemoglobin (COHb) level was obtained 3 hours after completing half of an 8-hour workday; the result was later reported as 7.5% (less than 2% is normal for nonsmokers).8 The patient was instructed to discontinue his weekend alcohol consumption and recreational oil painting, and a repeat liver function panel and COHb were scheduled for 1 month later.

An industrial hygiene inspection of the patient's work site identified the use of uncovered buckets for recycling the stripping solution, a nonoperative slot ventilation system with no overhead ventilation, the draining of paint sludges within the work area, and the performance of water-blasting in an unenclosed portion of the work area. Air sampling ("spot" samples using detector tubes (National Draeger, Inc, Pittsburgh, Pa). In the employee's breathing zone) showed methylene chloride levels of 350 ppm in the production area during manual stripping (the patient's primary activity), and 100 ppm in the break room. A review of the clinic's industrial hygiene records revealed that a previous inspection of the same workplace 4 years earlier had demonstrated similar methylene chloride levels, despite the fact that the slot ventilation system had been working at the time. Recommendations made at that time regarding the installation of a general ventilation system had not been heeded.

The employer agreed to request an industrial hygiene consultation and to make respirator cartridges available for frequent changes.

One month after the initial visit, the patient reported a slight improvement in his symptoms of sore throat, cough, and dizziness after partial modification of the work practices, including covering solvent reservoirs between use, at least weekly changes of the respirator cartridges, and consistent use of a respirator during water-blasting. The ventilation system, however, remained unchanged. Weekend beer-drinking and oil painting had been curtailed since the last visit, and repeat liver function test results were within normal limits. A follow-up COHb level test was scheduled, but was delayed several times because of the patient's concern with lost work time.

Two months later, the patient reported that he had been unable to obtain replacement respirator cartridges for more than 3 weeks and had begun to experience headaches at work. The repeat carboxyhemoglobin level at that time was 10.4% (approximately 40 minutes after completing 6.5 hours of work). Symptoms persisted into the following working day, and the patient was placed on temporary total disability pending reduction of methylene chloride exposures. The employer was informed of the decision and was again urged to effect rapid abatement of the workplace hazard, in accordance with the industrial hygiene recommendations of his consultant. After 3 days off work, the patient reported nearly complete resolution of symptoms, and a repeat physical examination showed normal results. A baseline COHb level obtained at that time was 1.1%, well within the normal range. Over the subsequent year the patient has been unemployed, his employment having been terminated by the employer, ostensibly for reasons unrelated to health. His symptoms have completely resolved.

Discussion

This patient's symptoms of intermittent eye and throat irritation, lethargy, and (eventually) headaches were consistent with repeated episodes of acute methylene chloride intoxication. The patient's single, marginally elevated liver function test results (and possible hepatomegaly), on the other hand, were difficult to ascribe specifically to his methylene chloride exposure,
given his weekend ethanol consumption, occasional oil painting, and the borderline nature of the findings. There were no clinical indications of cardiovascular compromise or chronic neurotoxicity, as evidenced by the absence of chest pain, neurological abnormalities, or mental status changes.

The formation of carboxyhemoglobin from CO generated in vivo from the metabolism of methylene chloride can be used to estimate methylene chloride exposure levels. Such estimates presume that there are no important confounding exposures (e.g., to vehicular exhaust or cigarette smoke) that also raise COHb.10 Methylene chloride is well absorbed during both inhalation and cutaneous exposure.11,12 Approximately 70% of an inhaled dose is retained; of this retained fraction, about 5% is excreted unchanged in alveolar air and 25% to 34% is eliminated from the lungs as CO. The remainder (up to 70% of the absorbed dose) is metabolized to carbon dioxide.11 The conversion to CO occurs via a saturable hepatic microsomal pathway, although saturation probably does not occur at exposure levels below 350 to 500 ppm.13 Several investigators have examined the kinetics of methylene chloride conversion to carbon monoxide, under either controlled conditions or working conditions.6-8,11,13,14 In these experiments, COHb levels typically plateau for a period of 2 to 3 hours post-exposure before beginning to drop, presumably reflecting mobilization of solvent from fat stores and continued conversion to carbon monoxide.6 The subsequent half-time of COHb clearance is approximately 13 hours—3 times longer than is observed in CO intoxication alone.4,10

This plateau and prolonged elimination phase of methylene chloride-derived COHb extends the critical period for biological monitoring. This is fortuitous in the present case, because the two post-exposure COHb levels were obtained after differing unexposed intervals. After initially concluding that the upward trend in COHb levels in this patient represented a worsening of the industrial hygiene situation, we realized that the difference might be related to duration of exposure. The three COHb determinations were subsequently graphed as a function of hours of work exposure (Fig. 1), with the data points being consistent with a linear relationship between the two variables (r = .997; slope = 1.45% per hour). Based upon extrapolation from these data, it was estimated that an 8-hour exposure in this workplace would be expected to produce a COHb level of 12.7%, assuming continued linearity with exposure time.

Figure 2, by comparison, represents the COHb levels achieved in two separate studies of 7.5-hour controlled exposures of sedentary human subjects at various methylene chloride levels. In these studies, a high degree of linearity is apparent between exposure concentration and COHb level (r = .997).14,18 Extrapolating from the highest exposure level in these experiments (350 ppm) to the measured concentration in the production area of the furniture-stripping shop described here (350 ppm), one would predict a COHb level of 11.8% at the end of 7.5 hours (or 13.1% after 8 hours, because COHb levels rose linearly with time in these experiments). The 12.1% figure corresponds closely to the above-cited 8-hour estimate of 12.7%, based upon measurements of the patient described.

The degree of concordance between predicted and measured COHb levels is remarkable, particularly given various potential complicating factors in this analysis. For example, because the patient was wearing an organic vapor cartridge respirator, one would expect some diminution of exposure compared with ambient measurements. However, this source of error is likely to be small, given the fact that organic vapor cartridges become saturated and breakthrough occurs within 30 minutes of exposure to methylene chloride at concentrations of 500 ppm.15 On the other hand, the fact that methylene chloride is not only well absorbed through the skin but also penetrates most glove materials (including neoprene) may have compensated for any deficit in presumed inhalation exposure.15 Likewise, the fact that the patient was involved in vigorous physical activity would have raised his methylene chloride uptake relative to the sedentary exposures applicable to the experimental studies cited above.16 Finally, the exposure estimates in this case were based on "spot" samples which, although consistent over more than one sampling date, were not true time-weighted averages.
The in vivo conversion of methylene chloride to CO constitutes an important component of its short-term health risks. The acute health effects of CO are well known, with headaches and lightheadedness typically reported in the 10% to 20% COHb range, and decreased perceptual motor performance, dyspnea on exertion, nausea, obtundation, coma, convulsions, and death occurring at progressively higher levels. At the opposite end of the spectrum, COHb levels as low as 2% have been associated with decreased exercise tolerance in coronary artery disease patients exposed to CO alone. Such levels would have been expected in the current workplace after only 1 hour of exposure. The American Conference of Governmental Industrial Hygienists' 8-hour time-weighted average Threshold Limit Value for methylene chloride has been set at 50 ppm; exposure at this concentration produces COHb levels of about 1.9% in experimental subjects. By comparison, the federal Occupational Safety and Health Administration's 8-hour permissible exposure level (PEL) at the time of this writing is 10 times higher—500 ppm—which is the standard is currently being reviewed by that agency. Eight-hour exposure to methylene chloride at its current 500 ppm PEL would be expected to produce COHb levels in excess of 12%—about twice those produced by exposure to carbon monoxide itself at its 8-hour PEL of 50 ppm.

This case illustrates the correlation between measured COHb and duration of methylene chloride exposure in a worker who was symptomatic because of his exposure. Because workplace air monitoring data may not always be available to the clinician, COHb measurements may be considered as a preliminary means of estimating airborne exposures to methylene chloride as well as acute risk from in vivo CO formation. It is likely that the workplace characterized in this case report is representative of many that merit intensive corrective action to reduce methylene chloride exposures.

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References


