

Hypersensitivity Pneumonitis Associated with Exposure to Quaternary Ammonium Compounds in Detergent Products: A Three-Case Series with Divergent Outcomes

Mehdi Maaroufi^{1*}, Imane El Hamdani¹, Hanaa Harraz¹, Khadija Chaanoun¹, Wafaa Jalloul¹, Zakaria Laklaai¹, Hanane Benjelloun¹, Nahid Zaghba¹

¹Department of Respiratory Diseases, Ibn Rochd University Hospital, Casablanca, Morocco

DOI: <https://doi.org/10.36347/sjmcr.2026.v14i07.025>

| Received: 01.06.2026 | Accepted: 23.07.2026 | Published: 25.07.2026

*Corresponding author: Mehdi Maaroufi

Department of Respiratory Diseases, Ibn Rochd University Hospital, Casablanca, Morocco

Abstract

Case Report

Background: Hypersensitivity pneumonitis is an immune-mediated interstitial lung disease caused by repeated inhalation of inciting antigens. Quaternary ammonium compounds are widely used in detergents and disinfectants and may cause respiratory toxicity through irritant, sensitizing, or adjuvant mechanisms. Their role in parenchymal lung disease remains insufficiently recognized. **Case presentation:** We report three women with hypersensitivity pneumonitis probably associated with repeated exposure to quaternary ammonium compound-containing detergent products, after exclusion of other identifiable inciting antigens. The first patient, a 63-year-old professional cleaner, developed a typical nonfibrotic pattern after two months of intense exposure. High-resolution computed tomography showed mosaic attenuation, expiratory air trapping, and centrilobular micronodules, and bronchoalveolar lavage fluid contained 30% lymphocytes. Strict exposure avoidance and oral corticosteroids were followed by favorable clinical, functional, and radiological outcomes. The second patient, a 60-year-old homemaker, developed typical fibrotic hypersensitivity pneumonitis after three years of exposure. She showed clinical and functional improvement after strict exposure avoidance, corticosteroids, and azathioprine, while residual fibrosis remained stable on computed tomography. The third patient, a 66-year-old professional cleaner with prolonged exposure, had typical fibrotic hypersensitivity pneumonitis. Despite repeated education and counseling regarding strict exposure avoidance, she continued to be exposed to the implicated detergent products. Her symptoms improved under corticosteroids and azathioprine, but lung function and imaging findings worsened; antifibrotic therapy was subsequently approved after multidisciplinary discussion. **Conclusion:** These cases illustrate a spectrum of detergent-associated hypersensitivity pneumonitis ranging from reversible nonfibrotic disease to progressive fibrotic disease. Detailed occupational and domestic exposure assessment, effective and sustained exposure avoidance, and multidisciplinary management are essential and may help limit irreversible fibrotic progression.

Keywords: Hypersensitivity pneumonitis; quaternary ammonium compounds; detergents; cleaning products; fibrotic hypersensitivity pneumonitis; progressive pulmonary fibrosis; antifibrotic therapy.

Copyright © 2026 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

INTRODUCTION

Hypersensitivity pneumonitis (HP), historically termed extrinsic allergic alveolitis, is an immune-mediated bronchioloalveolar and interstitial lung disease resulting from repeated inhalation of a sensitizing environmental, domestic, or occupational antigen in susceptible individuals. The diagnostic approach has evolved from the older acute, subacute, and chronic terminology toward a more clinically relevant distinction between nonfibrotic and fibrotic HP, because the

presence of fibrosis strongly influences prognosis and management [1,2].

The diagnosis relies on an integrated assessment of exposure history, clinical presentation, high-resolution computed tomography (HRCT), bronchoalveolar lavage (BAL), and, when needed, histopathology. Identification of the inciting antigen remains central, but it is often difficult because exposures may be multiple, intermittent, poorly labeled, or underreported. Detailed occupational and domestic

Citation: Mehdi Maaroufi, Imane El Hamdani, Hanaa Harraz, Khadija Chaanoun, Wafaa Jalloul, Zakaria Laklaai, Hanane Benjelloun, Nahid Zaghba. Hypersensitivity Pneumonitis Associated with Exposure to Quaternary Ammonium Compounds in Detergent Products: A Three-Case Series with Divergent Outcomes. Sch J Med Case Rep, 2026 Jul 14(7): 1718-1723.

exposure assessment is therefore a core component of the diagnostic work-up [3].

Quaternary ammonium compounds (QACs), also called quats, are cationic surfactants widely used for their detergent, disinfectant, antiseptic, and preservative properties. They are present in many household and professional cleaning products, wipes, sprays, disinfectants, cosmetics, and healthcare products [4]. The respiratory effects of QACs are best documented in occupational asthma and immediate or delayed hypersensitivity reactions, particularly among professional cleaners and healthcare workers [4,5]. Experimental studies have also shown that didecyldimethylammonium chloride (DDAC), a representative QAC, can induce pulmonary inflammation and fibrosis in animal models, supporting the biological plausibility of parenchymal lung injury after inhalational exposure [6,7].

However, HP associated with QAC-containing detergent products is likely underrecognized in clinical practice. We report three cases of HP probably associated with repeated exposure to detergent products containing QACs, with three divergent outcomes: favorable nonfibrotic disease, fibrotic disease with radiological stability, and progressive fibrotic disease requiring consideration of antifibrotic therapy.

METHODS

This retrospective descriptive case series was conducted in the Department of Respiratory Diseases, Ibn Rochd University Hospital, Casablanca, Morocco. We included three patients in whom the diagnosis of HP was established after multidisciplinary discussion. All patients had repeated exposure to detergent products containing QACs, compatible clinical and HRCT findings, BAL analysis, and no alternative inciting antigen identified after clinical and ancillary evaluation.

Radiological patterns were classified according to current HP terminology as typical nonfibrotic or typical fibrotic HP. Therapeutic decisions were individualized and discussed in a multidisciplinary setting.

CASE PRESENTATIONS

Case 1: nonfibrotic HP with a favorable outcome

A 63-year-old woman working as a professional cleaner had no relevant medical history. She reported intense and repeated exposure to detergent products containing QACs over a two-month period. Following exposure, she developed wheezing, respiratory discomfort, and dry cough. Physical examination revealed bilateral crackles.

HRCT showed mosaic attenuation with expiratory air trapping and centrilobular micronodules, indicating bronchiolar involvement (Figure 1).

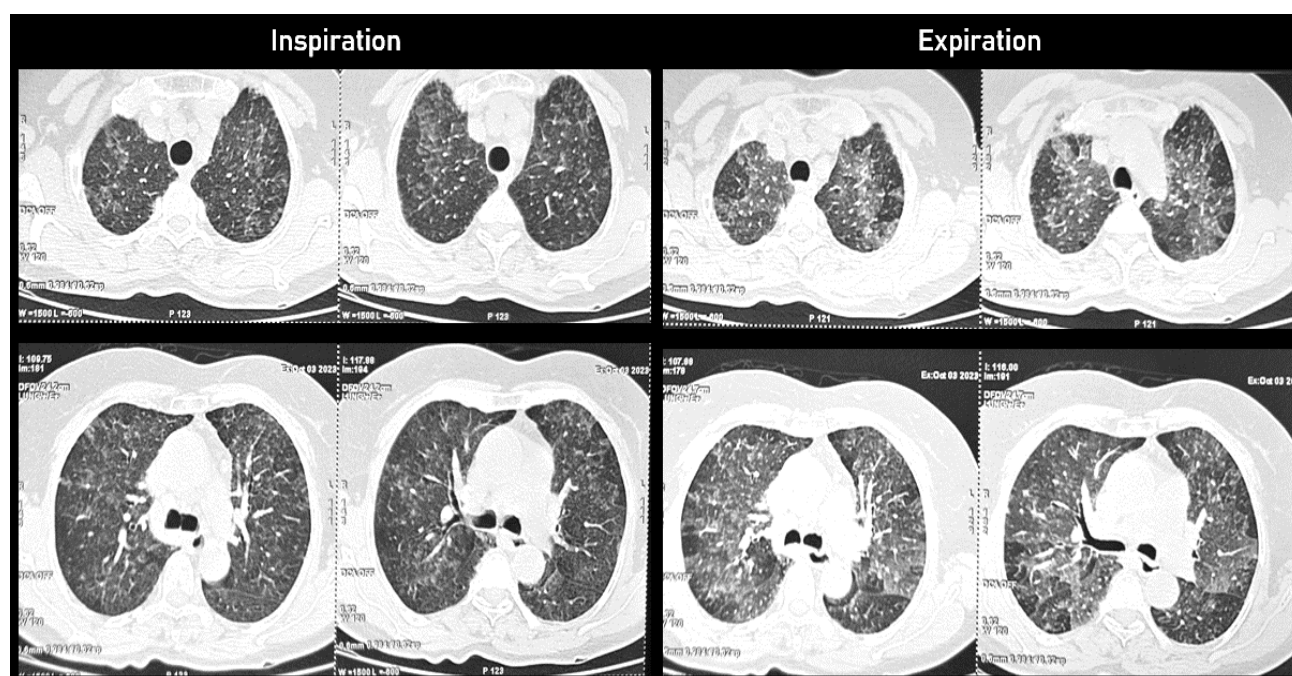


Figure 1: High-resolution computed tomography of Case 1. Inspiratory and expiratory acquisitions show mosaic attenuation, expiratory air trapping, and centrilobular micronodules, consistent with typical nonfibrotic hypersensitivity pneumonitis

BAL fluid differential cell count showed 30% lymphocytes. After exclusion of other identifiable inciting antigens and alternative diagnoses, typical

nonfibrotic HP probably associated with exposure to QAC-containing detergent products was considered.

The patient successfully avoided further exposure and received oral corticosteroid therapy. The clinical, functional, and radiological course was favorable. Forced vital capacity increased from 70% to 82% predicted between 2023 and 2024.

Case 2: fibrotic HP with clinical and functional improvement and radiological stability

A 60-year-old homemaker with no relevant medical history reported repeated and prolonged

exposure to detergent products containing QACs for three years. She presented with progressively worsening exertional dyspnea and chronic dry cough. Physical examination revealed digital clubbing and bilateral crackles.

HRCT showed a typical fibrotic HP pattern, including the three-density pattern and fibrotic abnormalities (Figure 2).

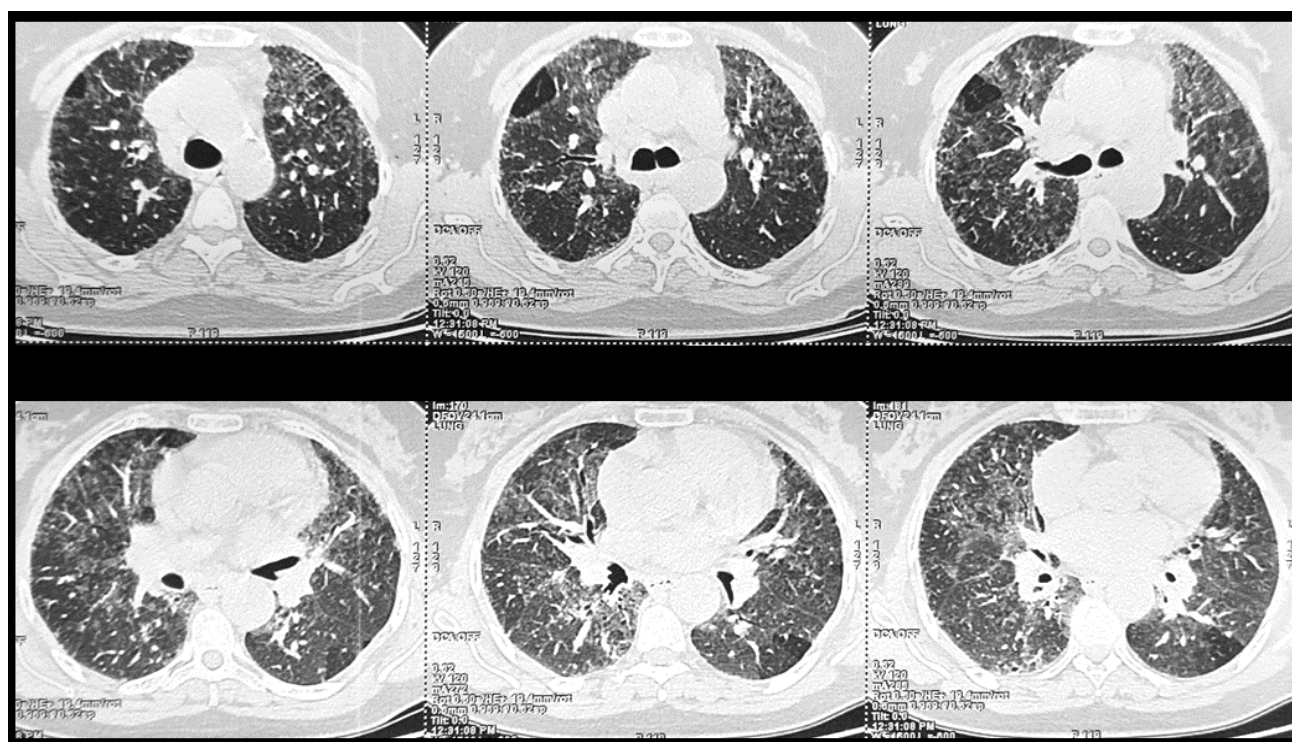


Figure 2: High-resolution computed tomography of Case 2. Typical fibrotic hypersensitivity pneumonitis showing the three-density pattern and fibrotic abnormalities

BAL fluid differential cell count showed 20% lymphocytes and 15% neutrophils. After exclusion of other suspected inciting antigens and alternative diagnoses, fibrotic HP probably associated with detergent exposure was considered.

Management included strict exposure avoidance, oral corticosteroids, and azathioprine after multidisciplinary discussion. The patient's symptoms and lung function improved, with forced vital capacity increasing from 56% to 71% predicted between 2024 and 2025. Follow-up HRCT, however, showed stable residual fibrotic abnormalities.

Case 3: progressive fibrotic HP with persistent exposure despite repeated counseling

A 66-year-old professional cleaner had a history of arrhythmogenic heart disease with supraventricular tachycardia and underwent placement of a subcutaneous implantable cardioverter-defibrillator in 2024. She reported repeated and prolonged exposure to detergent products containing QACs for three years. She

developed progressive exertional dyspnea and chronic dry cough. Physical examination revealed bilateral crackles.

HRCT showed typical fibrotic HP with a three-density pattern and fibrotic abnormalities (Figure 3).

BAL fluid differential cell count showed 25% neutrophils. After exclusion of other identifiable inciting antigens and alternative diagnoses, fibrotic HP probably associated with prolonged detergent exposure was considered.

Despite repeated education and counseling regarding strict exposure avoidance, the patient continued to be exposed to the implicated detergent products. Under oral corticosteroids and azathioprine, her respiratory symptoms improved; however, functional and radiological progression occurred. Forced vital capacity decreased from 77% to 57% predicted between 2024 and 2025. After a second multidisciplinary discussion, antifibrotic therapy was approved to limit

further respiratory functional decline, while efforts to achieve complete exposure cessation were reinforced.

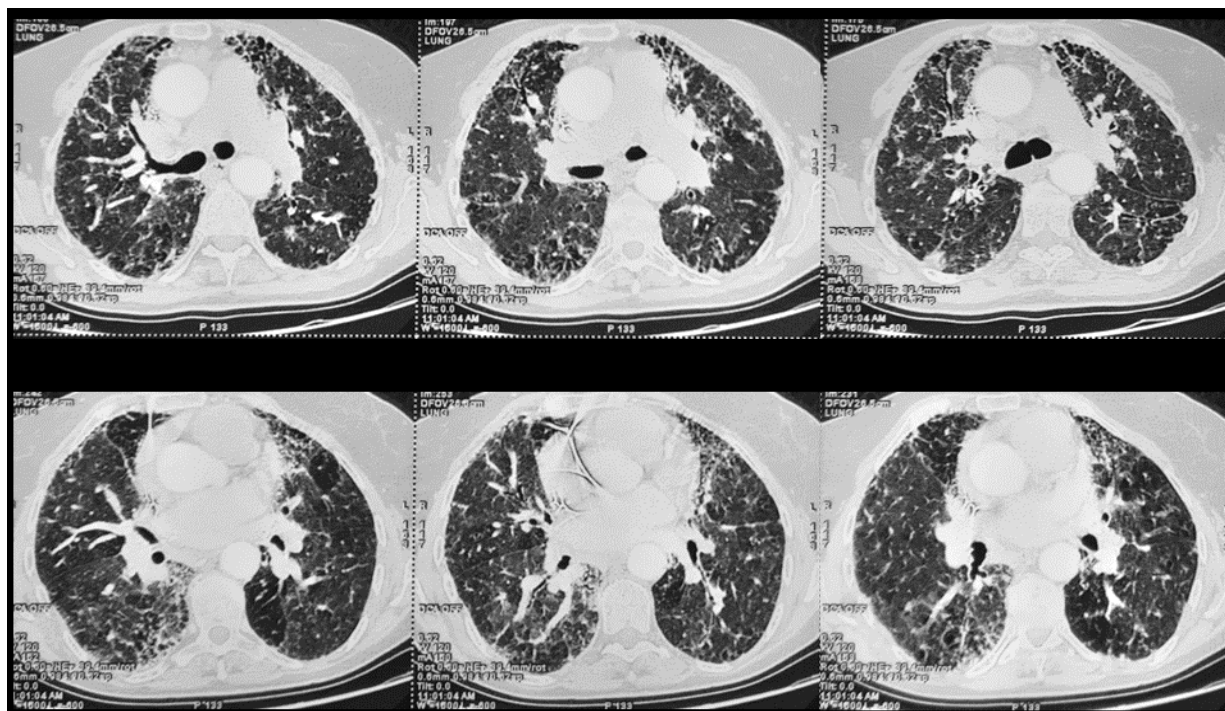


Figure 3: High-resolution computed tomography of Case 3. Fibrotic hypersensitivity pneumonitis with radiological progression during follow-up in the setting of persistent exposure despite repeated counseling and ongoing immunosuppressive therapy

Table 1: Summary of clinical, radiological, BAL, treatment, and outcome data

Variable	Case 1	Case 2	Case 3
Age/sex/occupation	63-year-old woman, professional cleaner	60-year-old woman, homemaker	66-year-old woman, professional cleaner
Relevant history	No relevant medical history	No relevant medical history	Arrhythmogenic heart disease with supraventricular tachycardia; subcutaneous defibrillator implanted in 2024
Exposure	Intense repeated exposure to QAC-containing detergent products for 2 months	Repeated prolonged exposure to QAC-containing detergent products for 3 years	Repeated prolonged exposure to QAC-containing detergent products for 3 years; exposure continued during follow-up despite repeated education and counseling
Clinical presentation	Wheezing respiratory discomfort and dry cough after exposure; bilateral crackles	Progressive exertional dyspnea, chronic dry cough, digital clubbing, bilateral crackles	Progressive exertional dyspnea, chronic dry cough, bilateral crackles
HRCT pattern	Mosaic attenuation, expiratory air trapping, centrilobular micronodules	Three-density pattern with fibrotic abnormalities	Three-density pattern with fibrotic abnormalities
HP classification	Typical nonfibrotic HP	Typical fibrotic HP	Typical fibrotic HP with progressive pulmonary fibrosis
BAL	BAL fluid: 30% lymphocytes	BAL fluid: 20% lymphocytes and 15% neutrophils	BAL fluid: 25% neutrophils
Treatment	Strict exposure avoidance and oral corticosteroids	Strict exposure avoidance, oral corticosteroids, and azathioprine	Repeated counseling regarding strict exposure avoidance; oral corticosteroids and azathioprine; antifibrotic therapy approved after progression
Outcome	Favorable clinical, functional, and radiological outcome; FVC increased from 70% to 82% predicted	Clinical and functional improvement with radiological stability; FVC increased from 56% to 71% predicted	Clinical improvement but functional and radiological progression in the setting of persistent exposure; FVC decreased from 77% to 57% predicted

DISCUSSION

This case series highlights three clinically important messages. First, QAC-containing detergent products should be considered during exposure assessment in patients with suspected HP, particularly in professional cleaners and individuals with repeated domestic use of disinfectant detergents. Second, the same suspected exposure may be associated with different radiological and clinical phenotypes, ranging from reversible nonfibrotic disease to progressive fibrotic disease. Third, effective exposure cessation is central to management but may be difficult to achieve; persistent exposure may contribute to progression despite anti-inflammatory and immunosuppressive treatment.

The diagnosis of HP is probabilistic and depends on concordance among exposure history, imaging, BAL findings, and clinical course. In the present series, the first case showed a typical nonfibrotic pattern, with mosaic attenuation, air trapping, and centrilobular micronodules, together with BAL lymphocytosis and a favorable response after exposure removal and corticosteroids. The second and third cases showed fibrotic HP with a three-density pattern, a radiological sign considered highly suggestive in an appropriate clinical context. Established fibrosis probably explains the incomplete reversibility in the second patient, while both fibrosis and persistent exposure may have contributed to progression in the third patient.

QACs are used extensively because of their stability, surfactant properties, and broad antimicrobial activity. Their respiratory effects are complex. The literature supports irritant, sensitizing, and possible adjuvant mechanisms, while the boundary between irritant-induced disease and true immunological sensitization remains difficult to define [4]. Occupational asthma caused by QACs is better described than HP, but experimental evidence that DDAC can induce pulmonary inflammation and fibrosis in mice provides biological plausibility for parenchymal involvement after inhalational exposure [6,7].

The divergent outcomes in our cases may reflect differences in exposure intensity and duration, host susceptibility, timing of diagnosis, effective exposure cessation, and the presence or absence of established fibrosis. The first patient had a shorter but intense exposure and a nonfibrotic phenotype, followed by a favorable course after exposure avoidance and corticosteroids. The second and third patients had longer exposure histories and fibrotic disease; the second achieved exposure avoidance and remained radiologically stable, whereas the third continued to be exposed despite repeated counseling and developed functional and radiological progression under treatment.

Management of HP begins with identification and elimination of the inciting antigen. Corticosteroids may improve inflammatory manifestations, and immunosuppressive therapy may be used in selected fibrotic cases when an inflammatory component is suspected. For progressive fibrotic interstitial lung disease, current international guidance supports consideration of antifibrotic therapy, particularly nintedanib, in patients who progress despite appropriate management [8,9]. The third patient demonstrated both physiological and radiological progression; after multidisciplinary discussion, antifibrotic therapy was therefore approved, and counseling regarding complete exposure cessation was reinforced.

The public health implications are relevant. Cleaning products are often perceived as harmless, especially in domestic settings, and product labels may not always be reviewed in clinical practice. A structured history should address occupation, cleaning frequency, spray or aerosolized product use, mixing of detergents, ventilation, protective equipment, and product labels or safety data sheets. Prevention should prioritize reducing aerosolization, improving ventilation, using appropriate protective measures in occupational settings, and substituting products when feasible. Education should be followed by verification that exposure has effectively ceased, particularly when exposure is occupational or embedded in daily routines.

This report has several limitations. The series is small and retrospective. Quantitative exposure measurements were not available, and the precise QAC molecules and concentrations could not be confirmed from the available product data. Therefore, causality cannot be considered definitive. Nevertheless, the repeated exposure histories, exclusion of other identifiable inciting antigens, compatible HRCT patterns, BAL findings, multidisciplinary assessment, and improvement or stabilization after avoidance in two patients support a probable association.

Learning points

- Quaternary ammonium compound-containing detergent products should be actively investigated as potential inciting exposures in patients with suspected HP.
- HRCT with inspiratory and expiratory acquisitions is essential for detecting mosaic attenuation and air trapping, particularly in nonfibrotic HP.
- Effective exposure cessation should be explicitly verified during follow-up; education alone may not be sufficient to eliminate persistent occupational or domestic exposure.
- Patients with fibrotic HP require repeated functional and radiological reassessment and multidisciplinary discussion, including consideration of antifibrotic therapy when progressive pulmonary fibrosis develops.

CONCLUSION

These three cases illustrate the heterogeneous clinical spectrum of HP probably associated with repeated exposure to detergent products containing QACs. Early recognition is essential because nonfibrotic disease may be reversible, whereas established fibrosis may persist or progress. Clinicians should systematically assess domestic and occupational cleaning exposures, confirm that exposure has effectively ceased during follow-up, and discuss diagnosis and management in a multidisciplinary setting.

DECLARATIONS

Consent for publication: Written informed consent was obtained from all three patients for publication of this case series and the accompanying anonymized clinical and radiological images.

Competing interests: The authors declare that they have no competing interests.

Funding: No specific funding was received for the preparation of this article.

Authors' contributions: Mehdi Maaroufi and Imane El Hamdani were responsible for the conception, drafting, and critical revision of the manuscript. Hanaa Harraz, Khadija Chaanoun, Wafaa Jalloul, Zakaria Laklaai, Hanane Benjelloun, and Nahid Zaghba contributed to the review and validation of the scientific content. All authors read and approved the final manuscript.

Data availability: All data relevant to this case series are included in the manuscript. Additional anonymized information may be provided by the corresponding author upon reasonable request, subject to institutional and ethical requirements.

REFERENCES

1. Raghu G, Remy-Jardin M, Ryerson CJ, Myers JL, Kreuter M, Vasakova M, *et al.*, Diagnosis of hypersensitivity pneumonitis in adults. An official ATS/JRS/ALAT clinical practice guideline. *Am J Respir Crit Care Med.* 2020;202(3):e36-e69. doi:10.1164/rccm.202005-2032ST.
2. Fernández Pérez ER, Travis WD, Lynch DA, Brown KK, Johansson KA, Selman M, *et al.*, Diagnosis and evaluation of hypersensitivity pneumonitis: CHEST guideline and expert panel report. *Chest.* 2021;160(2):e97-e156. doi:10.1016/j.chest.2021.03.066.
3. Johansson KA, Barnes H, Bellanger AP, Dalphin JC, Fernández Pérez ER, Flaherty KR, *et al.*, Exposure assessment tools for hypersensitivity pneumonitis. An official American Thoracic Society workshop report. *Ann Am Thorac Soc.* 2020;17(12):1501-1509. doi:10.1513/AnnalsATS.202008-942ST.
4. Peyneau M, de Chaisemartin L, Gigant N, Chollet-Martin S, Kerdine-Römer S. Quaternary ammonium compounds in hypersensitivity reactions. *Front Toxicol.* 2022; 4:973680. doi:10.3389/ftox.2022.973680.
5. Miguères N, Debaille C, Walusiak-Skorupa J, Lipinska-Ojrzanowska A, Munoz X, van Kampen V, *et al.*, Occupational asthma caused by quaternary ammonium compounds: a multicenter cohort study. *J Allergy Clin Immunol Pract.* 2021;9(9):3387-3395. doi:10.1016/j.jaip.2021.04.025.
6. Yoshida T, Tuder RM. Pulmonary fibrosis in response to environmental cues and molecular targets involved in its pathogenesis. *J Toxicol Pathol.* 2011;24(1):9-24. doi:10.1293/tox.24.9.
7. Ohnuma-Koyama A, Yoshida T, Tajima-Horiuchi H, Takahashi N, Yamaguchi S, Ohtsuka R, *et al.*, Didecyltrimethylammonium chloride induces pulmonary fibrosis in association with TGF- β signaling in mice. *Exp Toxicol Pathol.* 2013 ;65(7-8):1003-1009. doi:10.1016/j.etp.2013.02.005.
8. Raghu G, Remy-Jardin M, Richeldi L, Thomson CC, Inoue Y, Johkoh T, *et al.*, Idiopathic pulmonary fibrosis (an update) and progressive pulmonary fibrosis in adults: an official ATS/ERS/JRS/ALAT clinical practice guideline. *Am J Respir Crit Care Med.* 2022;205(9):e18-e47. doi:10.1164/rccm.202202-0399ST.
9. Flaherty KR, Wells AU, Cottin V, Devaraj A, Walsh SLF, Inoue Y, *et al.*, Nintedanib in progressive fibrosing interstitial lung diseases. *N Engl J Med.* 2019;381(18):1718-1727. doi:10.1056/NEJMoa1908681.