Yonsei Medical Journal
Vol. 44, No. 1, pp. 169 - 173, 2003

Dental Technician's Pneumoconiosis: Mineralogical Analysis of Two Cases

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Pneumoconiosis was diagnosed by open lung biopsy in two dental technicians who had interstitial lung disease. Mineralogical analysis was performed to investigate the origin of the dust that had been inhaled. A marked accumulation of silicon and phosphorus was found in both cases. The hard metals chromium and cobalt were also found. Dental technician's pneumoconiosis is a complex pneumoconiosis in which such dust and hard metals may play a role.

Key Words: Pneumoconiosis, dental technician

INTRODUCTION

Pneumoconiosis, sometimes seen in dental technicians, may be due to inorganic dusts such silica, silicon carbide and asbestos, and metallic dusts such cobalt, molybdenum, beryllium and nickel.[1,2] Acrylics also are known to cause pneumoconiosis.[3]

In this study, two dental technicians who had interstitial pulmonary diseases were investigated by open lung biopsy, in order to diagnose the etiologic minerals.

CASE REPORT

Case 1

A 21-year old man, a soldier for 5 months, presented with symptoms of cough and exercise induced breathlessness for 5 months. He had previously been working as a dental technician in a dental prosthesis laboratory for 8 years. He had no relevant medical history before, nor was there any family history of illness, but he had a history of 5 pack/years of cigarette smoking.

No abnormalities were found on physical examination. The laboratory investigations for total counts of RBC, WBC, platelets and urine analysis were all normal. Other haematological and biochemical data on admission were within normal ranges. He had BCG scars, PPD was positive, AFB in sputum was negative, the serological tests for VDRL, HIV and RF were negative, and CRP was positive.

Chest X-ray showed diffuse micro and reticulonodular infiltration in both hemithoraces (r type, 2/2), and CT scan showed diffuse linear infiltration with patchy soft tissue densities in both hemithoraces, and calcified lymph nodes at both hila and in the subcarinal and mediastinal spaces (Fig. 1A and 1B).

Pulmonary function testing presented the following values: VC, 3.55 L (69% pred); FVC, 2.95 L (60% pred); FEV_1, 2.79 (67% pred); and PEFR, 7.25 L/sec (75% pred). Diffusion capacity was lowered (4.4 mmol/min/kPa, 52% pred).

Plasma immunoglobulin and protein electrophoresis were within normal ranges. Fiberoptic bronchoscopy showed no pathology, while no atypical changes were evident on transbronchial biopsies (TBB) or bronchoalveolar lavage (BAL) specimens taken from the entrance to the right

Received April 17, 2002
Accepted June 24, 2002

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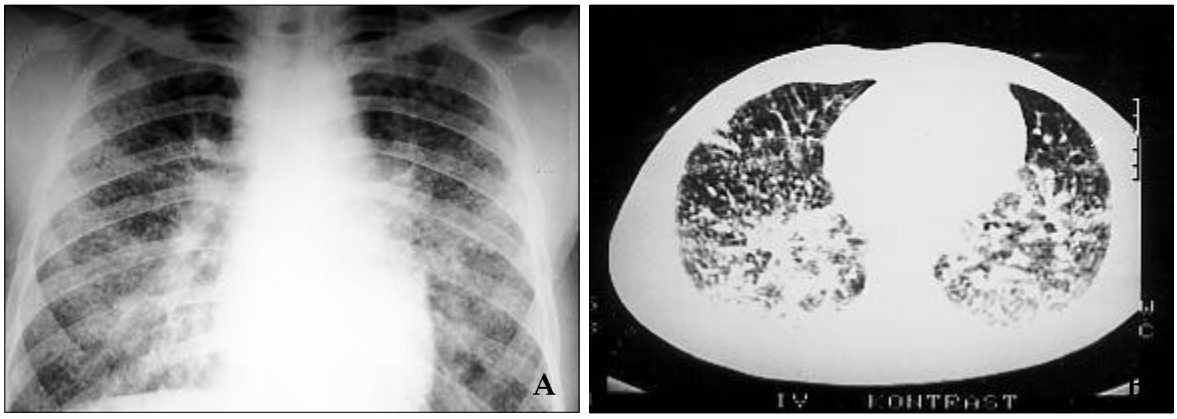

Fig. 1. A. Chest X-ray showing micronodular and reticulonodular infiltrates. B. CT scan showing diffuse linear infiltration with patchy soft tissue densities. (Case 1)

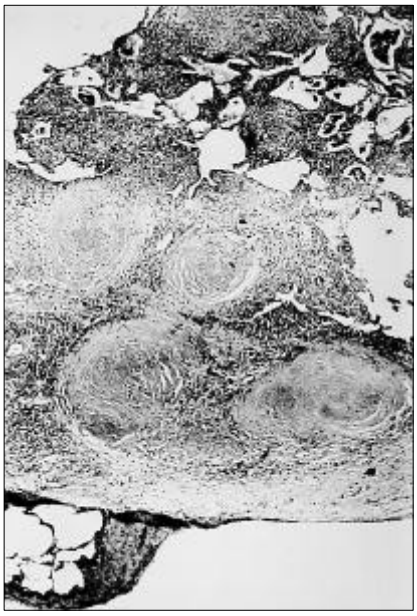

Fig. 2. Histological section from the open lung biopsy specimen of case 1 showing a lymphoplasmacytic infiltrate and nodules. (Haematoxylin and eosin stain, original magnification: ×40).

middle lobe and lingula BAL fluid cell distribution was normal.

Although interstitial fibrosis was considered, transbronchial and transthoracic aspiration biopsies on two occasions did not give any diagnostic findings. Thus open lung biopsy was performed, and histopathological examination showed "silicosis" (Fig. 2). The specimens then were incinerated for mineralogical examination at the TUBITAK (Turkish Science Research Association) Marmara Research Centre by scanning electron

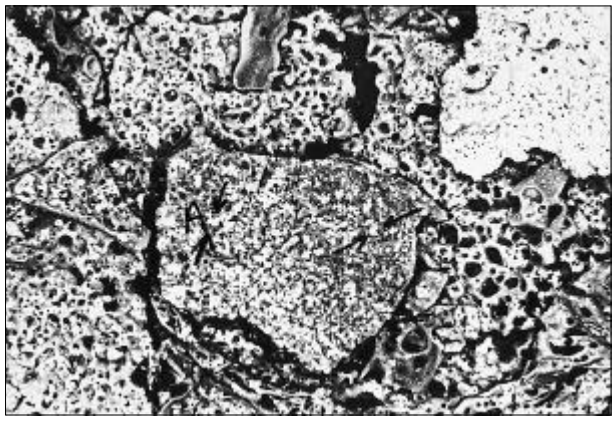

Fig. 3. Scanning electron microscopy (SEM) and energy dispersive X-ray spectrometer (EDS) images of case 2 showing light contrast areas (arrow and 'A'). These areas are rich in minerals.

microscopy (SEM) and energy dispersive X-ray spectrometer (EDS) methods. These revealed silicon (Si) and other elements such as phosphorus (P), potassium (K), calcium (Ca), iron (Fe), and aluminium (Al) in the specimens. Cobalt (Co), chromium (Cr), and cadmium (Cd) were also shown in the light contrast producing elements (Fig. 3 and 4).

Pneumoconiosis was diagnosed and the patient was discharged.

Case 2

A 20-year old man, a soldier for 3 months, had symptoms of exercise-induced breathlessness for 3 months. He had previously been working as a dental technician in a dental prosthesis laboratory for 8 years. He had no previous medical history,

a healthy family, and a history of 1 pack/year of cigarette smoking.

No pathology was found on physical examination. The laboratory investigations for total counts of RBC, WBC, platelets and urine analysis were all normal. Other haematological and biochemical data on admission were within normal ranges. He had BCG scars, PPD was positive, AFB in sputum was negative, and the serological tests for VDRL, HIV, CRP and RF were all negative. Plasma immunoglobulin and protein electrophoresis were within normal ranges.

Pulmonary function testing presented the following values: VC, 3.62 L (77% pred); FVC, 2.83 L (67% pred); FEV_1, 2.83 L (73% pred); and PEFR, 5.67 L/sec (63% pred). Diffusion capacity was slightly lowered (5.7 mmol/min/kPa, 71% pred).

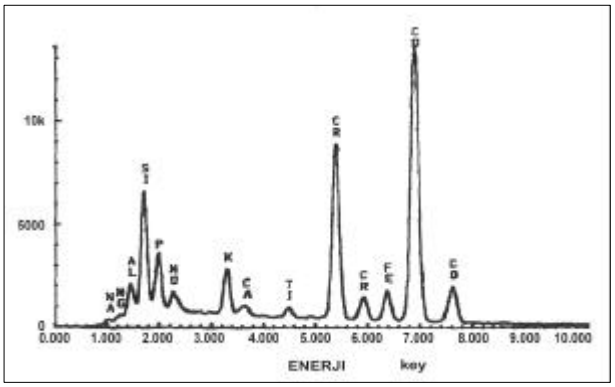

Fig. 4. Mineralogical analysis of case 1. Energy dispersive X-ray analysis of the lung of case 1 revealed the presence of silicon (Si), chromium (Cr), cobalt (Co), potassium (K) and other elements.

Chest X-ray showed diffuse micronodular infiltration in both hemithoraces (q type, 3/3) and CT scan showed millimetric nodular densities in the left lower lobe, scattered densities in the right upper lobe apical segment reaching to the pleura, and peribronchial infiltration in the right parahilar region, mediastinal and bilateral hilar lymph nodes (Fig. 5A and 5B).

Fiberoptic bronchoscopy showed no pathology. BAL fluid specimens were taken from the right middle lobe and lingula, and TBB showed no atypical changes. BAL fluid cell distribution was normal.

Although interstitial fibrosis was considered, transbronchial and transthoracic aspiration biopsies did not give any diagnostic findings. Thus open lung biopsy was done, and pathological examination showed "interstitial fibrosis". For mineralogical examination the same methods (SEM-EDS) were used as in case 1, and P, Si, Cr, Co, Mo and other elements were detected (Fig. 6).

Pneumoconiosis was diagnosed and the patient was discharged.

DISCUSSION

Since 1962, a few reports have been published on dental technician's pneumoconiosis due to exposure to heavy metals and Si, Co-Cr-Ni, and beryllium.[4] In addition to pneumoconiosis, chronic bronchitis, carcinoma and asthma have been reported in dental technicians.[5-7] If air filtration

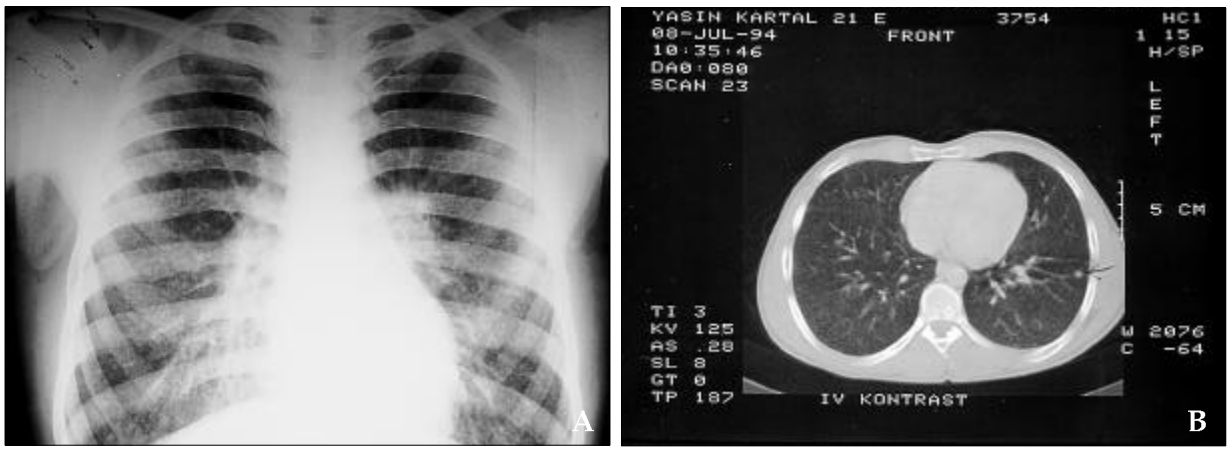

Fig. 5. A. Chest X-ray demonstrating diffuse micronodular infiltrations. B. CT scan showing nodular density (arrow) in the left lower lobe and peribronchial infiltration. (case 2)

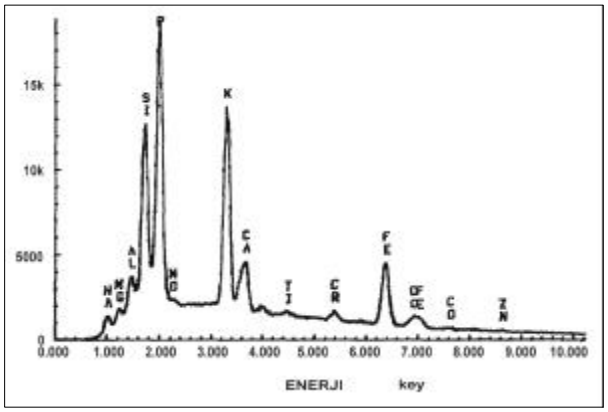

Fig. 6. Mineralogical analysis of case 2. Energy dispersion analysis revealed the presence of silicon (Si), phosphorus (P), potassium (K), iron (Fe) and other elements.

systems are not effective in the laboratory, dust particles may reach dangerous limits.[8] Although cleaning systems prevent diffusion of dust into the laboratory, most laboratories do not have these systems. A local ventilation system should be considered in dental laboratories to prevent airborne contaminants.[9]

The pathogenesis of dental technician's pneumoconiosis is complex and some particles, which are considered to cause pulmonary fibrosis, may be detected in BAL and biopsy specimens. Although Si is the most frequently suspected element, causing a typical nodular and fibrotic process, some other elements are also responsible for similar changes.[4] Case 1, with simple silicosis, is an example of this Si exposure injury. An open lung biopsy specimen demonstrated lymphoplasmacytic infiltration around the silicotic nodules. There were no giant cells or epitheloid cells. Furthermore, case 2 had interstitial fibrosis. Anthracotic pigments were found in alveolar histiocytes. Although the nodular process was not described, the histopathologic aspect was observed as areas of extranodular infiltration. It was reported that these cases had severe interstitial fibrosis with or without anthracotic pigment.[2,9]

The BAL fluid had a normal cell distribution in our cases. BAL fluid findings were normal or contained black pigmented alveolar macrophages.[10] In some cases, TBBs were diagnostic,[10] but BAL fluids were not diagnostic in all cases.

Berylliosis is also reported[11] to cause epitheloid granulomas. Although we were unable to search for this element by the EDS method, because of its low atomic weight, no granulomas were evident in our cases. Nevertheless, the absence of granulomas is insufficient to allow differential diagnosis for chronic berylliosis.

Asbestos caps are usually used in making gold dental prosthesis, and where these caps are broken, asbestos fibers are liberated in large amounts.[12] Malignant mesothelioma caused by asbestos has been reported in prosthesis technicians.[6] In our cases the typical radiological appearance of asbestosis was absent and asbestos was not detected by mineralogical examination.

Chest roentgenograms showed a diffuse bilateral reticulonodular infiltration in our cases. It has been demonstrated that these appearances are mainly in the upper lobes[2,13] or hilar level[10] in some cases. In high-resolution CT, extensive fibrotic changes are shown with occasional emphysematous blebs in the upper parts and compensatory emphysema in the lower parts of the lungs.[10] In our cases, high-resolution CT scan revealed the presence of bilateral diffuse micronodules and fibrotic changes without emphysematous blebs.

The physiopathology of fibrosis caused by Co-Cr-Mo (or Ni) particles is not yet known. It was thought that Co causes fibrosis by lymphocyte stimulation,[14] but we did not detect any lymphocyte dominance in the BAL fluid. On the other hand, mediators from macrophage stimulation may cause fibrosis.[2] Co is suspected to cause damage in heavy metal pneumoconiosis, but because of its high solubility it is very hard to detect in BAL and other specimens.[13] Cr-Co has been found in high amounts in quantitative studies.[2]

It has been suggested that the lung function of dental technicians is significantly lower, and that this reduction in lung function is more pronounced in smokers.[15] Some functional abnormalities were observed in our cases. In silicosis, restrictive as well as obstructive patterns have been described.[15] Tobacco smoke may have played a contributory role in the development of airway obstruction. In addition, the reduced lung function may indicate a component of bronchospasm representing a parallel phenomenon of Co-induced obstructive lung function.[16] In our cases,

lung function studies disclosed a mix type (restrictive and obstructive pattern) abnormality with a low diffusion capacity.

In conclusion, dental technicians are exposed to various dusts. We demonstrated silicon and some other elements in the lungs of two dental technicians. Other groups have made similar clinical and mineralogical observations. It was suggested that Cr-Co-Mo alloys might play a major role in the genesis. It is thought to be a real entity distinct from other pneumoconioses. However, further studies, including epidemiological investigations, are needed to determine the etiopathogenesis. For prevention, local exhaust ventilation should be encouraged in dental laboratories.

REFERENCES

1. Rom WN, Lockley JE, Lee JS, Kimball AC, Bang KM, Leaman H, et al. Pneumoconiosis and exposure of dental laboratory technicians. Am J Public Health 1984;74:1252-7.
2. Sherson D, Maltbaek N, Heydorn K. A dental technician with pulmonary fibrosis: a case of chromium-cobalt alloy pneumoconiosis? Eur Respir J 1990;3:1227-9.
3. Barrett TE, Pietra GG, Maycock RL, Rossman MD, Minda JM, Johns LW. Acrylic resin pneumoconiosis: report of a case in a dental student. Am Rev Respir Dis 1989;139:841-3.
4. Choudat D. Occupational lung disease among dental technicians. Tuber Lung Dis 1994;75:99-104.
5. Choudat D, Triem S, Weill B, Vicrey C, Ameille J, Brochard P, et al. Respiratory symptoms, lung function, and pneumoconiosis among self employed dental technicians. Br J Ind Med 1993;50:443-9.
6. Reid AS, Causton BE, Jones JSP, Ellis IO. Malignant mesothelioma after exposure to asbestos in dental practice. Lancet 1991;338:696.
7. Losewicz S, Davidson AG, Hopkrik A. Occupational asthma due to methyl metacrylate and cyanoacrylates. Thorax 1984;39:712-3.
8. Moffa JP, Guckes AD, Okawa MT, Lilly GE. An evaluation of nonprecious alloys for with porcelain veneers. Part II. Industrial safety and biocompatibility. J Prosthet Dent 1973;30:432-41.
9. Nayebzadeh A, Dufresne A, Harvie S, Begin R. Mineralogy of lung tissue in dental laboratory technicians' pneumoconiosis. Am Ind Hyg Assoc J 1999;60:349-53.
10. Selden A, Sahle W, Johansson L, Sorenson S, Persson B. Three cases of dental technician's pneumoconiosis related to cobalt-chromium-molybdenum dust exposure. Diagnosis and follow-up. Chest 1996;109:837-42.
11. Kotloff RM, Richman RS, Greenacre JK, Rossman MD. Chronic beryllium disease in a dental laboratory technician. Am Rev Respir Dis 1993;147:205-7.
12. Burn D, Beltesbrekke H. Levels of methylmetacrylate, formaldehyde and asbestos in dental workroom air. Scand J Dent Res 1981;89:111-6.
13. De Vuyst P, Weyer RV, De Coster A, Marchandise FX, Dumortier P, Ketelbant P, et al. Dental technician's pneumoconiosis: a report of two cases. Am Rev Respir Dis 1986;133:316-20.
14. Demendts M, Ceuppens JL. Respiratory diseases from hard metal or cobalt exposure. Chest 1989;95:2-3.
15. Selden A, Persson B, Bronberger-Dankvardt S, Winstrom LE, Bodin LS. Exposure to cobalt chromium dust and lung disorders in dental technicians. Thorax 1995;50:769-72.
16. Balmes JR. Respiratory effects of hard-metal dust exposure. Occup Med State Art Rev 1987;2:327-44.