Received: 30 April 2023 | Revised: 18 December 2023 | Accepted: 21 December 2023

DOI: 10.1002/ajim.23564

CASE REPORT

AMERICAN JOURNAL OF INDUSTRIAL MEDICINE WILEY

Interstitial pulmonary disease and aluminum trihydrate exposure: A single case report and detailed workplace analysis

Claudia Corwin MD, MPH[1] | Hillary Waterhouse MPH, CIH, CPH[2] | Jerrold L. Abraham MD[3] | Soma Sanyal MD[3] | Judith A. Crawford PhD, CIH[3] | Matthew Caddell DO, MPH[4] | Michael J. Hodgson MD, MPH[5]

[1]Department of Internal Medicine, Division of Pulmonary, Critical Care and Occupational Medicine, University of Iowa, Iowa City, Iowa, USA

[2]New York State Department of Health, Albany, New York, USA

[3]State University of New York Upstate Medical University, Syracuse, New York, USA

[4]Occupational and Environmental Health Center, Albany, New York, USA

[5]Occupational Safety and Health Administration, United States Department of Labor, Washington, District of Columbia, USA

Correspondence
Claudia Corwin, MD, MPH, Department of Internal Medicine, Division of Pulmonary, Critical Care and Occupational Medicine, University of Iowa, 200 Hawkins Dr., C33 GH, Iowa City, IA 52242, USA.
Email: claudia-corwin@uiowa.edu

Abstract

Exposure to aluminum compounds is clearly associated with pulmonary function decrements, and several animal models document possible mechanisms of aluminum-compound-induced pulmonary toxicity. Nevertheless, disagreements remain about the precise mechanism by which exposures lead to damage. We present a strong case for attributing a case of interstitial pulmonary disease to occupational exposure to aluminum trihydrate. This report follows a 2014 publication of another case of interstitial pulmonary disease following a similar exposure. Our patient eventually underwent double lung transplantation nearly 5 years postexposure. Detailed pulmonary particulate elemental analysis suggested that aluminum metal, including aluminum trihydrate, was the most likely cause. A detailed assessment of the worker's relevant occupational exposures accompanies this case report.

KEYWORDS

aluminum trihydrate, Corian, industrial, occupational, pulmonary disease

1 | INTRODUCTION

Pulmonary disease was first attributed to aluminum exposure in 1941 when Goralewski and Jaeger described lung disease in workers at an aluminum and brass stamping plant, initially attributed to the metal dusts.[1] In 1947 Goralewski[2] and Shaver and Riddell[3] described, independently, a series of human cases of lung disease attributable to aluminum exposure. Shaver's name has since been associated with the eponymous Shaver's disease, a form of pulmonary fibrosis believed to be related to occupational exposure to bauxite fumes containing aluminum and silica. Since then, numerous case reports and case series have attributed pulmonary disease to aluminum exposure in a variety of industrial processes, including welding, grinding, shipbuilding, and mining.[4–6] Several disease mechanisms have been postulated, including direct toxicity of metal,[7] immune adjuvant effects of aluminum,[8] induction of sarcoid-like granulomatous disease,[9] and genetic susceptibility.[10] Although exposures in certain aluminum industries, including bauxite mining and milling, are clearly associated with decrements in pulmonary function,[11] disagreements remain over how aluminum in its various forms contributes to the degree and type of pulmonary pathology. While epidemiologic studies lack detailed exposure information, both animal and epidemiologic studies report the association between aluminum exposure and pulmonary fibrosis.[7] In addition, some case series

suggest that interstitial lung disease in workers with aluminum exposure is associated with elevated pulmonary levels of aluminum.[12]

In 2014, a case of lung disease was attributed to work with Corian®,[13,14] a solid-surface material composed of two-thirds (by weight) aluminum trihydrate and one-third polymethyl methacrylate.[15] The authors now present a second case report of a worker exposed to acrylic solid surface products including Corian®. This second case report includes estimated exposure levels, pulmonary pathology, and lung tissue analysis that further suggest an association between interstitial pulmonary disease and aluminum trihydrate exposure. The worker filed a complaint with the U.S. Occupational Safety and Health Administration (OSHA) after receiving a diagnosis of interstitial lung disease. The resulting OSHA inspection consisted of a walk-through inspection and qualitative and quantitative sampling on two occasions. The walk-through results supported aluminum exposure. As a result of the findings, OSHA issued citations under the Respiratory Protection Standard for failure to conduct medical evaluations and fit-testing.

2 | CASE REPORT

A 48-year-old man presented with pulmonary fibrosis after working in cabinet and countertop production for 26 years. During the first 24 years of his career, he worked in a shop that manufactured cabinets as well as wood and stone countertops. Subsequently, he began working for a new employer who added aluminum trihydrate-containing composite countertop material to the production lines. Within 6 months of beginning work, the worker developed Raynaud's syndrome. Over the course of the next year, he developed progressive dyspnea, and was diagnosed with diffuse pulmonary fibrosis, which was initially attributed to systemic sclerosis.

The patient was referred to an occupational medicine physician for clinical evaluation of possible work-relatedness for his disease and impairment assessment. On initial exam, the patient was found to have exertional dyspnea with minimal ambulation and resting pulse oximetry of 95% on room air. Physical examination revealed bibasilar crackles, sclerodactyly, and finger-tip ulceration. There was no family history of autoimmune diseases. The patient was a former smoker with a 20-pack year history and reported he had to quit 2 years prior.

Laboratory investigation supported a diagnosis of systemic sclerosis with positive anti-smooth muscle (anti-Sm) antibody, positive antinuclear antibody (ANA) 1:640, positive RO-52 antibody, a negative cyclic citrullinated peptide (CCP) antibody, and weakly positive rheumatoid factor.

Pulmonary lung function testing revealed evidence of moderate to severe restrictive lung defect (Table 1).

A contrast-enhanced chest computed tomography (CT) scan revealed a ground glass appearance in the upper lobes, increased interstitial markings in the lower lobes, and right-sided hilar adenopathy (Figure 1).

A repeat CT scan 4 months later revealed progression of ground glass changes and fibrotic changes in lower lung zones. Despite empiric treatment with prednisone, no improvement was demonstrated on a third CT scan.

A lung biopsy was performed soon after the second CT. Light microscopy of lung tissue revealed areas of dense scarring and honeycomb change (Figure 2). In addition, there was focal

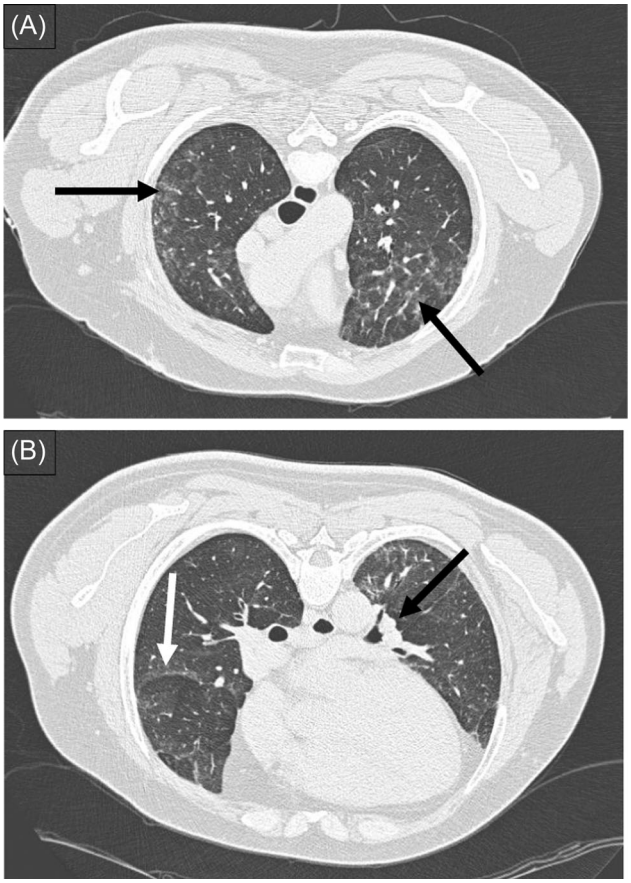


FIGURE 1 CT scans showing (A) ground glass appearance in the upper lobes and (B) increased interstitial markings in the lower lobes (white arrow) and right-sided hilar adenopathy (black arrow).

TABLE 1 Pulmonary function studies.

	FEV1 (L) (% predicted)	FVC (L) (% predicted)	FEV1/FVC	Total lung capacity (L) (% predicted)	DLCO mL/min/mm Hg (% predicted)	DLCO/VA
February 2012	1.55 (49%)	2.2 (50%)	77%	3.5 (63%)	10.2 (36%)	76%
November 2013	1.39 (43%)	1.71 (41%)	81%	2.12 (36%)	7.1 (25%)	67%

Note: source of predictive values not available.

Abbreviations: DLCO, diffusing capacity of the lungs for carbon dioxide; DLCO/VA, diffusing capacity divided by alveolar volume; FEV1, forced expiratory volume; FVC, forced vital capacity.

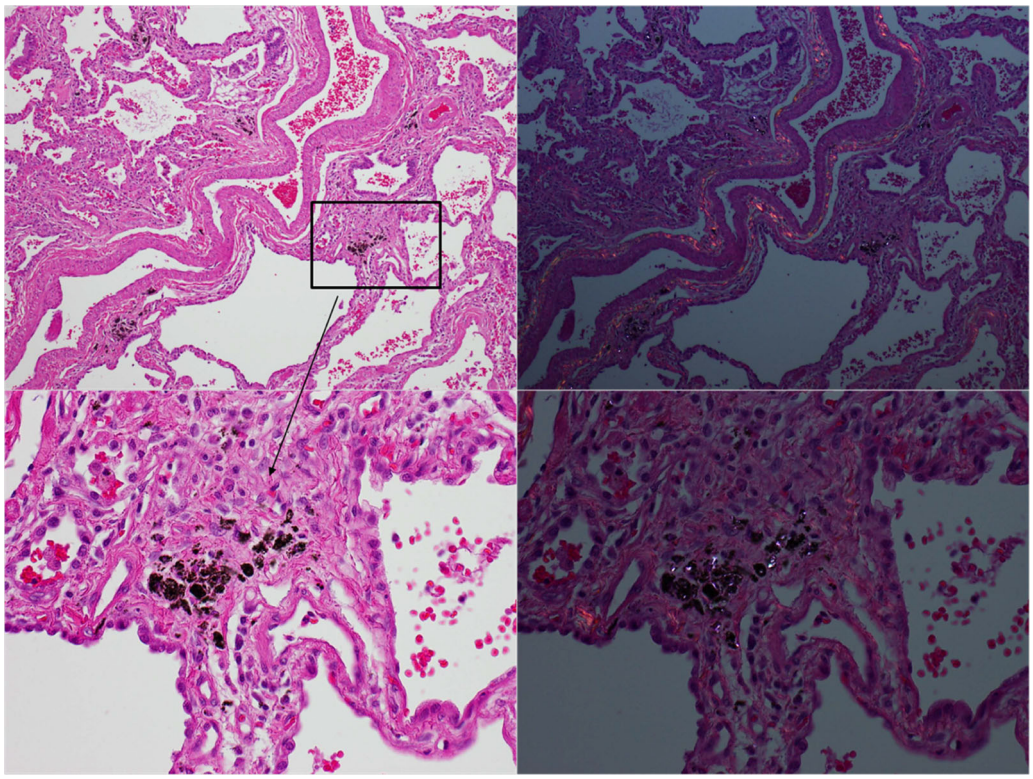


FIGURE 2 Interstitial fibrosis and focal mixed opaque and birefringent dust in lung biopsy sample. Brightfield, left; polarized light images, right.

accumulation of macrophages containing mixed opaque and birefringent dust. Silicotic nodules and asbestos bodies were not seen. Iron stains were examined.

Scanning electron microscopy with energy dispersive X-ray spectroscopy (SEM/EDS)[16] (Figure 3) showed macrophages containing a mixture of particles retained in the lung tissue, with aluminum (i.e., aluminum metal or oxides) as the predominant type, followed by silica and aluminum silicates. Some titanium was also present. SEM/EDS elemental analysis cannot differentiate between aluminum oxide and aluminum trihydrate.

Shortly after referral to the occupational medicine provider, the patient ceased working to prevent further exposures at his work site. Nine months later, pulmonary function had further declined, necessitating home oxygen support for activities of daily living.

Nearly 5 years after initial evaluation, the patient underwent double lung transplantation. The explant pathology showed areas of severe interstitial fibrosis (end stage, honeycombing) and the presence of macrophages containing a mixture of opaque and birefringent particles (Figure 4). Elemental analysis of the explanted lungs revealed aluminum particles as the most prevalent type (Figure 5).

Quantitative analysis of the lung dust burden in both the biopsy and lung explantation tissue showed particle concentrations of aluminum were the most clearly elevated compared to background cases with no known occupational exposures, and showed only minor increases of other particle types, such as aluminum silicates, silica, and other types (Figure 6).

3 | OCCUPATIONAL HISTORY

The patient had a 26-year history of working in cabinet and countertop production. Primary work materials included wood and stone. During his last 2 years of work, the production lines included a new solid surface countertop material, composed of aluminum trihydrate embedded in a polymethyl methacrylate base. The worker became a production supervisor and worked an average of 10-h days after the production line changed 2 years prior. Typically, he spent 2 days per week in the office and 3 days in the production environment. He described exposure to silica and other stone dusts, wood dusts, methyl methacrylate (liquid used to join solid surface seams), acrylic solid surface dust (aluminum trihydrate and poly methyl methacrylate), and aluminum oxide (an abrasive on sandpaper). Processes used in production included grinding, cutting, and drilling. He was not directly involved in painting or finishing and rarely came into contact with toluene and other solvents.

4 | WORKPLACE AND EXPOSURE ASSESSMENT

4.1 | Walk-through evaluation

A walk-through occurred as part of the OSHA enforcement inspection. Citations must be issued within 6 months of the opening

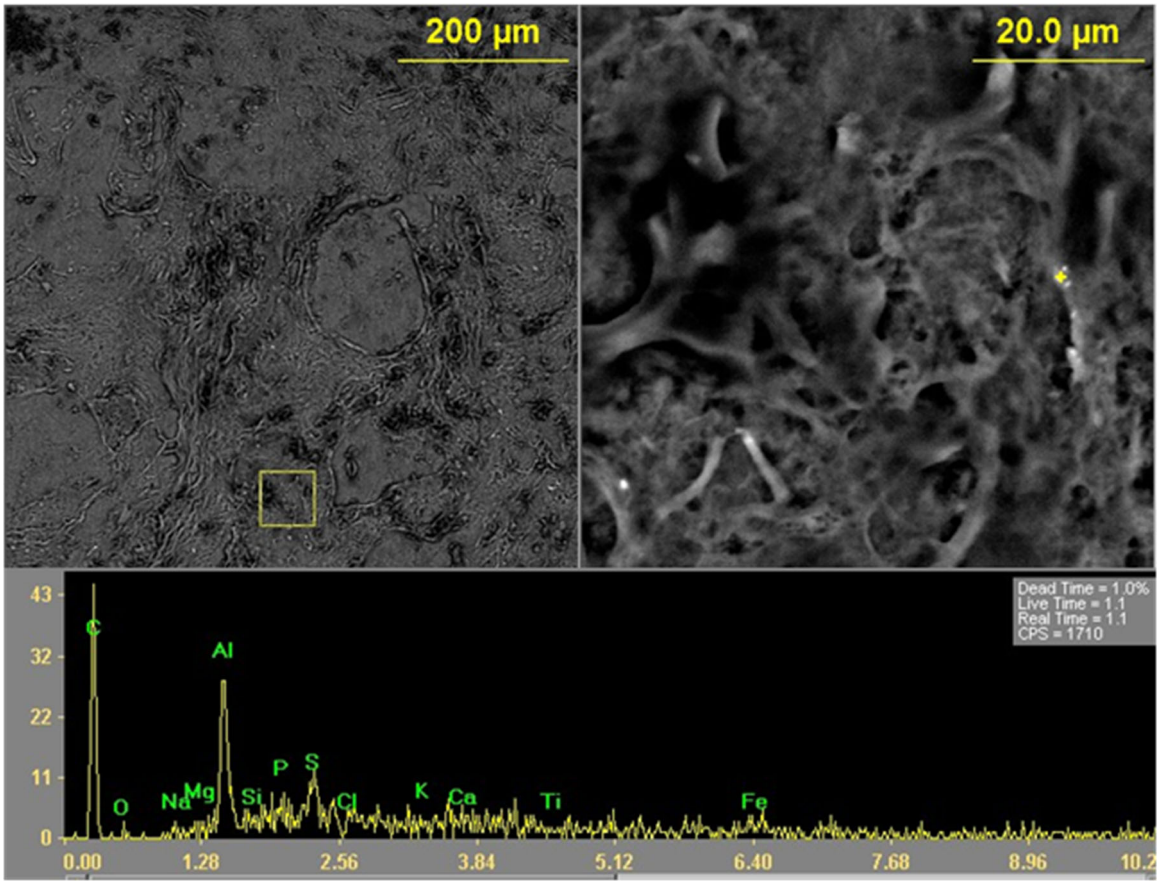


FIGURE 3 Energy dispersive X-ray spectroscopy (EDS) spectrum of aluminum particle from a field showing several particles; other spectra not shown. Lung biopsy sample.

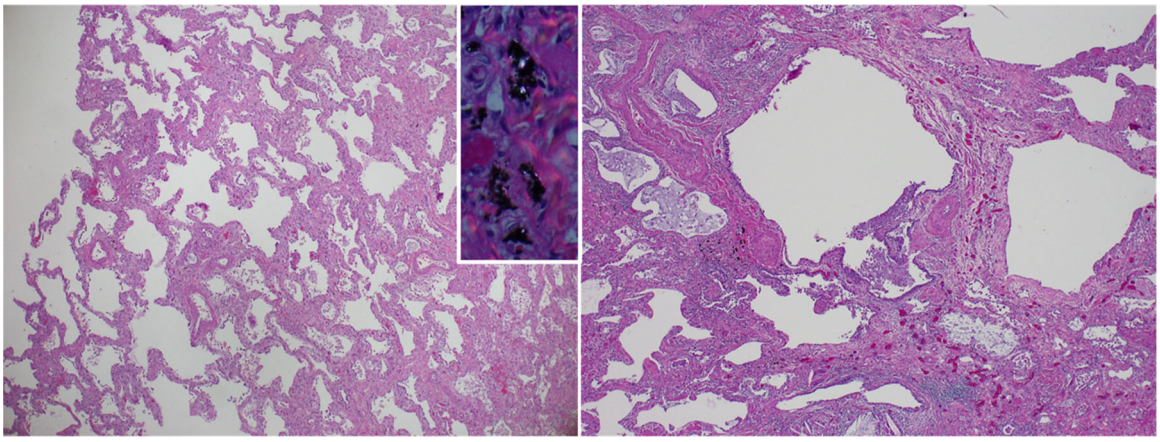


FIGURE 4 Light micrograph of lung explant showing extensive uniform interstitial fibrosis (left) and area of honeycombing (right). Inset shows focus of mixed dust with opaque and birefringent particles seen with polarized light microscopy.

date. OSHA's compliance safety and health officer observations follow. Approximately 70 employees worked within an open work-space that included four distinct work areas (quadrants) of approximately 1000 square feet each. The quadrants included a stone/quartz area; a solid-surface, non-quartz area; a wood cabinetry area; and a finishing, spray booth area.

4.1.1 | Quadrant 1, stone/quartz

This quadrant was divided into two separate areas: one for work on engineered stone countertops including Zodiaq© quartz solid surfaces[17] and one for natural stone. Silica and silicates were primary components of these materials. Activities included cutting, grinding, and drilling.

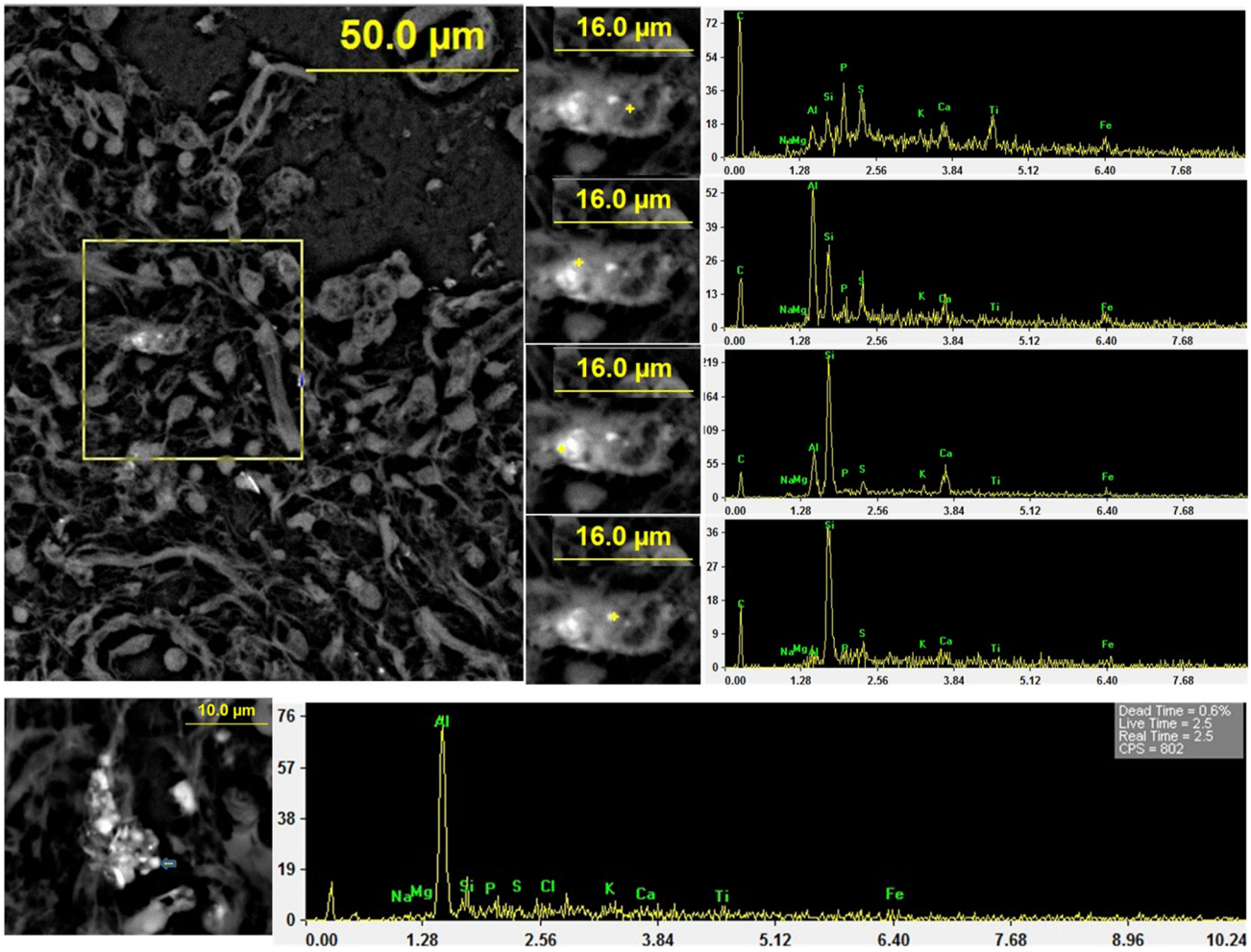
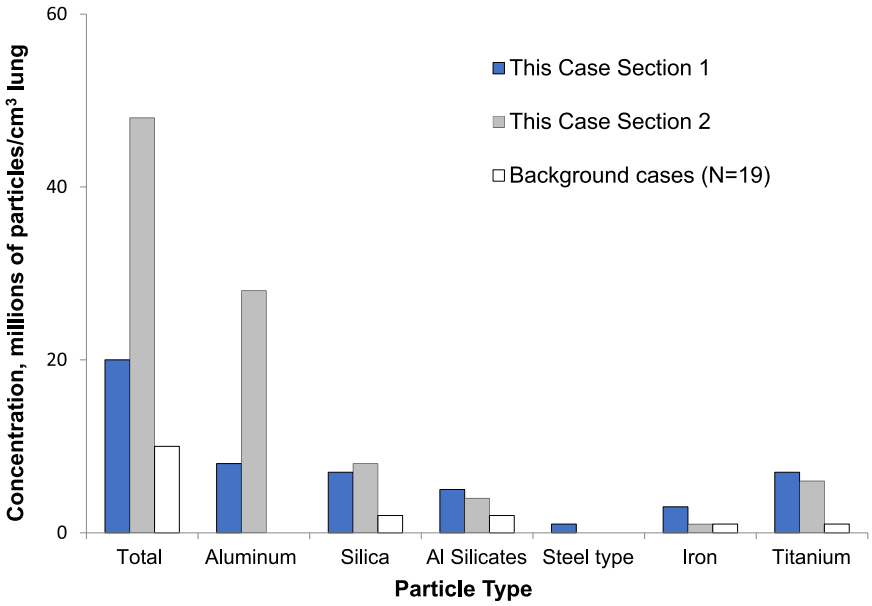


FIGURE 5 Lung explant sample Images showing macrophages containing multiple particles and selected examples of energy dispersive X-ray spectroscopy (EDS) spectra of individual particles, showing four spectra. Upper right top to lower: titanium (Ti), aluminum (Al), aluminum silicate, and silica. Bottom: Al.

FIGURE 6 Particle concentrations in biopsy lung tissue in this case and comparison with background cases, which have no known occupational exposure. Concentrations in millions of particles per cubic centimeter of lung tissue as determined by scanning electron microscopy with energy dispersive X-ray spectroscopy (SEM/EDS). Steel-type particles contain iron and chromium and/or nickel. Concentration shown is the median value of $N = 19$ comparison cases. Al, aluminum.



Wet-method, dust-control processes were used in accordance with the manufacturer's specifications. Processes were also controlled with appropriate local capture devices, with dust collected in a cyclone that was cleaned approximately every 3 months. With these engineering controls, work processes produced minimal airborne dust in the worker-breathing zone. Workers wore full-length, trench-coat-style coverings and N-95 respirators for personal protection. The employer had discontinued fit testing and other respiratory protection program elements that were previously in place.

4.1.2 | Quadrant 2, acrylic solid surface/non-quartz

Synthetic, solid-surface countertop fabrication was performed here including cutting, grinding, routing, drilling, and sanding of Corian® and other countertop products. Methyl methacrylate-containing joint adhesive was used to join together slabs of products. Primary exposures in this quadrant included countertop dust (aluminum trihydrate and polymethyl methacrylate). Large quantities of airborne fine white dust were visible during manufacturing and assembly. A thick layer of dust covered virtually every surface in this quadrant, as well as many of the surfaces in other work areas. The airborne dust created a haze effect visible throughout the shop. This quadrant was poorly ventilated, with inadequately maintained dust filters in overhead (general dilution) exhaust systems suspended from the ceiling. Airborne dust moved in a turbulent pattern, without a directed or organized airflow pattern. Employees did not wear personal protective equipment. N-95 respirators were available, but fit testing was not performed (Figure 7).

4.1.3 | Quadrant 3, wood cabinetry

This quadrant contained processes related to the manufacture of wood cabinetry where robotic functions performed some tasks. Employees were not required or trained to wear personal protective equipment, and most employees did not wear respirators. Dust haze from the synthetic countertop area was visible.

4.1.4 | Quadrant 4, finishing and offices

This quadrant included a finishing area, with two separate processes, and a separate formal office area. One finishing process fused large pieces of countertop through the application of methyl methacrylate. Another included polishing and spraying within a spraying booth. Large quantities of toluene were used for cleaning. Workers carried toluene-soaked cloths, sometimes tucked into their trouser pockets. The work area held open, uncovered containers of toluene and smelled of toluene and methyl methacrylate vapors. Workers in the spraying booth were required to use respiratory protection; in this case filtering facepieces not designed to be effective for this hazard. However, a respiratory protection program was not in place. OSHA cited the employer for the failure to provide medical clearance and fit-testing.

The office space included that of the case employee. The surfaces in this quadrant, including the office space, were covered with the same fine white dust seen in other work areas throughout the plant.

Given the occupational history and walk-through evaluation, exposure assessment focused initially on silica, toluene, and particulates. Aluminum was not identified as a component of the solid surface countertop until after the inspection was in process. Two days of sampling were performed to assess personal exposures to respirable dust and organic solvents as well as to characterize bulk samples.

4.2 | Exposure assessment: sampling

4.2.1 | Bulk dust samples

Bulk sample analysis was performed on dust mixtures collected from acrylic solid surface and quartz processing areas. Analysis indicated

FIGURE 7 Acrylic solid surface fabrication, Quadrant 2, with ineffective overhead filtration units and white dust visible on surfaces (left). Visible airborne dust and haze; dust-covered surfaces (right).

that up to 17% of the weight of samples collected in the solid surface/non-quartz area was aluminum (Table 2). This measurement method does not distinguish elemental aluminum from other forms (e.g., oxide or trihydrate). No other metals were detected in settled dust with the exception of iron (<1%). Samples of the silica slurry, collected from stone/quartz area, showed 30%–50% crystalline silica. Slurry (water and processed stone mixture) was sampled because wet methods and other controls prevented the accumulation of dry dust on surfaces in the stone/quartz area. Samples obtained were first analyzed for silica (quartz), and a secondary metals analysis was conducted for samples 1–3 and 6.

4.2.2 | Air samples

OSHA completed full-shift, personal air monitoring 8 months after the ill worker had been removed. An employee engaged in acrylic solid surface fabrication was sampled twice for solvent exposure, respirable dust, and respirable silica (quartz). The quartz shop supervisor was also sampled for respirable dust and silica. The following month, OSHA conducted full-shift solvent sampling of both the solid surface fabricator and cabinetry supervisor. Although analyses revealed substantial respirable dust, measurements remained below OSHA's Permissible Exposure Limit (PEL) and the American Conference of Governmental Industrial Hygienists (ACGIH) guideline for PNOR (particles not otherwise regulated; Table 3). OSHA did not sample directly for airborne aluminum; instead, the fraction of aluminum in settled dust was used to estimate relative quantities. Use of the mean proportional concentration of aluminum to overall respirable dust (16%) led to an estimate of 0.176 mg/m^3 airborne exposure, which is substantially below the current recommended ACGIH Threshold Limit Values (TLV) of 5 mg/m^3, as well as the OSHA PEL and National Institute for Occupational Safety and Health (NIOSH) Recommended Exposure Limit (REL) of 5 mg/m^3 for respirable aluminum metal dust, that is, below all existing occupational exposure limits. Toluene samples showed exposure below limits of detection. Personal sampling documented respirable silica (quartz) exposures for the fabricator and supervisor below limits of detection.

5 | DISCUSSION

The worker presented in this case report experienced three occupational exposures potentially associated with lung disease, including silica, methyl methacrylate, and aluminum trihydrate. The worker was also exposed to toluene, which has known associations with systemic sclerosis.[19] Systemic sclerosis was considered in the differential diagnosis early in clinical evaluation. Given the documented occupational history and the potential public health

TABLE 2 Bulk sample analysis for silica and metals results, Spring 2013.

		Sample number, type and area					
OSHA analytical method	Analyte(s)	1: Settled dust, solid surface area	2: Settled dust, solid surface area	3: Settled dust, solid surface area	4: Slurry, stone/quartz area	5: Slurry, stone/quartz area	6: Slurry, stone/quartz area
		Percent detected (by weight)					
Silica ID-142[26]	Silica (quartz)	ND	ND	ND	30%	50%	40%
					Not analyzed		
Metals	Copper	ND	ND	ND			0.19%
ID-125G[27]	Molybdenum	ND	ND	ND			ND
	Zinc	ND	ND	ND			ND
	Beryllium	ND	ND	ND			ND
	Lead	ND	ND	ND			ND
	Cadmium	ND	ND	ND			ND
	Antimony (compounds)	ND	ND	ND			ND
	Chromium	ND	ND	ND			ND
	Manganese	ND	ND	ND			0.34%
	Iron	MD	ND	0.93%			1.52%
	Cobalt	ND	ND	ND			0.17%
	Aluminum	15%	16%	17%			0.74%

Note: The ID-142 sample detection limit for quartz is 2.84 µg/sample. For an itemized list of qualitative and quantitative detection limits for ID-125, please see the table in the method documentation. Qualitative limits for the metals range from 0.013 to 8.9 µg/sample.

Abbreviations: ND, not detected; OSHA, Occupational Safety and Health Administration.

TABLE 3 Air sample results, Spring 2013.

Contaminant	OSHA (PEL)	NIOSH (REL)	ACGIH (TLV)	OSHA analytical method	Job type	Time sampled (minutes)	Exposure level (TWA)[a]
Toluene	200 PPM	100 PPM	20 PPM	111[18]	Non-quartz fabricator, Day 1 Non-quartz fabricator, Day 2 supervisor	173 416 454	1.6 PPM 1.0 PPM 0.55 PPM
PNOR/respirable fraction	5 mg/m^3	(Does not use PNOR construct)	3 mg/m^3[b]	PV2121[28]	Non-quartz fabricator	443 412	1.1 mg/m^3 ND(< 94 $\mu g/m^3$)
Respirable silica (quartz)[c]	0.05 mg/m^3[d]	0.05 mg/m^3	0.025 mg/m^3	ID-142[26]	Non-quartz fabricator Quartz supervisor	443 412	ND(< 13 $\mu g/m^3$) ND(< 14 $\mu g/m^3$)

Abbreviations: ACGIH TLV, American Conference of Governmental Industrial Hygienists Threshold Limit Value; ND, not detected; NIOSH REL, National Institute for Occupational Safety and Health Recommended Exposure Limit; OSHA PEL, Occupational Safety and Health Administration Permissible Exposure Limit; PNOR, particulates not otherwise regulated; TWA, total weight average.

[a]Based upon 480-min exposure.

[b]Guideline, not a TLV.

[c]Quantitation limit for this method is 9.76 µg/sample (12 $\mu g/m^3$).

[d]Current PEL, as of 2018.

implications of his occupational exposure, we were compelled to dig deeper to identify the most plausible exposure to explain his pulmonary disease.

Early during the patient's evaluation, clinical findings were consistent with systemic sclerosis (Raynaud's syndrome) and laboratory studies were suggestive of an autoimmune disorder. Knowing the associations of silica and toluene with systemic sclerosis and pulmonary fibrosis, both exposures were considered as potential etiologies for the patient's pulmonary disease and the focus of the workplace investigation. However, according to the exposure analysis, silica was well controlled in the workplace and not detectable with air sampling analysis—and the engineering design of the shop had not changed with the new employer. While silica particles were detected on SEM/EDS lung tissue analysis of both the pre-transplant biopsy and the post-transplant explant, the concentration of silica particles was only minimally above the background and far below the concentration of silica particles seen in cases of recognized silicosis.[20] Also importantly, the patient's clinical disease (including the histopathology findings) did not resemble forms of silicosis commonly described. With respect to toluene, ambient exposures were substantially below occupational exposure limits on repeated personal ambient toluene sampling (Table 3). With respect to the possibility that the patient's lung disease was related to a diagnosis of nonoccupational primary systemic sclerosis, he lacked a robust multisystem presentation for systemic sclerosis. For example, imaging studies did not reveal gastrointestinal anomalies, such as esophageal dilation on CT scan. Furthermore, the patient's demographic is also not consistent with the strong skew toward the female population.[21]

With respect to a third known workplace exposure, methyl methacrylate, although there are some reports of interstitial disease related to methacrylate, these document fundamentally different histologic appearances.[22,23,27]

Given these findings that argue against silica, toluene, and methyl methacrylate as a cause of the patient's clinical disease, the authors turned to acrylic solid surface including Corian®, an aluminum trihydrate-containing composite, as a potential cause of the patient's lung disease. The patient's history and workplace assessment details revealed substantial exposure to uncontrolled airborne and surface dust generated during cutting and sanding of acrylic solid surfaces. Initial attention focused on "airborne dust" (PNOR), that is, nuisance dust, and did not initially consider airborne aluminum, as the Corian® safety data sheet makes no mention of aluminum. As a result, OSHA did not include a direct or specific method for sampling airborne aluminum during the walk-through investigation and environmental sampling procedures. Subsequent research revealed the Dupont™ Corian® Product Overview,[15] which notes that Corian® composition is approximately two-thirds aluminum trihydrate derived from bauxite ore. Subsequent additional sampling was not possible due to the unique nature of OSHA investigations, including OSHA's limited access to the workplace after the initial walk-through assessment. Elemental analysis of the patient's initial lung biopsy tissue did reveal substantial aluminum concentrations, with a predominance of metal particles compared to other particle types. In addition, SEM/EDS analysis of tissue obtained from the worker's explanted lungs after transplantation revealed a similar profile of particle composition, with aluminum particles being the most prominent. In comparison with the case reported by Raghu et al.,[14] the worker here had fewer years of exposure, and the amount of fibrosis and the amount of dust visible by light microscopy was less. Still, the SEM/EDS quantitative analysis done in the present case (a method not employed in the Raghu et al. case) confirmed a concentration of aluminum particles above background.

In addition, a NIOSH study recently confirmed that aluminum, and specifically aluminum trihydrate, was the most abundant metal compound present in respirable dust generated during laboratory testing of Corian® sawing.[24]

With respect to uncertainty regarding other potential workplace sources of pulmonary aluminum, one may consider the contribution

TABLE 4 Aluminum inhalation and lung disease.

Study	Study type	Exposure type	Exposure length (years)	Chest CT	Pathology	SEM analysis/particle analysis[a]	Lung function	Other
Chew et al. (2016)[29]	Case report	Aluminum welder, boat building	7	Bilateral patchy infiltrates, calcified mediastinal lymph nodes	Electron microscopy of lavage fluid lamellar bodies in macrophages	Mediastinal lymph node with aluminum and aluminum oxide	Suggestive of restrictive disease	
Raghu et al.[13]	Case report	Dust inhalation; Corian® worker	16	Fibrosis, severe usual interstitial pneumonia	Usual interstitial pneumonia	Aluminum trihydrate[b]		
Ansari et al (2009)[30]	Case report	Fume inhalation; welder	30	Apical fibrosis, mediastinal adenopathy				
Cai et al. (2007)[31]	Case report	Dust inhalation; metal reclamation	15	Bilateral ground glass, consolidation, traction bronchiectasis	Non-caseating granulomas, multinucleated giant cells, no fibrosis or honeycombing	Aluminum	Restrictive	
Kraus et al. (2006).[32]	Cross-sectional ($N = 62$)	Inhalation; aluminum powder workers	Median 10	24% with upper lung rounded opacities, early thickened interlobular septae			Decreased vital capacity in 24%	Plasma and urine aluminum elevated
Hull[5]	Case report 1	Dust inhalation; aluminum ship welder/grinder; no mask/ventilation	24		Dense fibrosis, macrophages with fine particulate matter	100% aluminum-containing particles	Primarily restrictive	Chest X-ray: severe bilateral upper lobe fibrosis, lower lobe emphysematous changes
Hull[5]	Case report 2	Fume inhalation; aluminum ship welder, no mask/ventilation	22		Macrophages with granular pigment, emphysema	98% aluminum-containing particles	Obstructive	Chest X-ray: diffuse bilateral small densities upper lung fields
Vahlensiec et al. (2000)[33]	Case report	Fume inhalation; aluminum worker	12	Mediastinal adenopathy with increased HU density			Normal	
Jederlinic et al. (1990)[34]	Case report $N = 9$	Dust inhalation; Al_2O_3 abrasive production from aluminum ore	Mean 25		Interstitial fibrosis and honeycombing	Aluminum-containing particles/Al_2O_3 and aluminum alloys	Restrictive	International Labour Organization profusion ≥1/0
De Vuyst et al.[9]	Case report	Dust inhalation; metallic and oxide aluminum powders	8		Sarcoid-like non-caseating granulomas	Aluminum-bearing particles (metal or oxide) within granulomas	Mild restrictive	

(Continues)

TABLE 4 (Continued)

Study	Study type	Exposure type	Exposure length (years)	Chest CT	Pathology	SEM analysis/particle analysis[a]	Lung function	Other
De Vuyst et al. (1986)[35]	Case report	Inhalation; aluminum polisher	24		Dust-laden macrophages in fibrous lung parenchyma	11 minerals detected[a]; "major" minerals: Al, Al_2O_3, SiO_2	Restrictive	Chest X-ray: bilateral basilar honeycombing and emphysema, dense infiltrates in mid lung zones
Miller et al. (1984)[36]	Case report	Dust inhalation	6		Alveolar proteinosis, no fibrosis	Aluminum	Moderate restrictive, impaired diffusion	
Herbert (1982)[37]	Case report	Fume inhalation; aluminum and magnesium welder	16		Desquamative interstitial pneumonia	Electron dense aluminum particles within interstitial macrophages	Restrictive, impaired diffusion	Chest X-ray with wedge-shaped unilateral basilar infiltrate
Vallyathan et al. (1982)[4,38]	Case report	Fume inhalation; aluminum ship welder	17		Diffuse and focal fibrosis, macrophages with metallic material	Aluminum (exclusively) containing macrophages	Mixed restrictive/obstructive	
Musk (1980)[39]	Case report	Dust inhalation; dried alunite residue (crystalline aluminum silicate [cat litter])	1.5		Fibrosis, granulomas, multinucleated giant cells	Aluminum silicates	Decreased TL and T_L/V_A	Chest X-ray: bilateral small opacities throughout
Chen et al. (1978)[40]	Case report	Fume/dust inhalation; aircraft welder	5		Granulomas, birefringent crystalline structures	Aluminum		
McLaughlin et al. (1962)[41]	Case report	Dust inhalation; aluminum powder factory	13.5		Fibrosis, greater in upper lobes, and "aluminum dust"			Encephalopathy
Mitchell (1959)[42]	Case report	Dust inhalation; aluminum powder factory	2		Diffuse fibrosis, greater in upper lobes, "histochemical reactions for aluminum"			Chest X-ray: "pulmonary fibrosis"

Abbreviations: Al, aluminum; Al_2O_3, aluminum oxide; HU, Hounsfield Unit; SiO_2, silica; TL/VA, transfer factor for carbon monoxide/alveolar volume.

[a]Energy dispersive X-ray spectroscopic analysis (EDS).

[b]Definitively identified as aluminum trihydrate by Raman spectroscopy.

from sandpaper used in many of the work processes. Again, the limited access to the workplace unique to OSHA investigations limited our ability to completely characterize the type of sandpaper used and to analyze possible aluminum oxide exposure arising from the use of sandpaper materials. However, the dramatic and uncontrolled exposure to airborne surface dust created by grinding and sanding acrylic solid surface in our patient's workplace points to the resultant aluminum-containing dust as the source of the vast majority of the intrapulmonary aluminum particles in this worker. A recent report from NIOSH researchers demonstrated that the vast majority of the respirable aluminum generated by sanding Corian® material was from the aluminum trihydrate, independent of whether the sanding material was sandpaper containing aluminum oxide or other (nonaluminum containing) abrasives.[25] We remain unable to explain the sampling results and the degree of pulmonary loading. Did the sampling miss intermittent substantially higher exposure levels? Does aluminum have such potentially immunogenic activity, as acknowledged in its adjuvant functions for vaccination, that relatively mild exposures can create such dramatic reaction? Is there some genetic component, such as has been identified for beryllium, another divalent agent, that might explain the disease? There are limitations to this case study and analysis related to the reality that the authors were operating within the context of an OSHA workplace investigation. Follow-up data collection within the work place itself was not possible, due to limited access to the workplace itself. The authors also acknowledge there is latency between the worker's removal from the exposure at work and the initiation of the OSHA investigation. This is at largely related to the complex interplay between workers' compensation system, enforcement and patient care.

Follow-up with other workers was not possible and the authors have no knowledge of where these workers are or what their work exposures have been since this investigation. Considering the generalizability of workplace exposures, in the name of future prevention, OSHA did issue an exposure alert letter (HAL) to the employer detailing the hazard and control recommendations.

Despite the limitations inherent to determine work-related exposures and disease within the context of an OSHA investigation, the data presented here, including the exposure assessment, histopathology, and tissue analysis, as well as the temporal relationship between exposure and development of disease, all suggest a link between the patient's severe lung disease and his exposure to the aluminum-containing dust. The concern about aluminum exposure from solid surface composite materials is not limited to Corian®. Other commercially available solid surface products with the same major components include Aristech Surfaces, Avonite Acrystone®; Formica Corporation, Formica® brand Acrylic Solid Surfaces; Lion Chemtech Co., Tristone Solid Surface; Hanwha Corp., Hanex®; Kolpa, Kerrock®; Systempool S.A., Krion™; LG Hausys, LG HI-MACS®; LOTTE Advanced Materials, Staron® Solid Surface; Meganite Inc., Meganite®, and Wilsonart LLC, Wilsonart® Solid Surface.

Despite 80 years of intermittent publication, controversy remains regarding which forms or levels of inhaled aluminum cause, or contribute to, the development of pulmonary fibrosis. Taiwo reviewed the more recently published literature and characterized the discrepancies between cross-sectional studies that show relatively little disease and case reports that are quite persuasive.[7] Our own review of the literature reveals a predominance of case reports and highlights the link between aluminum inhalation and lung disease (Table 4).

6 | CONCLUSION

The case presented here is now the second case reported with a possible link between aluminum-containing solid surface dust exposure and pulmonary disease. The data presented in this single case report cannot confirm causality between such dust exposure and pulmonary fibrosis; however, case studies such as the one presented here highlight the significance of ongoing occupational aluminum exposure in the modern-day workplace, and the need for continued awareness of the potential association between aluminum exposures and lung disease. Furthermore, the investigation detailed in this case report points to the importance of basic primary prevention as the ultimate mission of governmental agencies such as OSHA, and continued optimization of workplace industrial hygiene practices to promote the safety and well-being of the modern-day industrial worker.

AUTHOR CONTRIBUTIONS

Claudia Corwin participated in the conception, aggregation of data, and primary drafting of the work. Michael J. Hodgson participated in the conception, acquisition of data, and drafting of the work. Hillary Waterhouse participated in acquisition and interpretation of industrial hygiene data at the worksite. Jerrold L. Abraham participated in the conception, acquisition of pathology data, and critically reviewing for important intellectual content. Soma Sanyal participated in the acquisition of the pathology data and editing of the work. Judith A. Crawford participated in the interpretation of industrial hygiene data and editing of the work. Matthew Caddell provided services as a care provider for the patient and served to communicate with the patient when necessary. He also approved the final version of the work to be published, ensuring that questions related to the accuracy or integrity of any part of the work related to the patient's healthcare experience were appropriately investigated and resolved. Michael J. Hodgson, Claudia Corwin, and Hillary Waterhouse worked at OSHA while the case was actively under investigation by OSHA.

ACKNOWLEDGMENTS

The authors appreciate the support of the Industrial Hygiene Chemistry Division at the Salt Lake Technical Center in OSHA's Directorate of Technical Support and Emergency Management in analyzing physical and chemical samples.

CONFLICT OF INTEREST STATEMENT

The authors declare that there are no conflicts of interest.

DISCLOSURE BY AJIM EDITOR OF RECORD

John Meyer declares that he has no conflict of interest in the review and publication decision regarding this article.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS APPROVAL AND INFORMED CONSENT

The patient described herein has provided written, signed consent.

ORCID

Claudia Corwin http://orcid.org/0000-0002-0389-4965
Jerrold L. Abraham http://orcid.org/0000-0002-4885-0050

REFERENCES

1. Goralewski G, Jaeger R. Zur Klinik, Pathologie und Pathogenese der Aluminiumlunge. *Arch Gewerbepathol Gewerbehyg Berlin*. 1941;11(1):102-105. doi:10.1007/bf02123167
2. Goralewski G. The aluminum lung: a new industrial disease (Abstract of Goralewski, 1947). *Br J Ind Med*. 1948;6:53-54.
3. Shaver CG, Riddell AR. Lung changes associated with the manufacture of alumina abrasives. *J Ind Hyg Toxicol*. 1947;29(3):145-157.
4. Vallyathan V, Bergeron WN, Robichaux PA, Craighead JE. Pulmonary fibrosis in an aluminum arc welder. *Chest*. 1982;81(3):372-374.
5. Hull MJ, Abraham JL. Aluminum welding fume-induced pneumoconiosis. *Hum Pathol*. 2002;33(8):819-825.
6. Donoghue AM, Frisch N, Olney D. Bauxite mining and alumina refining: process description and occupational health risks. *J Occup Environ Med*. 2014;56(5 suppl):S12-S17. doi:10.1097/JOM.0000000000000001
7. Taiwo OA. Diffuse parenchymal diseases associated with aluminum use and primary aluminum production. *J Occup Environ Med*. 2014;56(5S):S71-S72.
8. Peng Y, Chang W, Zhou H, Hu H, Liang W. Factors associated with health-seeking behavior among migrant workers in Beijing, China. *BMC Health Serv Res*. 2010;10:69. doi:10.1186/1472-6963-10-69
9. De Vuyst P, Dumortier P, Schandené L, Estenne M, Verhest A, Yernault JC. Sarcoidlike lung granulomatosis induced by aluminum dusts. *Am Rev Respir Dis*. 1987;135(2):493-497. doi:10.1164/arrd.1987.135.2.493
10. Arnaiz NO, Kaufman JD, Daroowalla FM, Quigley S, Farin F, Checkoway H. Genetic factors and asthma in aluminum smelter workers. *Archiv Environ Health*. 2003;58(4):197-200. doi:10.3200/aeoh.58.4.197-200
11. Townsend MC, Enterline PE, Sussman NB, Bonney TB, Rippey LL. Pulmonary function in relation to total dust exposure at a bauxite refinery and alumina-based chemical products plant. *Am Rev Respir Dis*. 1985;132(6):1174-1180. doi:10.1164/arrd.1985.132.6.1174
12. Abraham J, Burnett B, Hunt A. Quantification of non-fibrous and fibrous particulates in human lungs: twenty-year update on pneumoconiosis database. *Ann Occup Hyg*. 2002;46(1):397-401.
13. Raghu G, Collins BF, Xia D, Schmidt R, Abraham JL. Pulmonary fibrosis associated with aluminum trihydrate (Corian) dust. *N Engl J Med*. 2014;370(22):2154-2156. doi:10.1056/NEJMc1404786#SA1
14. Raghu G, Xia D, Abraham JL. More on pulmonary fibrosis associated with aluminum trihydrate (Corian) dust. *N Engl J Med*. 2014;371(10):973-974. doi:10.1056/NEJMc1407658
15. Dupont. *Corian® solid surface material composition*. 2018. https://www.corian.com/IMG/pdf/k-30025-corian-solid-surface-material-composition-bulletin_sec.pdf
16. Abraham JL, Burnett BR. Quantitative analysis of inorganic particulate burden in situ in tissue sections. *Scanning Electron Microsc*. 1983;(Pt 2):681-696.
17. Phillips ML, Johnson DL, Johnson AC. Determinants of respirable silica exposure in stone countertop fabrication: a preliminary study. *J Occup Environ Hyg*. 2013;10(7):368-373. doi:10.1080/15459624.2013.789706
18. Occupational Safety and Health Administration. *OSHA method PV 111*. Toluene. 1998. Accessed April 20, 2023. https://www.osha.gov/sites/default/files/methods/osha-111.pdf
19. Zhao JH, Duan Y, Wang YJ, Huang XL, Yang GJ, Wang J. The influence of different solvents on systemic sclerosis: an updated meta-analysis of 14 case-control studies. *J Clin Rheumatol*. 2016;22(5):253-259. doi:10.1097/RHU.0000000000000354
20. Abraham JL, Wiesenfeld SL. Two cases of fatal PMF in an ongoing epidemic of accelerated silicosis in oilfield sandblasters: lung pathology and mineralogy. *Ann Occup Hyg*. 1997;41(Inhaled Particles VIII, suppl 1):440-447.
21. Bergamasco A, Hartmann N, Wallace L, Verpillat P. Epidemiology of systemic sclerosis and systemic sclerosis-associated interstitial lung disease. *Clin Epidemiol*. 2019;11:257-273. doi:10.2147/CLEP.S191418
22. Kim YH, Chung YK, Kim C, Nam E, Kim HJ, Joo Y. A case of hypersensitivity pneumonitis with giant cells in a female dental technician. *Ann Occup Environ Med*. 2013;25(1):19. doi:10.1186/2052-4374-25-19
23. Piirilä P, Hodgson U, Estlander T, et al. Occupational respiratory hypersensitivity in dental personnel. *Int Arch Occup Environ Health*. 2002;75(4):209-216. doi:10.1007/s00420-001-0302-4
24. Qi C, Echt A, Murata TK. Characterizing dust from cutting Corian®, a solid-surface composite material, in a laboratory testing system. *Ann Occup Hyg*. 2016;60(5):638-642. doi:10.1093/annhyg/mew005
25. Kang S, Liang H, Qian Y, Qi C. The composition of emissions from sanding Corian® with different sandpapers. *Aerosol Air Qual Res*. 2021;21(2):200377. doi:10.4209/aaqr.2020.07.0377
26. Occupational Safety and Health Administration. OSHA method ID-142. Quartz and Cristobalite. 2016. Accessed April 20, 2023. https://www.osha.gov/sites/default/files/methods/osha-id142.pdf
27. Occupational Safety and Health Administration. OSHA method ID-125G. Metal and Metalloid Particulates in Workplace Atmospheres. 2002. Accessed April 20, 2023. https://www.osha.gov/sites/default/files/methods/id125g.pdf
28. Occupational Safety and Health Administration. OSHA method PV 2121. Gravimetric Determination. 2003. Accessed April 20, 2023. https://www.osha.gov/sites/default/files/methods/osha-pv2121.pdf
29. Chew R, Nigam S, Sivakumaran P. Alveolar proteinosis associated with aluminium dust inhalation. *Occup Med (Lond)*. 2016;66(6):492-494. doi:10.1093/occmed/kqw049
30. Ansari HA, Al-Bahrani G, Vishwanath M, Prescott M, James J. Thoracic scintigraphy in aluminosis: lymph node uptake on bone scan. *Clin Nucl Med*. 2009;34(1):24-26. doi:10.1097/RLU.0b013e31818f441a
31. Cai HR, Cao M, Meng FQ, Wei JY. Pulmonary sarcoid-like granulomatosis induced by aluminum dust: report of a case and literature review. *Chin Med J (Engl)*. 2007;120(17):1556-1560.
32. Kraus T, Schaller KH, Angerer J, Hilgers RD, Letzel S. Aluminosis--detection of an almost forgotten disease with HRCT. *J Occup Med Toxicol*. 2006;1:4. doi:10.1186/1745-6673-1-4
33. Vahlensieck M, Overlack A, Müller KM. Computed tomographic high-attenuation mediastinal lymph nodes after aluminum exposition. *Eur Radiol*. 2000;10(12):1945-1946. doi:10.1007/s003300000534
34. Jederlinic PJ, Abraham JL, Churg A, Himmelstein JS, Epler GR, Gaensler EA. Pulmonary fibrosis in aluminum oxide workers.

Investigation of nine workers, with pathologic examination and microanalysis in three of them. *Am Rev Respir Dis*. 1990;142(5):1179-1184. doi:10.1164/ajrccm/142.5.1179

35. De Vuyst P, Dumortier P, Rickaert F, Van de Weyer R, Lenclud C, Yernault JC. Occupational lung fibrosis in an aluminium polisher. *Eur J Respir Dis*. 1986;68(2):131-140.
36. Miller RR, Churg AM, Hutcheon M, Lom S. Pulmonary alveolar proteinosis and aluminum dust exposure: Case reports (research support, Non-U.S. Gov't). *Am Rev Respir Dis*. 1984;130(2):312-315.
37. Herbert A, Sterling G, Abraham J, Corrin B. Desquamative interstitial pneumonia in an aluminum welder: Case reports. *Hum Pathol*. 1982;13(8):694-699.
38. Vallyathan V, Bergeron WN, Robichaux PA, Craighead JE. PUlmonary fibrosis in an aluminum arc welder. *Chest*. 1982;81(3):372-374. doi:10.1378/chest.81.3.372
39. Musk AW, Greville HW, Tribe AE. Pulmonary disease from occupational exposure to an artificial aluminium silicate used for cat litter. *Br J Ind Med*. 1980;37(4):367-372.
40. Chen WJ, Monnat RJ, Jr. Chen M, Mottet NK. Aluminum induced pulmonary granulomatosis. *Hum Pathol*. 1978;9(6):705-711. doi:10.1016/s0046-8177(78)80053-7
41. McLaughlin AI, Kazantzis G, King E, Teare D, Porter RJ, Owen R. Pulmonary fibrosis and encephalopathy associated with the inhalation of aluminium dust. *Br J Ind Med*. 1962;19:253-263.
42. Mitchell J. Pulmonary fibrosis in an aluminium worker. *Br J Ind Med*. 1959;16(2):123-125. doi:10.1136/oem.16.2.123

How to cite this article: Corwin C, Waterhouse H, Abraham JL, et al. Interstitial pulmonary disease and aluminum trihydrate exposure: A single case report and detailed workplace analysis. *Am J Ind Med*. 2024;1-13. doi:10.1002/ajim.23564