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Review

Pneumoconiosis among miners in coal mines

Bojan Joksimović[1], Biljana Mijović[1], Biljana Čančar[2], Milena Božinović[1], Zlatko Maksimović[3], Slobodan Stanić[4], Djordje Jevtić[5]

[1]University of East Sarajevo, Faculty of Medicine, Foca, The Republic of Srpska, Bosnia and Herzegovina

[2]University hospital Foca, The Republic of Srpska, Bosnia and Herzegovina

[3]PHI Hospital "Sveti vračevi" Bijeljina, The Republic of Srpska, Bosnia and Herzegovina

[4]Institute of Public Health of Republika Srpska, The Republic of Srpska, Bosnia and Herzegovina

[5]University of Belgrade, Faculty of Medicine, Belgrade, Serbia

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Corresponding author:
Biljana Mijović, MD, PhD
Durmitorska 5, 73 300 Foča
biljana.mijovic@ues.rs.ba

Summary

Pneumoconiosis of workers in brown coal mines is an occupational disease, a global public health problem and a serious disease of the lung parenchyma. If it is not prevented, it leads to irreversible changes in the lungs with complications. The disorder occurs after prolonged exposure to coal dust containing high concentration of free crystalline silica. Data in literature regarding its health impact on people working in coal mines are relatively scarce. Recently, there has been an increase in miners' pneumoconiosis, which requires a stricter policy to protect workers in the mines. There are two classical types of CWP: simple and complicated. The main diagnostic method of CWP is based on a specific X-ray finding, and the auxiliary method of choice is spirometry. The pathophysiological mechanism of CWP formation is not fully known, although it has been shown that damage to the lung parenchyma goes through three phases caused by effect of lung cells exposure to coal dust. Studies show that cytokines play an important role in inflammation and the immune response as mediators of toxic and pathogenic effects in CWP. A link between exposure to coal dust in brown coal mines and the development of CWP has also been demonstrated, with a consequent reduction in the physical and psychological quality of life of workers in the mines.

Key words: pneumoconiosis, coal dust, miners

Introduction

Miners' pneumoconiosis in coal mines is a chronic and irreversible disease that represents a global public health problem. A large number of research is associated with environmental problems caused by mining in coal mines, processing, combustion and similar problems such as acid rain, smog, gas emissions and the like. In contrast, there is insufficient data in the literature on the direct impact of coal on the health of people who work in coal mines and who use coal as an energy source [1].

Inhalation of coal dust during blasting in brown coal mines leads to the development of coal mine dust lung disease (CMDLD). The most common manifestation of CMDLD is pneumoconiosis of the lungs. Coal Workers' pneumoconiosis (CWP) is a parenchymal disease caused by the accumulation of coal dust in the lungs and the consequent reaction of lung tissue, the formation of fibrous nodular lesions [2, 3], and the main cause is prolonged exposure to coal dust which contains high concentrations of free silica crystals [4]. Workers in brown coal mines are at high risk for respiratory morbidity and premature death. Given the global prevalence of mining in coal mines and the potential for severe respiratory diseases, the respiratory health of workers in coal mines is an important problem that should be prevented, given that CWP is an incurable disease.

The aim of this paper is to summarize research on pneumoconiosis among workers in coal mines.

Prevalence and mortality from pneumoconiosis

Considering the chronic nature of CWP, the assessment of the prevalence of this disease is mainly examined through prevalence studies. Blackely and collaborators estimated the prevalence of pneumoconiosis in U.S. miners working for at least 25 years. They conducted a prevalence study using radiograms collected from 1970 to 2017. Each radiogram was classified according to international standards. The prevalence was over 10%, and in central Appalachia as much as 21% [5].

In a study conducted in the Czech Republic, the authors compared the total and specific mortality of miners working in brown coal mines with and without pneumoconiosis, and the mortality of the general male population in the period from 1992 to 2013. The mortality of miners with CWP was higher compared to the general male population [6].

Coal mining and coal processing involves multiple dust generation processes, including cutting, transport, crushing and grinding of coal, etc. Coal dust is one of the main sources of health hazards for miners. Exposure to coal dust can be prevented by administrative and engineering controls. Ineffective control of coal dust exposure can harm miners' health. Although many efforts have been made to eliminate these threats, an unexpected increase in miners' pneumoconiosis (CWP) in the U.S. Appalachian Basin has been recorded in recent years. The authors hypothesized that nano-sized coal dust has contributed to an increase in the prevalence of CWP in recent years [7].

In Colombia, a study was conducted to assess the exposure to coal dust and the prevalence of pneumoconiosis in underground mining in three Colombian mines. The prevalence of pneumoconiosis was 33.8% (95% CI: 27.0 - 41.3%). Pneumoconiosis was significantly associated with the level of severe exposure to coal dust (PR=2.055, 95% CI: 1.043 - 4.048; p = 0.038) and work in underground mining for 25 years or more (for those with 25.0-29.9 years: PR = 2,199, 95% CI: 1,449 - 3,338; p = 0.001) [8].

A meta-analysis of the prevalence of pneumoconiosis among miners in China concluded that the prevalence of CWP remains high in China compared to the UK (0.8%, during 1998-2000) and the US (3.2% in 2000.). In addition, conditions in privately owned mines caused more CWP (9.86%; 95% CI: 1.25-25.17%) than in state-owned mines (4.83%; 95% CI: 2.35-8.13%) (P <0.05). The data clearly showed that regulatory agencies in China need to step up their efforts to implement more rigorous policies to protect miners, especially those in privately owned mines [9].

According to a study to determine the prevalence of CWP in China, in Jiangsu Province, from 2015, it was determined that out of the total number of respondents covered by CWP screening, 5.5% of workers had CWP. Out of the total number of workers working

in brown coal mines with a diagnosis of CWP, 71.1% are still in the first stage of CWP, and 90.7% of this number of workers work directly in blasting or in tunnels. Also, 7.3% of the total number of patients with CWP had a complication in the form of pulmonary tuberculosis, and the mortality of patients with stage III was the highest and amounted to 69.7%. According to the Chinese National Registry for Occupational Health and Poisoning, 23,152 new cases were diagnosed in 2013 [10].

Data on the frequency of CWP in the world indicate that it is necessary to introduce a stricter policy in order to protect workers in brown coal mines, especially for workers working in tunnels or directly in mining.

The introduction of preventive measures in brown coal mines can reduce the incidence of CWP. Thus, in the United States, after the introduction of preventive measures in brown coal mines, the prevalence of CWP was reduced from 30% to 2% [11].

Pathophysiology of pneumoconiosis

There are two classical types of CWP: simple and complicated.

The difference between these two is primarily in the severity of the disease. In simple CWP, fibrous lesions are centered around the respiratory bronchioles, especially in the upper lobes of the lungs, with radiological shadows 1–10 mm in diameter. With increased exposure to coal dust, macro-nodules are formed, which can be 1 to 2 cm in size, and severe symptoms appear at this stage. This stage is called complicated CWP or progressive massive fibrosis. The main diagnostic method of CWP is based on a specific X-ray finding, and the auxiliary method of choice for estimating the severity of obstruction is spirometry [12, 13].

Miners working in brown coal mines are generally divided into two basic groups. Miners who work in tunnels and directly on blasting belong to the group of underground miners. The second group consists of miners working at the surface of the mine. Underground miners are at a significantly higher risk for the development of CWP, due to higher and longer exposure to coal dust, compared to workers working at the surface of the mine [14].

A study conducted by Kurth and collaborators found a significant association between impaired airflow and pneumoconiosis. Impaired airflow was present in 7.7% of non-smoking miners, while in miners with pneumoconiosis it was present in 16.4% of them. The survey was conducted among non-smoking miners participating in the National Institute for Occupational Safety and Health (NIOSH) Coal Workers' Health Surveillance Program (CWHSP) [15].

Although the exact pathophysiological mechanism of CWP formation is not fully known, it has been established that coal dust components interact with cells in the lungs causing cell membrane damage accompanied by lipid peroxidation.

The pathogenic mechanism of CWP takes place in three phases. Initially, there is an accumulation and activation of inflammatory cells in the lungs. Damaged cells release intracellular enzymes, which provoke further tissue damage, resulting in scarring or destruction of alveolar septa. Coal dust phagocytosed by alveolar macrophages stimulates the formation of reactive oxygen species which then stimulate the secretion of cytokines and chemokines. These inflammatory cytokines act as chemoattractants that attract polymorphonuclear leukocytes and macrophages from the pulmonary capillaries in the alveoli and result in chronic inflammation [16].

The second phase of this pathological process consists of alveolar macrophages that stimulate the secretion of fibrogenic factors, which induce fibroblast proliferation and/or stimulate collagen synthesis resulting in the development of pulmonary

fibrosis, which is the main pathological indicator of CWP [17].

The third phase in the pathogenetic mechanism of CWP formation is the increased synthesis of extracellular matrix components [18].

Current concepts of CWP pathogenesis suggest that alveolar macrophages play a key role because of their ability to release various mediators such as proteolytic enzymes and growth and differentiation factors. In the chronic phase of CWP leading to pulmonary fibrosis in pneumoconiotic lungs, cytokines produced by alveolar macrophages play a significant role in the pathogenesis of CWP [19].

Wang et al. did a review of the literature to evaluate the association between IL-1 gene polymorphism and susceptibility to pneumoconiosis. The study included 10 case-control studies. The conclusions of this review suggest that IL-1RA (+2018) may modify worker sensitivity to pneumoconiosis and silicosis. New large-scale replication studies need to be conducted and the link between IL-1RA (+2018) and the risk of pneumoconiosis and silicosis of coal workers needs to be reassessed [20].

Han and collaborators conducted research to investigate the genetic association between single nucleotide polymorphisms (SNPs) of IL-17A and CWP in the Chinese population. A total number of 1391 subjects were included in this study, including 694 subjects in the control group and 697 in the case group. TaqMan qRT-PCRs were performed for genotype rs2275913, rs3748067, rs4711998 and rs8193036 within the IL-17A gene. Luciferase assays were used to determine the effects of the rs8193036 C> T allele on IL-17A expression. The rs3748067 G> A and rs8193036 C> T polymorphisms reduce CWP risk. These findings could be helpful in identifying people at reduced risk of CWP, and further studies justify their validity [21].

Cytokines are also known to play a very important role in a wide range of CWP biological processes such as inflammation and immune responses and are crucial mediators of toxic and pathogenic effects in observed CWP patients [22].

It is well known that various cytokines and growth factors secreted from macrophages/monocytes play a key role in the pathogenesis of pneumoconiosis. They can act as biosensors to predict pneumoconiosis. Kim and collaborators measured tumor necrosis factor-alpha (TNF-alpha), interleukin-8 (IL-8) and platelet-derived growth factor-AA to assess which cytokines can be used as sensitive biomarkers in pneumoconiosis, monocytes with or without coal dust (5 mg / ml) and serum in 42 miners with pneumoconiosis from coal mines and ten healthy controls. The release of carbon-stimulated TNF-alpha and IL-8 from monocytes in the blood was significantly increased in patients with pneumoconiosis compared with controls. Serum TNF-alpha and IL-8 levels were higher in subjects with pneumoconiosis than in control groups [23].

The genes IL-4, IL-4 (IL4R) and IL-13 are key immune factors and can influence the course of various diseases. Wang et al. investigated the association between potential functional polymorphisms in IL-4, IL-4R, and IL-13 and the risk of CWP in the Chinese population. Six polymorphisms (C-590T in IL-4, Ile50Val, Ser478Pro and Gln551Arg in IL-4R, C-1055T and Arg130Gln in IL-13) were genotyped and analyzed in a case control study of 556 individuals with CWP and 541 individuals from the control group. The results of this study suggest that the IL-4 C-590T polymorphism is involved in the etiology of CWP and susceptibility to this disease. Larger studies are needed to confirm these findings [24].

Cytokines produced by macrophages, IL-1, and TNF-α are involved in coal dust-induced inflammation as proinflammatory cytokines. The presence of a constant stimulus and chronic secretion of these cytokines can result in the development of inflammatory diseases such as silicosis and CWP [17].

Inter-individual differences in spontaneous and stimulated IL-1 and TNF-α production give us data indicating that CWP severity is associated with the production of these cytokines. According to the research of Ates and collaborators, performed at the Turkish brown coal mines, the association of the gene polymorphism of the cytokines IL-1, TNF-α, IL-6 and TGF-β in patients with CWP and the severity of this disease was estimated. The results of this study showed that TNF-α (-238) is a risk factor in both the development and severity of CWP, while TNF-α (-308) is important only in the severity of CWP. In contrast, IL-6 had a protective effect on the development and severity of the disease in patients with CWP [25, 26]. According to data in recently published studies, it is clear that the inflammatory cytokines IL-1 and TNF-α are associated with the formation and development of CWP [27].

According to research by Vanhee et al. in vitro exposure of alveolar macrophages to coal dust, coal dust particles caused significant secretion of TNF-α and IL-6 [19].

However, there are few data in the literature on the concentration of these and other important anti and proinflammatory cytokines such as IL-2, 4, 5, 9, 10, 13, 17A, 17F, 21, 22 and IFN-γ, measured in the serum of patients with CWP.

In a study conducted in Foca in the Republic of Srpska, among the miners in the Brown coal mine in Ugljevik, the production of anti and proinflammatory cytokines IL- 2, 4, 5, 9, 10, 13, 17A, 17F, 21, 22 and IFN-γ was examined. It was observed that the average values of anti-inflammatory cytokines IL-6 ($p = 0.03$), IL-10 ($p = 0.02$), IL-4 ($p = 0.02$), IL-17A ($p = 0.02$), were significantly higher in the control group of subjects compared to the group exposed to coal dust [28].

The association between exposure to coal dust in brown coal mines and the occurrence of respiratory symptoms, lung function deficit and the development of CWP has been proven [29].

Cumulative exposure to coal dust has been associated with a decrease in FEV1 among workers in brown coal mines in the United States without radiographically proven CWP [30].

In a study performed on 3,380 British miners with documented CWP, exposure to coal dust was associated with abnormal pulmonary function with FEV1 <65%. Longitudinal studies performed in England [31] and in the United States [32] showed similar results, linking coal dust exposure with FEV1 values.

Differences in risks from exposure to dark and brown coal

It is known that the structure of dark coal is dominated by less sustainable woody structure, higher carbon content (65–80%) and higher calorific value (12.6 to 23.8 MJ/kg), compared to the carbon content (60–65%) and the calorific value (6-12.5 MJ/kg) of brown coal (lignite). There are a small number of papers in the literature that have examined the relationship between work in lignite mines and the occurrence of pulmonary dysfunction.

However, in one study, in a group of 904 young miners in lignite mines in Sardinia exposed to relatively low levels of coal dust, individual exposure to coal dust was associated with a significant decrease in FEV1 and FVC, and the ratio of carbon dioxide diffusion capacity to alveolar volume [33].

These data show that there is a risk of pulmonary dysfunction in lignite mines.

According to data, 4.7 million underground workers in coal mines worked in China between 2010 and 2014, who were constantly exposed to a large amount of occupational hazards, including image dust, coal dust, noise, vibration and heat, which can lead to a large number of occupational physical diseases, the most common of which is CWP [34]. Also, underground miners usually work in much worse conditions, with higher

risk and higher intensity, which together can increase occupational stress [35].

Therefore, the combination of all these factors cannot only lead to the occurrence of physical diseases in miners, but also to disorders in their mental health, which is why it is important to pay attention to the quality of life of underground miners working in coal mines.

Miners' quality of life

Quality of life is a multidimensional concept that encompasses the physical, psychological and social components of health, and is widely accepted as an important parameter of medical care [36].

The 36-item Short-Form Health Survey (SF-36) is widely used to assess the quality of life of people in many fields and studies [37], including the quality of life of workers in brown coal mines [35].

According to a study by Han et al. from 2017 in China on 612 underground miners working in brown coal mines, it was found that the physical component of quality of life was significantly better in surface miners compared to underground miners. Compared to other populations without exposure to mine pollutants, the physical and psychological component of quality of life was significantly better in the group of respondents who do not work in the mines [38].

Also, longer work experience in mines showed significantly poorer quality compared to miners who have shorter work experience [38].

Mijović and collaborators conducted a survey of the quality of life of workers working in the Brown coal mine in Ugljevik between two groups of respondents, one of whom was exposed to coal dust, and the other, controlled, was not. Subjects working in the brown coal mine have significantly lower average values of the domains of physical functioning ($p = 0.005$), general health ($p = 0.001$) and mental health ($p = 0.041$) compared to the control group of subjects.

Also, the average values of the common physical component of quality of life were significantly ($p = 0.007$) lower in the group of miners compared to the control group of respondents. Differences in other domains of the SF-36 questionnaire between groups of respondents were not observed [39].

Conclusion

Review shows that coal workers' pneumoconiosis (CWP) is an insufficiently researched disease. The input of data is especially warranted in fields of immunology and pathophysiology. It is known that there is a disorder at the level of the lung parenchyma in the form of pulmonary fibrosis, with a consequent violation of lung function and diffuse lung capacity, which leads to respiratory failure. Common complications include pulmonary tuberculosis and even lung cancer.

The physical and psychological component of the quality of life of these workers is further impaired in relation to the population of people who do not work in the mine, which imposes the need to work on prevention and improvement of their health.

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Pneumokonioza radnika u rudnicima mrkog uglja

Bojan Joksimović[1], Biljana Mijović[1], Biljana Čančar[2], Milena Božinović[1], Zlatko Maksimović[3], Slobodan Stanić[4], Đorđe Jevtić

[1]Univerzitet u Istočnom Sarajevu, Medicinski fakultet, Foča, Republika Srpska, Bosna i Hercegovina

[2]Univerzitetska bolnica Foča, Republika Srpska, Bosna i Hercegovina

[3]JZU Bolnica "Sveti vračevi" Bijeljina, Republika Srpska, Bosna i Hercegovina

[4]Institut za javno zdravstvo Republike Srpske, Republika Srpska, Bosna i Hercegovina

[5]Univerzitet u Beogradu, Medicinski fakultet, Beograd, Srbija

Pneumokonioza radnika u rudnicima mrkog uglja je profesionalno oboljenje, globalni javnozdravstveni problem i ozbiljno oboljenje plućnog parenhima. Ukoliko se ne prevenira, dovodi do ireverzibilnih promjena na plućima sa komplikacijama. Nastaje nakon produženog izlaganja ugljenoj prašini sa visokom koncentracijom slobodnih kristala silike o čemu je malo podataka u literaturi o direktnom uticaju na zdravlje ljudi koji rade u rudnicima uglja.

U posljednje vrijeme zabilježen je porast pneumokonioza rudara, što zahtijeva striktniju politiku zaštite radnika u rudnicima. CWP se dijeli na jednostavnu i komplikovanu u zavisnosti od težine bolesti. Glavna dijagnostička metoda CWP se zasniva na specifičnom rendgenskom nalazu, a pomoćna metoda izbora je spirometrija. Patofiziološki mehanizam nastanka CWP nije u potpunosti poznat, mada je pokazano da oštećenje plućnog parenhima prolazi kroz tri faze usljed reakcije ugljene prašine i ćelija u plućima. Istraživanja pokazuju da citokini imaju važnu ulogu u inflamaciji i imunskom odgovoru kao medijatori toksičnih i patogenih efekata u CWP. Dokazana je i povezanost između izloženosti ugljenoj prašini u rudnicima mrkog uglja i razvoja CWP sa posljedičnim smanjenjem fizikalnog i psihološkog kvaliteta života radnika u rudnicima.

Ključne riječi: pneumokonioza, ugljena prašina, rudari