PNEUMOCONIOSES
DIAGNOSIS, DIFFERENTIAL DIAGNOSIS AND TREATMENT

Carlos Robalo Cordeiro

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Diagnosis

1 History of sufficient exposure to dust, in time and intensity/either occupational or environmental/markers of exposure

2 Compatible radiological features/structural pathology consistent

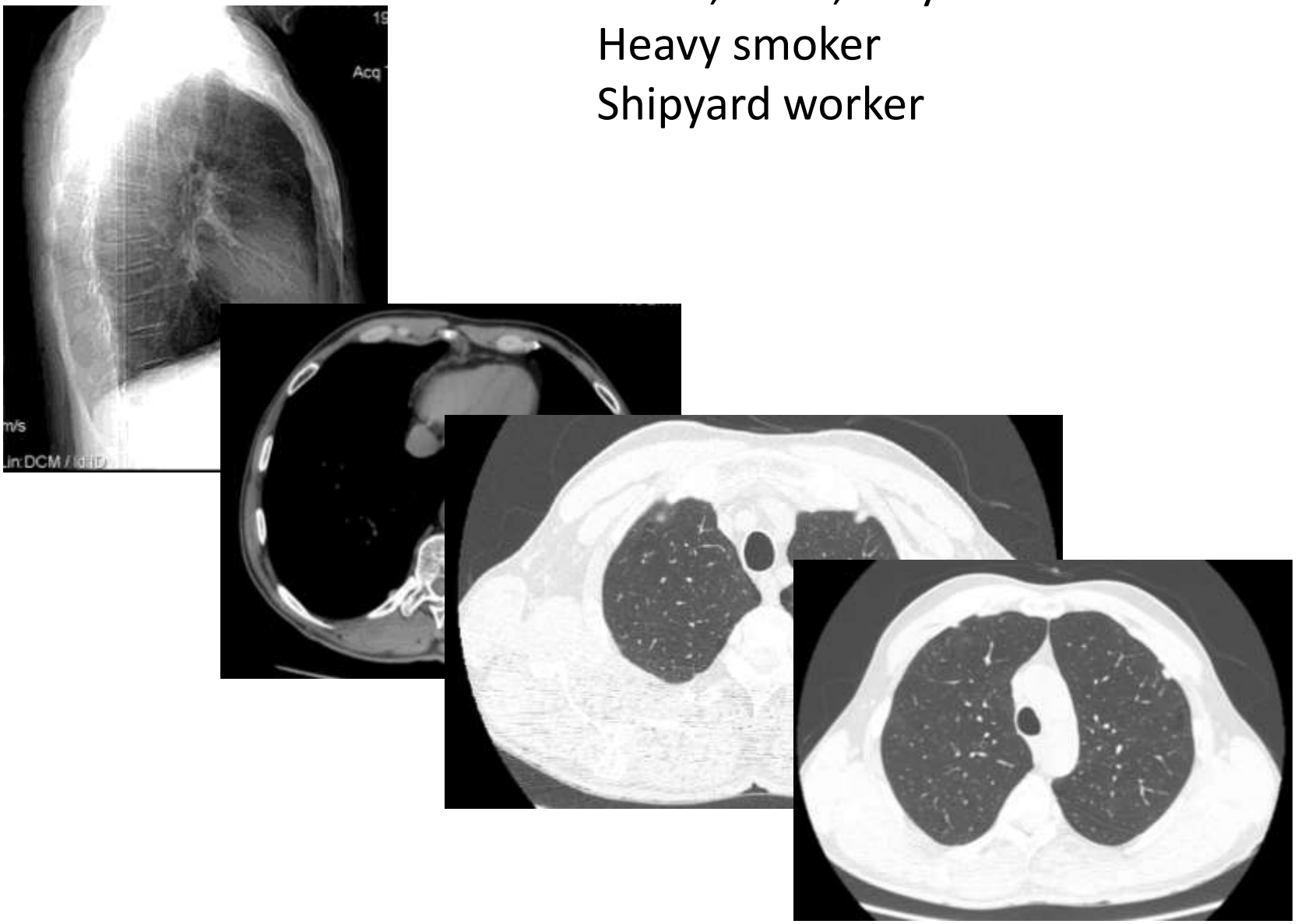
3 Exclusion of other competing diagnosis /confounders/diseases that mimic pneumoconiosis

Diagnosis

1 History of sufficient exposure to dust, in time and intensity/either occupational or environmental/markers of expousure

- Detailed occupational history
- Parents' and spouses'occupations
- Products and manner of manipulation
- Poor appreciation/exageration (compensation)
- Hobbies
- Objective evidence of exposure (pleural plaques/asbestos sputum,BAL)

JMRR, male, 59 years-old
Heavy smoker
Shipyard worker



Bronchoalveolar Lavage in the diagnosis and management of Interstitial Lung Disease

TABLE 5. BAL Cell Patterns in ILD

Lymphocytic Pattern (≥15%)	Neutrophilic Pattern (≥5%)	Eosinophilic Pattern (≥3%)	Plasma Cells	Mast Cells
Sarcoidosis	Infection	EP	HP	HP
HP	ARDS	Drug-induced ILD	Drug-induced ILD	Drug-induced ILD
CBD	AIP	Churg-Strauss syndrome	Chronic EP	IPF
CVD-associated ILD	COP	Hypereosinophilic syndrome	Malignancy	CVD
Drug-induced ILD	DIP	Parasitic infestations	COP	COP
Radiation pneumonitis	IPF	IPF	Infection (Legionella, Pneumocystis)	EP
IIP (NSIP-cellular, COP, IPF)	CVD-associated ILD	CTD-associated ILD		Malignancy
Inflammatory bowel disease	Drug-induced ILD	Pneumocystis pneumonia		Sarcoidosis
Occupational lung disease	HP			
Mycobacterial infection	Occupational lung disease			
Viral pneumonia	Sarcoidosis			
	Aspiration pneumonia			

Keith C Meyer. Clin Pulm Med 2007, 14: 148-156

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Keith C Meyer. Clin Pulm Med 2007, 14: 148-156

Significance of Bronchoalveolar Lavage for the Diagnosis of Idiopathic Pulmonary Fibrosis

Shinichiro Ohshimo[1], Francesco Bonella[1], Ai Cui[1], Martin Beume[2], Nobuoki Kohno[3], Josune Guzman[4], and Ulrich Costabel[1]

Rationale: According to the 2002 ATS/ERS Consensus Classification, a confident diagnosis of idiopathic pulmonary fibrosis (IPF) without surgical lung biopsy is made with consistent clinical/physiological findings and the typical features on high-resolution computed tomography (HRCT). Bronchoalveolar lavage (BAL) and/or transbronchial biopsy, one of four major criteria in the 2000 ATS/ERS IPF Statement, was no more essential in the diagnostic algorithm of 2002 ATS/ERS Consensus Classification.

Objectives: To evaluate the additional utility of BAL for the diagnosis of IPF.

Methods: A total of 101 patients with suspected IPF on HRCT were studied. Twenty-seven patients were excluded because of lack of functional impairment (n = 20), an underlying condition causing fibrosis (n = 5), or a clinical history inconsistent with IPF (n = 2). The remaining 74 patients met all the criteria recommended in the 2002 ATS/ERS Consensus Classification for making a diagnosis in the absence of surgical biopsy. The final diagnosis was made with further examinations, including pathological analysis, in patients who showed inconsistent findings for IPF on BAL.

Measurements and Main Results: A cut-off level of 30% for lymphocytes in BAL demonstrated a favorable discriminative power for the diagnosis of IPF. Six of the 74 patients (8%) showed a lymphocytosis of 30% or greater in BAL. Their final diagnoses were idiopathic nonspecific interstitial pneumonia (n = 3) and extrinsic allergic alveolitis (n = 3). The change in perception of the diagnosis was validated by a surgical biopsy in two cases and by subsequent outcome in four cases.

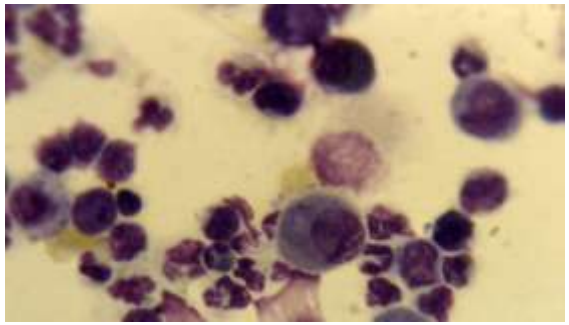
Conclusions: BAL lymphocytosis changed the diagnostic perception in six of 74 patients who would have been misdiagnosed as having IPF without BAL.

AJRCCM 2009, 179: 1043-1047

Bronchoalveolar Lavage in Occupational Lung Diseases

Table 5 Bronchoalveolar Lavage Data on Pneumoconioses—Pulmonology Research Centre

Total Cells $\times 10^4$	Macrophages %	Lymphocytes %	Neutrophils %	Eosinophils %	CD4:CD8
20.2 ± 12.3	68.2 ± 22	22 ± 12.6	12.8 ± 15.2	0.2 ± 0.6	1 ± 0.9



C Robalo Cordeiro et al. Sem Respir Crit Care Med 2007, 28 : 504-513

Bronchoalveolar Lavage in Interstitial Lung Disease

Table 2. Diagnostic bronchoalveolar lavage findings

Bronchoalveolar lavage finding	Diagnosis
Pneumocystis carinii, fungi, CMV transformed cells	Opportunistic infections
Milky effluent, PAS-positive noncellular corpuscles, amorphous debris, foamy macrophages	Alveolar proteinosis
Hemosiderin-laden macrophages, intracytoplasmic fragments of red blood cells in macrophages, free red blood cells	Alveolar hemorrhage syndrome
Malignant cells of solid tumors, lymphoma, leukemia	Malignant infiltrates
Dust particles in macrophages, quantifying asbestos bodies	Dust exposure
Eosinophils >25%	Eosinophilic lung disease
Positive lymphocyte transformation test to beryllium	Chronic beryllium disease
CD1 positive Langerhans cells increased	Langerhans cell histiocytosis

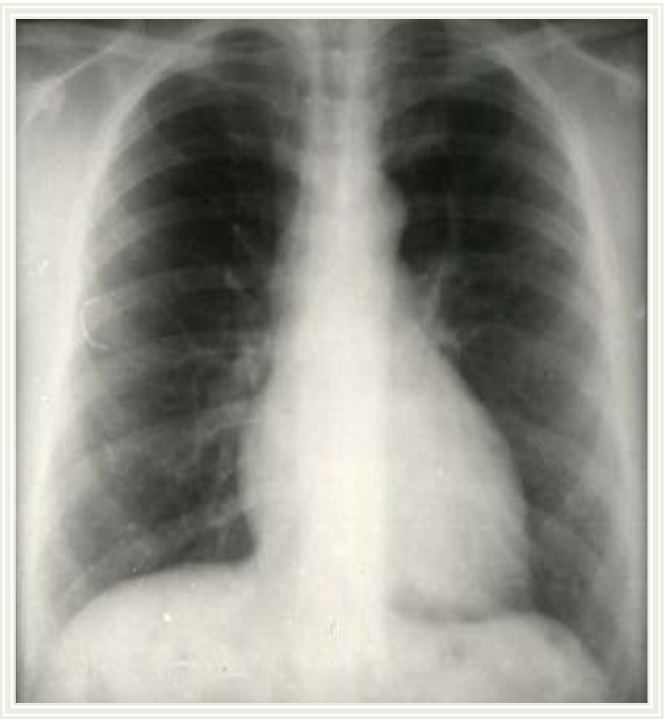
CMV, cytomegalovirus; PAS, periodic acid-Schiff.

Costabel U et al. Curr Opin Pulm Med 2001, 7: 255-261

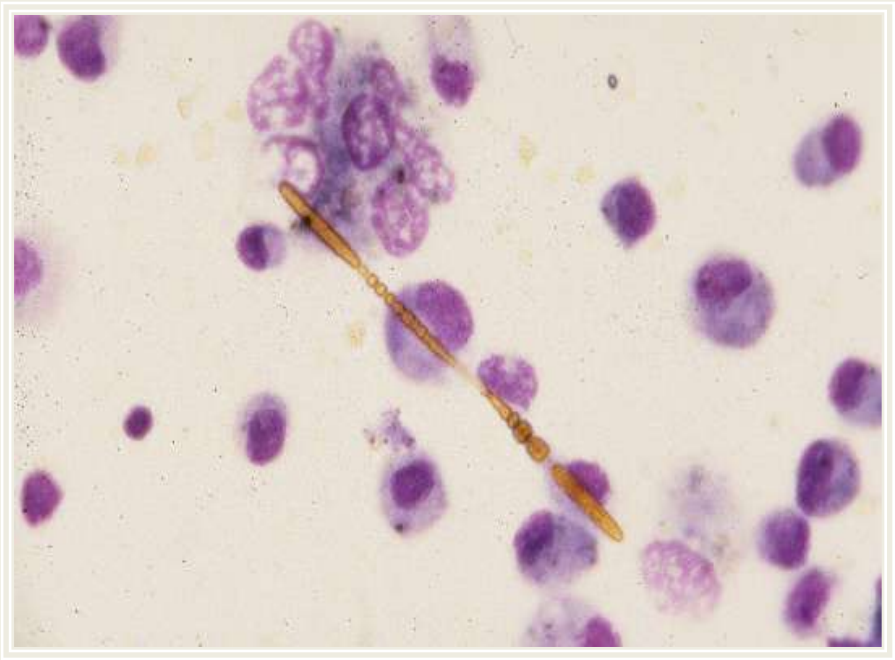
Occupational Diseases

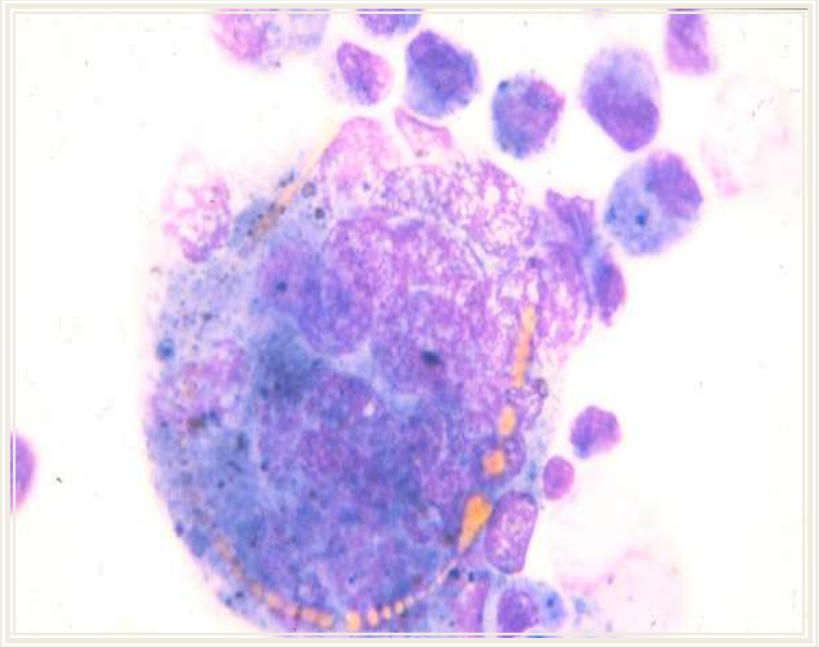
J. C. S., male, 62 years-old
Progressive dyspnea and fatigue
Restrictive ventilatory defect

Professional contact with Asbestos



Occupational Diseases





Identification of asbestos bodies > 1/ml

C Robalo Cordeiro et al. Sem Respir Crit Care Med 2007, 28 : 504-513

Diagnosis

2 Compatible radiological features/structural pathology consistent

- ILO X-Ray classification

Silicosis

Chi Chiu Leung, Ignatius Tak Sun Yu, Weihong Chen

	Notes and further scale divisions
Small opacities (<1 cm)	
Four-point major scale for profusion	
0	0/–, 0/0, 0/1
1	1/0, 1/1, 1/2
2	2/1, 2/2, 2/3
3	3/2, 3/3, 3/+
Round shape and size	
p	≤1·5 mm
q	1·5–3 mm
r	3–10 mm
Irregular shape and size	
s	≤1·5 mm
t	1·5–3 mm
u	3–10 mm
Large opacities (>1 cm)	
A	≤5 cm
B	5 cm to the size of the right upper zone
C	Bigger than the right upper zone

Grades given on the basis of comparison with standard films. Classifications from the International Labour Organization.[85]

Table 2: **Radiographical classification of silicosis**

Lancet 2012; 379: 2008–18

Diagnosis

2 Compatible radiological features/structural pathology consistent

- ILO X-Ray classification

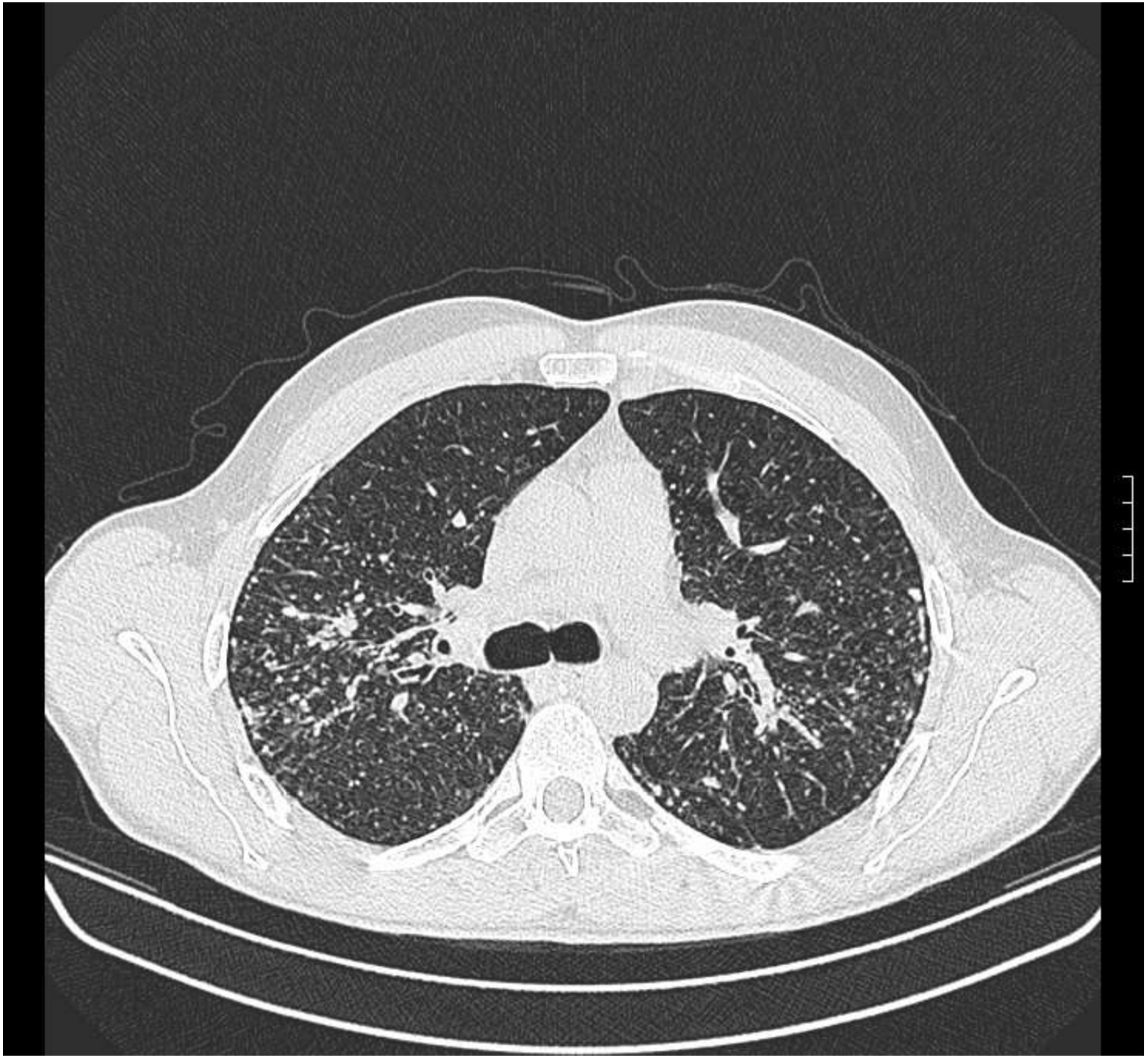
  B reader

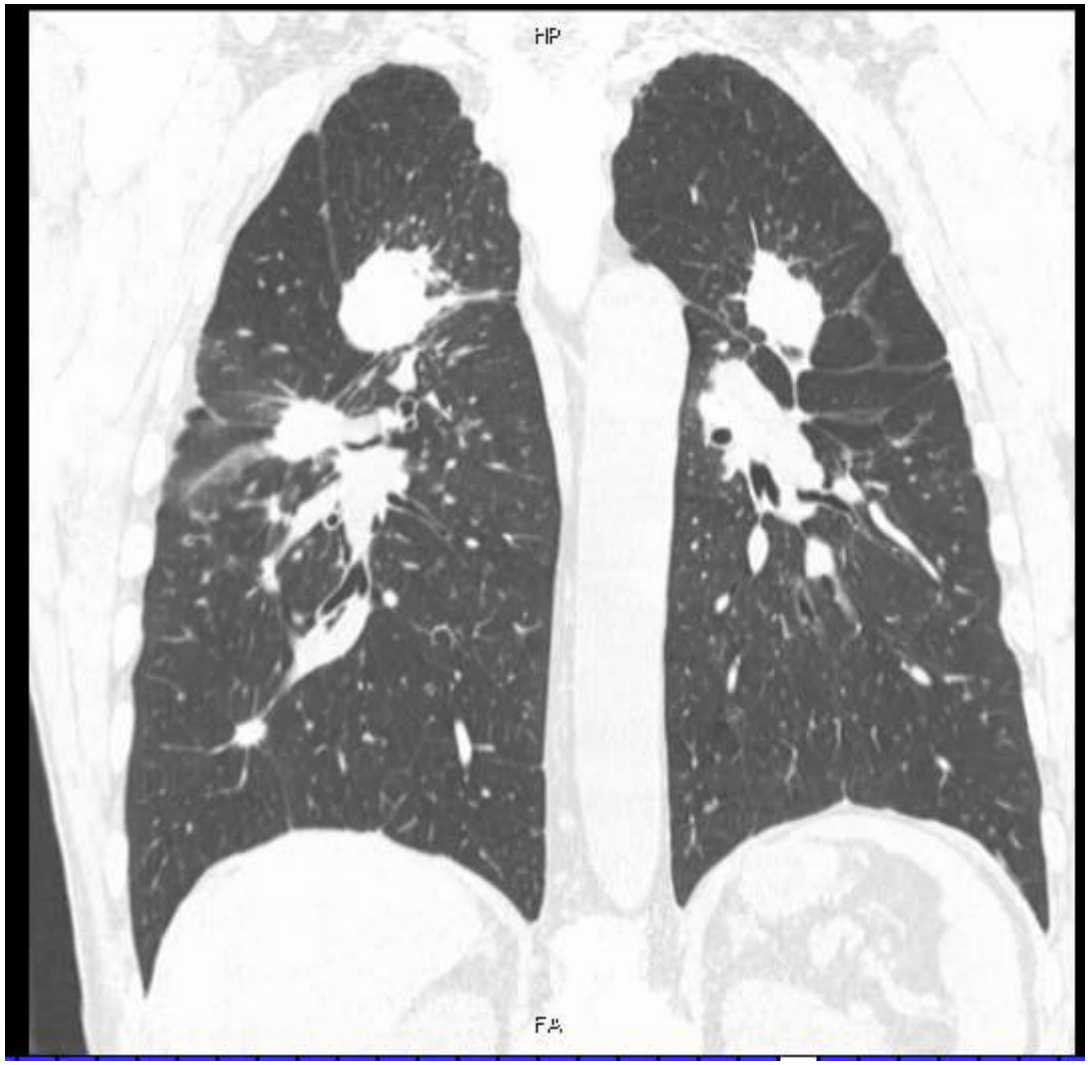
- Digital chest images

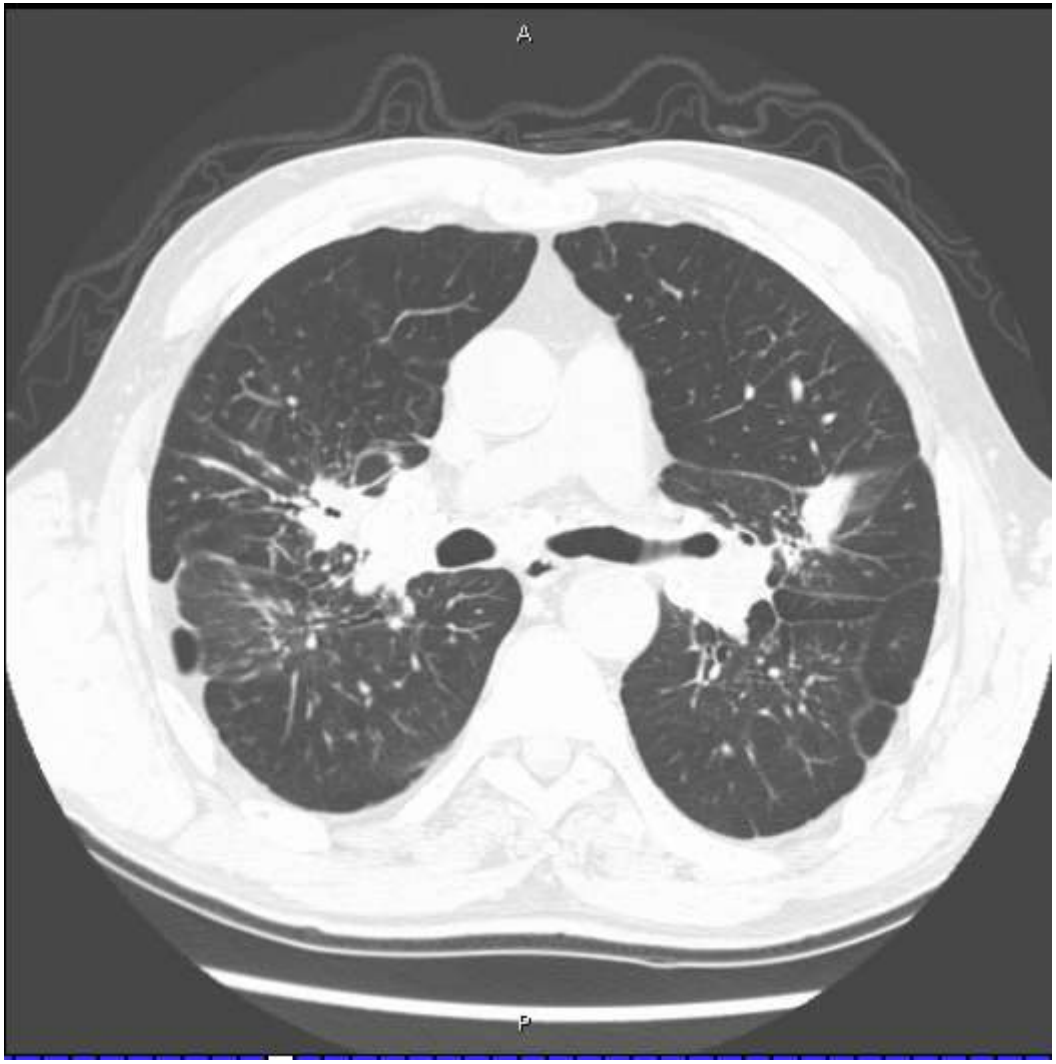
  ILO classification?

- HRCT scan

  more sensitive in specific features, differential diagnosis




HP
FA


A
P


A

Comparison of chest radiography and high-resolution computed tomography findings in early and low-grade coal worker's pneumoconiosis

Ahmet Savranlar[a,*], Remzi Altın[b], Kamran Mahmutyazıcıoğlu[a], Hüseyin Özdemir[a], Levent Kart[b], Tülay Özer[a], Sadi Gündoğdu[a]

Table 1
Comparison between chest radiography and HRCT according to profusion and shape of the lesions

Profusion	Shape	Number of patients	HRCT +	HRCT −	Discordance rate
Normal					
0/0	Total	10	6	4	6/10 (60%)
Early pneumoconiosis					
0/1	Rounded	4	2	2	
	Irregular	2	1	1	
	Total	6	3	3	
1/0	Rounded	6	4	2	
	Irregular	2	1	1	
	Total	8	5	3	
1/1	Rounded	14	10	4	
	Irregular	5	3	2	
	Total	19	13	6	
Total		33	21	12	12/33 (36%)
Low-grade pneumoconiosis					
1/2	Rounded	12	11	1	
	Irregular	4	3	1	
	Total	16	14	2	
2/2	Rounded	6	6	0	
	Irregular	2	2	0	
	Total	8	8	0	
Total		24	22	2	2/24 (8%)

European Journal of Radiology 51 (2004) 175–180

Diagnosis

2 Compatible radiological features/structural pathology consistent

- Multidetector CT

  Denim-sandblasting-induced silicosis

- Low dose HRCT

  Lung cancer screening

- MRI

  Distinguish between progressive massive fibrosis and lung cancer

- PET scan

  Differentiate active inflammation and lung cancer from chronic changes

MDCT Findings of Denim-Sandblasting-Induced Silicosis: a cross-sectional study

Cihan Akgul Ozmen*[1], Hasan Nazaroglu[1], Tekin Yildiz[2], Aylin Hasanefendioglu Bayrak[1], Senem Senturk[1], Gungor Ates[2] and Levent Akyildiz[2]

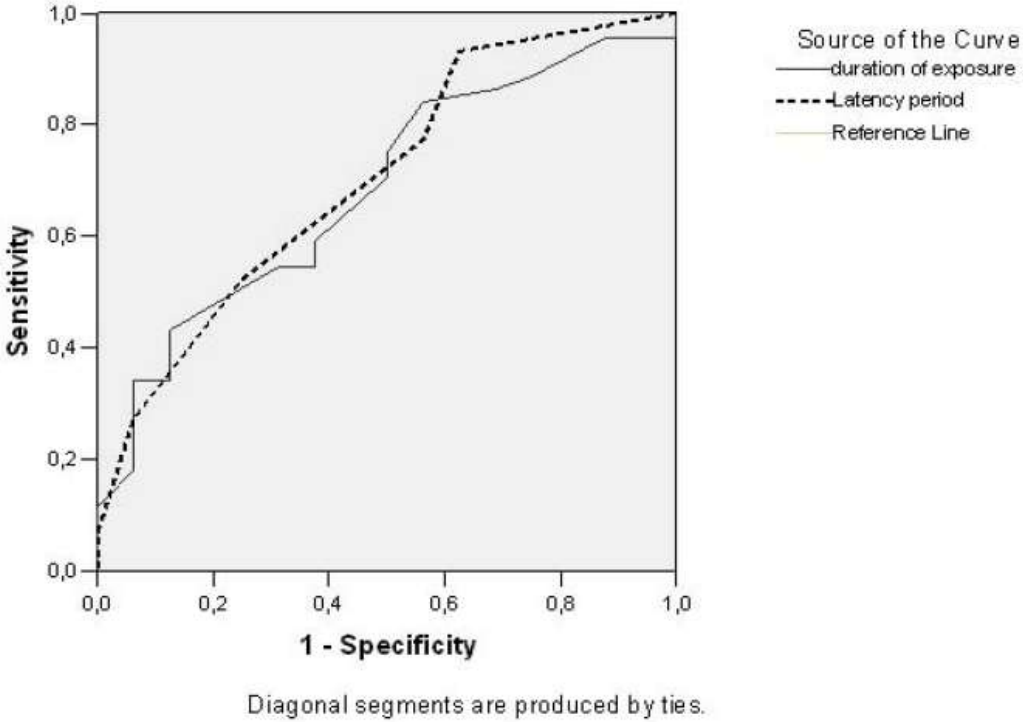


Figure 1 The areas under the curve (AUC) for the duration of exposure to denim sandblasting (line) and the latency period (dashed line) were 0.703 and 0.683, respectively.

Conclusions: The duration of exposure and the latency period are important for development of silicosis in denim sandblasters. MDCT is a useful tool in detecting findings of silicosis in workers who has silica exposure.

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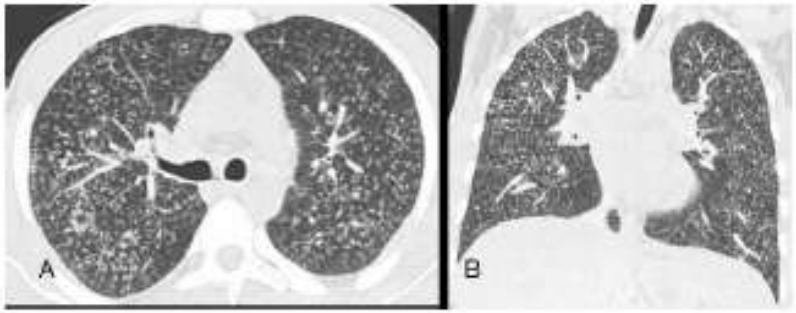


Figure 2 a, b - Diffuse centrilobular nodules on 5-mm-thick reconstructed axial (a) and coronal maximum intensity projection (MIP) images (b) in a 24-year-old man who worked as a denim sandblaster for 48 months.

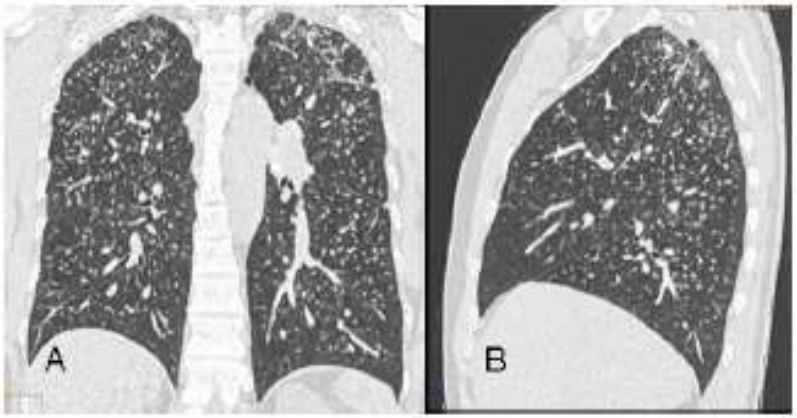


Figure 3 a, b - Coronal (a) and sagittal (b) reconstructed computed tomography images showing the predominance of nodules located in the upper lobes and peripheral regions of the lungs in a 29-year-old man who worked as a denim sandblaster for 20 months.

Conclusions: The duration of exposure and the latency period are important for development of silicosis in denim sandblasters. MDCT is a useful tool in detecting findings of silicosis in workers who has silica exposure.

Diagnosis

3 <u>Exclusion of other competing diagnosis</u> /confounders/diseases that mimic pneumoconiosis

- IPF (smoking related and other ILD), CTD, Infections (miliary tuberculosis, fungal), Carcinomatosis,…

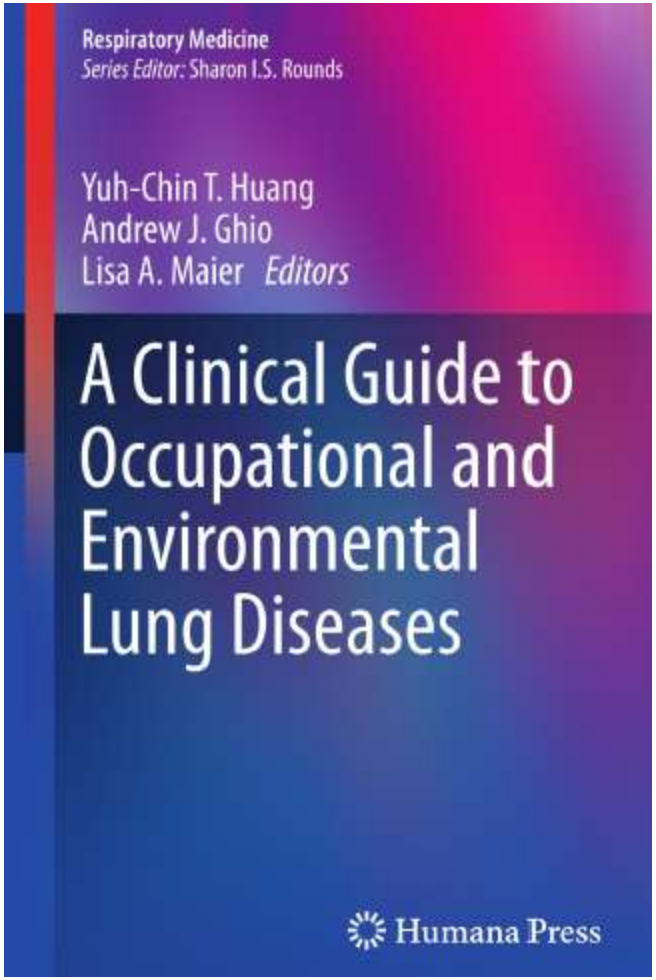
- Confounders

  Cigarette smoking

  Granulomatous disease

  …

Diagnosis



Medical history

Physical examination

Pulmonary function tests → Spirometry, lung volumes, DLCO

Arterial blood gas

Chest X-ray- posteroanterior and lateral

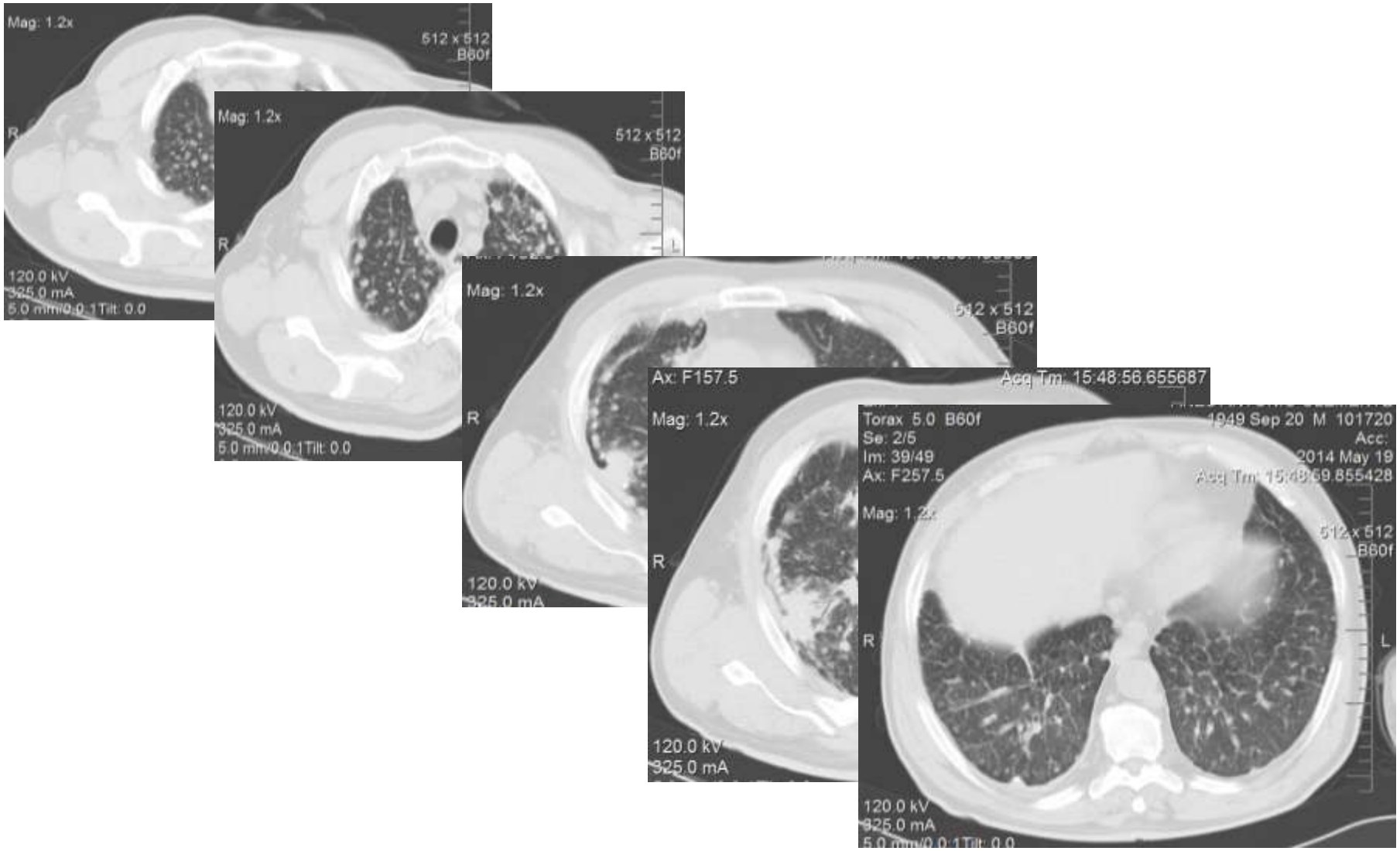
↓

CT scan of the chest

Respiratory illness
Past medical history
Occupational history
Smoking history

Clinical Case

- A.C.E, male, 64 years-old
- Granite/stone work
- Diagnosis 12 years ago
- Dispnoea
- Crackles on inspiration
- FVC 51%, DLCO 58%
- Clinically stable
- No treatment


Mag: 1.2x
512 x 512
B60f
120.0 kV
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Mag: 1.2x
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B60f
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Ax: F257.5
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512 x 512
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Complications/Conditions associated

- Silicosis

  Tuberculosis, …

- Coal-worker's pneumoconiosis

  "Rheumatoid shadows" (Caplan's syndrome)

- Asbestosis

  Lung cancer, Pulmonary Hipertension

Silicosis

Chi Chiu Leung, Ignatius Tak Sun Yu, Weihong Chen

Panel: **Conditions that have been associated with silica exposure**

Silicosis
- Chronic silicosis[16,25-29]
- Accelerated silicosis[16]
- Silicoproteinosis[16]

Infections
- Tuberculosis (pulmonary and extrapulmonary)[16,35-39]
- Other mycobacterial, fungal, and bacterial lung infections (usually with silicosis)[16,35]

Airway disease
- Chronic obstructive pulmonary disease[16,40-44]

Malignant disease
- Lung cancer[16,45-55]
- Gastric, oesophageal, and several others (possible association)[16]

Autoimmune diseases
- Scleroderma[16,56]
- Rheumatoid arthritis[16,56]

Renal diseases
- Chronic renal disease[16]

Lancet 2012; 379: 2008–18

Silicosis

Chi Chiu Leung, Ignatius Tak Sun Yu, Weihong Chen

	Notes
LTBI periodic screening*	
Tuberculin skin test[36]	Cutoff of 10 mm Possible interference from BCG vaccination Booster effect on serial testing
Interferon-γ release assay (eg, T-SPOT.TB)[110]	T-SPOT.TB predicted tuberculosis more accurately than did the tuberculin skin test in patients with silicosis in one study[110]
LTBI treatment[111]	
Isoniazid for 6–12 months	Recommended regimen
Rifampicin for 3–4 months	Alternative regimen
Isoniazid and rifampicin for 3 months	Alternative regimen
Tuberculosis screening	
Periodic chest x-ray screening in areas with high prevalence[112]	Compare serial films and look for features such as cavity, effusion, consolidation, and rapid or focal deterioration
Bacteriology when clinically suspected	Smear not sensitive enough Culture takes time, but more sensitive than is smear Identification required to exclude other mycobacteria Drug susceptibility assays when drug resistance suspected
Rapid molecular testing	For rapid diagnosis and detection of rifampicin resistance
Tuberculosis treatment	
Usual anti-tuberculosis drugs with directly observed therapy	Extended duration of 8 months recommended (to reduce chance of relapse)[113]

LTBI=latent tuberculosis infection. *Frequency depends on risk of infection.

Table 3: **Recommended measures for detection and treatment of LTBI and tuberculosis in patients with silicosis**

Lancet 2012; 379: 2008–18

Treatment

- No proven curative treatment for silicosis exists
- No evidence that any intervention alters the course of asbestosis

Silicosis - Treatment

- Inhalatory Therapy

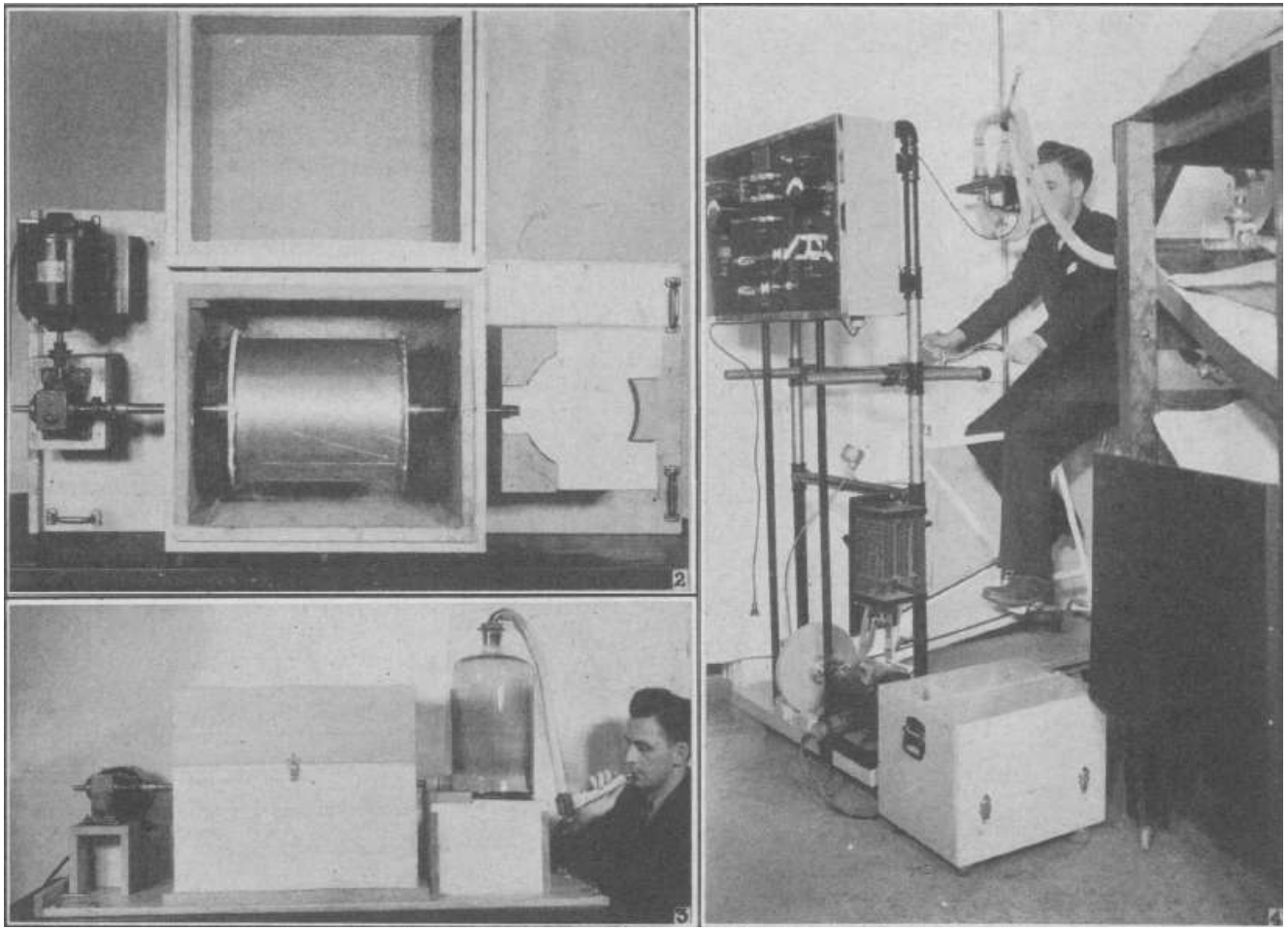
  Alluminium

  Tetrandrine

  Polivinyl-pyridine-N-oxide
- Whole lung lavage
- Steroids/Immunossupressors/Pirfenidone (?)
- 02
- Lung Transplant

THE TREATMENT OF SILICOSIS BY ALUMINUM POWDER*

By D. W. Crombie, M.D., C.M.,
J. L. Blaisdell, B.Sc., M.D. and
G. MacPherson, M.A.



[Can. M. A. J.
April 1944, vol. 50

AMERICAN JOURNAL OF PUBLIC HEALTH July, 1945

ALUMINUM VS. SILICA

IT is now seven years since Denny, Robson, and Irwin at Toronto [1] first pointed out that the addition of metallic aluminum prevented the solubility of siliceous materials *in vitro* and reported that, when rabbits were exposed to quartz dust, they could be protected against silicosis by the addition of 1 per cent of aluminum. Two years later,[2] the same authors presented more detailed and convincing evidence of the importance of this discovery. They demonstrated that metallic aluminum, when hydrated, reduces the toxicity of quartz by flocculation and by adsorption, but chiefly by covering the quartz particles with an insoluble and impermeable coating. They exposed a large series of rabbits for 12 hours a day to natural quartz dust (containing a variable but generally small amount of aluminum); to a mixture of quartz dust and aluminum; and to natural quartz dust for 12 hours plus a 40 minute dusting with aluminum. The percentage of aluminum actually present in the lung was determined.

ALUMINUM VS. SILICA

of silicotic lesions and cause retrogression in immature tissue responses. Inhalation is probably the only effective method of administration. In the doses recommended for human therapy (5 to 30 minutes a day or less frequently), no harmful results are anticipated. Aluminum therapy should be administered only under close medical supervision and should not be used to the exclusion of recognized methods of dust control. Aluminum hydroxide possesses certain theoretical advantages over metallic aluminum and deserves serious clinical trial."

ALUMINIUM POWDER INHALATIONS IN THE TREATMENT OF SILICOSIS OF POTTERY WORKERS AND PNEUMOCONIOSIS OF COAL-MINERS

BY

M. C. S. KENNEDY

Brit. J. industr. Med., 1956, **13**, 85.

ALUMINIUM POWDER INHALATIONS IN THE TREATMENT OF SILICOSIS OF POTTERY WORKERS AND PNEUMOCONIOSIS OF COAL-MINERS

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TREATMENT GROUPS A AND B COMPARED BEFORE TREATMENT

Index	Group A	Group B
Number of patients (male and female)	60	60
Number of female patients	8	7
Mean age (in yr.)	57·1 (35–73)*	56·0 (39–70)
Mean weight (lb.)	144·0 (108–228)	137·6 (98–209)
Mean clinical grade of dyspnoea ..	2·8 (0–4)	3·0 (1–4)
Mean E.T.T. grade†	3·3 (1–7)	3·2 (1–7)
Mean vital capacity (l.)	2·73 (1·50–4·25)	2·68 (1·41–4·06)
Mean M.V.V. (l./min.)	59·1 (22–111)	52·8 (17–98)
Mean E.S.R. (mm./hr. Westergren) ..	12·9 (2–33)	11·2 (2–27)
Number of patients with clinical bronchospasm	15	15
Coalworkers	20	24
Pottery workers	40	36
Simple pneumoconiosis	16	23
Complicated pneumoconiosis	44	37

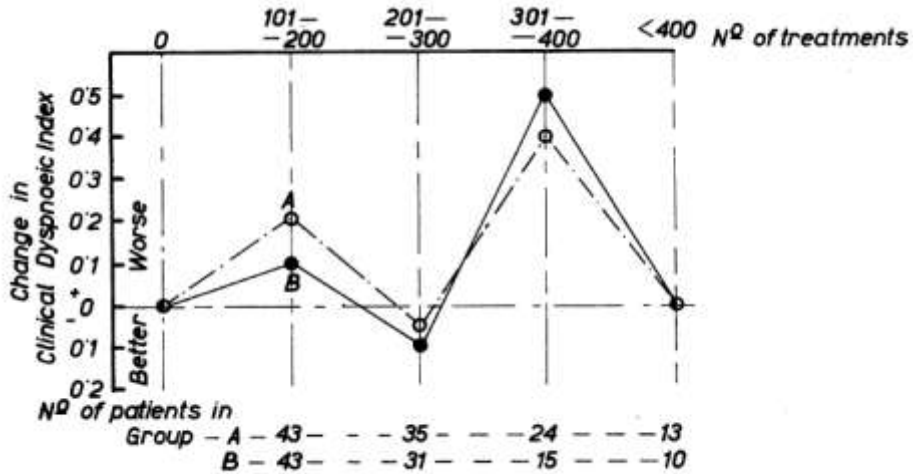
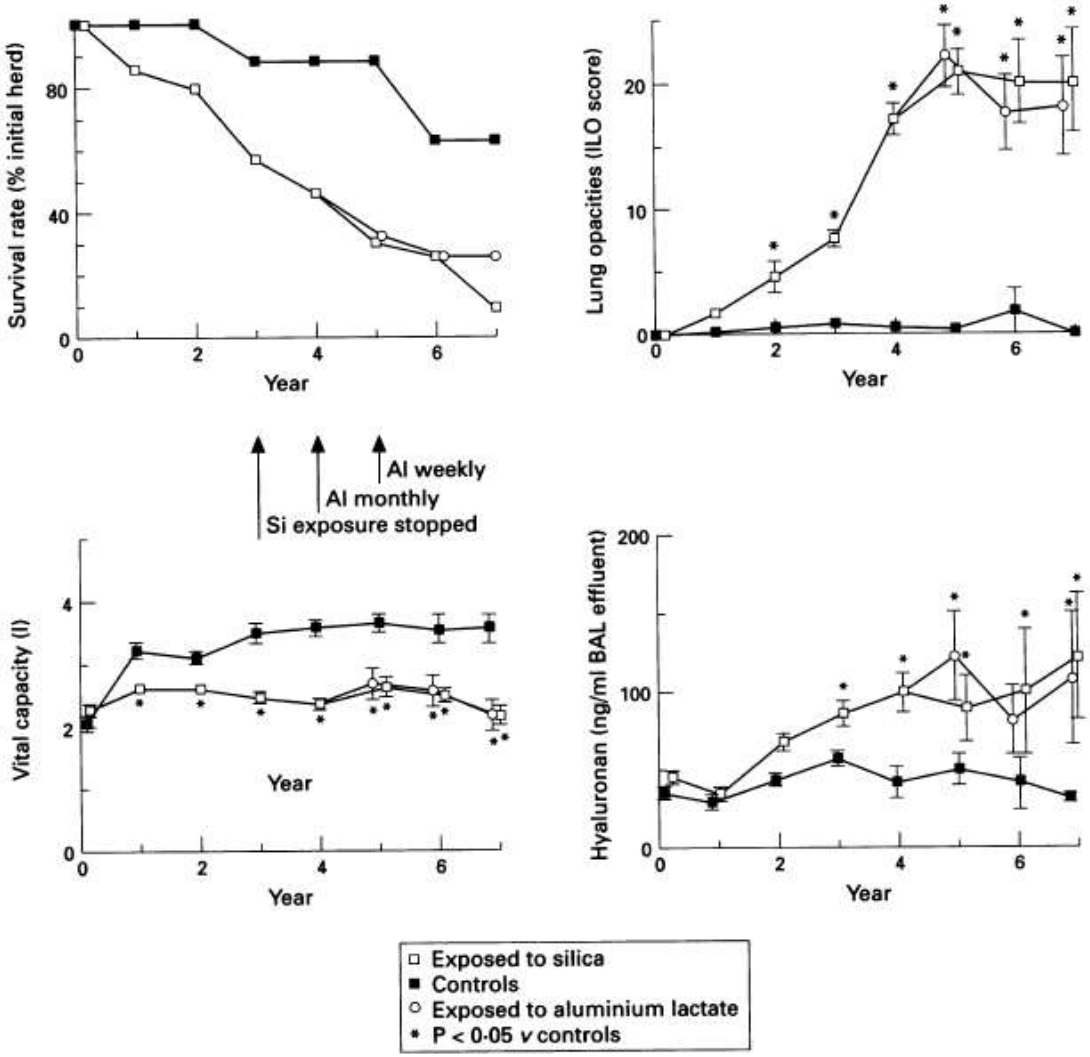


FIG. 3.—Change in clinical grade of dyspnoea in relation to treatment.

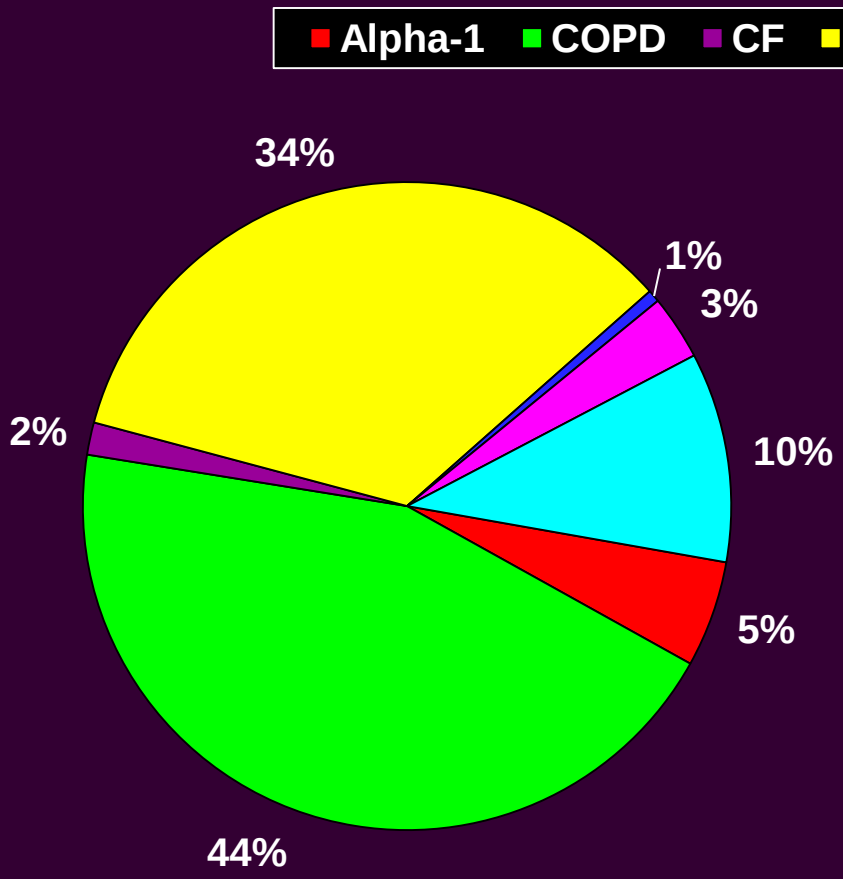
Occupational and Environmental Medicine 1995;**52**:778–780

Further information on aluminium inhalation in silicosis

Raymond Bégin, Serge Massé, André Dufresne

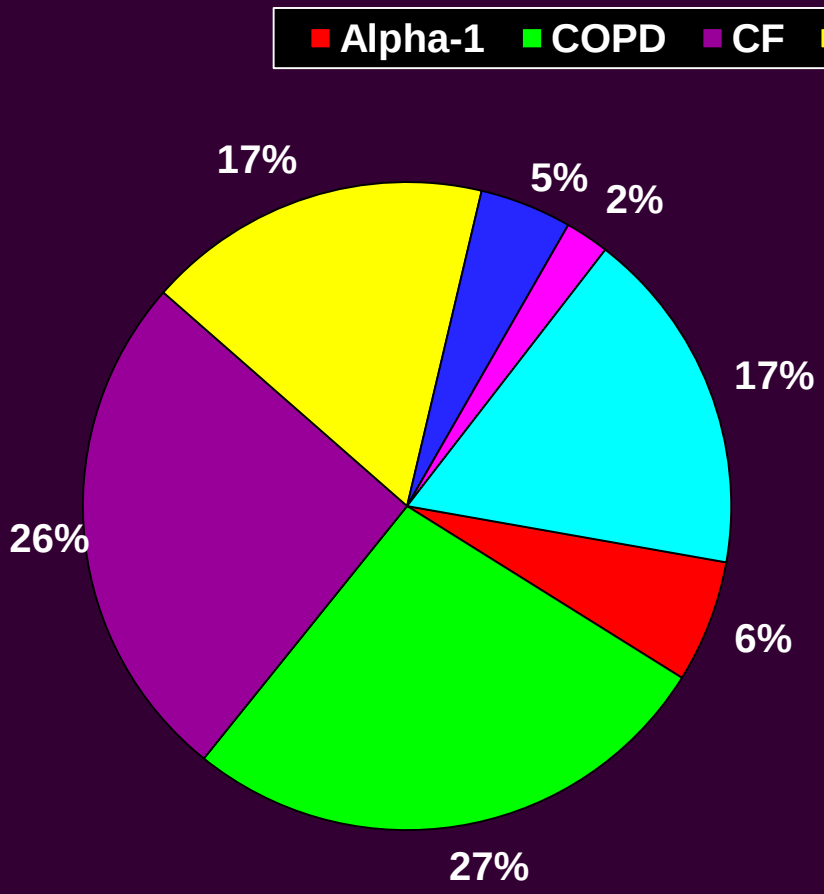


Adult Lung Transplants
Indications for Single Lung Transplants
(Transplants: January 1995 – June 2012)



*Other includes:	
Pulmonary Fibrosis, Other:	4.0%
Bronchiectasis:	0.4%
Sarcoidosis:	1.9%
Connective Tissue Disease:	1.1%
OB (non-ReTx):	0.7%
LAM:	1.0%
Congenital Heart Disease:	0.4%
Miscellaneous:	1.1%

Adult Lung Transplants
Indications for Bilateral/Double Lung Transplants
(Transplants: January 1995 – June 2012)



*Other includes:	
Pulmonary Fibrosis, Other:	3.5%
Bronchiectasis:	4.1%
Sarcoidosis:	2.9%
Connective Tissue Disease:	1.4%
OB (non-ReTx):	1.3%
LAM:	1.1%
Congenital Heart Disease:	1.2%
Miscellaneous:	1.8%

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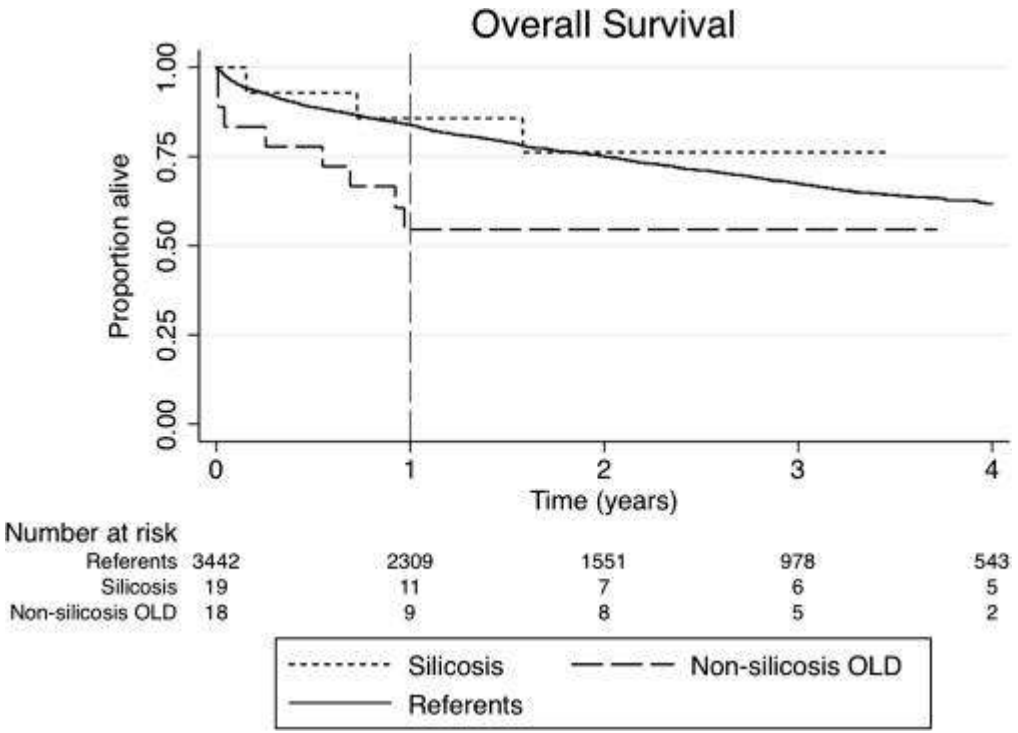
Survival following lung transplantation for silicosis and other occupational lung diseases

Singer JP1, Chen H, Phelan T, Kukreja J, Golden JA, Blanc PD



Occup Med. 2012 Mar;62(2):134-7

Survival following lung transplantation for silicosis and other occupational lung diseases

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Key points

- Lung transplantation for occupational lung diseases is relatively rare in the USA, representing 0.5% of all transplants performed.
- Subjects undergoing lung transplantation for occupational lung diseases appear to be at risk for poorer survival in the first post-transplant year.
- This first post-transplant year risk appears to be restricted to those subjects undergoing lung transplantation for non-silicotic occupational lung diseases.

Occup Med. 2012 Mar;62(2):134-7

Silicosis - Prevention

	Suggested measures
Primary prevention	
Silica exposure control at source	Substitution of materials; modification of processes and equipment; wet methods; silica warning sign; work practices
Control silica dust emission or transmission	Isolation of the source or workers; enclosed processes; air curtain; water spray; local exhaust ventilation; general ventilation system; enclosed cabs; air supply system
Control silica dust at worker level	Training and education about work practices; personal protection; personal hygiene; personal protective equipment; health promotion
Secondary prevention	
Surveillance of working environment	Establish concentration of silica dust; assess health risk for workers exposed to silica dust
Surveillance of worker health	Periodic health examination, such as chest radiography; early detection of the disease; research into biomarkers for early stages of silicosis
Tertiary prevention	Removal from environment; prevention of complications; modification of work processes; rehabilitation

Information taken from National Institute of Occupational Safety and Health.[126]

Table 4: **Suggested preventive measures**

Chi Chiu Leung, Ignatius Tak Sun Yu, Weihong Chen

Lancet 2012; 379: 2008–18