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Occupational Interstitial Lung Diseases (Part I)

Enfermedades del intersticio pulmonar de causa laboral. (Parte I)

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The possibility exists that the global workforce, which is approximately 140 million people, may suffer from an occupational disease in practically every task it performs.[1] However, despite that risk in the working population, few published studies have estimated the incidence of these lesions or diseases, their final cost in terms of medical expenses, the health of the affected individual and economic losses related to hours of work. Methodological failure, inconsistency between the members of the research population or poor records are factors that cause differences in the notification of occupational diseases and probably contribute to this situation.[2]

As a consequence of changes in manufacturing practices and the use of new materials, occupational medicine specialists continue describing associations between new types of exposure and chronic forms of diffuse parenchymal lung disease (DPLD). In order to understand the association between exposure and disease, specialized physicians must see a high index of suspicion regarding the potential toxicity of occupational and environmental exposure.

The diffuse interstitial lung disease (DILD) related to the inadequate exposure or poor protection of workers in different tasks, such as the usufruct of coal and asbestos deposits, construction, manufacturing, work in shipyards and quarries, and agricultural work, has been recognized as an occupational risk a long time ago.

Interstitial lung diseases caused by exposure to substances present in the workplace (OCCUP DILDs) are a group of significant, preventable diseases. Many different agents cause the OCCUP DILD, some of which are correctly defined, and some have been poorly characterized. The list of causative agents keeps growing. Once considered as “pneumoconiosis”, the list of known causes of OCCUP DILD extends far beyond coal, silica and asbestos. Clinical, radiological and pathological presentations of OCCUP DILD are similar to the non-work-related forms of the disease due to the complex list of pulmonary depuration and repair of the lesion. The attending physician must have a high index of suspicion and keep a detailed occupational record in order to look for potential exposure whenever he/she sees a patient with a DILD. Recognizing an OCCUP DILD is especially important due to the implications related to primary and secondary prevention of diseases among the exposed co-workers of the index case.

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A study of the American Thoracic Society published in 2019 collected a literature review and data summary regarding the occupational contribution to the burden of the main non-malignant respiratory diseases. The purpose of the study was mainly to inform about diffuse interstitial fibrosis, hypersensitivity pneumonitis, sarcoidosis and other non-infectious, granulomatous diseases.

The result indicated that exposure in the workplace contributes essentially to the presence of multiple chronic respiratory diseases, for example: asthma (16%), COPD (14%), chronic bronchitis (13%), **idiopathic pulmonary fibrosis** (**26%**), **hypersensitivity pneumonitis** (**19%**), **granulomatous diseases**, including **sarcoidosis** (**30%**), **pulmonary alveolar proteinosis** (**29%**) and tuberculosis (2.3% in cases of silica exposure).

The conclusion of the study establishes the urgent need to improve clinical suspicion and knowledge of the public health regarding the contribution of occupational factors.[3]

Litow et al classify the OCCUP DILDs into four conditions (frequently overlapped from the clinical point of view):

A. *Pneumoconiosis,* defined as non-cancer pulmonary reaction to inhaled mineral or organic dusts and resulting modification of the parenchyma structure.

B. *Hypersensitivity pneumonitis (HN),* also known as "extrinsic allergic alveolitis". Substantial number of disorders of the organism's immune response to the inhalation of organic or chemical antigens, associated with histopathological, granulomatous-like changes.

C. *Granulomatous diseases with reaction of foreign body or chronic, immune diseases*.

D. *Interstitial diffuse fibrosis*, as a reaction to a severe pulmonary lesion, including the inhalation of irritants.[4]

The following table shows some of the numerous examples of OCCUP DILDs. Given the fact that a lot of cases in the literature are referred to as exceptional, we should consider individual propensity, thus it may occur that one causative agent causes different pulmonary interstitial responses.

The most widely known paradigms of OCCUP DILDs are SILICOSIS and ASBESTOSIS. The acute form of silicosis may adopt a pattern similar to pulmonary alveolar proteinosis (PAP), whereas the non-tumor clinical form of asbestosis can be manifested as an idiopathic pulmonary fibrosis (IPF).

OCCUP DILD SIMILAR TO PAP

PAP is an exceptional disease. Both the surfactant secreted by the huge number of type II pneumocytes and the imperfect removal of the surfactant by alveolar macrophages play a role in the physiopathology of this condition.

PAP is classified into two essential forms: **congenital** or **acquired**. The most common variant is the acquired, and is divided into two sub-classes, **autoimmune** and **secondary**, related to a causal factor. Acquired autoimmune PAP is the most common. Secondary PAP is related to chronic inflammatory processes, tumors, hematologic diseases,

Type	Occupational cause
Pulmonary alveolar proteinosis (PAP)	Silica. Cotton. Chlorine. Indium. Marble dust
Bronchiolitis obliterans (BO)	Acramine. Diacetyl
Non-specific interstitial pneumonia (NSIP)	"Baker's" lung. "Hairdresser's" lung. "Nylon spinner's" lung
Organizing chronic pneumonia (OCP)	Anilines
Idiopathic pulmonary fibrosis (IPF)	Asbestos Aluminum
Giant cell interstitial pneumonia (GIP)	Hard metals. Cobalt. Tungsten
Lipoid pneumonia	Fuels. MWF (metalworking fluids, liquid to work with metals)

immunosuppressor syndromes, and inhalation of inorganic or organic particles.

Abraham and McEuen studied twenty-four cases of PAP through optical and electron microscopy to check if the PAP was associated with silica exposure. A large number of birefringent particles was found in 78% of the cases, as opposed to control groups.

The environmental history that was researched correlated well with the results of analyzed particles, for example, silica "sandblasting", metal fumes in welders and cement particles.[5]

Silicosis is generally a chronic pneumoconiosis. It is not common for the silicosis to adopt an **accelerated** or **acute** form; there aren't many publications due to its low frequency of presentation.

The accelerated variant occurs between 5 to 10 years after exposure to dust with high concentrations of silica and if the person didn't wear respiratory protection.

The acute variant is developed a few years after very intense, brief exposure. It is similar to PAP, due to the accumulation of lipoprotein PAS (periodic acid-Schiff) positive material in the alveolar spaces; and the chest CT shows the radiographic crazy paving pattern. It is difficult to differentiate silicoproteinosis from PAP; the right thing to do is to have good occupational anamnesis. A whole-lung lavage may delay the unfortunate evolution of this form of silicosis.[6-8]

Attached is a case report of a worker who performed sandblasting of metal pieces for five years without personal protection (he was unaware of the risk). Dyspnea FC IV and severe hypoxemia were observed, and the chest CT showed the crazy paving pattern. The evolution was fatal (Figures 1 and 2).

There are some publications in the literature where the form of OCCUP DILD similar to PAP is induced by the inhalation of indium (used in the

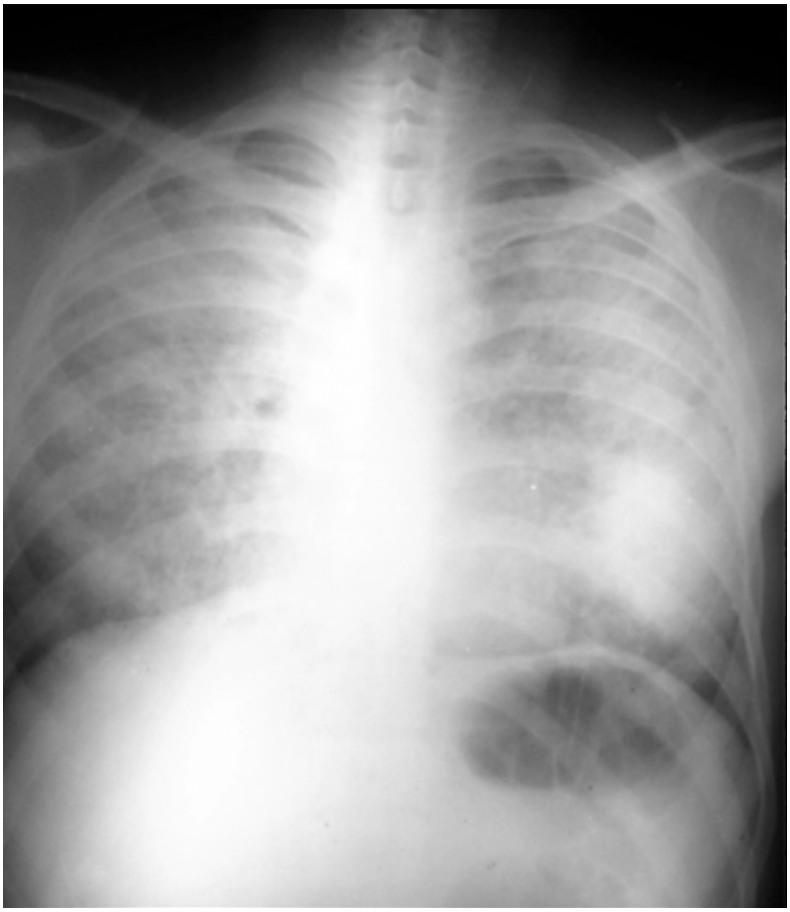

Figura 1. Sandblasting of metal pieces for 5 years without personal protection. Severe hypoxemia. Dyspnea FC IV. Fatal evolution. Silicoproteinosis (postmortem-confimed through lung histology).

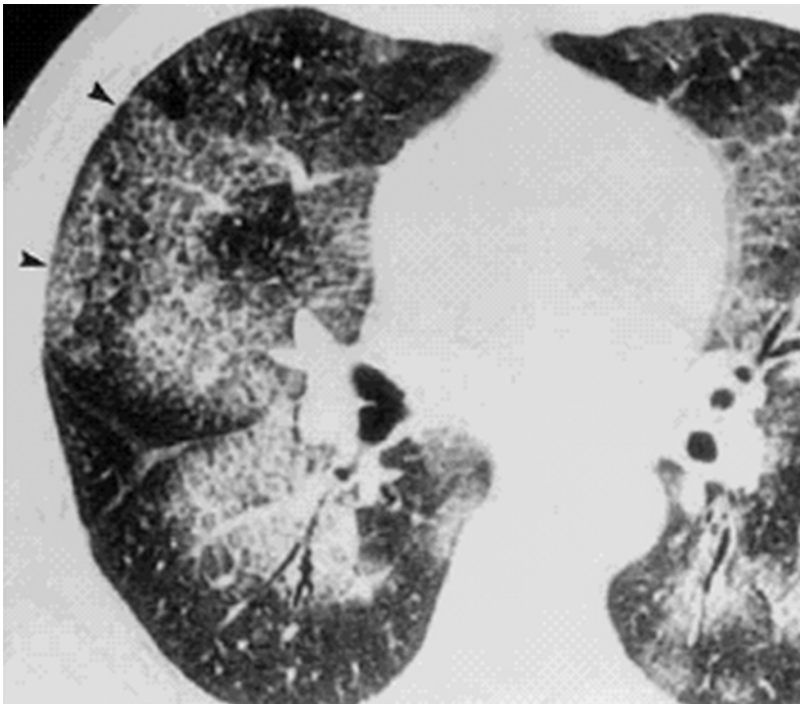

Figura 2. Chest CT shows crazy paving pattern.

electronic industry), aluminum (for the industry of metallization, ship construction, used as dust or for welding), cotton dust (it can be used in cotton bale carding machines), titanium (used by painters, mechanics, artificial jewelry manufacturers and titanium dioxide producers); and there is a case report published in 2018 regarding years of chronic inhalation of chlorine (used for tanning leather in a tannery).[9-15]

OCCUP DILD SIMILAR TO IPF (*IDIOPATHIC PULMONARY FIBROSIS*)

IPF is a chronic interstitial pulmonary disease of progressive and irreversible evolution, with high mortality. The histological pattern is of an usual interstitial pneumonia (UIP). The life expectancy is of 3 to 5 years, once the diagnosis has been established. The therapeutic use of pirfenidone and nintedanib has allowed prolonging the patients' lives, but the best option continues to be lung transplantation.[16] Recent epidemiological research suggests that occupational and environmental exposure, as well as the inhalation of irritants, contribute to cases of IPF, which then ceases to be idiopathic.

Exposure to asbestos may create radiographic and histopathological changes similar to IPF.[17] We should also include smoking, sawdust or wood dust, silica, aluminum, and agricultural activities. The genetic variation among the population may explain the differences in the susceptibility of the presentation of varied patterns of interstitial diseases, even with the same causative agent.

Between 1997 and 2000, Ekström et al investigated the effects of smoking in occupational exposure and found that the fact of being a smoker represented a great danger of developing severe forms of IPF, as well as a direct relationship between the smoking load and higher risks.[18]

It is possible that in many IPF patients, the fibrosis isn't idiopathic because it was caused by occupational exposure. Misdiagnosis can be due to inadequate anamnesis or lack of evident suspicion in the causative agent. Some examples of the types of exposure that contribute to the IPF pathogenia are: organic dust in agriculture, livestock farming, metal, wood and asbestos dust.

One study conducted in Italy in 2008 found two groups of activities with a particularly high risk of developing UIP that increased as long as the occupational exposure continued: gardeners,

veterinarians, farmers and metal workers. People who had their own experience with exposure to the inhaled agent had a higher risk of developing UIP.[19]

Siderosis is the accumulation of excess iron oxide in the alveolar macrophages, and was described in 1946 by Buckell et al.[20] Gothi et al published a case report of a welder with IPF associated with siderosis who had been exposed for 40 years to metal fumes; and confirmed that it was originated by siderosis through transbronchial biopsy, with moderate functional loss one year after diagnosis, but without major changes in the chest CT.[21] Another similar report is that of McCormick et al, in which a patient showed IPF secondary to siderosis even though he had worn a welder's protective mask for 20 years.[22]

For a long time, it was thought to be pneumoconiosis with "benign" evolution, given the lack of signs, symptoms or fibrosis. It is also known as "arc-welders disease" because of the aspiration of welding fumes with poor respiratory protection or without any protection at all.[23]

Aluminum dust has also been an IPF producer; there are publications about it in the literature. In 1990, Jederlinic et al published 9 cases of workers from a factory of abrasives, with a mean exposure of 25 years. Radiographic and functional findings were followed up with biopsy, confirming pulmonary aluminosis.[24]

In 2014, Raghu et al published a similar case of IPF caused by aluminum with bad evolution in a worker who had mechanized, sanded, drilled and rectified Corian for 16 years. This is a synthetic material consisting of one third of acrylic resin and two thirds of aluminum trihydrate. Due to the fact that it is thermoformable and acid-resistant, it is used for the manufacture of kitchen and bathroom countertops and counters.[25]

Sawdust, in the form of inhalable particles, can deposit on the pulmonary parenchyma and damage the workers' health.

It is known that sawdust is carcinogenic to humans. Hancock et al carried out a meta-analysis of 85 studies in order to identify researches. The authors showed an elevated risk in workers exposed to sawdust, and a lower risk in those who worked with soft wood. The study provided important certainty of this occupational cancer, suggesting a different effect between hard and soft woods.[26] The study of Gustafson et al established that wood dust could contribute to the incidence of IPF. To that end, they provided a 30-question questionnaire to 757 individuals with this disease, which was answered by 181 of them. Through statistical studies they found a higher risk among workers who had been exposed to the dust of hard woods and birch wood.[27]

Attached is a case report of IPF caused by asbestosis in an individual who worked as an electrician and manipulated insulation materials for 25 years without personal protection. The chest CT shows pleural calcifications and peripheral signs of "honeycombing" (Figure 3).

OCCUP DILD SIMILAR TO COMBINED PULMONARY FIBROSIS AND EMPHYSEMA (CPFE)

In 1990, Wiggins et al published nine cases of CPFE, but the most important publication about this interstitial disease was presented by Cottin et al with a cohort of 61 cases in which they emphasized the predominance of the condition in men, its relationship with smoking, acceptably preserved lung volumes and severely decreased DLCO (diffusing capacity for carbon monoxide). Chest CT: fibrosis or "honeycombing" in lower lung fields and emphysema in upper fields, with extremely bad prognosis associated with pulmonary hypertension. Survival is lower in CPFE, compared to IPF or COPD.[28, 29]

Workers who manufacture tires may suffer from pleural or pulmonary diseases because they are exposed to the powder used to prevent adherence of the vulcanized surfaces of rubber. Vinaya et al published the case of a worker with CPFE who had been exposed to powder aspiration for 26 years. The development of this condition has also been associated with the use of agrochemicals[30, 31].

OCCUP DILD SIMILAR TO BRONCHIOLITIS OBLITERANS

Bronchiolitis obliterans (BO) is an exceptional obstructive pulmonary disease of the small airways, eventually fatal. It is characterized by the fibrosis of terminal and distal bronchioles and an obstructive airflow pattern with progressive reduction of the respiratory function. It is mostly seen as a non-infectious complication and chronic rejection after lung transplantation. From the occupational point of view, cases have been published of BO caused

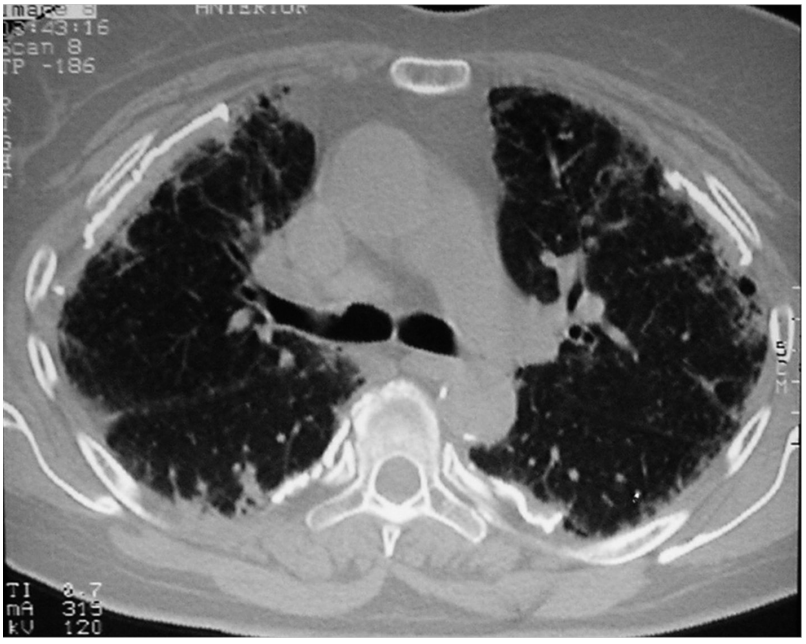

Figura 3.

by artificial flavors of popcorn, where diacetyl stands out. Other causes of BO include exposure to toxins and inhaled gases, nitrogen oxides, sulfur mustard, fiberglass.

American service members who served in Iraq and Afghanistan suffered from BO after being exposed to a fire in a sulfur mine, with elevated levels of SO_2, and possibly to emissions from open burn pits in military bases, where batteries, plastic and waste had been disposed of by burning with jet fuel. Doujaiji and Al-Tawfiq, published the case of a patient with BO caused by SO_2 exposure in an oil refinery in the Persian Gulf.[32, 33]

In 1992 there was a series of 22 cases of BO among 257 textile workers in Spain who had been spraying the fabrics with what seemed to be non-toxic dyes. Similar products caused a minor situation in Algeria, with one death and 2 severe cases.[34, 35]

Experimental studies showed that the textile paint that was used (Acramin FWR and Acramin FWN) was highly detrimental to the respiratory system.[36]

Nanoparticles (NPs) are widely used at present, and, due to their size, they are included in the range of breathable elements, because they remain in aerial suspension as aerosols. They are used in the paper, pharmaceutical, paint, and cosmetic industries. Cheng et al published the case of an individual who developed BO after exposure to titanium NPs in paint. Recent experimental research revealed inflammatory phenomena and formation of granulomas after unprotected exposure to NPs.[37]

Cullinan et al published in 2012 six cases of BO in fiberglass workers, five of which built vessels with that material, and the remaining worker was building a cooling tower. The procedure was as follows: they had to build glass reinforced plastic using resin mixed with styrene and phthalate plus methyl acetone peroxide. The evolution was bad, with one death caused by respiratory failure, two lung transplants and three survivors with poor lung function.[38]

On May 2000, the Bureau of Occupational Health of Missouri, U.S.A, was informed about eight individuals with BO who worked in a popcorn manufacturing plant. The national entity (NIOSH, National Institute of Occupational Safety and Health) was notified of this, and 135 workers were evaluated through questionnaires and spirometry. 117 workers finished the studies, and had a respiratory obstruction rate 3.3 times higher than expected, and 2.6 times higher than expected chronic cough rate.[39] Diacetyl is a volatile, water-soluble compound that vaporizes with heat, and

is a natural component of several foods, including beer and wine. Concentrated formulae of diacetyl are used in the food industry, as flavoring agents, and it is estimated that by 1995, 95 tons had been used per year.[40] If inhaled without respiratory protection or an adequate ventilation, it can cause BO. Studies have been published in this regard.[41]

Diacetyl-induced BO among workers of the food industry is the most widely known and published. After reviewing the literature, a publication has been found of four cases in Brazil: young men, previously healthy, non-smokers. After 1 to 3 years of work without protection, there was obstructive spirometric impairment between 25% and 44% of what was expected, and tomographic studies showed air trapping, representative of BO, confirmed through biopsy.[42] Just like Gulati and Kreiss, the BO diagnosis must include inspiratory and expiratory chest CT, spirometry, and confirmatory biopsy can be avoided in case of compatible exposure. The identification of the causative agent and its cessation is still fundamental for the index case, as well as prevention among co-workers who participate in similar types of exposure. Hygiene and safety include the monitoring of environmental ventilation, the use of respiratory masks and covering food containers in order to avoid leaks.[43, 44]

The notification, study and follow-up of similar cases produced by diacetyl or analogous substances in coffee processing plants have been reported by Reid-Harvey and Bailey.[45, 46]

OCCUP DILD SIMILAR TO PLEUROPARENCHYMAL FIBROELASTOSIS (PPFE)

PPFE is an exceptional pulmonary disease, with rare radiological and histopathological clinical characteristics. Since the 2013 classification, it has been recognized as an idiopathic interstitial pneumonia resulting from the combination of visceral pleura fibrosis and fibroelastosis changes at the subpleural pulmonary parenchyma. The chest CT provides characteristic images that raise the suspicion of this disease. Present as a cause of various diseases, there isn't a unique triggering factor for PPFE; reports have been published of chemotherapy sequelae, bone marrow transplant, collagenopathy or as a consequence of lung transplant rejection.[47-52]

Despite its uncommon incidence and prevalence, there are some publications that relate the PPFE with exposure to toxic substances in the workplace. In 2011, Piciucchi et al published the case of an individual with PPFE who had high exposure to asbestos; and in 2018, Xu et al published a similar case report in which both asbestos and silica were associated with this condition.[53, 54]

The publication of Okamoto et al reports the case of a patient with PPFE who works as a dental technician. These artisans are exposed to an unlimited number of materials such as silver, cobalt, chrome, nickel, silica, indium and titanium. The prevalence of pneumoconiosis among them is estimated at 4.5%-23.6%, with a mean exposure of 12.8 to 28.4 years.[55-57]

However, regarding this particular disease, cases have been published caused by chronic inhalation of aluminum in individuals without respiratory protection, such as the reports of Huang, Chino and Yabuchi. This metal generally produces OCCUP DILD similar to IPF, but the repair mechanisms of the pulmonary parenchyma show individual sensitivity that causes this response.[58-60]

OCCUP DILD SIMILAR TO DESQUAMATIVE INTERSTITIAL PNEUMONIA (DIP)

First described by Averil Liebow et al, the main characteristic of DIP is the accumulation of macrophages both in the lumen and walls of the alveoli. Patients with this disease are usually heavy smokers; and the masculine gender is predominant. Clinical symptoms are non-specific, and due to the fact that patients generally respond to treatment with steroids, this disease has a better prognosis than IPF.[61]

The literature has published case reports about work-related DIP. In many cases, the patient is a heavy smoker.

Abraham and Hertzberg studied 62 samples of DIP confirmed by biopsy using electronic microscopy and X-ray scattering analysis, in search of inorganic particles. Seven out of seventeen analyzed cases had an occupational history of exposure to that type of elements, and high concentrations of those were found in their biopsies. The authors certified specific classes of particles in 92% of the patients, that is why they suggested a relationship between DIP and the worker's profession.[62]

Interstitial lung diseases are not commonly associated with primary aluminum production. OCCUP DILD induced by aluminum is very rare, but the experience suggests that exposure to metal

fumes and dust may cause diffuse changes in the parenchyma, such as granulomas, PAP or DIP.[63]

Lijima et al published the case report of a worker with a smoking load of 60 packs per year who developed DIP caused by aluminum exposure and showed excellent evolution after corticosteroid treatment and smoking cessation. The oriented anamnesis revealed certain tasks of aluminum processing that include melting the metal in molds for engine covers and polishing the pieces.[64]

Blin et al published the case report of a patient with DIP who had been exposed to asbestos working as a plumber and then worked as a caster, with potential exposure to copper, bronze, iron, aluminum and zirconium alloys. He had a smoking history of 30 packs per year, and had quitted 10 years before the consultation.[65]

Zirconium is a corrosion-resistant material used in the aerospace, airline, and nuclear power industries. Thanks to scientific research tests, it is known that after repeated use, it may cause a hypersensitivity skin reaction, forming granulomas of epitheliolid cells. Experimental studies showed lung alterations such as granulomas and interstitial fibrosis after exposure to zirconium. There are few publications in humans regarding pulmonary diseases related to zirconium.[66]

Kawabata et al studied 31 patients with DIP of varied etiologies, where 93% of the patients were male smokers. These patients had been followed-up for more than 99 months. 14 patients who had been monitored for a longer period showed 5 cases of IPF and 4 of lung cancer. The conclusion of this study was that, with time, DIP can progress to IPF, despite the treatment.[67]

Coal power plants are still an important source of power supply worldwide, so miners will continue suffering from pneumoconiosis and dying because of it. After the approval of the Federal Coal Mine Health and Safety Act of 1969 in the U.S.A., measures were adopted to restrict exposure, thus causing a persisting reduction in the prevalence of pneumoconiosis among coal workers in the U.S.A. from 1970 to 2000.[68]

Jelic et al studied, in 25 autopsies with microscopy and polarized light, the number of intramacrophageal silica particles and the degree of fibrosis in antracosilicosis, and in 21 cases, respiratory bronchiolitis associated with smoking. The proportion of particles found was significantly higher in the cases of occupational disease (331:4 $p < 0.001$).

The presence of intra-alveolar macrophages full of particles of different fibrosis levels indicated a new disease: chronic DIP, as a predecessor of diffuse fibrosis and emphysema related to stone coal. Among smoking miners, smoking-related fibrosis wasn't significant in relation to occupational DIP.

Counting the number of particle-laden macrophages in the BAL (bronchoalveolar lavage) of miners can predict the disease and suggest prevention measures.[69]

OCCUP DILD SIMILAR TO LIPOID PNEUMONIA (LP)

LP, caused by the presence of lipids in the alveoli, is a rare condition. It is classified into two main groups, depending on the source of the oily substance: exogenous or endogenous. It has an insidious onset and non-specific respiratory symptoms, such as dyspnea, fever or cough. Tomographic findings include nodules, ground glass, crazy paving or consolidation. Since this condition is not suspected, the diagnosis is delayed or unnoticed. It may simulate many other lung diseases, including carcinoma. LP is a chronic foreign body reaction, identified with lipid-laden macrophages. The diagnosis requires a high index of suspicion and may be confirmed through respiratory samples showing the macrophages. Treatment guidelines are not properly defined. The aim of the diagnosis in the exogenous form of the disease is to avoid exposure. Steroids as a therapeutic option seem promising.[70]

Normally, exogenous LP is associated with the accidental inhalation of oil laxatives to treat constipation, as can be seen in the series of 44 cases of Gondouin et al, where only 4 patients had an occupational origin.[71] Fire-eaters are a workgroup inside the entertainment industry. Possible complications of their work must be taken into consideration. The normal procedure consists in blowing the pyrofluid against a burning stick, so there's the risk that the fire may propagate to the mouth.[72]

The first description in the literature about this occupational hazard was related to accidental aspiration and was reported in 1971. Since then, there were other publications of LP in fire-eaters. It is easier to make the diagnosis of the clinical condition after a good patient inquiry.[73-76]

According to Gentina et al, fire-eaters use different pyrofluids, the most common being the

Kerdan, an oil product of reduced viscosity that unfortunately spreads rapidly through the bronchial tree after accidental inhalation.

Between October 1996 and January 2001, these authors reviewed seventeen subjects, ten of which had been working as fire-eaters for years. Their mean age was 24.5 years, and they showed pleural pain (100%), dyspnea (93.3%), fever of more than 38.5°C (93.3%), cough (66.6%) and hemoptysis (26.6%). One patient suffered an hemodynamic shock and had to be treated at the ICU, where he/she developed a pleuropulmonary infection. Upon discharge, he/she had a good evolution but with sequelae (opacity in the middle lobe and pleural scar). Complete pulmonary resolution occurred after 14 days in fifteen patients.[77]

Apparently, according to consulted publications, it is very common for the employees of gas stations to practice fuel siphonage, which may be a potential risk of aspirating liquids or vapor. Directed inquiry allows us to suspect the cause of this particular LP and obtain diagnosis and treatment success.[78-82]

Sometimes, suspicion requires a thorough inquiry. The patient from the study of Dhouib et al lubricated cars for eight years with an oil *spray* without any respiratory protection; this delayed his/her etiologic diagnosis for 2 years.[83]

Han et al reported three cases of LP caused by the use of a paraffin aerosol. Their diagnosis was confirmed through biopsy and through the study of the samples by x-ray diffraction. The workers had long-term occupational exposure to paraffin, and the environmental concentration of the product in the workplace was specifically higher than the levels measured outdoors.[84] Very exceptional cases of LP were caused by herbicides or solvents used for dry cleaning in the laundry.[85, 86]

In short, a good patient occupational inquiry is extremely useful because it generally provides us the key to guide and elucidate the diagnosis.

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