The Pneumoconioses

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1. Introduction

1.1 Overview on pneumoconioses

Pneumoconiosis is an occupational lung disease caused by the exposure to dust. This section summarizes the generalities on pneumoconioses, including definitions, epidemiology and clinical manifestations of those occupational and environmental lung diseases. The most important step in the diagnosis of pneumoconiosis is to question the subject regarding specifics of the actual job and the minerals or materials involved in case of history of dust exposure. It is very important to seek a detailed account of workers’ past employment, too; as some pneumoconioses develop after only a brief but intense dust exposure.

Lately, many cases have been linked to environmental exposure to dust. This suggests that pneumoconiosis is no more a pathology exclusively related to work. Clinicians and epidemiologists should keep in mind the fact that cigarette smoking has a devastating impact of the health of dust-exposed individuals. Rates of cigarette smoking as high as 80% have been recorded among miners and other dust-exposed populations (Baum et al, 1998; Hammond et al, 1979). Asbestos-related diseases, silicosis and coal worker’s pneumoconiosis (CWP) are most predominant and widely investigated pneumoconioses.

Pneumoconioses have relatively specific radiologic features that are not well-known to most physicians. Radiological imaging plays an important role in the diagnosis of those occupational lung diseases, including asbestos-related diseases, Silicosis and coal worker’s pneumoconiosis (Ngatu et al, 2010; Blum et al, 2008). This review is aimed at providing health care workers, especially clinicians, with basic and accurate knowledge on principal radiologic features often found on a pneumoconiotic chest radiograph that characterize each of the above mentioned lung diseases related to occupational or environmental exposure to dust.

1.2 Asbestosis and other asbestos-related diseases (ARDs)

1.2.1 Definitions and epidemiology

Asbestosis refers to the pneumoconiosis caused by inhalation of asbestos fibers. It is characterized by a slowly progressive, diffuse pulmonary fibrosis. Asbestos is a fibrous
mineral whose specific properties have encouraged its use since ancient times, in particular for industrial applications since the 19th century (Tarres et al, 2009; American Thoracic Society [ATS], 2001). Few natural materials used in industry have been the subject of more epidemiological and pathological research than the fibrous mineral, asbestos. Asbestos fibers are divided into two categories based on fiber’s shape:

- serpentine fibers (curly, long strands) of which chrysotile (white asbestos) is the most commercialized; it accounts for more than 90 percent of the asbestos produced in the United States;
- amphibole fibers (long and straight) which include amosite (brown asbestos), tremolite, crocidolite and other types of asbestos.

Of the different asbestos fibers used in industries, crocidolite (blue asbestos) is generally considered to be less toxic. The amphibole and amosite are reported to carry the greater risk of causing other asbestos-related diseases such as pleural plaque, mesothelioma and lung cancer. Exposed workers who have developed asbestosis are at risk of fatal complications (Wagner, 1997; Sichletidis et al, 2009; Mossman et al, 1990).

In patients with asbestosis, the chest radiograph usually shows small bilateral parenchymal opacities with a multinodular or reticular pattern that may be associated with pleural abnormalities. However, in some individuals with histopathologic evidence of pulmonary fibrosis, no interstitial abnormalities are found on the chest radiographs (Larson et al, 2010; Kipen et al, 1987). Other asbestos-related disorders are: pleural plaques, mesothelioma, diffuse pleural fibrosis, rounded atelectasia, benign asbestos-related pleural effusion and chronic bronchitis. In the course of asbestosis development, other radiographic features such as honeycombing changes and ground-glass opacities can be seen on the radiograph; they are well visualized on computed tomography (CT) and high resolution computed tomography (HRCT) scans.

It is estimated that approximately 27 millions workers in the United States received a significant exposure to asbestos during the middle of the last four decades of the twentieth century.

**Pleural disease** is one of the characteristics of asbestos exposure. Pleural plaques are present in about 50 percent of asbestos exposed individuals and most commonly involve the parietal pleura, while involvement of the visceral pleura is scarce. The histological examination of pleural plaque specimens shows the presence of predominant acellular bundles of collagen (Yang et al, 2010).

The prevalence of both pleural plaques and asbestosis is associated with “time since the first exposure” (TSFE) to asbestos, the “exposure intensity level”, the “duration or cumulative exposure” to asbestos. Meanwhile, time since the first exposure seems to be the best predictor for pleural plaques related to asbestos exposure, whereas cumulative exposure to asbestos is reported to be a major determinant for asbestosis (American Thoracic Society [ATS], 2004; Schart, 1991). In a study conducted in Finnish construction, shipyards and asbestos industry workers by Koskinen and coworkers (1998), a strong relationship was found between pleural plaques and time since the first exposure to asbestos. A recent study by Paris et al. (2009) showed for the first time, in a multivariate analysis, strong and independent correlations between time since first exposure and pleural plaques, and between cumulative or level of exposure with both pleural plaques and asbestosis, indicating that time and dose parameters should be included in the definition of high-risk populations in screening programs.

**Malignant mesothelioma** is an aggressive tumor that develops from the mesothelial cell of the pleura; it may also develop from the peritoneum, pericardium, or testicular tunica vaginalis.
The association between malignant mesothelioma and asbestos exposure has been well-known worldwide since the 1950s. Approximately, 90% of malignant mesothelioma cases are related to earlier exposure to asbestos and the risk of developing the disease is greater in case of exposure to the amphibole fibers in crocidolite and amosite, but serpentine fibers in chrysotile can also cause the disease with a relatively long latency period (30 years or more) (Fujimoto et al, 2010; Snashall et al, 2001). In Japan, one of the biggest consumers of asbestos in the last four decades of the twentieth century, the number of cases of malignant mesothelioma is shown to correlate with the amount of asbestos consumption and the country has been expected to confront an epidemic of asbestos-related malignancy (Suganuma et al, 2001; Takahashi, 1999). The incidence of malignant pleural mesothelioma, an asbestos-related tumor, is increasing, with a median survival of seven to ten months; its clinical pattern usually involves substantial pain and dyspnea. The disease causes approximately 15,000 to 20,000 deaths per year worldwide (Pass et al, 2008).

**Lung cancer** can be consecutive to asbestos exposure. Clinician’s search during risk assessment in patients with possible dust exposure may be hindered by the long latency period between the inhalation and the appearance clinical manifestations. In addition to its fibrogenic properties, asbestos is a first-level carcinogen and the most accepted oncological model is the dose-response without safety level (Rosell-Murphy et al, 2010; Goldberg et al, 1999). According to some previous studies, smoking increases the risk of lung cancer in asbestos exposed individuals by 50 – 90 times more than non-smokers (Valavanidis et al, 1996; Hartly et al, 2000).

A variety of occupational and nonoccupational settings can be source of exposure to asbestos:
- Manufacture of asbestos products
- Thermal and fire insulation
- Construction and demolition work
- Shipbuilding and repair
- Building maintenance and repair
- Plumbers and gasfitters
- Vehicle body builders
- Electricians
- Carpenters
- Mining...

Since a while, there is an increasing number of reports on cases of asbestos-related diseases in workers whose occupations have not been traditionally on the lists of occupational health specialists. Asbestosis and pleural plaques have been found in dentists and mechanics in Europe and the United States. And in some cases, these asbestos-related abnormalities are of unusual source of exposure.

Torbica and Krstev (2006) suggested that asbestos-related diseases in dentists might be linked to exposure to inorganic dust in the manufacturing of cobalt-chromium-molybdenum- based dental protheses. With the use of white asbestos-made lining material for casting rings in dentistry, dental technicians and dentists are potentially exposed to asbestos; and some cases of pleural plaques and malignant mesothelioma have been reported in a number of dentists after a relatively long period of work (35 to 45 years) (Choel et al, 2001; Reid et al, 1991; Sichletidis et at, 2009; Radi et al, 2002).

Similarly, pneumoconiosis may occur in mechanics. Most motor vehicles, from passenger cars to heavy duty trucks, have disc brakes on the front wheels and drum brakes on the rear...
wheels which help control their movement. Asbestos containing brake lining is generally found in those vehicles, ranging from 35% to 65% of chrysotile contents. Their repair or replacement can be a source of exposure to asbestos (Gilles, 2005; Paustnebach et al, 2004; Kakooei et al, 2004).

According to previously published reports, the incidence of lung cancer and malignant mesothelioma in auto mechanics is higher than in the general population. In the Unites States, concentrations of airborne asbestos more than 300 times higher than the current permissible exposure limit (Occupational Safety and Health Agency, OSHA) of 0.1 fiber/cc were found (Levin et al, 1999; Lorimer et al, 1976; Rohl et al, 1976).

1.2.2 Clinical manifestations of asbestos-related diseases
For asbestosis, in general, the latency period between exposure and the appearance of symptoms is inversely proportional to the intensity of asbestos exposure. And the majority of patients remain asymptomatic for 20-30 years after the first exposure, even though pleural plaque is present. The first symptom of asbestosis to appear is usually breathlessness with exertion. Dyspnea will follow as the disease progresses; however, cough, wheezing, sputum production are often present when the patient has a history of cigarette smoking. With the progression of the disease, the patient will develop bibasilar crackles heard mostly at the end of inspiration, and clubbing. Pleural disease occurs earlier mostly within 15 years since the initial exposure to asbestos (Epler et al, 1982; Paris et al, 2009).

1.3 Silicosis
1.3.1 Definition and epidemiology
Silicosis is an interstitial pulmonary disease secondary to the inhalation of crystalline silica (silicon dioxide), usually in the form of quartz, and less commonly as cristobalite and tridymite. It is one of the world’s oldest known occupational diseases characterized by irreversible, progressive lung disease. Crystalline silica has long been considered as the toxic form of silica; however, very little was known about the toxicity of amorphous silica until the very recent toxicological and mechanistic study in animals by Constantini and coworkers that revealed the fact that both crystalline and amorphous (which has been regarded as non-fibrogenic) silica are phagocytosed and are both toxic to alveolar macrophages and have similar pathway that lead to apoptosis.

Silica, the second most abundant element that forms the quarter part of the earth’s crust, is an ubiquitous mineral in human environment. Thus, exposure to silicon dioxide and salts of silicic acid is a fact of life. However, intense exposure can lead to silicosis (Pascual et al, 2011; Wagner, 1995; Constantini et al, 2011; Baum et al, 1998). Silica is also bound to other minerals; it is then called silicate. Silicates are used in some industries; they include asbestos, talc (Mg3SiO10 (OH)2), and kaoline (Al2Si2O5(OH)4) [Gamble, 1986].

New cases of Silicosis are annually recognized worldwide. In the United States, up to 200,000 miners and 1.7 million non-mining workers have experienced significant occupational exposure to inhaled silica; and every year the country register between 3600 and 7300 new cases of Silicosis. The overall mortality attributable to Silicosis has decreased in the United States thanks to the improvement of workplace protection, compared to that of 30 years ago (3000 deaths a year) [Rosenman et al, 2003; CDC, 2008; NIOSH, 1999]. Silicosis can occur in many industries and work settings with evident dust exposure such as:

- Surface mining, underground mining.
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- Quarrying
- Construction work, masonry.
- Sandblasting
- Foundry work
- Ceramics, etc.

The list is not complete; there are numerous other work sites with silica exposure, as free silica can be found anywhere. Mining and construction work that involve drilling, cutting, grinding or crushing the earth’s crust or rocks are associated with high level of silica exposure. In several developing countries, in Africa and Asia particularly, mining and minerals processing industries represent the main source of income. It is common to find employers who fail to provide necessary personal protective materials to workers, putting them at high risk of occupational lung disease such as silicosis.

It is also obvious that, in some occupational settings, workers may be exposed to both silica and asbestos or other dust and develop what is called a mixed-dust pneumoconiosis (MDP). MDP is pathologically defined as an occupational lung disease that shows dust macules or mixed-dust fibrotic nodules, with or without silicotic nodules, in an individual with a history of exposure to mixed dust [Honma et al, 2004]. Radiologic features of MDP may include those related to exposure to silica and other fibrogenic dusts such as silicates, metals, carbon.

1.3.2 Clinical manifestations of Silicosis

Clinical forms of Silicosis are described according to both their clinical and radiological manifestations. They are divided into:

- Simple Silicosis
- Acute Silicosis
- Chronic silicosis
- Accelerated silicosis

Simple Silicosis is characterized by the presence of small (less than 10 mm in diameter) opacities that are rounded in general, on the chest radiograph, and mostly without symptoms.

Acute Silicosis often occurs after weeks or a few years of an intense exposure to silica. The presence of silicotic nodules on the chest radiograph or computed tomography scan is the main radiologic manifestation.

Chronic silicosis usually appears 10 – 30 years after the initial exposure to silica and develops slowly. This clinical form often share the same radiographic feature with simple Silicosis; but in some cases silicotic nodules may coalesce and form a large opacity which represent a progressive massive fibrosis (PMF). Both accelerated and chronic silicosis have similar radiographic features. The only trait that differentiates them is the interval between the exposure to silica dust and the development of the disease symptoms.

Accelerated Silicosis occurs within ten years after initial exposure and is associated to high-level exposure to silica. Both chronic and accelerated Silicosis may be asymptomatic with the patient having only an abnormal chest radiograph consistent with Silicosis. However, symptomatic patients would present with a chronic cough and dyspnea on exertion. Later, symptoms will become severe with the worsening of the radiographic abnormalities. Crackles, rhonchi and or wheeze may be present in some patients.

The presence of large opacity or progressive massive fibrosis is always accompanied by the aggravation of patient’s status and respiratory impairment. The presence of emphysema, air trapping on chest radiograph may be observed (Munakata et al, 1985). Progressive massive
fibrosis (PMF) occurs when small rounded opacities enlarge progressively and coalesce to generate larger opacities (more than 10 mm in diameter). PMF, when it is present, its location is either the upper or the middle zone of the lung.

Malignancy represents one of the complications of Silicosis. The international Agency for Research on Cancer (IARC) determined in 1997 that there was evidence of carcinogenicity of crystalline silica (Scarselli et al, 2011). The relationship between silica exposure and lung cancer risk have been demonstrated in several studies. More than ten cohort studies have shown a standardized mortality ratio (SMR) between 1.37 and 3.70, and few reports showed stronger associations with highest risks and excess mortality from lung cancer among silicotics (Scarselli et al, 2011; Lacasse et al, 2005; Erren et a, 2009; Pelucchi et al, 2006). This suggests that clinicians should keep in mind the relationship between a workplace with exposure to silica dust and the development of lung cancer. Ignoring this fact may mistakenly lead the clinician towards a wrong diagnosis of the patient’s condition.

1.4 Coal worker’s pneumoconiosis (CWP)
1.4.1 Definition and epidemiology

Coal worker’s pneumoconiosis is defined as an occupational lung disease caused by the deposition of coal mine dust in the lung parenchyma and the reaction of tissues to its presence. Coal mining has been used as a source of fuel for hundreds of years, and it still remains a major industry in countries such as the United States, France, Germany, Australia, China, India and South Africa. Recent researches suggest changes in terms of the epidemiology and clinical features of pneumoconiosis among underground coal miners that are characterized by an increase in the severity and rapid disease progression. Factors such as over-exposure to silica dust in coal mines, the increase in coal production and increasing hours of work are thought to be responsible of the current high prevalence and increased severity of the coal worker’s pneumoconiosis (CDC, 2007; Laney, 2009; Antao, 2005).

Laney and Attfield’s more recent work, conducted in the Unites States, demonstrated the role of size of mine as another factor that contribute to the high prevalence and fatality of disease observed the last decades. In their study, workers from small mines had a greater risk of coal worker’s pneumoconiosis and progressive massive fibrosis than their large mines counterparts. Smaller mining operations often have limited capital to upgrade safety equipment, and dedicated safety and personnel are less likely to be available to workers. This influences the implementation of dust monitoring and control activities in the mines (Laney & Attfield, 2010).

1.4.2 Clinical forms of CWP

CWP comprises two clinical forms: the simple and complicated Coal worker’s pneumoconiosis. Generally, simple CWP is not associated with symptoms. It is often characterized by a history of underground exposure of more than 10 years. As for silicosis, the early radiological features are small rounded opacities that first appear on upper lobes of the lung.

In contrast, complicated CWP is associated with symptoms and a marked impairment of lung function. Shortness of breath, cough and sputum production are common symptoms (Caplan, 1953). The pattern and severity of respiratory impairment in coal worker’s pneumoconiosis are related to the levels of coal mine dust exposure, geologic factors, exposure to other respiratory hazards, and the immunologic response to dust (Shen et al, 2004).
2. Classification of High Resolution Computed Tomography (HRCT) scans and chest radiographs of pneumoconioses

2.1 Overview on the classification of pneumoconiotic HRCT scans

The use of Computed Tomography (CT) has revolutionized the radiologic diagnosis of chest diseases as compared with conventional radiography. However, the ability of CT scanners to evaluate pulmonary parenchymal abnormalities is limited because of their resolving power. With the introduction of High-Resolution Computed Tomography (HRCT) techniques, it became possible to perform images of the lung with excellent spatial resolution, providing anatomic detail similar to that available from gross pathologic specimens (Kusaka et al., 2005). Thus, HRCT increases the specificity in the diagnosis of lung diseases, including pneumoconioses. In addition, the early diagnosis of these chronic diseases allows an early care and avoidance of further exposure to the hazardous dusts. For that reason, the proposed International Classification of HRCT of pneumoconiosis is used in some developed countries (such as Japan, Germany and Finland) in the screening and surveillance programs; however, its acceptance by international health institutions might take a time.

The chest radiograph, which is easily accessible and cheaper, presently remains the unique internationally accepted diagnostic tool for pneumoconiosis. For this practical reason, in this chapter, we only present different parameters that are taken into account in the international classification of HRCT of pneumoconiosis.

This section summarizes briefly the information on the proposed international classification of HRCT of pneumoconiosis. Though it is not yet accepted by the International Labor Office (ILO), clinicians should have in mind the existence of this tool which, sooner or later could be of use worldwide.

The purpose of the international HRCT classification is to describe and code parenchymal and pleural manifestations of diffuse non-malignant occupational and environmental respiratory diseases. It is used for the screening (for early detection of pneumoconiosis) and surveillance and follow-up of the exposed individuals (Kusaka et al., 2005).

In asbestosis, the most common HRCT findings are centrilobular nodules on branching areas of high attenuation, and the thickened interlobular and intralobular lines, subpleural dot-like or curvilinear opacities and honeycombing predominantly distributed in the bases of the lungs. The interstitial fibrosis may also be manifested as traction bronchiectasis, honeycombing.

On the other hand, in case of silicosis or CWP, the common characteristic radiologic features on HRCT scan are small opacities predominantly distributed in the upper zones of the lung and, sometimes, images of progressive massive fibrosis (large opacity) (Aberle et al., 1988; Akira et al., 2002).

The reading sheet used in the coding system for CT or HRCT films of pneumoconiosis has the following main parameters to be reported: film quality, parenchymal abnormalities (small opacities, large opacity, emphysema, honeycombing, inhomogenous attenuation of lung parenchyma, pleural abnormalities) (Kusaka et al., 2005).

- **quality of the film**: four grades are considered, similarly to the ILO/ICRP (grade 1, 2, 3, 4; see ILO/ICRP).
- **parenchymal abnormalities**: the absence (No) or presence (Yes) of small opacities (rounded or irregular) has to be reported on the reading sheet; their overall distribution is recorded in a grading system for each side (right, left), according to each zone of the thorax (upper [U], middle [M] or lower [L]). In addition, the severity of the disease is also reported as follows:

\[ 0 \text{ = no definite opacities; } \]
1=mild (present but only few small opacities);
2=moderate (numerous small opacities);
3=severe (very numerous small opacities and normal anatomical lung structure are poorly.

A number of additional parenchymal abnormalities have to be checked:

a. **inhomogenous attenuation** (absence [No] or presence [Yes] which may be due to the
   presence of **ground glass opacities** (GGO). When present on HRCT scan, the
   inhomogenous attenuation should be graded for each side and each zone for its extent
   as follows: 1=focal; 2=patchy; 3=diffuse.

b. **honeycombing** (HC): absence (No) or presence (Yes);

c. **emphysema**: absence (No) or presence (Yes) of emphysema has to be reported, and
   graded for right (R) and left (L) side, and for all zones as 1 (mild [up to 15% of area of
   one zone]), 2(moderate [15-30%]), 3(severe [more than 30%]).

d. **large opacity**: absence (No) or presence (Yes).

- **pleural abnormalities**: absence (No) or presence (Yes); two types of pleural abnormalities
  should be differentiated considering the CT appearance (parietal and visceral type). The
  parietal type refers to the flat thickening of the pleura, whereas the visceral type
  (diffuse pleural thickening) is associated with subpleural fibrosis or parenchymal bands
  (PB) and rounded atelectasis (RA). Their extent, width and location (medias- tinal,
  diaphragmatic,..) are also reported.

- **pleural calcifications**: their absence (No) or presence (Yes) should also be reported on the
  reading sheet.

Detailed information on the international classification of HRCT of pneumoconioses is
available elsewhere (Kusaka et al., 2005).

### 2.2 The ILO International Classification of chest Radiographs of Pneumoconiosis (ILO/ICRP) and ILO/WHO global program for the elimination of Silicosis (ILO/WHO GPES)

The ILO 2000 International Classification of Radiographs of Pneumoconiosis (ILO/ ICRP) is
used worldwide by experts to categorize conditions that are consistent with
pneumoconiosis, with the use of a set of ILO standard films.
This international classification system was created with the purpose of coding anterio-
posterior chest radiographs in a simple and reproducible manner; it provides a means for a
systematic description and recording of radiographic abnormalities in the chest caused by
the inhalation of dusts. It is useful in epidemiologic research, surveillance and medical
checks of dust-exposed workers (Suganuma, 2001; International Labor Office [ILO], 2002).
Chest radiographs of patients with silicosis, coal worker’s pneumoconiosis asbestosis and
other asbestos-related diseases can be interpreted and classified the same way anywhere in
the world using the ILO/ICRP system. In this section, basics on the principal radiologic
features of pneumoconioses and their characteristics are summarized. In order to fully
understand the system, a special training is of utmost importance.
To classify a chest radiograph of a dust-exposed individual, there are four main points to be
considered:

- Technical quality;
- presence (or absence) of parenchymal abnormalities consistent with pneumoconiosis;
- presence (or absence) of pleural abnormalities
- presence (or absence) of other abnormalities (represented by symbols on the reading
  sheet).
Technical quality There are four grades:
1. Good (grade 1);
2. Acceptable, with no technical defect (grade 2);
3. Acceptable, with some technical defect but still adequate for classification purposes (grade 3);
4. Unacceptable for classification purposes (grade 4).

A technical defect may be the presence of artifact, with poor contrast, under (light) or overexposed (dark) film or due to an improper position of the subject (overlapping of scapula, for example) or other defect. Artifacts can be interpreted as pathological abnormalities in case they mimic opacities, while the overlap of scapula (Fig. 1) can easily be interpreted as in-profile or face on plaque. An abnormal feature related to the lung parenchyma or pleura may be present on the chest radiograph, but hidden by the overlapping scapula. When a chest radiograph is qualified as unreadable, it is necessary that another one be indicated.

Fig. 1. Chest radiograph of poor quality showing overlapping scapula (white arrows) looking like pleural plaques on both right and left lung fields.
[Courtesy of Prof. Dr. Yukinori Kusaka, Asian Intensive Reading of Pneumoconiosis radiographs (AIR PNEUMO)]

2.2.1 Parenchymal abnormalities
They include two types of opacities: small and large opacities.

Small opacities are described by profusion, affected lung zones, the shape (rounded or irregular) and size.

- Profusion refers to the concentration of small opacities in the affected zones of the lung; the are four categories of profusion (0, 1, 2, 3) and 12 sub-categories of profusion (0/-; 0/0; 0/1; 1/1; 1/2; 2/1; 2/2; 2/3; 3/2; 3/3; 3/+), where category 0 means the absence of small opacities or the presence of small opacities with a profusion lower than category 1, and category 3 has the highest profusion.

To classify the dust-exposed individual’s chest radiograph, it has to be compared with the standard radiographs from ILO (known as ILO standard radiographs) which profusion
seems to be closer to that in the worker’s film. Standard radiographs provide examples of appearances classifiable as subcategory 0/0; 1/1; 2/2; 3/3.

- **Affected lung zone** is the area the lung where small opacities are seen; each lung field is divided into three zones (upper, middle, lower) by horizontal lines.

- **Shape and size** of small opacities are also recorded; for the shape, there are *rounded* and irregular (reticular) opacities. For *small rounded opacities*, there are three size ranges designated as $p$ (diameter up to 1.5 mm), $q$ (diameter exceeding 1.5 mm and up to 3 mm), $r$ (diameter exceeding 3 mm and up to 10 mm). Similarly, for *small irregular opacities*, the three sizes are denoted by the letters $s$ (opacities with a width up to 1.5 mm), $t$ (width exceeding 1.5 mm and up to 3 mm), $u$ (with a width exceeding 3 mm and up to 10 mm). On each standard radiograph, the type of small opacities and their profusion are shown (for example: $p/p, 2/2$ which means ‘small opacities of $p$ type and profusion 2/2’). However, some chest radiographs may show both small rounded and irregular opacities; this often happens in case the worker was exposed to silica and asbestos, for example (mixed-dust pneumoconiosis).

**Large opacities** are opacities whose longest diameter exceeds 10 mm. There are three categories of large opacities:

- **category A**: one large opacity with longest dimension up to 50 mm, or several large opacities with the sum of their longest dimension not exceeding about 50 mm;

- **category B**: longest diameter exceeding 50 mm, but not exceeding the equivalent area of the right upper zone; or several large opacities with the sum of their longest dimensions not exceeding the equivalent of the right upper zone (Fig. 2);

- **category C**: one large opacity which exceeds the equivalent area of the right upper zone, or several large opacities which, when combined, exceed the equivalent area of the right upper zone.

![Fig. 2. Silicotic chest radiograph showing a large opacity of category B (black arrows) on the right upper zone. Numerous rounded opacities (q type) are present in all zones of the lung field. (Courtesy of Dr Hisao Shida)](image_url)
Fig. 3. Chest radiograph showing calcified diaphragmatic pleural plaques (white arrows) and face-on plaques (blue arrows) bilaterally. In this film, pleural plaques can also be seen in other site (mediastinal region bilaterally).
(Courtesy of Prof. Dr Yukinori Kusaka, AÎR PNEUMO)

Fig. 4. Chest radiograph showing an in-profile pleural plaque (arrows) in asbestos-exposed worker.
(Courtesy of Dr Hisao Shida)

2.2.2 Pleural abnormalities
They comprise pleural plaques (localized pleural thickening), costophrenic angle obliteration and diffuse pleural thickening (DPT).
Pleural plaques may be located on the diaphragm, on the chest wall (in-profile or face-on plaques) and at other sites; they generally represent a thickening of the parietal pleura and are recorded as present or absent. Other details regarding the site (right or left lung), presence or absence of calcification, width and extent of the plaque are recorded.

2.2.3 Costophrenic angle obliteration and diffuse pleural thickening

Costophrenic angle obliteration is recorded as present or absent. However, with the ILO classification, diffuse pleural thickening extending up to the lateral chest wall is recorded only in case of an obliterated costophrenic angle. All details are summarized in a single chest radiograph reading sheet (ILO, 2002).

The global program for the elimination of Silicosis (GPES) was adopted in 2003, Geneva, by a panel of experts in occupational health. It is aimed at establishing a wide international cooperation so as to eliminate Silicosis by the year 2030 (World Health Organization [WHO], 2003). GPES was established following recommendations of the twelfth session of the joint ILO/WHO Committee on occupational health in 1995, which indentified the global elimination of silicosis as a priority area for action in occupational health. The committee wanted countries to place it high on their agendas, with the belief that the experience gained through implementation of this program would provide a prevention model for other pneumoconioses and a proven system for the management of exposure to mineral dust.

The GEPS has two different approaches; the primary prevention of Silicosis that emphasizes the control of silica hazard at source using engineered methods of dust control, while the secondary prevention includes surveillance of the work environment to assess the efficacy of the implemented measures for dust exposure control, exposure evaluation by assessing health risk for workers and surveillance of workers’ health for the early diagnosis of the disease.

In most industrialized countries, following recommendation of the ILO/WHO Global Program for the Elimination of Silicosis, pneumoconiosis prevention programs have been initiated to protect workers from dust exposure and the actual trend of show a decrease in number of new cases. On the other hand, in the majority of developing countries millions of workers continue to be exposure to dust. In order to reach the goal by 2030, each country should have its own national program. Unfortunately, the reality shows that there are still obstacles that might make it difficult to reach the GPES goal by 2030: one is the non adherence of some countries to the ILO/WHO program and, the insufficiency of qualified individuals in the interpretation of pneumoconiotic chest radiographs.

Under the ILO/WHO GPES, experts focused mainly on secondary prevention, upgrading skills of occupational physicians in developing countries in using the ILO 2000 Classification of Radiographs of Pneumoconiosis (ILO 2000 ICRP) and strengthening national Silicosis elimination programs (Luton, 2007).

3. Short training on pneumoconiosis chest radiographs reading

3.1 Objective

The ILO classification system is an important tool for training occupational physicians in the diagnosis of pneumoconioses. Based on this ILO system, some countries have developed their own national program and produced necessary teaching materials. In the United States, the National Institute for Occupational Health (NIOSH) provides certification tests physicians, radiologists and other professionals in the field of occupational health through
the B-readers system. And, since the year 2008, the Asian Intensive Reading of Pneumoconioses chest radiographs (AIR-PNEUMO) program started, organizing annual workshops and certification test for Asian countries and also Brazil. These programs contribute a lot for the achievement of the ILO/WHO Global Program for the Elimination of Silicosis, and also those dedicated to the elimination of asbestosis. However, the number of physicians who have required skills to interpret pneumoconiotic radiographs remains insignificant in many regions of the world, as participation to ILO training course by physicians is quite costly and, moreover, the absence of local trainers constitutes a real obstacle. Countries that do not yet get involved in the ILO/WHO program are numerous. Despite the existence of institution such Air-pneumo, NIOSH and others, the elimination of silicosis may not be achieved by the year 2030 without involvement of governments and health authorities of all nations. Providing basic knowledge on pneumoconiosis to clinicians and those who are about to graduate from medical school would also be helpful for the diagnosis of pneumoconiosis, particularly in regions where occupational or environmental exposure to dust represent a real public health issue. Here is presented a short training on pneumoconioses and the evaluation of its potential to improve physicians’ ability to detect main pneumoconiotic radiographic features on chest radiographs of dust-exposed individuals.

3.2 Differentiating normal and pneumoconiotic radiographs
Radiological imaging plays an important role in the diagnosis of pneumoconiosis. Although chest radiography has some limitations in terms of detecting earliest parenchymal and pleural abnormalities, it remains the most useful and universally accepted method for assessing pneumoconiosis; it is an easily accessible, cheap diagnostic tool (Takashima et al., 2007). On a normal chest radiograph (Fig. 5), there are anatomical structures that resemble abnormal features and may lead the reader to wrong interpretation. The presence of lymph

Fig. 5. Normal chest radiograph showing normal anatomical features. Note the presence of opacities that represent lymph nodes and blood vessels. (Courtesy of Dr Hisao Shida)
nodes, muscles (serratus anterior, oblique externus abdominis...), pleural fat’s shadow and normal cross-section or tangential views of blood vessels can be interpreted as opacities or pleural plaques related to pneumoconiosis, for example. This poses a challenge during chest radiograph reading process and may lead to misclassification of conditions consistent with pneumoconioses (Ngatu et al, 2010; Jinkai et al, 2008).

For individuals who present with pneumoconiotic-like radiographic features, establishing the history of dust exposure is an important step towards the confirmation of the diagnosis. A silicotic or coal worker’s pneumoconiosis’ chest radiograph will often present small rounded opacities (opacities having a diameter up to 10 mm) as parenchymal abnormalities (Fig. 6). When they coalesce, small opacities form a large opacity (having a diameter > 10 mm). In addition, a silicotic radiograph may also show calcified lymph nodes (eggshell calcifications). In silicosis, small opacities are first localized on the upper zone of the lung, then will progress towards the middle and lower zones.

In contrast, a chest radiograph from an asbestosis patient will often present small irregular opacities (opacities having a longest dimension up to 10 mm) as parenchymal abnormalities (Fig. 7) and/or pleural plaque(s). Asbestosis small opacities appear on the lower zone of the lung, then later progress towards the middle and the upper lung zones. Other features may also be present such as diffuse pleural thickening, honeycombing changes.

The table below summarizes frequently found pneumoconiotic radiographic features for mineral dust-exposed subjects: their localization according to the type of pneumoconiosis, and their shape and size for small opacities (Table 1). It is important to know that individuals exposed to both silica and asbestos may develop a “Mixed Dust Pneumoconiosis (MDP); the radiograph may show a combination of features of silica, asbestos or other fibrogenic dusts related diseases.
Fig. 7. Chest radiograph of asbestos-exposed worker showing the presence of small irregular opacities (of “t” type primarily and “s” type secondarily), predominantly on lower and middle lung zones. The right upper lobe is also involved. The profusion is 2/3. In addition, diffuse pleural thickening (DPT) is evident on the right and in-profile plaque on the left; also note the abnormal cardiac size. The film quality is of grade 2 (scapula overlap).
(Courtesy of Prof. Dr Yukinori Kusaka, AIR PNEUMO)

<table>
<thead>
<tr>
<th>Radiographic features</th>
<th>Silicosis</th>
<th>Asbestos / Asbestos-related disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>First localisation</td>
<td>Upper zone</td>
<td>lower zone</td>
</tr>
<tr>
<td>Rounded opacities</td>
<td>often present</td>
<td></td>
</tr>
<tr>
<td>Irregular opacities</td>
<td></td>
<td>often present</td>
</tr>
<tr>
<td>Large opacity</td>
<td>may be present (category: A, B, C)</td>
<td></td>
</tr>
<tr>
<td>Pleural plaque</td>
<td></td>
<td>may be present (face-on plaque and/ or in-profile plaque)</td>
</tr>
<tr>
<td>Diffuse Pleural Thickening</td>
<td></td>
<td>may be present</td>
</tr>
</tbody>
</table>

Table 1. Summarized main radiographic findings related to silicosis and asbestos-related lung diseases
3.3 Implementation of the two-hour training session

3.3.1 Participants and methods

For the first reported training held in 2008, 102 Japanese physicians (72 males and 28 females; 2 to 44 years of experience) having different background (preventive medicine, internal medicine, psychiatrists, surgery, pediatrics) from different cities within the country. Participants were invited to the 2008 training session held in Kochi and Ehime prefecture through the staff of Japanese medical corporations, and the training session was conducted by a NIOSH-B reader from Kochi University Medical School, Japan.

We used three sets of chest radiographs were used, namely ILO/ICRP standard films, dust-exposed workers’ chest radiographs and a set of twelve test films. ILO standard films were used to describe pneumoconiotic lung abnormalities (shape, size, profusion of small opacities; large opacities, pleural plaques) during the lecture. Dust-exposed workers’ chest radiographs, having different radiographic features of pneumoconioses, were provided by the Japan Pneumoconioses Study Group (JPSG). In order to read them, each film was put side by side with the ILO standard whose profusion, for example, seems to be close to that of the patient.

Our intervention was aimed at improving inexperienced physicians’ skill in reading pneumo-coniotic chest radiographs. Participants had to listen to a pre-session talk to remind them the abnormalities to be checked on each of patients’ radiographs (before the main lecture); then, they take a pre-test as described in the protocol (Fig. 8). Each participant received a reading form that had a list of 12 radiograph numbers (from 1 to 12) and they had to check for the presence (Yes) or the absence (No) of parenchymal or pleural abnormalities consistent with pneumoconiosis (opacities and pleural plaques) for each chest radiograph. Classifying the radiographs using categories of profusion of small opacities, location, width or extent of pleural plaques was not indicated. The main lecture was given after the pre-test for two-hours; afterwards a post-test was organized using the same test films that were displayed on viewboxes in the lecture room.

3.3.2 Evaluation of physicians’ reading skill and observed results

The reading forms were collected and managed by s staff who no relationship with any of the participant and all participants were anonymously coded for the analysis of the tests

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Fig. 8. Protocol of the intervention study  (CXR: chest radiograph; JPSG: Japan pneumoconiosis study group)
(pre-test and post-test) results. The difference in the physicians’ reading skill before and after the intervention was evaluated using McNemar’s chi-square test.

The general trend was towards improvement in terms of physicians’ ability to detect pneumoconiotic parenchymal and pleural abnormalities on the test-radiographs. Regarding the detection of the presence of pneumoconiotic small opacities, a significant increase in the specificity score was observed, 65% in the pre-test and 73% in post-test (p<0.0001); whereas the sensitivity score remained high, 84% and 81% in pre- and post-test (p>0.05), respectively (Table 2). It is noticeable that the high sensitivity score in the pre-test was due to a high proportion of physicians who considered the normal radiographs to have pneumoconiotic opacities (false positive or over reading) in the pre-intervention test. The over reading of test radiographs was noticeable for the pre-intervention test for most of the readers.

<table>
<thead>
<tr>
<th>Lung abnormality</th>
<th>Sensitivity</th>
<th>p</th>
<th>Specificity</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>pre-test</td>
<td>post-test</td>
<td></td>
<td>pre-test</td>
</tr>
<tr>
<td>Small opacity</td>
<td>84%</td>
<td>81%</td>
<td>0.204</td>
<td>65%</td>
</tr>
<tr>
<td>Pleural plaque</td>
<td>46%</td>
<td>60%</td>
<td>&lt;0.0001</td>
<td>77%</td>
</tr>
</tbody>
</table>

p: p-value by McNemar’s chi-square test.

Table 2. Overall distribution of the sensitivity and specificity scores of the participants for detecting pneumoconiotic small opacities and pleural plaques on chest radiographs.

<table>
<thead>
<tr>
<th>Group of readers</th>
<th>Lung abnormality</th>
<th>Sensitivity</th>
<th>p</th>
<th>Specificity</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>pre-test</td>
<td>post-test</td>
<td></td>
<td>pre-test</td>
</tr>
<tr>
<td>1. Internists</td>
<td>Small opacity</td>
<td>83%</td>
<td>78%</td>
<td>0.208</td>
<td>67%</td>
</tr>
<tr>
<td></td>
<td>Pleural plaque</td>
<td>41%</td>
<td>61%</td>
<td>&lt;0.0001</td>
<td>83%</td>
</tr>
<tr>
<td>2. Other specialties</td>
<td>Small opacity</td>
<td>85%</td>
<td>83%</td>
<td>0.546</td>
<td>63%</td>
</tr>
<tr>
<td></td>
<td>Pleural plaque</td>
<td>50%</td>
<td>59%</td>
<td>&lt;0.05</td>
<td>72%</td>
</tr>
</tbody>
</table>

p: p-value by McNemar’s chi-square test.

Table 3. Distribution of sensitivity and specificity scores of readers for detecting pneumoconiotic abnormalities according to medical specialty.

For pleural plaques, a marked increase in the sensitivity score was noted, 46% in pre-test and 60% in post-test (p<0.0001), while an improvement was also observed in the specificity score but not significantly (77% and 79% in pre and post-test, respectively) (p>0.05) as shown in Table 2.

When compared according their medical specialties, a relatively similar reading skill improvement was observed between internists and physicians from working in other departments. Higher scores of sensitivity for the detection of small opacities and specificity for plaques were found in internists, and a similar trend was also noted in the group of physicians from other specialties in which a significant improvement of specificity score for pleural plaques was found (p<0.05) in the post-test (Table 3) (Ngatu et al, 2010).

The lack of training for medical doctors in the diagnosis of occupational diseases is the main factor leading to the misdiagnosis of pneumoconiosis as either chronic bronchitis or
pulmonary tuberculosis (Murlidhar et al, 2005). One of the strong points of this training program is that, despite being carried out in relatively short time, it results in a noticeable improvement of physicians’ ability to detect pneumoconiotic parenchymal and pleural abnormalities. Popularizing such program may contribute to the early diagnosis of pneumoconioses, improve their prognosis and give chances of survival for individuals with lung diseases related to dust exposure.

4. Conclusion

This review article provides basic knowledge on pneumoconioses and a practical approach that may help physicians to diagnose occupational lung disorders related to exposure to mineral dust. Meanwhile, a training with demonstrations on the pneumoconiotic chest radiograph reading process is of utmost importance. Holding regular short period training courses for physicians, and even medical students who are about to graduate, focusing on main radiological features of lung parenchymal and pleural abnormalities related to occupational or environmental exposure to dust, with the use of typical ILO standard films, will contribute to improving their skill in the diagnosis of pneumoconioses.

5. Acknowledgments

The authors thank Professor Yukinori Kusaka, Dr Hisao Shida and Dr Masanori Akira for providing materials used in this report.

6. References


The developments in molecular medicine are transforming respiratory medicine. Leading clinicians and scientists in the world have brought their knowledge and experience in their contributions to this book. Clinicians and researchers will learn about the most recent advances in a variety of lung diseases that will better enable them to understand respiratory disorders. This treatise presents state of the art essays on airways disease, neoplastic diseases, and pediatric respiratory conditions. Additionally, aspects of immune regulation, respiratory infections, acute lung injury/ARDS, pulmonary edema, functional evaluation in respiratory disorders, and a variety of other conditions are also discussed. The book will be invaluable to clinicians who keep up with the current concepts, improve their diagnostic skills, and understand potential new therapeutic applications in lung diseases, while scientists can contemplate a plethora of new research avenues for exploration.

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