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Inhalational Lung Injury Associated With Humidifier "White Dust"

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abstract

Humidifiers are commonly used in the community to relieve symptoms associated with acute respiratory infections in young children; however, clear benefits of these devices have not been documented. The Environmental Protection Agency has not found any adverse health effects related to humidifier use. We report here the case of a young infant with significant accidental inhalational lung injury related to dispersal of mineral dust from an ultrasonic home-use humidifier. The clinical consequences included prolonged hypoxemia, tachypnea, and failure to thrive. Radiography revealed pneumonitis, and pulmonary-function testing showed a nonreversible mild obstructive ventilatory defect. Because of persistent symptoms, evolution of failure to thrive, and nonresponse to inhaled and short courses of systemic glucocorticoids, an aggressive management approach was successfully pursued with high-dose pulse steroid therapy, which could be a potential therapeutic approach for similar patients. In addition, this case raises important questions about the safety of exposing infants and young children to humidifiers and emphasizes the need for further study. *Pediatrics* 2011;127:e000

Lung injury from inhaled irritants in children is being increasingly recognized as a source of pulmonary morbidity attributable to the organ's immaturity and vulnerability.[1] The literature attributes indoor and outdoor pollutants as major culprits.[2] Although humidifiers are popularly used, published literature on humidifier-associated lung injury has focused on bacterial and fungal infectious complications as well as hypersensitivity pneumonitis. There has been no report of adverse health effects in children related to inhalation of mineral "white dust" dispersed by humidifiers designed for home use. Studies by the Environmental Protection Agency have revealed that ultrasonic humidifiers can disperse materials such as microbes and minerals. However, the federal government has not determined that inhalation of such mineral white dusts poses a serious health risk.[3] We report here the case of a 6-month-old infant who was exposed to humidifier white dust and subsequently developed prolonged pulmonary morbidity.

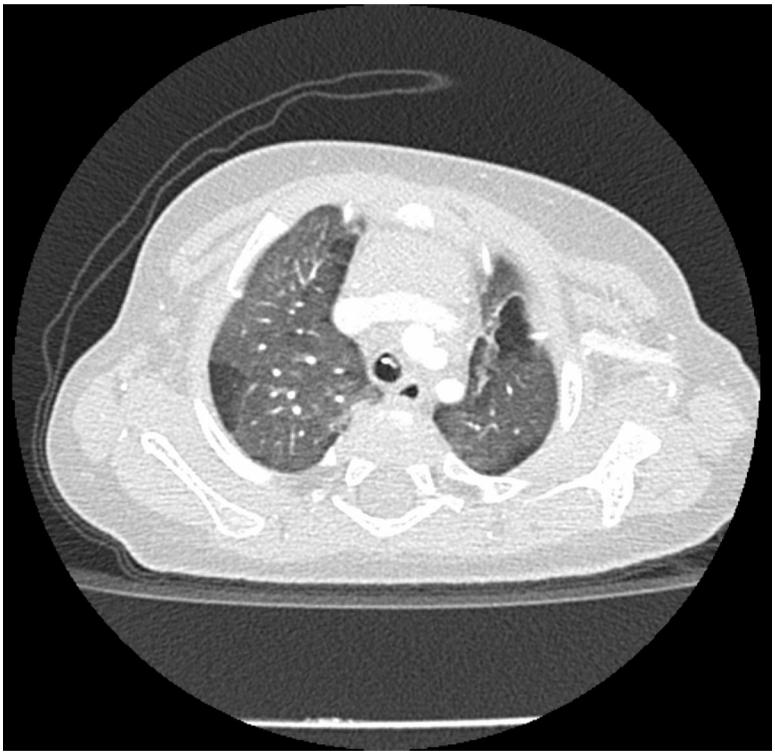

FIGURE 1
Diffuse ground-glass attenuation of the lung parenchyma from inhalational lung injury.

CASE REPORT

A 6-month-old boy born at term with no previous health concerns developed an upper respiratory tract infection. His parents placed a new ultrasonic humidifier within 4 feet of the child's crib to provide humidification and relieve symptoms of nasal congestion. Per manufacturer instructions, distilled water was used. The child's room was a relatively confined space with no open windows. After using the humidifier for ~2 hours, the child was heard coughing, which prompted a physical check. The parents discovered a dense cloud of white dust around the crib that had diffusely settled on all room surfaces. The child did not appear to be in any respiratory distress at the time, apart from having a dry cough. The room was thoroughly cleaned and aired before returning the infant to the room. The following day he was observed to have increased work of breathing, which prompted physician evaluation. An examination revealed tachypnea with a respiratory rate of 60 to 70 per minute and oxygen saturations in the upper-80% range (altitude at evaluation: 4500 ft above sea level). A chest radiograph showed peribronchial thickening with mild hyperinflation. Results of a viral analysis of respiratory secretions were positive for rhinovirus, which prompted a diagnosis of viral bronchiolitis that required supportive care in the hospital. The child remained afebrile, fed well, and was not particularly fussy through the illness. He was discharged from the hospital after a 48-hour stay on oxygen supplementation because of persistent hypoxemia. In view of the persistent tachypnea and hypoxemia for 2 weeks, a repeat chest radiograph was performed; it showed residual mild hyperinflation and peribronchial thickening. The child was treated with bronchodilators and 2 doses of dexamethasone 0.6 mg/kg without improvement. He was referred for pulmonology consultation, because inhalation lung injury was suspected.

He was evaluated with a high-resolution noncontrast chest computed-tomography scan, and the white dust was sent for analysis. The elements detected in the white dust included fluoride, nitrate ion, phosphate ion, sulfate, aluminum, copper, iron, chromium, manganese, nickel, selenium, thalium, titanium, and zinc in small quantities. Relatively larger quantities of calcium, magnesium, sodium, and chloride were identified. The chest computed-tomography scan showed diffuse ground-glass attenuation of the lung parenchyma and linear foci of increased attenuation in the right upper and lower lobes, as well as lingula (Fig 1). His erythrocyte sedi-

mentation rate was 0 mm/hour. Capillary blood gas analysis revealed a pH of 7.42, P_{CO_2} of 35 mm Hg, HCO_3 concentration of 23 mmol/L, and base deficit of 1 mmol/L. Results of a complete blood count and C-reactive protein level test were normal. Results of a repeat respiratory viral direct fluorescent antibody test on nasopharyngeal secretions were negative. Because of the persistent tachypnea and hypoxemia, a flexible bronchoscopy was performed, but it showed no macroscopic bronchitis, and results of bacterial, fungal, and viral cultures were negative. The bronchoalveolar lavage fluid showed 82% alveolar macrophages, 15% bronchial lining cells, 2% neutrophils, and 1% lymphocytes. No ingested particulate material or inclusions were identified within the macrophages by hemosiderin, hematoxylin, and eosin staining. In light of these reassuring results and the absence of literature to support aggressive intervention, the child was clinically monitored for the next 8 weeks. His tachypnea and hypoxemia persisted, and he developed failure to thrive. He was treated with a 7-day course of prednisolone at 2 mg/kg per day without benefit. Azithromycin 3 days/week was also initiated but was not tolerated by the patient. Airway clearance and a trial of inhaled steroids were ineffective. Twelve weeks into the illness, he was referred for infant pulmonary-function testing, which revealed mild nonreversible obstructive ventilatory defect with air-trapping (forced vital capacity: 110% predicted; forced expiratory flow: 0.5 second, 111% predicted; forced expiratory flow: 75%, 77% predicted; forced expiratory flow: 85%, 57% predicted; total lung capacity: 132% predicted; and residual volume/total lung capacity: 143% predicted). At this point, because of persistent symptoms and failure to thrive, he was treated with pulse methylprednisolone 30 mg/kg intravenously on 3 successive days. His tachypnea dramatically improved within days of treatment. A second set of pulse steroids was administered 1 month later, and the hypoxemia and tachypnea resolved. After a third set of pulse steroids, a repeat chest computed-tomography scan was performed, and the results were normal. The child's activity levels and feeding normalized after the above-described treatment, and he demonstrated an 800-g weight gain in the 3 months after commencing pulse steroid therapy.

DISCUSSION

Our case highlights the significant pulmonary disease that can result from inhaled chemicals that are of relatively low concern for adults. Many constituents found in humidifier white dust are reported as respiratory irritants when inhaled.[4,5]

Humidifier white dust can be generated as part of normal functioning of the devices and, therefore, may represent a real concern for inhalational lung injury in infants and young children, yet the true risks have not been well studied and could represent an underappreciated environmental risk for airway reactivity or lung injury in exposed children. The aerosolizing of minerals that can spew from specific humidifiers can generate particle sizes small enough to be inhaled deeply into an infant's developing lung and cause pulmonary injury. To our knowledge, this is the first reported case in an infant that strongly suggests prolonged pulmonary morbidity including pneumonitis and airflow limitation after a significant exposure to white dust. It is possible that rhinovirus may have increased the infant's susceptibility to particulate lung injury, but this is frequently the setting in which humidifiers are used by families. The absence of particulate debris in bronchoalveolar lavage macrophages was not surprising, because frequently, finely aerosolized minerals may not be seen with current cell-staining techniques, and many minerals were present in trace to small quantities.

The role of pulse glucocorticoids in the resolution of symptoms is unclear, but the temporal time line for symptom resolution suggests that they were helpful. Pulse steroids have been advocated for use in children's interstitial lung disease and bronchiolitis obliterans associated with lung transplantation, for which short courses of lower steroid dosing have not been as effective.[6] Because infant pulmonary-function testing demonstrated fixed peripheral airway obstruction, there were concerns that this child could have been evolving bronchiolitis obliterans, which prompted an aggressive management approach. It is fortunate that this treatment regimen seems to have resolved symptoms in our patient; however, it is unclear if there are future risks for asthma in the child.

CONCLUSIONS

Instructions on humidifiers recommend the use of distilled water to avoid white-mineral-dust aerosolization; indeed, our patient did use distilled water as recommended. Therefore, the exact source of the white dust in this case is unclear. Many young families with children may not read the fine print to understand how to avoid white dust; thus, these instructions and warnings are not effective. Furthermore, using humidifiers that could generate white dust in a closed space, in close proximity to an infant, seem to be hazardous and should not occur, especially because the benefits from humidifier use for small children are not well supported. On the basis of this case report, we strongly recommend that further investigation of these devices be pursued and that added warning be given to families about the potential risk of white-dust–induced lung injury in infants and young children.

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