LUNG FUNCTION IN SILICA EXPOSED WORKERS

R. BÉGIN, M.D. • G. Ostiguy, M.D. • A. Cantin, M.D. • D. Bergeron, M.D.
Université de Sherbrooke, Sherbrooke (QC)
Hôpital Maisonneuve-Rosemont, Montréal (QC)

INTRODUCTION

In long term silica-exposed workers, the attribution of changes in lung function to a direct effect of dust exposure or to the development of silicosis has been the subject of considerable debate in the literature and occupational lung conferences. The topic remains to this date a controversial issue, particularly in view of a recent pathological study whicb documented the presence at autopsy of fibrotic lesions and silicotic nodules in men who had normal pre-mortem chest radiographs.[7] Also, clinical studies[4-6] in silica exposed workers documented on lung lavage that a subclinical quartz-induced alveolitis may be present in silica-exposed workers with normal chest radiograph.

In that regard, we have documented that CT scan does not identify more patients with minimal parenchymal disease, although it images the disease more clearly in several cases with significantly higher CT scan score of disease. Also we have clearly demonstrated that CT scan identifies significantly more coalescence and/or large opacities in 33% of patients who were thought to have simple silicosis on the plain chest radiograph. To further investigate the clinical significance of these CT scan observations, we expanded our studied population to 94 long-term silica-exposed workers who were examined concomitantly by standard clinical, radiographic and pulmonary function tests.

SUBJECTS AND METHODS

Silica Exposed Workers

The 94 workers of this study had worked in either the granite or foundry industries or gold mines of Quebec for an average of 29 ± 3 years (range 14–42 years). Ninety percent were either current or former cigarette smokers and they had smoked on the average 21 ± 5 pack-years. Eighty of the 94 were granite workers, 10 were foundry workers and 4 were gold miners.

Pulmonary Function Tests

The lung volumes, pressure-volume curves, flow-volume curves and diffusing capacities were measured according to standard methods[1] as previously applied in our laboratory.[2]

Chest Radiograph

Standard high-kilovoltage posteroanterior, lateral, and oblique films were obtained at maximal inspiration. The radiograph was graded by three observers according to the International Labour Organization (ILO) 1980 classification.[8]

CT Scan of the Thorax

Eighty CT examinations were performed on a General Electric Model 8800 scanner in Sberbrooke (Canadian General Electric, Co., Montréal, Québec) and 14 were done on a Picker 600 scanner (Picker, New York, N.Y.). For each patient, at least 10 slices of 1-cm thickness were obtained with wide windows, and 10 with narrow windows, for adequate assessment of pulmonary, chest wall, and pleural changes. CT scans were obtained within 48–72 hr of the plain chest film.

Subsets of Workers Based on Diagnostic Criteria and CT Scan

The 94 silica exposed workers were divided into 4 categories on the bases of evidence of silicosis and the findings of CT scan of the thorax. Group 1 consisted of 21 workers who did not meet the diagnostic criteria for silicosis.

Group 2 consisted of 28 workers with simple silicosis on chest radiograph and CT scan of the thorax.

Group 3 consisted of 18 workers with simple silicosis on chest radiograph but with coalescence and/or conglomeration on CT scan of the thorax.

Group 4 consisted of 27 workers with complicated silicosis on chest radiograph and CT scan of the thorax.

Statistical Analysis

All results are expressed as the mean ± standard error of measurement. The data were tested by the Student t-test or Mann-Whitney U test for differences between groups, by the Wilcoxon matched-pairs signed-rank test for differences between radiologic methods, and by Spearman's correlation procedure when appropriate.[9,10]

RESULTS

The lung volumes, compliance and change in vital capacity were within normal prediction in group 1. Subjects of group 2 had no significant change in lung volumes but lung compliance was significantly lower than that of group 1. In group 3, the silicotics with coalescence and/or large opacity on CT scan, we found significant reduction in vital capacity, lung compliance and an increased loss of vital capacity/year. The subjects of group 4 had lower total lung capacity, vital capacity, lung compliance and a significantly increased loss of vital capacity per year.

Diffusing capacity was normal in group 1 and decreased gradually with increased disease severity. This was significant in groups 3 and 4. Exercise gas exchange parameters were also significantly reduced in groups 3 and 4. Group 2, patients with simple silicosis had gas exchange parameters between those without obvious disease, group 1, and patients with conglomerate disease, groups 3 and 4. These changes reached significance level for $\dot{V}_E/O_2$ ratio and exercise $\Delta(A\text{-}a)PO_2$.

In workers with radiographic silicosis, group 2, expiratory flow rates were lower than in group 1 and this reduction was more severe in groups 3 and 4, the workers with radiographic and/or CT scan coalescence/conglomeration. The lowest values were in group 4.

DISCUSSION

This study of lung function in long term silica exposed workers documents that the disease severity which is better defined radiographically by CT scan, is also reflected on lung function as restrictive changes. The disease severity also appears to be associated with excessive airflow limitation.

These data document that early coalescence/conglomeration in silicosis as seen often only on CT scan, is associated with worsened lung functions, a finding which strengthens our prior recommendation for the CT scan exam in radiographic simple silicosis.[3] These data also support the concept of a relationship of disease severity, loss of lung function and airflow limitation in silicosis.

REFERENCES

1. Bates, D.V., Macklem, P.T., Christie, R.V.: The normal lung: Physiology and methods of study. In: *Respiratory Function in Disease*, pp. 11–94, 276–280. W.B. Saunders Co., Philadelphia (1971).
2. Bégin, R., Bureau, M.A., Lupien, L., Bernier, J.P., Lemieux, B.: Pathogenesis of Respiratory Insufficiency in Myotonic Dystrophy: The Mechanical Factors. *Am. Rev. Respir. Dis.* 125:312–318 (1982).
3. Bégin, R., Bergeron, D., Samson, L., Boctor, M., Cantin, A.: CT Assessment of Silicosis in Exposed Workers. *Am. J. Roent.* 148:509–514 (1987).
4. Bégin, R., Cantin, A., Boileau, R., Bisson, G.: Spectrum of Alveolitis in Quartz-Exposed Human Subjects. *Chest* 92:1061–1067 (1987).
5. Calhoun, W.J., Christman, J.W., Ershler, W.B., Graham,. W.G.B., Davis, G.S.: Raised Immunoglobulin Concentrations in Bronchoalveolar Lavage Fluid of Healthy Granite Workers. *Thorax* 41:266–273 (1986).
6. Christman, J.W., Emerson, R.J., Graham, W.G.B., Davis, G.S.: Mineral Dust and Cell Recovery from the Bronchoalveolar Lavage of Healthy Vermont Granite Workers. *Am. Rev. Respir. Dis.* 132:393–399 (1985).
7. Craighead, J.E., Vallyathan, N.V.: Cryptic Pulmonary Lesions in Workers Occupational Exposed to Dust Containing Silica. *JAMA* 244:1939–1941 (1980).
8. *International Classification of Radiographs of Pneumoconiosis 1980.* No. 22 revised, International Labour Office/University of Cincinnati, Occupational Safety and Health series. Geneva (1980).
9. Siegel, S.: *Non-parametric Statistics.* pp. 195-240. McGraw-Hill, New-York (1956).
10. Snedecor, G.W., Cochran, W.C.: *Statistical Methods.* Iowa State Univ. Press, Ames, IO (1967).

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