LUNG SIDEROSIS; A REVIEW

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Abstract

With the development of industrialization, pneumoconiosis become one of the problems of occupational medicine. Known in antiquity as miners specific diseases, by the severity of evolution, this generates social and economic implications. In recent years, the frequency of these diseases has decreased significantly due to sustained efforts to prevent them. However, worldwide there are jobs with exposure to pneumoconiotic dust.

Keywords: siderosis, pneumoconiotic dust, welding fumes, lung cancer, occupational exposure

Introduction

Siderosis is an occupational lung disease (pneumoconiosis) caused by the inhalation of dust or fumes containing iron particles or iron oxide. This is most frequently observed in arc welders. The dust accumulation in the lungs, inert particles, which are formed during the technological process, causes tissue reactions and thus the occurrence of chronic lung diseases, known as pneumoconioses [1,2].

In 1867, Zenker was the first one who identified iron oxide in the lungs. Welder’s siderosis was first described in 1936 in a prospective study that included 16 welders working with an electric arc welding. They were clinically and radiographically checked, and 15 of them were followed for nine years [3]. Arc welder’s siderosis is the chronic inflammation of the lungs caused by the inhalation of dust containing iron particles. The workers from the iron mines, iron welding operations, manufacturing iron oxides, iron oxides pigments, polishing metals from foundries and cleaners can develop the disease.

The inhaled particles of iron oxide are removed by the sputum. Only a small part are phagocytized by macrophages and then transported by the lymphatic system. Iron is an essential metal in maintaining health. A matter of interest of occupational medicine is represented by the toxicological implications of short or long-term occupational exposure and cumulative exposure to iron dust, gas and welding fumes.

The exposure to iron powders, gases and welding fumes

Exposure to iron powders, gases and welding fumes in the steel and metallurgical industry is inevitable. Therefore, siderosis is a condition that affects a large number of professional workers. The particles of iron

General Review

Lung Siderosis; a Review

43
oxide are inhaled in aerosol form or breathable particles. Prolonged inhalation of these fumes can lead to the development of lung siderosis. Technological processes such as welding, foundry work, cleaners, grinding and polishing metals, manufacture of electrolytic iron oxides, iron ore exploitation, are widely used in industry.

Welding is one of the main technological processes generating iron oxides, gases and welding fumes. There are few industries that do not use electric welding during the technological process. Iron is a chemical element, vital for almost all living organisms, participating in a variety of metabolic processes, including oxygen transport, DNA synthesis and transport of electrons. The concentration of iron in body tissues must be strictly regulated because the excess of iron leads to tissue damage as a result of the formation of free radicals [4]. Electric arc welding generates gas and welding fumes that vary in the composition. The metals existing in the welding process are of toxicological interest, because of the potential effects on worker health [5]. In welding operations, there is a simultaneous exposure to irritants such as oxides of nitrogen, ozone, aerosols from the glaze firing the electrodes and impurities on the surface of metal parts [6]. The electrodes used in welding process are covered by a protective layer of oxides, carbonates and wrapped in a fine mesh of asbestos. Commonly used electrodes, produce an abundance of welding fumes. These include iron oxide particles having a diameter of about 0.5μ. The inhaled particles are accumulated in the lymphaticperibronchovascular spacecausing secondary fibrosis [7]. Besides iron oxide, welding fumes also contain aluminum, beryllium oxide, cadmium, chromium, copper, fluoride, iron oxides, manganese, molybdenum, nickel, vanadium and zinc oxide. Note that fumes from welding the steel contain the largest amount of iron [8]. Occupational exposure to the particles of iron oxide is not limited to the workers dealing with metals, miners and manufacturers of iron oxide, but because of other properties (pure color shade, the consistency and the ability to dye), the iron oxides are commonly used as pigments [9], widely used in construction, ceramics, paints, rubber, plastics and cosmetics, affecting other occupational groups [9]. Another aspect is the use of iron oxide as additives in fertilizers, catalysts, fluid markers, magnetic materials, absorbing water purification systems, biomedical imaging and therapeutic agents. Through these, another group of workers are potentially exposed iron oxide powders [9]. Siderosis cases have been reported in the grinder bearing part made of chrome-vanadium, chrome-molybdenum and steel containing 98% iron, ~2% the above-mentioned alloys and 0.2% silica [4].

Pathogenesis

Siderosis pathogenesis mechanism requires the knowledge of developing pneumoconiosis, namely identifying how penetration, deposition, removal and retention of pneumoconiotic dusts in the respiratory tract. The chemical structure of the inhaled particles, solubility, size and their concentration determine the character of lung lesions. The individual susceptibility of the human body and the duration of occupational exposure to dusts are all factors that are directly influencing the lung lesions. The most dangerous are the fine particles with a diameter less than five microns. The lesion capacity of the dust is greater the smaller the particle. Pneumoconiotic risk is amplified by cigarette smoking, especially on large exposures to dust [10]. The pathogenic conditions of the pneumoconiotic dusts are: the particle diameter must be small (<3μm,) and the concentration of dusts must be high to exceed the maximum allowable concentration [1]. Different factors influence the penetration of dust, in respiratory system or the temporary or permanent filing at different levels. The shape, hardness, diameter, density of the respirable particles, the anatomy of the respiratory system and exhaled air flow determines the initial detention and the place of deposit dust inhaled. Pneumoconiotic dust deposition mechanisms are inertial impacting, centrifugal precipitation gravity sedimentation and diffusion [6]. The air speed in the airways and the particle size directly influences their deposition in the lungs. The particles of inhalable dust fraction must cross the airways – the nose and nasopharynx. Large particles (diameter ≥10μm) are deposited by inertial impact (striking) to the nasal mucosa [11]. In the case of particles with a diameter up to 2μm, the main mechanism of deposition is by settling due to gravity. For particles with a diameter <0.5μm, sedimentation becomes less important and under 0.2μm insignificant, the main mechanism for submission being the diffusion [6]. Accumulation of dust particles in lungs represents the difference between the particles that were temporarily deposited and those that were removed by the phenomenon of lung clearance (bronchial and alveolar) during occupational exposure [11]. Other factors that influence the temporary deposition and retaining of dusts in the respiratory tract are the agglutination of small particles with hygroscopic properties, particle shape (particularly the sharp ones are retained in high percentages), rare and profound breaths, increase the particle deposition in the upper airways. Bronchodilators reduce dust deposition the main airways [12]. They are associated with the mucociliary clearance. Due to this, the particles deposited and retained temporarily in the bronchial and bronchiolar mucous (10-
3 μm) are disposed with the tracheobronchial secretions at a rate of 90% within the first hour. The rest are removed in 6-12 hours. By the alveolar clearance particles from the walls of the alveoli and alveolar ducts (3-0.5μm diameter) are eliminated in two ways: 50% by alveolar surfactant and the second way is the lymphatic one [6].

**Pathology**

Transbronchial lung biopsy specimen, in a hematoxylin and eosin-stained showed numerous hemosiderin-laden macrophages in alveolar lumina, shedding and hyperplasia of alveolar epithelial cells and marked alveolar capillary congestion. Varying interstitial fibrosis, fibrous thickening of the alveolar septa, intra-alveolar hemorrhage. No necrosis, no vasculitis, no granulomas and no lymphoid follicles [13].

**Clinical data**

The degree of damage to the health of workers due to welding fumes depends on its composition, concentration and exposure time [8]. Welder’s siderosis was considered over time as a lung tattoo. In lung siderosis specific reticular opacities appear on chest X-ray. These opacities are initially reticular and then reticulomicronodular, of high intensity due to the iron particles [14]. Siderosis is generally considered a benign condition associated with respiratory symptoms [15]. Clinical symptoms only appear when silico-siderosis occurs. Clinical signs and symptoms may occur due to concomitant exposure to other professional pollutants or the presence of smoking [6]. However, a review of the literature suggests that this assumption is incorrect and that siderosis can lead to both symptomatic and functional changes [15].

The first case was described by Enzer and Sanders, in a patient who presented changes on chest radiography and died in an accident [16,17]. Histological examination showed that, although in alveolar septum has been described a massive accumulation of iron powder, the degree of interstitial fibrosis has been decreased [16].

In 1936, Doig and McLaughlin described radiological changes to 16 electric welders apparently in a good state of health [3]. Later, in 1948 they reported that in the case of two of the patients from the previous study, the radiological changes have disappeared along with the removal of harmful environment [16,18].

Siderosis is a benign condition. Those welders, described in the literature, with obstructive ventilatory dysfunction and the retention of carbon dioxide, most likely have the harmful effects associated with concomitant bronchitis or emphysema [16].

In recent years, it was observed that siderosis is accompanied by pulmonary ventilatory function disorders [19]. In a study of 44 electric welder in Modakeke, Ile-Ife, Nigeria, they were compared for work-related symptoms and pulmonary ventilatory function. The most common symptoms described among these subjects were eye irritation, rhinitis and skin irritation. Respiratory functional parameters were decreased compared with the control group [20].

In 2008, McCormick et al communicated the case of an engineer aged 66, who worked for 20 years, actively participating in the process of welding but always wearing a face mask [21]. Clinical, radiological and histological aspect confirmed the diagnosis of siderosis, associated with pulmonary fibrosis. Ventilatory functional tests showed a moderate obstructive ventilatory dysfunction. Chest X-ray showed a diffuse reticulosclerosis drawing heightened and CT examination showed multiple micronodular opacities in both lungs [21].

**General Review**

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The idiopathic pulmonary sideroelastosis was first introduced by Ceelen (1931) and Gellerstedt (1939), described as the vascular alterations associated with a chronic lung congestion. Verhoff and Muller [24] studied if the existence of secondary sideroelastosis can be demonstrated in the lungs after previous exposure to welding fumes. In 43 patients with various periods of exposure to welding fumes, lung tissue samples have been investigated. In 6 cases (14%), vascular alterations have been observed with siderophileimpregnation in the area of the distorted elastic fibers. The comments corresponded to the previous research of Gellersted and Ceelen. Giant cells, with granulomatous reaction were exceptional seen. This phenomenon was particularly...
observed in cases with marked deposits of iron and reactive fibrosis. A correlation between increased dietary intake of iron welding powder inhalation and vascular walls sideroelastosis must be established [24].

**Treatment**

Siderosis is preventable but not treatable. Because it does not normally cause any symptoms or damage to body tissues, treatment is not required. Avoiding exposure to iron dust or fumes will prevent any further accumulation of particles in the lungs. In individuals with lung damage related to siderosis, treatment will be contingent on the particular complication.

**Prognosis**

Siderosis is usually a harmless (benign) disease. Most cases never produce symptoms of illness. In rare cases where lung damage does occur, avoiding further exposure to iron dust or fumes prevents progressive damage.

**Complications**

Complications do not generally occur, but may include pneumonia, lung cancer, and pulmonary fibrosis.

**Siderosis and lung cancer**

It is known that cigarettes smoking is the main risk factor in the development of lung cancer (70-90% of cases). However there is a significant fraction attributable to occupational factor in the development of these diseases [25,26]. Occupational exposure to certain toxic, radiation or dust from work, have a slightly increased risk of developing cancer in some people. Identification of these carcinogens in the workplace has always been an interest in occupational medicine.

Lung cancer is the most common in case of simple pneumoconiosis than among those complicated with progressive massive fibrosis [27,28]. The smelting activities of iron and steel are classified by the International Agency for Research on Cancer in Group I carcinogen for humans – produce lung cancer [29]. Regarding the carcinogenic risk of exposure to welding fumes and gases, the literature is controversial. The studies made so far are relatively few and intake of iron powders and iron oxides is relatively low as carcinogens, given that welding fumes contain other elements, such as ozone, carbon monoxide, carbon dioxide nitrous oxide. The welding smoke composition, depends on many factors including the type of metal to be welded and the type of electrode used. The most important iron ore is iron trioxide (Fe2O3 – hematite). There was an increase of lung cancer in miners hematite, as in the case of tuberculosis and interstitial fibrosis. Hematite miners are simultaneously exposed to silica and other minerals as well as radioactive materials. The etiology of lung cancer may be related to the influence of cigarette smoking or occupational carcinogen exposure [4].

Vallières et al [26] investigated the relationship between occupational exposure to gases and welding fumes in relation to the risk of developing lung cancer among exposed workers. They concluded that the risk of developing lung cancer among occasional smokers exposed to welding fumes and gases was increased, compared with moderate and heavy cigarette smokers, occupational exposed to these agents [26]. Epidemiological studies have reported an increased risk of lung cancer (30-40%) in workers exposed to welding fumes [30,31,32,33]. The results of these studies are difficult to compare between them. While some authors have concluded that the risk apparently linked to welding fumes is actually attributed to concomitant exposure to asbestos fibers or of smoking [34,35,36] other authors state that this risk can not be explained only by these assumptions [30,37,38].

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