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Pneumonitis Associated with Fluoropolymer Waterproofing Agents: Case Report

Pneumonite Associada a Impermeabilizantes com Fluoropolímeros: Caso Clínico

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ABSTRACT

Pneumonitis associated with fluoropolymer waterproofing agents, an entity with few reported cases, can result from occupational exposure. This condition has a rapid onset after exposure, usually resolves with supportive treatment but there could be chronic sequelae. The authors report the case of a 48-year-old male patient admitted to hospital with acute onset of dyspnea and chest pain after using an aerosolized fluoropolymer-containing waterproofing product. He presented tachypnea, leukocytosis, elevated C reactive protein, elevated serum lactate dehydrogenase and hypoxemic respiratory failure. Chest computed tomography revealed bilateral ground-glass opacities with peribronchovascular distribution. The patient was treated with oxygen and corticosteroid therapy, with clinical improvement. This chemical pneumonitis represents a diagnostic challenge since it implies a history of exposure to toxic agents and the pathophysiological mechanisms and safe exposure limits are still unknown.
Keywords: Fluorocarbon Polymers/poisoning; Inhalation Exposure/adverse effects; Occupational Diseases/chemically induced; Pneumonia/chemically induced

RESUMO

A pneumonite associada a impermeabilizantes com fluoropolímeros é uma entidade com poucos casos relatados e que pode resultar duma exposição ocupacional. Esta condição tem um início rápido após a exposição, que geralmente se resolve com tratamento de suporte, podendo resultar em sequelas crónicas. Os autores relatam o caso de um homem de 48 anos admitido no hospital com quadro agudo de dispneia e dor torácica após uso de impermeabilizante que continha fluoropolímeros em aerossol. Apresentava taquipneia, leucocitose, proteína C reativa elevada, níveis séricos de lactato desidrogenase elevados e insuficiência respiratória hipoxémica. A tomografia computadorizada do tórax revelou opacidades em vidro despolido bilaterais com distribuição peribroncovascular. O doente foi tratado com oxigenoterapia e corticoterapia com melhoria clínica. Esta pneumonite química representa um diagnóstico desafiante já que implica uma história de exposição a tóxicos, sendo que a fisiopatologia e os limites de segurança de exposição ainda são desconhecidos.
Palavras-chave: Doenças Ocupacionais/induzidas quimicamente; Exposição por Inalação/efeitos adversos; Pneumonia/induzida quimicamente; Polímeros de Fluorcarboneto/intoxicação

INTRODUCTION

Chemical pneumonitis can result from occupational exposure, either accidentally or by disregarding safety rules. Fluoropolymer-containing waterproofing agents are generally composed of a solvent, a propellant and a water repelling fluoropolymer,[1] i.e., a molecule consisting of carbon and fluorine that is an ultrafine and respirable particle. Fluoropolymers have been associated with outbreaks of respiratory diseases[2] and their mechanisms depend on the methods of application, as well as on personal and environmental factors.[1] Toxicity may also result from the solvent when it vaporizes and is inhaled and the hydrophobic agent remains on the surface.

Respiratory symptoms, leukocytosis, hypoxemia and radiological changes caused by this lung injury usually start minutes to a few hours after exposure.[1,3,4] Most cases have complete resolution in days under supportive treatment and supplemental oxygen.[3,4] However, there are reports of persistent symptoms and progression to fibrosis.[1,5]

CASE REPORT

We report the case of a 48-year-old male, light smoker (less than one pack/day) with no relevant previous medical history, that complained of progressively worsening dyspnea and chest pain which evolved over four hours, after using an aerosolized waterproofing product. He was a stone restoration technician with previous similar contacts with other formulations.

He applied a fluoropolymer-containing waterproofing product, *HS O,* a colorless volatile liquid. According to the manufacturer, the composition was 94% solvent with C9-C11 paraffinic hydrocarbon, 0.45% solution of 3-iodine-2-propnyl-butylcarbamate and 5.55% fluoroacrylate polymer with ethyl acetate. The product was applied with a trigger spray in an occupational setting with inadequate ventilation and inappropriate personal protective equipment. Based on technical information obtained from the manufacturer,[6] the product was developed to be applied by brush or roller, in order to avoid prolonged direct contact with the skin and inhalation and not to be used at temperatures above 35º C.

In the Emergency Department the patient presented tachypnea, leukocytosis (16.8 x 10^9/L), elevated C reactive protein (7.5 mg/dL) and elevated serum LDH (275 U/L).

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There was no fever, no wheezing and the cardiovascular examination was normal. The arterial blood gases analysis showed hypoxemic respiratory failure (pH 7.45, pO2 46 mmHg, pCO2 32 mmHg, oxygen saturation 85.6%). A chest computed tomography (CT) revealed centrilobular emphysema and bilateral ground-glass opacities with predominant peribronchovascular distribution in the upper lobes and associated with centrilobular micronodules (Fig. 1).

After hospital admission, oxygen therapy and prednisolone (1 mg/kg/day) were prescribed. The patient underwent a bronchoscopy with bronchoalveolar lavage (BAL) of the middle lobe and microbiological and cytological (immunocytology) investigations were unremarkable. The cellular analyses of BAL fluid revealed a macrophage dominant cell pattern (277 cells/mm^3 with 3% lymphocytes, 2% neutrophils, 87% macrophages and CD4/CD8 ratio of 2.7). Transbronchial lung biopsies of the right lower lobe were indeterminate (normal respiratory epithelium).

Blood serological markers for autoimmunity, immunoglobulins, angiotensin converting enzyme levels and precipitins for birds and fungi were performed with normal results.

Eight days after admission, respiratory function tests revealed volumes, flow rates, resistance and carbon dioxide lung diffusion within normal parameters [FEV1 3.84 l (109% predicted), FVC 4.60 l (106%), FEV1/FVC 83%, TLC 6.61 l (99%), RV 1.95 l (94%), DLCO 9.3 mmol/min/kPa (94%)].

Both clinical and radiological evolution were excellent. The patient was discharged on the tenth day, with no residual symptoms and maintained corticosteroid therapy and smoking cessation at home.

The patient received follow-up in the Pulmonology department and smoking cessation out-patient clinic, remained asymptomatic. At one month, the ground-glass opacities and micronodules had resolved completely.

DISCUSSION

These findings were consistent with the diagnosis of fluorocarbon aerosol pneumonitis supported by the onset of respiratory symptoms. The hydrocarbon aspiration pneumonitis was ruled out by the pulmonary manifestations instead of known central nervous system or cardiac effects.

Acute lung injury after inhalation exposure to fluoropolymer has several manifestations. This case, induced by aerosol exposure, illustrates an entity with few cases reported in outbreaks. It differs from the known polymer fume fever in the less often reported systemic symptoms, the BAL cellular pattern and it does not involve heating the polymer.

In order to identify this pneumonitis, it is important to obtain a history of exposure to toxic agents, their formulation, as well as personal and environmental factors. Even though this patient had previous similar contacts with other formulations, he did not have the experience of using it with a trigger spray, nor did he have the proper professional training or the personal protective equipment to use.

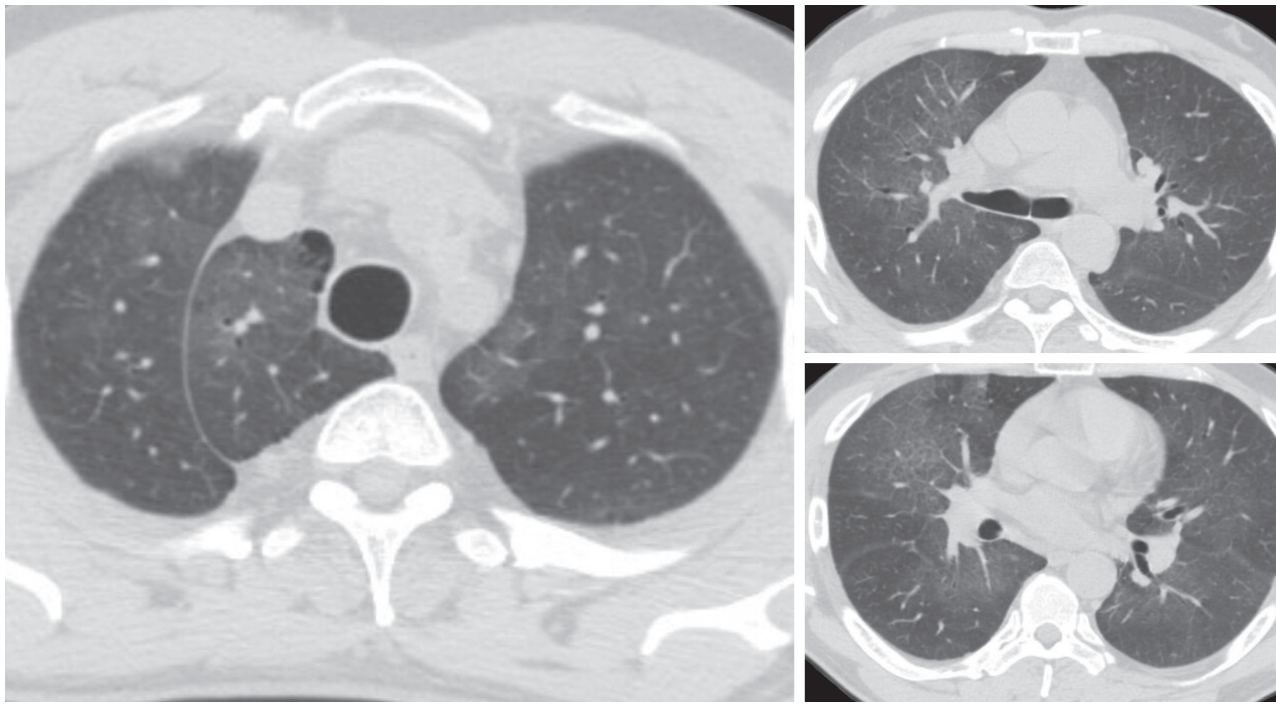

Figure 1 – Chest CT at the Emergency Department showing bilateral ground glass opacification with peribronchovascular distribution predominant in the upper lobe

National poison centers (like the Centro de Informação Antivenenos - CIAV) play a crucial role in the characterizationof the exposure and detection of these outbreaks. The pathophysiological mechanisms and safe exposure limits are unknown.

Workplace education should stimulate the use of these products according to the indications from the manufacturer and promote awareness of the potential hazards of fluorocarbons in order to prevent future toxic exposures.

AUTHOR CONTRIBUTIONS

JAB: Draft of the manuscript.

FF, AJF: Critical review of the manuscript.

PROTECTION OF HUMANS AND ANIMALS

The authors declare that the procedures were followed according to the regulations established by the Clinical Research and Ethics Committee and to the Helsinki Declaration of the World Medical Association published in 2013.

DATA CONFIDENTIALITY

The authors declare having followed the protocols in use at their working center regarding patients' data publication.

PATIENT CONSENT

Obtained.

COMPETING INTERESTS

All authors report no competing interests.

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REFERENCES

1. Hays HL, Spiller H. Fluoropolymer-associated illness. Clin Toxicol. 2014;52:848-55.
2. Henke D, Campbell A, Bradberry SM, Sandilands EA, Thomas SH, Thompson JP, et al. Toxicity from fluoropolymer-containing grout, tile and stone floor sealants reported to the UK National Poisons Information Service 2009–2015. Clin Toxicol. 2017;55:585-8.
3. Laliberté M, Sanfaçon G, Blais R. Acute pulmonary toxicity linked to use of a leather protector. Ann Emerg Med. 1995;25:841-4.
4. Blanc PD. Acute responses to toxic exposures. In: Broaddus VC, Mason RJ, Ernst JD, King TE, Lazarus SC, Murray JF, et al, editors. Murray and Nadel's textbook of respiratory medicine. Philadelphia: Elsevier; 2016. p.1343-53.
5. Wallace GM, Brown PH. Horse rug lung: toxic pneumonitis due to fluorocarbon inhalation. Occup Environ Med. 2005;62:414-6.
6. HSO, Hidrófugo e oleófugo de elevada qualidade. Marinha Grande: Labo Portugal; 2010.