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Silicosis in Artificial Stone workers – Spectrum of radiological High Resolution CT Chest findings

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Abstract and key words

Silicosis in artificial stone workers has become increasingly recognised in Australia over the last two years, with a large proportion of screened workers showing imaging features of the disease. The spectrum of findings has differed from the classical silicosis previously described, with many features of accelerated disease, including ground glass opacities and progressive massive fibrosis. This cohort of patients presents after exposure to a unique product high in silica and other binding agents, and the patterns of disease on imaging in this cohort are not previously described. This article reviews the radiological features seen in different forms of silicosis seen to date in this Australian cohort.

Keywords

Chest Imaging

Respiratory

Computed Tomography

High resolution CT

Silicosis

Introduction

Silicosis is a disease caused by inhalation of respirable crystalline silica (RCS), and is present throughout the world as one of the most common occupational lung diseases. It is found in workers involved in the cutting of stone and other industries where there is sustained exposure to RCS.

Silica produces a reaction within the lungs when inhaled in tiny fragments, typically less than 10 microns in diameter, and over a sustained period. The majority of cases of silicosis develop after decades of exposure to relatively low amounts of RCS, although heavy exposure over a short period may also cause disease. The natural defences of the lung become overwhelmed, and the burden of RCS in the small airways leads to accumulation in silica-laden macrophages which trigger an inflammatory response and release of radical oxygen species (1) and in some cases, fibrotic reaction around the silica conglomerates to form nodules.

Over time, in some individuals the nodules coalesce, drawing in the surrounding lung parenchyma and forming larger conglomerates. Progressive massive fibrosis (PMF) represents disease which has become progressive even in the absence of ongoing silica exposure, and has a poorer prognosis. Progressive massive fibrosis reduces overall available air exchange due to scarring and lung destruction, but can lead to downstream complications especially pulmonary hypertension and right heart strain.

Workers in mining, quarrying, stonecutting, foundry workers, ceramics and construction have been well documented as being at risk of silica-related lung disease. Outbreaks of silicosis continue to be reported, in industries such as oil fracking (2, 3) and in denim jean sandblasting where development of silicosis is almost inevitable (4). Over the last decade, exposure to RCS through work with artificial stone (also referred to as manufactured or engineered stone) has become a recognised cause of silicosis.

Many radiologists have limited experience in the diagnosis of silica-related lung disease as the prevalence of silicosis had declined over the last half century, due to improved safety programs and use of protective equipment. Excellent review articles have been published discussing the range of occupational lung disease radiological findings (5,6) however there is a recognised underreporting of occupational lung disease by radiologists across the world (7), partly due to disorganised screening programs and inconsistent referral for imaging. In many cases the relevant occupational history may not be stated and therefore not considered as a

potential cause for the patient's presentation. However, artificial stone workers have now been identified as a high risk cohort with a large number of cases identified across Australia in the last year. There has been very limited literature published on the radiological features of artificial stone-related silicosis on high resolution CT (HRCT). This article reviews the imaging features of silicosis, and demonstrates the range of atypical features seen in the artificial stone worker cohort.

Artificial stone

The popularity of artificial stone for kitchen and bathroom benchtops over the last 15 years in Australia has led to its widespread use. Artificial stone is non-porous, cheap, easy to cut and polish, robust and available in a wide array of colours and size. Unlike natural stone materials such as granite and marble, which have a relatively low silica content of 5-30%, artificial stone products contain over 90% silica (8). Polymeric resins are also present, binding the small pieces of silica-rich rock into slabs which are then heat cured. The danger to workers occurs when the slab is cut and ground with power tools, creating tiny respirable silica fragments. Use of dry-cutting methods creates much higher levels of RCS in the air compared to water bath methods to reduce respirable particles.

The relative ease of cutting, polishing and installing this product led to small, unregulated workshops and workers requiring little training prior to starting work. Many workers in this industry were unaware of the risks of RCS inhalation and there have been multiple reports of poor usage of personal protective equipment. Air concentration of RCS has been documented as hundreds or thousands of times the recommended maximum limits (9). In addition, the effects on the lung of short term, very high concentration exposure are thought to be three times greater than longterm lower concentration exposure to the same volume of RCS (10). Compared to natural stone and quartz products, artificial stone appears to trigger a greater free radical reaction in the lung, possibly due to the greater number of metal transition ions in the artificial stone product (11), which in conjunction with higher concentrations of RCS and lack of protective equipment has led to multiple outbreaks of silicosis around the world. Size of particle, including the submicron ultrafine particle, may also increase the risk of developing disease (12). Individual variation in protective equipment use, workshop safety measures and frequency of inspections contributes to difficulty in obtaining an accurate occupational history.

Case reports began emerging in the mid 2000s from around the world of artificial stone workers requiring lung transplant (13, 14). The first cases of artificial stone-related lung disease in Australia were reported in 2015 (15), with several subsequent case reports and small case series published by 2017 (16). Other reports from around the world (17-22) have raised further concerns about the safety of this product. In the last year, screening programs for stonemasons have been established in Australia after concerns were raised about silica-exposed artificial stone workers. Several hundred cases of silicosis have been identified in workers screened so far.

Imaging guidelines

The chest radiograph has been the mainstay of occupational lung disease screening programs across the world. This is particularly true for screening of miners, quarry workers, foundry workers, sandblasters, ceramics workers and stonemasons working with natural stone. The International Labour Organisation (ILO) classification of occupational lung disease on chest radiographs (23) has been regarded as the preferred template for screening, performed by radiologists accredited as B readers by the National Institute of Occupational Safety and Health in the United States.

Of concern with respect to the use of chest radiographs as a primary screening investigation, it has been noted that a large percentage (43%) of artificial stone workers with HRCT features of disease have normal chest radiographs (24). In addition, there is evidence that lung function testing lacks the sensitivity to identify early stages of silicosis (25).

The proportion of workers with accelerated or acute silicosis identified through Australian screening programs to date is high compared to previously screened workers in other industries, and the poorly formed nodules in the rapid form of the disease seem to be less identifiable on the chest radiograph, even when reported ILO standards by an accredited B reader.

For this reason, the use of HRCT as a primary screening examination in conjunction with a baseline ILO x-ray has been proposed. This would be particularly important in those at high risk or with equivocal x-rays, however has been proposed even for those with lower level exposures due to the false negative rates for chest radiographs for early disease. While

established disease cannot currently be reversed, the early detection of disease is of significant importance as elimination or reduction of subsequent exposures may result in slower overall disease progression.

HRCT protocols should include non-contrast supine inspiratory and expiratory acquisitions. Whilst the ALARA principle (as low as reasonably achievable) for radiation dose reduction should inform all protocols, dose minimisation must be balanced against image quality, especially in this setting, where depiction of subtle findings such as ground glass nodules is of paramount importance. Expiratory imaging has been used routinely in screening programs to identify air trapping which is incorporated into the diagnostic decision-making process.

Prone imaging has been felt to be of limited use, and not routinely used in screening programs as findings usually have an upper lobe predominance, with dependant changes uncommon. However prone imaging can be incorporated where necessary, at the discretion of the radiologist.

Patterns of disease

Acute silicosis

Acute silicosis, or silicoproteinosis, has been described rarely in the literature. It is associated with intense exposure to very high concentrations of RCS, such as in sandblasting, with a rapid onset of symptoms and HRCT findings within several years of onset of exposure (26). The pathological appearance of bronchoalveolar lavage often resembles alveolar proteinosis (5,26) and the prognosis has been reported as poor. Workers sandblasting denim jeans have been associated with high mortality rates and near-100% rate of silicosis development (4) with progression occurring without ongoing exposure.

Imaging findings in acute silicosis include widespread ground glass opacity, usually with extensive consolidation and variable presence of centrilobular nodules and crazy paving (5,26).

The artificial stone cohort has contained several patients with a predominantly ground glass appearance, which is typical for acute silicosis. Consolidation, considered typical in acute silicosis from other causes, has not been a feature.

In this cohort, following cessation of RCS exposure, temporal change in the ground glass pattern has followed one of two main courses to date - persistent unchanged ground glass opacities (Figure 1), or obvious improvement. The former is postulated to represent a mild form of acute silicosis.

Simple silicosis

This form of silicosis usually develops after 10-20 years of exposure, and is often asymptomatic. The HRCT parenchymal features of simple silicosis are small nodules, between 2mm and 5mm in diameter, predominantly centrilobular but with some peripheral migration or perilymphatic distribution (6). Some patients show both solid (Figure 2a, 2b) and ground glass (Figure 3a, 3b) nodules. The nodules have an upper zone predominance, often in the posterior aspects of the upper lobes, best appreciated on coronal reconstructions, and may be calcified. Peripheral nodules may coalesce into pseudoplaques, without representing true pleural based disease.

Pleural effusions occur in up to 11% of simple silicosis patients (27), and overlap of exposure with other mineral dusts including asbestos may lead to pleural thickening, pleural plaque disease, and occasionally rounded atelectasis (27, 28), along with features of fibrosis seen in asbestosis.

Up to 10% of affected patients will show features of interstitial fibrosis, most commonly with a usual interstitial pneumonia (UIP) pattern and a lower zone predominance (29), with the earliest signs of interstitial fibrosis unhelpful to predict future typical or atypical HRCT patterns for UIP (30).

In addition to coronal reconstructions, maximum intensity projection (MIP) images in the coronal plane can be extremely useful in detecting subtle disease, determining zonal distribution and assessing disease progression (Figure 2b).

Complicated silicosis

This form of silicosis occurs when small nodules in simple silicosis coalesce into larger conglomerates with diameter of 10mm or greater, also known as progressive massive fibrosis (PMF). The reporting of nodule coalescence on HRCT is important, even if not meeting criteria for PMF, as this demonstrates early development towards the complicated form of the disease.

PMF is more likely to occur in silicosis than other pneumoconiosis, such as coal workers pneumoconiosis, as silica demonstrates greater fibrogenic properties (5). PMF conglomerates typically occur in the posterior parts of the upper lobes, are relatively symmetric and may be heavily calcified (Figure 4a, b, 5 a, b). Except in end stage cases, there remain background nodules in the unaffected parenchyma. Severe PMF causes indrawing of large volumes of lung parenchyma and may incorporate nearly all the silicosis nodules.

Traction on adjacent parenchyma may lead to associated paracicatricial emphysema. The emphysematous changes may be more important in lung function decline than the size of the PMF lesion (31). Importantly however, symptomatology is not necessarily clearly related to radiological findings, especially in young workers.

PMF may show avidity on PET CT imaging (Figure 5 c) and therefore is not, of itself, useful in distinguishing PMF from lung cancer. There is no evidence for a reliable avidity level to distinguish between PMF and lung cancer in silica-exposed workers.

In the largest published dataset to date from the Australia artificial stone cohort, 21% of affected workers had PMF (24) although the reported proportion to screening programs has been lower (14%) (9).

Accelerated silicosis

This rapidly progressive form is defined as development of silicosis, either simple or complicated, within 10 years of onset of exposure. The artificial stone cohort has shown an alarming number of accelerated silicosis cases compared to natural stone workers who typically develop simple silicosis over a longer period (9, 24). In the currently screened cohort, it is unclear how long workers have had disease as no previous investigation had taken place. Cases of simple silicosis (classified on exposure time of greater than 10 years) may have demonstrated disease before 10 years of exposure (accelerated silicosis) had screening occurred earlier.

The distinction between accelerated and simple silicosis may be of importance as potential different rates of progression between the two cohorts may influence treatment, development of health policy and allocation of resources to complex and expensive therapeutic options such as lung transplantation.

Different stages of disease may be seen within the same HRCT study. The majority of accelerated silicosis cases seen in the Australian cohort to date have shown features of simple silicosis, with some showing additional ground glass opacities, either in a diffuse (Figure 6a) or centrilobular distribution. Ground glass opacities, adjacent to small discrete nodules and pseudoplaques have been demonstrated (Figure 6b, 6c) in the same scan. A small number (usually younger patients) have had PMF (Figure 6d).

It is postulated that this array of findings reflects the rapid nature of the disease, occurring after as little as 2 years of exposure.

Other reports from around the world corroborate the Australian experience and are consistent with the shorter latency and more rapid progression seen in artificial stone workers than in other stonemason groups (8, 13). In Israeli artificial stone workers, 31 of 82 cases represented accelerated disease (14) with the most common HRCT pattern being a combination of centrilobular +/- perilymphatic nodules, nodal enlargement with or without calcification, associated emphysema and a minority with PMF. The upper zone distribution has been seen in cohorts of workers around the world (13, 14, 17,18).

Auxiliary findings

Nodal changes

The thoracic nodes in the artificial stone workers cohort are highly variable. Nodes may be enlarged or normal in size. They may be of soft tissue density or display diffuse hyperdensity (which may represent early calcification) (Figure 7 a). Calcification may or may not be present; if present it may be egg-shell (Figure 7 b) or coarse (Figure 7 c).

At present, calcified nodes are insufficient for a diagnosis of silicosis. Follow up of this cohort will reveal whether nodal changes are predictive of subsequent parenchymal disease.

Emphysema

It is well recognised that emphysema may develop due to dust exposure in the absence of smoking (32). Anecdotal experience in the Australian artificial stone cohort is that emphysema in young workers with parenchymal nodules is relatively uncommon, except in patients with PMF where traction may contribute to the upper zone emphysematous changes.

When present, the emphysema tends to be upper zone, particularly in the apices, and may be paraseptal or centrilobular.

Pulmonary hypertension and Interstitial fibrosis

The estimated rate of interstitial fibrosis in workers with silicosis is 11% (29) with variable features including the UIP pattern of fibrosis. Reporting of fibrotic features is recommended for HRCT studies in these workers, although no data are yet available on their prevalence in this cohort.

In addition, because fibrosis confers a risk of pulmonary hypertension, main pulmonary artery diameter is recommended to be measured during HRCT reporting.

No data is yet available on the prevalence of UIP or other lower zone predominant fibrotic patterns in the Australian screening cohort.

Lung cancer

Exposure to inorganic dust is a risk factor for lung cancer (33). Workers exposed to decades of RCS have a higher likelihood of developing lung cancer compared to the general population, especially if also a smoker (33), often with background emphysematous changes alongside the silicosis changes. Although no cases of lung cancer have been identified in the artificial stone cohort to our knowledge, there is no reason to believe that this cancer risk will not be present in this group.

Limited information is available epidemiologically regarding the length of time post cessation of exposure that the worker remains at increased risk of developing malignancy. This information would assist in developing screening program guidelines.

Associated Diseases

Sarcoidosis

The nodal and parenchymal changes of silicosis can be difficult to distinguish from sarcoidosis, which also manifests as enlarged mediastinal and hilar nodes, sometimes calcified, on background parenchymal nodularity and scarring.

Sarcoidosis is an inflammatory systematic disease, forming granulomas in multiple organs. The cause is uncertain, postulated to be related to both genetic factors and environmental exposures; silica exposure may be a trigger in stone workers (34, 35). The presence of silica particles in sarcoid granulomas, and the regression of sarcoid symptoms upon cessation of silica exposure, suggests a link in susceptible individuals.

Radiologically, it is increasingly difficult to separate the features of the two diseases, especially in patients with nodal enlargement. Two patterns may help distinguish the two disease processes:

1. Centrilobular ground glass nodules are rare in sarcoidosis, and suggest silicosis.
2. Perilymphatic nodules are more common in sarcoidosis.

Ultimately, response to therapy may determine the final diagnosis.

Silicotuberculosis

The risk of developing tuberculosis infection in silica-exposed individuals is many times greater than in the general population (36, 37), although no data is yet available about TB infection in the artificial stone cohort. Non-tuberculous mycobacterial infection (NTMI) also has a higher prevalence. Quantiferon testing in patients with silicosis is therefore often performed prior to commencement of immunosuppressant therapy.

Hypersensitivity pneumonitis (HP)

The acute and subacute stages of silicosis may manifest radiologically as predominantly ground glass nodules in the upper zones, resulting in radiological overlap with hypersensitivity pneumonitis. Non-silica components of artificial stone, such as resins or binders, have been postulated to cause HP in these workers, however without supporting data. The variable response of GGO nodules to cessation of dust exposure (including resolution, persistence and progression) does not clarify the situation, as the pathological nature of these nodules is unknown, and probably differs from patient to patient. There is no reliable way to identify what, if any, of this pattern may be due to HP.

No strategy currently exists to identify those patients likely to respond to HP treatment.

Autoimmune diseases

The prevalence of autoimmune diseases such as rheumatoid arthritis and systemic lupus erythematosus (SLE) is increased in silica-exposed patients compared to the general population (38), and may be present without silicosis. There is emerging evidence that silica exposure may favour the development of certain subtypes of SLE over others (38). The rate of

tuberculous infection in silica-exposed patients with autoimmune disease has been reported as high as 36% (37), therefore TB testing in this subset of patients is recommended.

Conclusion

The artificial stone-related silicosis outbreak seen over the last year in Australia corroborates other reports worldwide about the risk of lung disease from artificial stone dust exposure. Familiarity with this disease process and the HRCT findings is vital to diagnosing workers, particularly in accelerated disease, and crucial to facilitating urgent referral to an appropriate respiratory and occupational physician team. Whilst some workers are being screened for silicosis, there are many other patients presenting through other pathways who are also at risk of disease.

HRCT features of silicosis in these workers may differ from those seen in workers exposed to natural stone dust, potentially showing a more rapid development and progression of disease, likely related to extreme exposure to crystalline respirable silica. Features such as ground glass opacity and nodal enlargement, which figure more prominently in this group than in traditional stone workers, carry a differential diagnosis and require further study.

Given the evolving knowledge base of radiological and clinical findings in this cohort, close collaboration with specialist physician colleagues and careful reporting of chest radiographs and HRCT in workers exposed to artificial stone are recommended.

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References:

1. Leso V, Fontana L, Romano R, Gervetti P, Iavicoli I. Artificial Stone Associated Silicosis: A Systematic Review. *Int J Environ Res Public Health*. 2019 Feb 16;16(4). pii: E568. doi: 10.3390/ijerph16040568.

- 338 2. Shtraichman O, Kramer MR: Artificial stone silicosis: The Israel epidemic, current view.
339 Harefuah 156:517-521, 2017
- 340 3. Esswein EJ, Breitenstein M, Snawder J, Kiefer M, Sieber WK. Occupational exposures to
341 respirable crystalline silica during hydraulic fracturing. J Occup Environ Hyg. 2013;10(7):347-56.
342 doi: 10.1080/15459624.2013.788352.
- 343 4. Akgun M, Araz O, Ucar EY, et al. Silicosis Appears Inevitable Among Former Denim
344 Sandblasters: A 4-Year Follow-up Study. Chest. 2015 Sep;148(3):647-654. doi:
345 10.1378/chest.14-2848.
- 346 5. Batra K, Aziz MU, Adams TN, Godwin JD. Imaging Of Occupational Lung Diseases. Semin
347 Roentgenol. 2019 Jan;54(1):44-58. doi: 10.1053/j.ro.2018.12.005. Epub 2018 Dec 27.
- 348 6. Cox CW, Rose CS, Lynch DA. State of the art: Imaging of occupational lung disease.
349 Radiology. 2014 Mar;270(3):681-96. doi: 10.1148/radiol.13121415.
- 350 7. Mazzei MA, Sartorelli P, Bagnacci G et al. Occupational Lung Diseases: Underreported
351 Diagnosis in Radiological Practice. Semin Ultrasound CT MR. 2019 Feb;. 40(1):36-50. doi:
352 10.1053/j.sult.2018.10.019.
- 353 8. Hoy RF, Baird T, Hammerschlag G et al. Occup Environ Med 2018;75:3–5.
354 doi:10.1136/oemed-2017-104428
- 355 9. Workcover Queensland. Managing respirable crystalline silica in bench top fabrication.
356 Injury prevention safety advice. Updated 22/10/2019. URL available at
357 [https://www.worksafe.qld.gov.au/injury-prevention-safety/workplace-hazards/managing-](https://www.worksafe.qld.gov.au/injury-prevention-safety/workplace-hazards/managing-respirable-crystalline-silica-in-bench-top-fabrication)
358 [respirable-crystalline-silica-in-bench-top-fabrication](https://www.worksafe.qld.gov.au/injury-prevention-safety/workplace-hazards/managing-respirable-crystalline-silica-in-bench-top-fabrication)
- 359 10. Buchanan D, Miller BG, Soutar CA. Quantitative relations between exposure to respirable
360 quartz and risk of silicosis. Occup Environ Med. 2003 Mar;60(3):159-64.
- 361 11. Pavan C, Polimeni M, Tomatis M, Corazzari I, Turci F, Ghigo D, Fubini B. Editor's Highlight:
362 Abrasion of Artificial Stones as a New Cause of an Ancient Disease. Physicochemical Features
363 and Cellular Responses. Toxicol Sci. 2016 Sep;153(1):4-17. doi: 10.1093/toxsci/kfw101. Epub
364 2016 Jun 2.

12. Ophir N, Bar Shai A, Korenstein R, Kramer MR, Fireman E. Functional, inflammatory and interstitial impairment due to artificial stone dust ultrafine particles exposure. *Occup Environ Med*. 2019 Sep 27. pii: oemed-2019-105711. doi: 10.1136/oemed-2019-105711
13. Kramer MR, Blanc PD, Fireman E, et al. Artificial stone silicosis [corrected]: disease resurgence among artificial stone workers. *Chest*. 2012 Aug;142(2):419-424. doi: 10.1378/chest.11-1321. Erratum in: *Chest*. 2012 Oct;142(4):1080.
14. Grubstein A, Shtraichman O, Fireman E, Bachar GN, Noach-Ophir N, Kramer MR. Radiological Evaluation of Artificial Stone Silicosis Outbreak: Emphasizing Findings in Lung Transplant Recipients. *J Comput Assist Tomogr*. 2016 Nov/Dec;40(6):923-927.
15. Frankel A, Blake L, Yates D. Complicated silicosis in an Australian worker from cutting engineered stone: an embarrassing first for Australia. *Eur Respir J* 2015;46:PA1144.
16. Matar E, Frankel A, Blake LKM, Silverstone EJ, Johnson AR, Yates DH. Complicated silicosis resulting from occupational exposure to engineered stone products. *Med J Aust*. 2017 May 15;206(9):385-386.
17. Pérez-Alonso A, Córdoba-Doña JA, Millares-Lorenzo JL, et al. Outbreak of silicosis in Spanish quartz conglomerate workers. *Int J Occup Environ Health* 2014;20:26–32.
18. Pascual S, Urrutia I, Ballaz A, et al. Prevalence of silicosis in a marble factory after exposure to quartz conglomerates. *Arch Bronconeumol* 2011;47:50–1.
19. Martínez C, Prieto A, García L, et al. Silicosis: a disease with an active present. *Arch Bronconeumol* 2010;46:97–100.
20. Paolucci V, Romeo R, Sisinni AG, Bartoli D, Mazzei MA, Sartorelli P. Silicosis in Workers Exposed to Artificial Quartz Conglomerates: Does It Differ From Chronic Simple Silicosis? *Arch Bronconeumol*. 2015 Dec;51(12):e57-60. doi: 10.1016/j.arbres.2014.12.010
21. Friedman GK, Harrison R, Bojes H, Worthington K, Filios M; Centers for Disease Control and Prevention (CDC). Notes from the field: silicosis in a countertop fabricator - Texas, 2014. *MMWR Morb Mortal Wkly Rep*. 2015 Feb 13;64(5):129-30.
22. Mao Ling, Zhou S, Chen Z, et al. Investigation of clinical features and working environment of silicosis patients caused by agglomerated quartz stone processing dust. *Journal of Environmental and Occupational Medicine*, 2019, 36(8): 744-749.
23. International Labour Organisation website
http://www.ilo.org/safework/info/publications/WCMS_168260/lang--en/index.htm

- 397 24. Newbigin K, Parsons R, Deller D, Edwards R, McBean R. Stonemasons with silicosis:
398 Preliminary findings and a warning message from Australia. *Respirology*. 2019 Aug 12. doi:
399 10.1111/resp.13672.
- 400 25. Gamble JF, Hessel PA, Nicolich M. Relationship between silicosis and lung function. *Scand J*
401 *Work Environ Health*. 2004 Feb;30(1):5-20
- 402 26. Marchiori E, Souza CA, Barbassa TG, Escuissato DL, Gasparetto EL, Souza AS Jr.
403 Silicoproteinosis: high-resolution CT findings in 13 patients. *AJR Am J Roentgenol*. 2007
404 Dec;189(6):1402-6
- 405 27. Arakawa H, Honma K, Saito Y, Shida H, Morikubo H, Suganuma N, Fujioka M. Pleural
406 disease in silicosis: pleural thickening, effusion, and invagination. *Radiology*. 2005
407 Aug;236(2):685-93.
- 408 28. Honma K, Shida H, Chiyotani K. Rounded atelectasis associated with silicosis. *Wien Klin*
409 *Wochenschr*. 1995;107(19):585-9.
- 410 29. Arakawa H, Johkoh T, Honma K et al. Chronic interstitial pneumonia in silicosis and mix-
411 dust pneumoconiosis: its prevalence and comparison of CT findings with idiopathic pulmonary
412 fibrosis. *Chest*. 2007 Jun;131(6):1870-6.
- 413 30. Arakawa H, Fujimoto K, Honma K et al. Progression from near-normal to end-stage lungs in
414 chronic interstitial pneumonia related to silica exposure: long-term CT observations. *AJR Am J*
415 *Roentgenol*. 2008 Oct;191(4):1040-5. doi: 10.2214/AJR.07.3871.
- 416 31. de Castro MC, Ferreira AS, Irion KL, Hochegger B, Lopes AJ, Velarde GC, Zanetti G,
417 Marchiori E. CT quantification of large opacities and emphysema in silicosis: correlations
418 among clinical, functional, and radiological parameters. *Lung*. 2014 Aug;192(4):543-51. doi:
419 10.1007/s00408-014-9590-9.
- 420 32. Marchetti N, Garshick E, Kinney GL, et al. Association between occupational exposure and
421 lung function, respiratory symptoms, and high-resolution computed tomography imaging in
422 COPDGene. *Am J Respir Crit Care Med*. 2014 Oct 1;190(7):756-62. doi: 10.1164/rccm.201403-
423 0493OC.
- 424 33. Kurihara N, Wada O. Silicosis and smoking strongly increase lung cancer risk in silica-
425 exposed workers. *Ind Health*. 2004 Jul;42(3):303-14

34. Beijer E, Meek B, Kromhout H, et al. Sarcoidosis in a patient clinically diagnosed with silicosis; is silica associated sarcoidosis a new phenotype? *Respir Med Case Rep.* 2019 Jul 10;28:100906. doi: 10.1016/j.rmcr.2019.100906.
35. Vihlborg P, Bryngelsson I-L, Andersson L, et al. Risk of sarcoidosis and seropositive rheumatoid arthritis from occupational silica exposure in Swedish iron foundries: a retrospective cohort study. *BMJ Open* 2017;7:e016839. doi:10.1136/bmjopen-2017-016839
36. Shafiei M, Ghasemian A, Eslami M, Nojoomi F, Rajabi-Vardanjani H. Risk factors and control strategies for silicotuberculosis as an occupational disease. *New Microbes New Infect.* 2018 Nov 15;27:75-77. doi: 10.1016/j.nmni.2018.11.002.
37. Mizutani RF, Siqueira Lombardi EM, de Paula Santos U, Terra-Filho M. Silica exposure, silicosis, autoimmune diseases, tuberculosis and nontuberculous pulmonary mycobacterial disease. *European Respiratory Journal* 2016 48: PA1171; DOI: 10.1183/13993003.congress-2016.PA1171
38. Pollard KM (2016) Silica, Silicosis, and Autoimmunity. *Front. Immunol.* 7:97. doi: 10.3389/fimmu.2016.0009

Figure Legends

Figure 1.

32 year old male artificial stone worker, presenting for screening after several years of exposure to artificial stone dust. HRCT demonstrated centrilobular upper zone ground glass nodularity. Follow up HRCT 6 months later after cessation of smoking showed no improvement. Case courtesy of Dr Mark Hansen.

Figure 2.

27 year old male with 12 years history of artificial stone dust exposure, identified through screening, asymptomatic. Initial HRCT showed multiple small soft tissue nodules (a), both centrilobular and perilymphatic, without coalescence. Coronal MIP reconstruction clearly demonstrates the upper zone predominance (b). Follow up at 6 months showed no change.

Figure 3.

49 year old stonemason, 19 years exposure including to artificial stone, identified through screening. HRCT showed both soft tissue and ground glass nodules (a, b) with an upper zone predominance. No nodule coalescence. No change on follow up HRCT four months later.

Figure 4.

56 year old male stonemason, several decades of exposure, including artificial stone. HRCT demonstrated upper zone predominant nodularity with coalescence into large mass-like conglomerates (a, b) consistent with PMF.

Figure 5.

34 year old stonemason, exposed to artificial stone for 10 years. HRCT performed as part of screening demonstrates upper zone predominant centrilobular and peripheral nodules (a) with pseudoplaque formation and PMF (b). PET imaging demonstrates avidity in the nodes and the PMF conglomerates (c).

Figure 6.

47 year old stonemason, with 10 years exposure to artificial stone identified through screening. HRCT shows multiple diffuse ground glass opacity (a), with additional solid nodules in an upper zone distribution (b). 29 year old male stonemason, with 7 years of exposure to artificial stone, presenting through screening. HRCT shows upper zone nodularity with formation of pseudoplaques (c) and coalescence of nodules into PMF conglomerates (d).

Figure 7.

Patterns of nodal calcification in artificial stone workers. Enlarged hyperdense nodes (a), eggshell calcification (b), scattered coarse nodal calcifications (c).

Figures

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483 Table

HRCT Feature	Interpretation (in patients with RCS exposure)	Differential diagnosis
Eggshell nodal calcification	Likely to reflect silica exposure	
Enlarged mediastinal and hilar nodes	May represent reaction to RCS exposure.	Sarcoidosis
Upper zone predominant air trapping	Suggestive of hypersensitivity pneumonitis	Respiratory bronchiolitis
Centrilobular ground glass nodules, upper zone predominant	Suggestive of acute/accelerated silicosis	Respiratory bronchiolitis, hypersensitivity pneumonitis
Solid nodules, centrilobular or peripheral, with upper zone predominance	Silicosis (simple or complicated)	Sarcoidosis
Nodule coalescence to >10mm	Progressive massive fibrosis	Lung malignancy

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ara_13015_f2b.jpg

ara_13015_f3a.jpg

ara_13015_f3b.jpg

ara_13015_f4a.jpg

ara_13015_f4b.jpg

ara_13015_f5a.jpg

ara_13015_f5b.jpg

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