

Yacht-maker's Lung: A Case of Hypersensitivity Pneumonitis in Yacht Manufacturing

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ABSTRACT

We present a case of hypersensitivity pneumonitis in a 46-year-old female working at a yacht manufacturing company. She reported a 2-month history of progressive dyspnea, chest tightness, and daytime, nocturnal, and exertional cough in temporal relationship to work where she was exposed to chemicals involved in the manufacture of yachts. Treatment with systemic antibiotic therapy, inhaled bronchodilators, and inhaled corticosteroids provided minimal relief of symptoms. Spirometry revealed a restrictive defect and a chest x-ray demonstrated a diffuse interstitial pattern. She improved on oral corticosteroids and with avoidance of her work environment had resolution of her symptoms and normalization of her spirometry. Among the various chemicals the patient was exposed to, the most likely causative agents for her symptoms were dimethyl phthalate and styrene. Although the specific chemical or antigen could not be determined, the history and objective findings are consistent with occupational hypersensitivity pneumonitis. This represents a case of hypersensitivity pneumonitis related to the manufacture of yachts.

INTRODUCTION

Hypersensitivity pneumonitis (HP), also referred to as extrinsic allergic alveolitis, is a clinical syndrome of variable respiratory signs and symptoms with or without systemic features resulting from an immune response to inhaled organic dust antigens. The immune response is non-IgE mediated with both humoral and cell-mediated processes and contributing host, antigen, and environmental factors.¹ Sensitization may take months to

years and, unfortunately, the exact mechanisms in the development of disease have not been fully elucidated although both humoral and cell mediated mechanisms are demonstrated. The numerous etiologic agents of HP are derived from a variety of sources including microbes; animal, plant and insect products; amoebae; chemicals; and medications.¹ While many exposures occur in an occupational setting, HP has not been described in individuals working in yacht manufacturing.

The majority of modern boats manufactured today are constructed with fiberglass-reinforced plastics in a multi-step, chemical intensive process.² We report the case of a worker who developed hypersensitivity pneumonitis from fume exposures while working in yacht manufacturing.

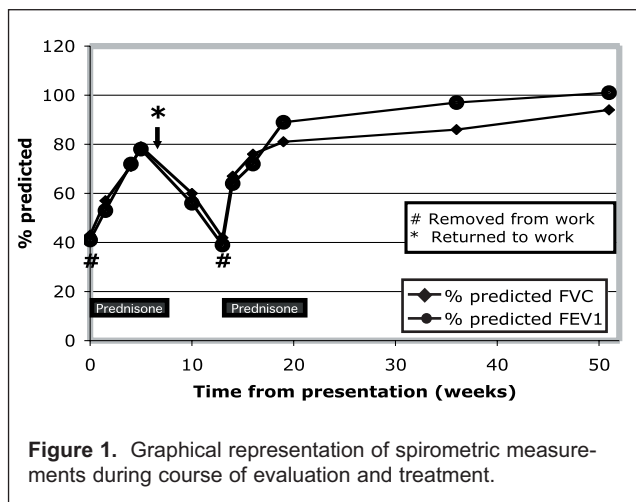
CASE REPORT

The patient is a 46-year-old Hmong female with a 2-month history of progressive dyspnea, chest tightness, and non-productive cough without wheezing. The cough awakened her nightly, occurred during the daytime, and worsened with exertion. She denied previous lower respiratory symptoms. A chest x-ray showed diffuse mild interstitial prominence. Treatment with antibiotics, nebulized levalbuterol, ipratropium bromide, and inhaled fluticasone with salmeterol had minimal effect on her symptoms.

Past medical history was unremarkable, and she reported no other medications. The family history was negative for asthma, atopy, tuberculosis, and other lung diseases. The social history revealed that she was born in Laos and lived in a Thai refugee camp for 4 years. She is a non-smoker but was exposed to her husband's second hand tobacco smoke until 1 month prior to presentation.

Environmental history revealed that she worked full-time at a yacht manufacturing company for the past 5 years rolling fiberglass. Specifically, she rolled out wet sheets of fiberglass impregnated with resin that

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were then placed into a mold for curing. The workplace was maintained at a constant temperature of 72° F to facilitate curing. There were no windows and the 2 doors were kept closed. Ventilation ducts are present but were noted to extrude hot air. She wore a full body suit with respiratory protection, initially a simple mask, then a properly fitted mask with a chemical filter canister. The air quality was frequently assessed and had been below current National Institute for Occupational Safety and Health and Occupational Safety and Health Administration guidelines for styrene, dimethyl phthalate, and airborne particulate matter. Her home environment was unremarkable. There were no pets (including birds) or mold incursions.

Review of systems was negative for weight loss, fatigue, fever, chills, night sweats, rash, headache, and rhinitis but she reported throat itching, sore throat with coughing, and finger paresthesias while working.

Physical examination revealed a blood pressure of 90/68, heart rate of 68 beats per minute, respiratory rate of 16 breaths per minute, and oxygen saturation of 97% on room air. Pertinent findings included her coughing with deep inspiration, an erythematous papular rash on the posterior neck, and dry, erythematous excoriated skin on the dorsum of her hands. Her trachea was midline and her breath sounds were normal. Her cardiac exam was normal. There was no digital clubbing or cyanosis, and jugular venous distention was not evident.

Initial spirometry demonstrated a significant restrictive defect with forced expiratory volume in the first second (FEV1) of 41% predicted and forced vital capacity (FVC) of 43% predicted (Figure 1). IgE radioallergosorbent testing to isocyanates and PPD skin testing for tuberculosis were negative. She was treated with prednisone 20 mg twice daily and removed from work.

After 4 weeks, her symptoms resolved and her spirometry improved. However, pulmonary function testing revealed lung volumes consistent with a mild restrictive ventilatory defect (Figure 1). Repeat chest x-ray was normal.

Due to the patient's eagerness to resume working, she returned to her work environment with the knowledge that her symptoms could recur. Her prednisone was tapered over 9 days. Three weeks after returning to work, she began coughing, and her spirometric measurements again declined (Figure 1). A repeat chest x-ray revealed small nodular densities consistent with granulomas. A complete blood count revealed 63% neutrophils, 28% lymphocytes, 8% monocytes, and 1% eosinophils by automated differential. Erythrocyte sedimentation rate (ESR) was elevated at 71 mm/hr. Her symptoms progressed to exertional dyspnea and nocturnal cough with continued work exposure. Her spirometry decreased to levels seen at presentation (Figure 1) and her diffusion capacity and lung volumes were consistent with a restrictive ventilatory defect. A chest computed tomography (CT) demonstrated nodular densities. A repeat complete blood count showed normal white blood cells, hemoglobin, hematocrit, and platelet count with an automated differential of 77% neutrophils, 16% lymphocytes, and 7% monocytes. A subsequent ESR was elevated at 48 mm/hr and lactate dehydrogenase (LDH) was 613 IU/L (normal 297-537). Retreatment with prednisone and avoidance of the work place again resulted in clinical improvement and improved spirometry. Exercise challenge was normal 9.5 months after removal from work.

Review of the material safety data sheets from her worksite identified 2 possible offending chemicals: dimethyl phthalate and styrene. Assay for serum precipitins was performed as previously described.³ Briefly, each chemical was mixed with an equal amount of distilled water and extracted by gentle shaking for 48 hours at 4°C. Each mixture was centrifuged at 3000 rpm for 10 minutes. The supernatant was extracted and tested against the patient's serum by agar gel diffusion. Serum precipitins to these chemicals could not be demonstrated.

DISCUSSION

Boat manufacturing is an ancient industry with occupational hazards including lead toxicity from paint, abrasions, acute and chronic musculoskeletal injuries, noise injury, burns, visual changes, and injury from fires and explosions.² Occupational diseases as a result of boat manufacturing include dermatitis, asbestos-related dis-

ease, neuropathy, encephalopathy, upper airway irritation, pulmonary edema, and toxic hepatitis.² In 2002, over 49,000 people were employed in over 1000 boat manufacturing companies in the United States.⁴ The current case represents occupational HP in this industry, and we coined it yacht-maker's lung.

Current boat manufacturing processes are dominated by the use of fiberglass-reinforced plastics. Chemicals used in this process include unsaturated polyester resins, epoxy resins, catalysts, curing agents, fillers, pigments, paints, lacquers, accelerators, inhibitors, and mold-release agents.⁵ Basic boat building using fiberglass-reinforced plastics entails spray coating a prepared wooden mold with a polyester resin. Then lamination follows, where layers of catalyzed resin and fiberglass are applied to the mold either manually or mechanically. Once these layers are dried, the mold is removed and the structure is sanded, painted, and attachments added to complete the boat structure.²

During her work rolling fiberglass, our patient was exposed to a number of chemicals. Of these, the 2 most likely causative agents for her occupational hypersensitivity pneumonitis were styrene and dimethyl phthalate. In boat manufacturing, styrene is a cross-linking agent and diluent of unsaturated polyester resins.⁶ It is well established that workers in boat manufacturing have the highest exposure levels to styrene.⁷ Of styrene used in this setting, 10%-15% can evaporate into the work environment.² Styrene irritates the mucous membranes and respiratory tract and is hepato- and neurotoxic.⁶ The patient's digital paresthesias may have been due to repetitive hand motions or neurotoxicity. Styrene exposure can induce wheezing, dyspnea, and chest tightness.⁸ However, a Medline search reveals no prior scientific literature confirming styrene as a causative agent of HP.

Dimethyl phthalate belongs to a family of immunologic sensitizers termed acid anhydrides that are used as curing or hardening agents for epoxy resins and in the production of plasticizers, polyester resins, and alkyd resins.⁶ Its role as a curing agent is the main use in boat manufacturing. Acid anhydride exposure can result in irritation such as skin burns, corneal ulcers, epistaxis, and hemoptysis. Furthermore, hypersensitivity to acid anhydrides can present with rhinitis, conjunctivitis, and urticaria.⁶ Inhalation of acid anhydrides can also cause direct irritation to the respiratory system, occupational asthma, late respiratory distress syndrome (characterized by myalgias, malaise, fever, chills, arthralgias, cough, wheezing, and dyspnea), and pulmonary disease anemia syndrome (characterized by dyspnea, hemopty-

Table 1. Signs and Symptoms of Occupational Hypersensitivity Pneumonitis in the Case Patient

Signs	Symptoms
<ul style="list-style-type: none"> • Lack of response to antibiotics, inhaled corticosteroids, and bronchodilators • Elevated erythrocyte sedimentation rate • Elevated lactate dehydrogenase • No detectable IgE to isocyanates • Pulmonary function: restrictive defect • Chest x-ray: mild interstitial prominence, nodular densities • Computed tomography of the chest: nodular densities 	<ul style="list-style-type: none"> • Progressive dyspnea • Chest tightness • Daytime, nocturnal, and exertional cough • Temporal relationship of symptoms to the work environment

sis, pulmonary infiltrates, restrictive lung disease, and hemolytic anemia).^{6,9,10} Acid anhydrides are reported to cause HP.¹ However, they are rarely conclusively investigated in the medical literature. Two cases of alveolitis have been linked to exposure to epoxy resin polyester paints containing phthalates.^{11,12}

No single historical symptom, physical examination finding, diagnostic laboratory value, or radiologic finding is diagnostic for HP. Rather the constellation of these features supports the diagnosis of HP. For the case patient, the features that are consistent with HP include progressive respiratory symptoms, interstitial infiltrates on chest x-ray, granulomas on chest CT, lack of response to antibiotics, inhaled corticosteroids, and bronchodilators, a restrictive pattern on pulmonary function testing and elevated ESR and LDH (Table 1).¹ Her occupational exposure history and temporal relationship to work further support the diagnosis of subacute HP.

Subacute HP is characterized by progressive respiratory symptoms over weeks to months but without acute systemic symptoms of fever, chills, myalgias, and malaise. Bibasilar rales may be heard or the exam may be entirely normal. In contrast, acute HP often mimics an acute respiratory illness with cough, dyspnea, fever, chills, myalgias, and malaise. In chronic HP, progressive dyspnea, cough, malaise, weakness, anorexia, and weight loss are seen. Fine dry crackles may be heard on exam.¹ While the "gold" standard for confirming an antigen for HP is intentional inhalational challenge of the antigen in a hospital setting, it was deemed that the risks were too great in this patient.

The main treatment modality of HP is avoidance of the inciting antigen. Systemic corticosteroids may decrease acute symptoms but do not change the long-term outcome.¹ Improved ventilation and/or removal from the suspect environment can decrease exposure. With complete avoidance, most patients with HP become asymptomatic and have complete recovery.

The patient has not returned to work, her lung function has normalized, and her symptoms have resolved. Unfortunately, her inability to work under these conditions has become a significant financial burden as she requires retraining.

CONCLUSIONS

This is a case of occupational HP in the setting of yacht manufacturing. Although in vitro testing could not identify the exact causative agent, the symptoms, objective clinical findings, and temporal relationship to the work environment supports the diagnosis of HP. Likewise, agents known to cause HP were found among the chemicals to which the patient had known exposure. A diagnosis of HP should be considered in patients with intermittent respiratory complaints with or without systemic symptoms or progressive unexplained pulmonary symptoms and interstitial lung disease.

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