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Profound chemical pneumonitis sparked off by common household cleaning products: An uncanny and perplexing respiratory repercussion

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Abstract

Our clinical case represents a classic case of acute hypersensitivity pneumonitis which developed secondary to short term exposure to cleaning products. Although not very common, it transpires in the individuals who develop heightened immune response to chemicals used in common household cleaning products. Patients might be asymptomatic, although some of them might present with shortness of breath, chest pain, cough or acute respiratory failure. The pathophysiology is centered around macrophage induced activation of B&T lymphocyte, with subsequent myofibroblast differentiation, and collagen deposition in the extracellular matrix. High resolution CT scan might reveal ground glass opacities, centrilobular nodules with or without honey combing, and fibrosis. Treatment options include avoidance of irritants, immunosuppression and anti-fibrotic agents. Pertinent clinical history regarding occupation and identification of temporal relationship between irritants and lung pathology is critical in early identification of clinical syndrome, so that effective management can be tailored for preventing fibrotic hypersensitivity pneumonitis.

Keywords: hypersensitivity pneumonitis, pathophysiology, COVID-19, influenza.

Clinical Case

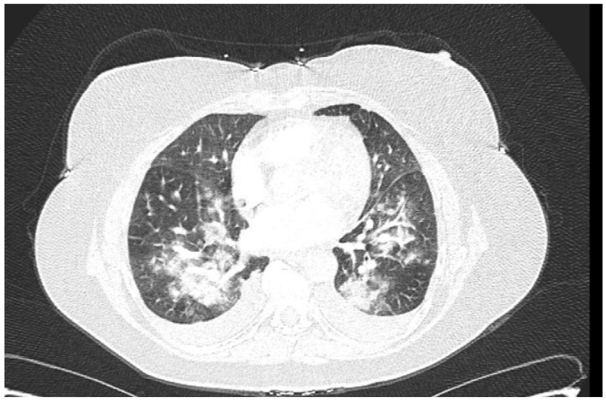
The patient is a 66-year-old female with no known significant past medical history who presented with a one-week history of worsening shortness of breath and chest pain. She was found to have acute hypoxic respiratory failure, likely secondary to chemical pneumonitis related to occupational exposure from cleaning products. Chest X-ray on 09/28/2026 demonstrated multifocal patchy airspace opacities, greater on the right, and CTA chest showed bilateral small pleural effusions and patchy bilateral pulmonary ground glass opacities with zonal predilection (**Figures 1-4**). COVID-19, influenza, and RSV testing was negative. She was treated with supplemental oxygen and empiric ceftriaxone and azithromycin, with oxygen requirement improving on room air.

Hospital course was also complicated by elevated troponins consistent with type 2 myocardial infarction, felt to be secondary to acute pulmonary illness. Troponin peaked at 1,016 and subsequently downtrended, with BNP 624 and ECG showing subtle V1-V2 ST changes. Echocardiogram demonstrated preserved LVEF of 55-60% with moderate mitral and tricuspid regurgitation. She was treated with aspirin, high-intensity atorvastatin, and a heparin infusion, with cardiology following. Ischemic evaluation was planned outpatient once the acute pulmonary process resolved. She was discharged with instructions to take all home medications as prescribed which includes aspirin and atorvastatin and follow up with her primary care provider and cardiologist in 1-2 weeks after discharge.

Discussion

Our patient presented with classic symptoms of shortness of breath and chest pain and subsequently developed acute hypoxic respiratory failure following exposure household cleaning products. Our clinical case represents a classic clinical case of acute hypersensitivity pneumonitis following exposure to household chemical products. Although household products are safe to use, some patients develop respiratory complications particularly in those with pre-existing atopy and hypersensitivity to environmental allergens or toxins

There are few reports of patients developing asthma, chronic bronchitis, bronchial hyperresponsiveness, acute respiratory distress syndrome, and reactive airway dysfunction syndrome (RADS) following short term exposure to household cleaning products [1-4]. Furthermore, additional pulmonary abnormalities that can materialize following exposure to chemicals include bronchitis, bronchiolitis, chemical pneumonitis, pulmonary edema, acute respiratory distress syndrome, organizing pneumonia, hypersensitivity pneumonitis, acute eosinophilic pneumonia, and sarcoid-like granulomatous lung disease [5].



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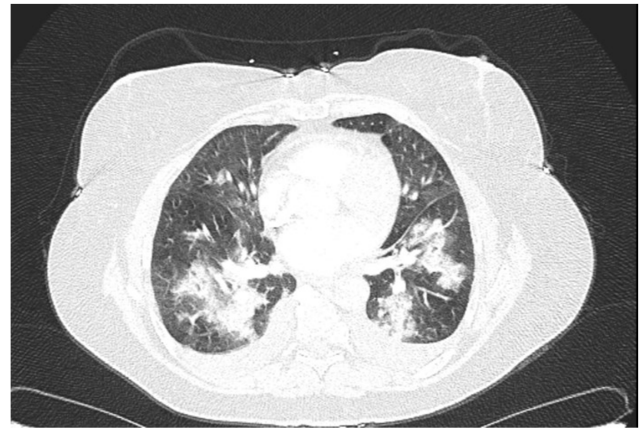

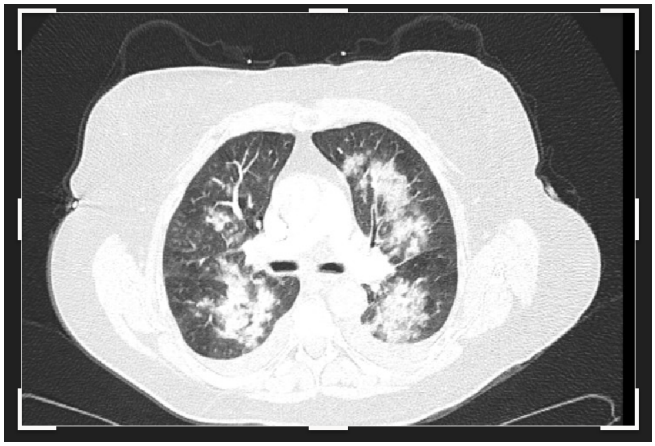

Figures 1-4.

Our clinical case represents a classic clinical case of hypersensitivity pneumonitis (HP). Its reported prevalence is estimated to be 11.5 cases per 100,000 population[6]. It is found to be more common in farmers, bird breeders and mushroom workers [6]. Approximately 12-28% of HP are known to be associated with occupational exposure to environmental irritants [7]. Inhaled irritants after reaching smaller airways including smaller bronchi and alveoli interact with macrophages to induce Th1 lymphocytes and B-lymphocytes [6]. This leads to production of chemokines, cytokines, IgG antibodies as well as activation of complement cascade [6]. Subsequently, macrophages fuse with multinucleated giant cells to form granulomas which produce chemotactic factors [6]. In the final step, these chemotactic factors in combination with CD8+ T-lymphocytes, and Th17 lymphocytes triggers fibroblast proliferation, and myofibroblast differentiation, thus forming collagen and extracellular matrix [6]. The pathology of hypersensitivity pneumonitis is mainly classified in non-fibrotic and fibrotic HP. In non-fibrotic HP, findings include non-specific interstitial pneumonia (NSIP) with cellular infiltration with predominant lymphocytes, cellular bronchiolitis, peri-bronchiolar lymphocyte inflammation, poorly formed non-necrotizing granulomas [8]. In fibrotic HP, changes include Chronic fibrosing interstitial pneumonia, airway-centered fibrosis with Peribronchiolar metaplasia and poorly formed non-necrotizing granulomas [8].

The diagnosis of hypersensitivity pneumonitis can be made by combination of clinical history, high resolution CT scan, bronchoalveolar lavage and lung biopsy[8]. CT scan in acute chemical pneumonitis can reveal diffuse ground glass opacities with no zonal predilection, irregular nodular abnormalities and centrilobular nodules [9,10]. In the contrary patient with fibrotic HP, CT scan might reveal bronchiectasis and honey combing [8]. Bronchoscopy and bronchoalveolar lavage for assessing lymphocyte counts, markers such as KL-6, SP-D and CXCL-17 has been previously used for gauging prognosis [6,11-13]. Treatment options in HP includes avoidance of irritants, immunosuppression with corticosteroids, mycophenolate mophetil, azathioprine and rituximab and antifibrotic agents (Ninetanib)[14-18].. These agents work through depleting proinflammatory cytokines, inhibiting B & T lymphocytes as well as inhibiting multiple growth factor receptors [14-18]. In addition to these therapies, pulmonary rehabilitation, oxygen use, and lung transplantation might be used in case-by-case basis [19-21]. Some of the prognostic factors for poor prognosis can include older age, males, reduced pulmonary function and pulmonary hypertension [6,22,23].

Conclusion

Identification of chemical pneumonitis is important in patients presenting with shortness of breath or other respiratory symptoms in patient exposed to chemical irritants. CT scan lung might reveal non-fibrotic vs non-fibrotic HP which be useful in tailoring therapies. Bronchoalveolar lavage can be performed for assessing biomarkers that can be helpful in assessing the prognosis. Appropriate education for further avoidance of chemical allergens is very crucial in preventing further episodes of lung injury as well to prevent fibrotic type of HP.

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