Dental Technicians' Pneumoconiosis: Mini-Review of a Neglected Work-Related Disease

Mara M Tiraboschi[1], Emma Sala[2], Emanuele Sansone[1], Giuseppe De Palma[1,2*]

[1]Department of Medical and Surgical Specialties, Radiological Sciences, and Public Health, Unit of Occupational Health and Industrial Hygiene, University of Brescia, Brescia, Italy;[2]Occupational Health, Occupational Hygiene, Toxicology and Prevention Unit, University Hospital "Spedali Civili di Brescia", Brescia, Italy

Received: 09-Feb-2022, Manuscript No. OHDM-22-15617; **Editor assigned:** 11-Feb-2022, Pre QC No. OHDM-22-15617 (PQ); **Reviewed:** 25-Feb-2022, QC No. OHDM-22-15617; **Revised:** 02-Mar-2022, Manuscript No. OHDM-22-15617 (R); **Published:** 09-Mar-2022, DOI: 10.35248/2247-2452.22.21.981.

ABSTRACT

Working as a dental technician exposes to an increased risk of developing lung diseases, especially pneumoconiosis, caused by the inhalation of metallic and non-metallic dusts produced by some working processes, in particular grinding, sandblasting, casting, and polishing of ceramic, acrylate, and metal alloys. In this mini review, we describe clinical and radiological findings of this occupational disease, also examining the different characteristics of pneumoconiosis caused by specific exposures. Knowledge of the illness is important to plan effective strategies for prevention and monitoring in the workplace.

Key Words: Dental technician, Pneumoconiosis, Lung disease, Occupational disease.

Introduction

Dental technicians' job profile implies the construction of mobile and fixed dentures and mobile orthodontic appliances made of various materials. It is a highly specific work realized in collaboration with the dentist to design individual plans for each patient. Dental staff is exposed to various physical, chemical and biological harmful conditions in the working environment. In particular, dental technicians are exposed to several hazardous dusts that may affect the respiratory tract and cause occupational lung illnesses. The work process is used worldwide, but occupational exposure can vary according to the working conditions and materials used [1,2].

The aim of this mini-review is to describe dental technician's pneumoconiosis, a serious occupational lung disease caused by the accumulation of inhaled particles in the lungs, which can lead to chronic respiratory failure. Knowledge of this illness is important to plan effective strategies for prevention and monitoring in the workplace.

Dental Technicians' Pneumoconiosis

Pneumoconiosis is any lung disease caused by the inhalation of organic or nonorganic airborne dust and fibers. These particles cause inflammation and fibrosis in the lung, resulting in irreversible lung disease. It exposes patients to an increased risk of lung cancer and pulmonary infections. It is known as an occupational disease because patients usually encounter these inhalants in the workplace environment [3]. Pneumoconiosis in a dental technician was reported in 1939. The case was diagnosed as "silicotuberculosis" and was probably caused by a polishing powder with approximately 50% free silica [4].

The prevalence of pneumoconiosis among dental technicians is estimated to be approximately 9.8%-23.6%. Studies show that the disease seems to be more frequent after many years of exposure [5]. Rom et al. found a prevalence of 16.7% in workers with more than 20 years of employment duration, in contrast with one of 1.4% in those with less than 20 years of employment duration [6]. Choudat et al. studied 105 dental technicians and determined a pneumoconiosis prevalence of 3.5% in those with less than 30 years of employment duration and a prevalence of 22.2% in those with more than 30 years employment duration [7]. However, a case of dental technician's pneumoconiosis that arose only a few months after the beginning of work is reported. Some unrecognized individual susceptibility traits are likely to be involved in the acceleration of the development of lung damage [8].

Dental technicians' pneumoconiosis is a complex pneumoconiosis and sometimes can be designated mixed dust pneumoconiosis. Exposure during grinding, sandblasting, casting, and polishing of ceramic, acrylate and metal alloys are the most hazardous. If no ventilation or poor ventilation is used during these operations, the intensity of exposure might be very high. Ergün et al. suggested that exposure to sandblasting is a major risk factor that carries a 77-fold-increased risk of pneumoconiosis [9].

Metal alloys, such as vitallium, wisil, duralium and vironite, are used in the production of crowns, bridges, and dental prostheses. These alloys are made of cobalt (35%-65%), chromium (20%-30%), nickel (up to 30%) and small amounts of molybdenum, silica, beryllium, boron, tantalum, and other elements. Gold-palladium alloys are currently seldom used [2].

Porcelain is used extensively and is composed of varying amounts of kaolin, silica and alumina (Al2O3). The three types of abrasives used in dentistry may be classified as finishing, polishing and cleansing abrasives. Aluminum oxide, quartz particles, diamond and silicon carbide are the most important abrasives used in dental laboratories. In the past, liners with asbestos were used in casting procedures [10].

Silicosis and pneumoconiosis associated with exposure to cobalt-chromium-molybdenum are the most common dental technicians' pneumoconiosis [11,12]. However, nickel-chromium, asbestos, beryllium, aluminum, tungsten carbide, and other components of dental metals are reported to be causes of pneumoconiosis [9,13]. Few cases due to exposure to indium and zirconium have also been described in the literature [8,14].

Personal history of work exposure, physical examination, and chest radiographs (CXR), high-resolution CT evaluations (HRCT) and pulmonary function tests (PFT) are used for the diagnosis of pneumoconiosis.

Cimrin et al. analyzed 214 cases and found that 46.7% of them had at least one symptom (26.6% had cough, 30.4% had sputum, 18.2% had dyspnea, and 15.0% had wheezing). The influence of cigarette smoking has a significant relation to the

Corresponding author: De Palma G, Department of Medical and Surgical Specialties, Radiological Sciences, and Public Health, Unit of Occupational Health and Industrial Hygiene, University of Brescia, Brescia, Italy, E-mail: giuseppe.depalma@unibs.it

presence of respiratory symptoms [15,16].

In the early stages of pneumoconiosis, Pulmonary Function Tests (PFTs) may be normal. As the disease progresses, obstructive, restrictive or mixed abnormalities can be found, especially obstructive abnormalities in those with low-grade pneumoconiosis [17]. The disease leads to airflow limitation through various mechanisms: bronchial stenosis secondary to peribronchial fibrosis, enlargement of lymph nodes and centrilobular emphysema [18]. Radi et al. showed that FVC%, FEF25% and FEF50% were significantly lower in male dental technicians than in controls [19]. Wang et al. found that workers with pneumoconiosis had significantly lower VC, FEV1, FEV1/FVC and DLCO values than workers with the same occupational exposure but without radiographic evidence of pneumoconiosis [20]. Furthermore, lower values of DLCO were found in smokers and in those with emphysema. Lung function abnormalities correlated better with emphysematous changes than with nodular changes in pneumoconiosis [21].

Chest radiographs are evaluated according to the International Labor Organization (ILO) classification, but this method is known to have limited utility and accuracy compared to HRCT, which is more sensitive than chest radiography for detecting small opacities, interstitial fibrosis and emphysema, especially in the case of low-grade pneumoconiosis [22-24]. For these reasons, the effectiveness of ILO classification is controversial.

There are few studies demonstrating radiological and functional correlations in dental technicians. Berk et al. identified parenchymal opacities in 31% of workers by CXR and in 69% by HRCT. HRCT was shown to be more valuable in showing possible lung pathologies other than pneumoconiosis and detecting pneumoconiosis at early stages. Opacities, shown by CXR and HRCT, had a positive correlation with age, employment duration and smoking duration and a negative correlation with FVC, FVC% and FEV1 on PFTs [5].

Lopes et al., in their study, established that there was a correlation between an increase in damage detected with HRCT and a decrease in air flow. Therefore, it was stressed that the degree of parenchymal involvement is an important indicator of the severity of functional impairment [25].

Ergün et al. found a close correlation between lung radiography (ILO score) and the extent of parenchymal involvement in HRCT and functional parameters. This relationship was more marked in HRCT. Functional loss increased with radiological progression. The functional parameter mirroring the degree of pulmonary involvement most accurately was FEV1 [26].

All these data show and agree that, compared to chest radiography; HRCT offers improved correlations between histopathologic and clinically relevant imaging findings.

Pneumoconiosis Caused By Specific Exposures

Silicosis is caused by the inhalation of crystalline silica dust. Chronic silicosis develops several decades after exposure and is characterized radiologically by nodules prevalent in the upper lobes and eggshell calcifications in the lymph nodes, not always associated with clinical manifestations. Exposure to large quantities of silica, on the other hand, can lead to an acceleration of disease or acute silicosis with worsening symptoms and a high mortality rate. Progressive forms generally evolve into massive fibrosis, with coalescence of the nodules and early onset of respiratory failure [27].

Hard metal pneumoconiosis may be variable. In the early stages of the disease, the HRCT scan shows ground-glass opacities and centrilobular nodules [28]. Some patients show perilymphatic distribution of the nodules, mimicking sarcoidosis [29]. The ground-glass opacities show diffuse, patchy or lobular distribution. Traction bronchiectasis and architectural distortion are seen in advanced stages. Clustered cystic lesions are also seen and consist of traction bronchiectasis and bronchiolectasis. Peripheral cysts, lymph node enlargement and spontaneous pneumothorax spaces are also reported in some cases [30]. The progression of fibrosis is very different from case to case. Ground-glass opacities are reported to improve following cessation of exposure to hard metals and treatment [31,32].

Workplace exposure to indium compounds, including indium oxide and indium-tin oxide, causes several lung diseases [33]. In cases with interstitial lung disease, the major HRCT findings are ground-glass opacities and centrilobular opacities [34]. The parenchymal abnormalities show diffuse, upper or mid-lung predominance. Interstitial opacities with volume reduction and traction bronchiectasis in the upper lungs are reported. Paraseptal emphysema, small cysts at both apices and subpleural honeycomb can be seen [35]. Cessation of exposure to indium can reduce radiographic interstitial changes, whereas emphysema may progress among those with a history of heavy exposure [31,33].

The HRCT appearances of aluminum pneumoconiosis may be variable and include nodular, reticular, and upper lung fibrosis patterns [31]. In the early stages of aluminosis, the HRCT findings are small rounded centrilobular opacities mainly in the upper lobes [36,37]. In addition, the nodular pattern includes nodules in lymphatic distribution, mimicking sarcoidosis, probably reflecting a granulomatous lung reaction. In advanced stages, subpleural bullous emphysema with an increased risk of spontaneous pneumothorax can be found [38].

Bronchoalveolar lavage (BAL) and lung biopsy are not typically performed but can be useful in difficult and doubtful cases to reach an exact diagnosis.

For example, berylliosis has clinical and radiological findings almost entirely comparable to sarcoidosis. The most frequently affected organ is the lung, with predominantly reticulo-nodular involvement in the upper lobes and the presence of adenomegaly. Alternative radiological findings include the presence of centrilobular nodules or ground glass opacities. Generally, the disease is slowly progressive, reaching fibrosis in the late stages. Berylliosis can also have multiorgan involvement with the formation, rarely, of granulomas, for example, in the liver. The definitive diagnosis of berylliosis is therefore based on a careful anamnestic collection and on the positivity of the beryllium lymphocyte proliferation test, as well as on the finding of granulomatous inflammation on a lung biopsy. The beryllium lymphocyte proliferation test has a high sensitivity and can be performed on both peripheral blood and BAL [27].

Discussion

Prevention for dental technicians is very important due to occupational exposure to several hazardous dusts for the respiratory tract, as we have seen thus far. This implies careful risk assessment through airborne and biological monitoring of pneumotoxic dusts and the use of personal and general protective precautions, such as local and general ventilation and individual masks. For example, the efficient use of masks decreases the respiratory uptake of dust by 70.95% [39]. Nevertheless, several studies have reported that the number of precautions related to the prevention of dust in dental technicians' workplace was rather low. For example, the use of ventilation was reported to be 33% and 45% in previous studies [40]. Bozkurt et al. described that vacuum use was found in 66% in 2008 and increased to

78% in 2013, showing a slight increase over the course of time [41]. Investing in prevention is extremely important and a duty to safeguard dental technicians' health.

Conclusion

Dental technicians' laboratories are workplaces that are exposed to the risk of developing pneumoconiosis. It is important to plan careful risk assessment and improve the use of personal and general protective precautions to guarantee workers' safety. Health screenings should be performed regularly on these workers. Furthermore, in patients with suspected pneumoconiosis, a history of occupational exposure should be taken carefully, and clinical and radiological evaluations should be performed to reach an early diagnosis of the disease.

Acknowledgments

Author's Contribution

All authors read and approved the final version of the manuscript.

Funding

Not applicable.

Availability of Data and Materials

Data sharing is not applicable to this article, as no datasets were generated or analyzed during the current study.

Ethics Approval and Consent to Participate

Not applicable.

Consent for Publication

Not applicable.

Competing Interests

The authors declare that they have no competing interests.

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