

A dental technician with pulmonary fibrosis: a case of chromium-cobalt alloy pneumoconiosis?

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ABSTRACT: A 45 yr old male developed pulmonary fibrosis after 29 yrs of employment as a dental technician. He subsequently developed adenocarcinoma of the lung. Diffuse interstitial fibrosis was seen using light microscopy. Neutron activation analysis of non-neoplastic lung tissue demonstrated high levels of chromium and cobalt suggesting the possibility of a chromium-cobalt alloy pneumoconiosis.

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Dental laboratory technicians are apparently at risk to develop pneumoconioses. A number of presumed aetiological agents have been described: beryllium [1, 2], silica [3, 4], alginate [5] as well as various metals (cobalt, chromium, molybdenum and tungsten) with or without silica exposure [6-11]. Acrylic resin has been described as a possible cause of pneumoconiosis in a dental student [12].

Case report

A 45 yr old male was examined in November 1983, in connection with an epidemiological study of dental technicians in the greater Copenhagen area [11]. He had smoked 10 cheroots daily for 30 yrs and had been employed full-time as a dental technician for 29 yrs. He spent approximately 20 h a week producing and polishing chromium-cobalt prostheses as well as a few hours a week producing gold prostheses. The patient did not work with acrylate or porcelain. No hygienic monitoring was performed. He complained of grade 2 dyspnoea without chronic bronchitis. Lung function studies demonstrated the following: forced vital capacity (FVC) 3.75 l (80%), forced expiratory volume in one second (FEV₁) 2.53 l (67%), diffusing capacity of the lung DLco 22.79 ml·min⁻¹·mmHg⁻¹ (83%), residual volume (RV) 2.71 l (126%) and total lung capacity (TLC) 6.74 l (99%). His chest X-ray demonstrated diffuse bilateral small opacities and was classified in a blind fashion as 1/1 p using ILO's international classification of radiographs of pneumoconiosis from 1980.

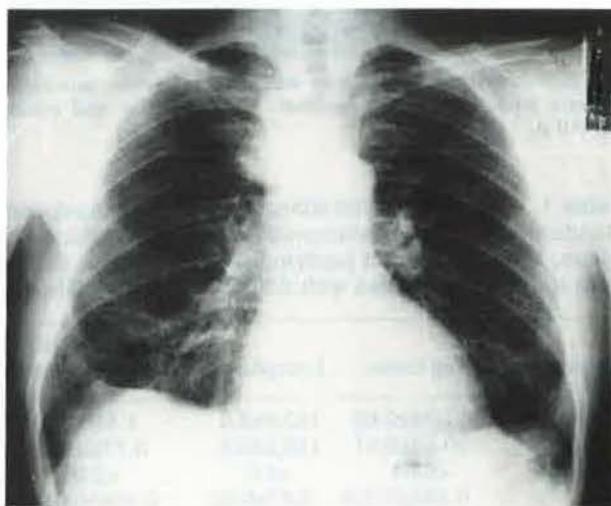


Fig. 1. - PA chest X-ray demonstrating diffuse small opacities classified as 1/1 p as well as a new medial infiltrate in the right upper lobe. PA: posteroanterior.

A chest X-ray performed 3 yrs later demonstrated a new medial infiltrate in the right upper lobe (fig. 1). Bronchoalveolar lavage was performed with a total cell count of 216×10⁶ cells·ml⁻¹. Differential cell count showed 82% macrophages, 6% lymphocytes and 12% neutrophils. Superoxide anion release, O₂⁻, after phorbol myristate acetate was 4.16 nmol·min⁻¹·10⁶ cells⁻¹ in alveolar macrophages and 2.00 nmol·min⁻¹·10⁶ cells⁻¹ in blood monocytes. An inoperable poorly differentiated adenocarcinoma in the right upper lobe with spread to

paratracheal lymph nodes was seen at thoracotomy. A surgical biopsy specimen of lung tissue without gross evidence of cancer demonstrated severe interstitial fibrosis. Alveoli were of varying size with frequent destruction of alveolar walls. Minimal inflammation was seen with a predominance of lymphocytes. Anthracotic pigment was described in fibrotic connective tissue (fig. 2). Anthracosis as well as localized fibrosis associated with fine particles were seen in lymph nodes. Only a few refractile bodies were identified under polarized light in lung and lymph node tissue (signed A. Aru). Quantitative levels of chromium, cobalt, tungsten and gold were determined in surgical biopsy specimens of lung and lymph node tissue by means of neutron activation analysis [13] (table 1).



Fig. 2. - A surgical lung biopsy demonstrating severe interstitial fibrosis with anthracotic pigment. Haematoxylin and eosin. Bar=10 μ .

Table 1. - Mean concentrations ($\mu\text{g}\cdot\text{g}^{-1}$ wet tissue) and standard deviation of elements in non-neoplastic lung tissue, lymph node and perilymphatic connective tissue from a dental technician with diffuse pulmonary fibrosis

Element	Lung tissue	Lymph node	Perilymphatic connective tissue
Chromium	11.74 \pm 0.08	142.0 \pm 2.0	1.6 \pm 0.3
Cobalt	20.54 \pm 0.07	160.2 \pm 0.6	0.77 \pm 0.02
Tungsten	<0.04	<8.0	<2.0
Gold	0.493 \pm 0.003	3.87 \pm 0.02	0.196 \pm 0.002
Total wet specimen weight mg	946.2	5.04	5.98

Discussion

Results from the neutron activation analysis demonstrated extremely high levels of cobalt and chromium. Wet lung tissue specimens from controls using neutron activation analysis have been described in the literature: mean chromium concentrations ($\mu\text{g}\cdot\text{g}^{-1}$) 0.117, 0.39 and 0.327 as well as cobalt concentrations ($\mu\text{g}\cdot\text{g}^{-1}$) 0.0128 and 0.05 [14-16]. The range of lymph node concentrations of chromium

(0.307-2.884 $\mu\text{g}\cdot\text{g}^{-1}$) and cobalt (0.0147-0.242 $\mu\text{g}\cdot\text{g}^{-1}$) have also been described [14]. A 23-123 fold increase in pulmonary gold concentration was also found in this patient [14, 16]. The high concentrations found in our patient suggest significant occupational exposure. However, no air monitoring was done.

Other possible causes of pneumoconiosis seem unlikely. He was not exposed to beryllium or asbestos. The absence of both refractile bodies under polarized light and typical histological changes rules out silicosis. The absence of multinucleated cells in lavage fluid argues against a typical case of hard metal lung disease. Cobalt alone is highly soluble and, thus, can be difficult to recover [10]. The large concentration of both cobalt and chromium that we measured suggests that cobalt is much less soluble in the form of an alloy with chromium.

The pathophysiological mechanism leading to pulmonary fibrosis in association with exposure to cobalt and chromium is uncertain. An immunological basis for cobalt induced fibrosis with lymphocyte stimulation has been suggested [17]. However, the absence of lymphocytes in lavage fluid from our patient as well as from other cases [10] is inconsistent with this type of hypersensitivity. On the other hand, fibrosis may result from macrophage stimulation with the release of macrophage-derived mediators. Alveolar macrophage stimulation in our patient as defined by superoxide anion release was more than double the activation in his blood monocytes. This relative increase in alveolar macrophage activation suggests that factor(s) in the lung may produce localized cellular stimulation.

The aetiology of our patient's lung cancer is uncertain. Smoking is related to all major cell types [18] and most likely the primary risk factor. Chromium has been described as a carcinogen [19], but has been associated with a predominance of squamous cell carcinoma [20]. Another possible aetiological factor could be an association between fibrosis and a scar cancer. Scar cancers are often adenocarcinomas in the upper lobes [21]. However, the present case cannot satisfy the diagnostic criteria as no histological scar was seen.

In conclusion, we have demonstrated extremely high lung levels of chromium and cobalt in a dental technician with diffuse pulmonary fibrosis. Other groups [7, 10] have made similar clinical observations, which suggest that the accumulation in the lung of chromium-cobalt alloy may be associated with diffuse pulmonary fibrosis. However, this theory should be supported by animal studies as well as epidemiological investigations before causation can be certain.

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Un Technicien dentaire avec fibrose pulmonaire: un cas d'alliage chrome-cobalt pneumoconiose? D. Sherson, N. Maltbaek, K. Heydorn.

RÉSUMÉ: Une fibrose pulmonaire est apparue chez un homme de 45 ans après 29 ans de travail comme technicien dentaire. Par la suite est apparu un adénocarcinome du poumon. Une fibrose interstitielle diffuse a été observée en microscopie optique. Une analyse après activation neutronique a montré des taux élevés de chrome et de cobalt évoquant une pneumoconiose liée à un alliage chrome-cobalt. *Eur Respir J.*, 1990, 3, 1227–1229.