BYSSINOSIS

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Byssinosis as a nonspecific chronic respiratory disease in textile workers exposed to cotton, flax, jute, hemp and sisal is described. Opinions about aetiopathogenesis are presented. The following chapters are focused on clinical symptoms and functional lung changes. Classification of the disease is described afterwards. Finally, the necessity of medical and technical measures to prevent the development of this disease in textile workers is emphasised.

CHARACTERISTIC

Byssinosis is a chronic nonspecific respiratory disease in textile workers exposed to dust of cotton, flax, hemp, jute and sisal. It mostly arises after more than 10 years of exposure to dust. It is characterized by chest tightness and breathlessness, which the worker experiences periodically most severe on the first day of the working week after the days of absence from work. In the following days the symptoms are less troublesome and subsequently they disappear at all. In the following course of the disease the symptoms persist in the course of the whole working week, later they do not disappear not even on the nonworking days. Subsequently – as a rule – the symptoms of chronic bronchitis and emphysema associate to byssinosis. In the late period the disease causes the respiratory insufficiency and development of cor pulmonale.

EPIDEMIOLOGY AND OCCUPATIONAL RISK

Byssinosis occures in different countries of the world. The data about its prevalence are very different. For example in Great Britain, the country where cotton processing has the longest tradition, the prevalence has been falling. While the original principal studies recorded a rate of around 50% in workers in dusty parts of spinning mills[1, 2], this number continued to fall. Recent studies have reported about 10% prevalence rates and current studies state prevalence 3–4% in spinning mills; a far lower (0.3%) rate has been found in weaving mills[3, 4, 5, 6]. The reduction in prevalence has been explained by a reduction in dust exposure. In the USA technological improvement has resulted in large reduction in dust exposure and the disease has practically disappeared[7]. Similar prevalence rates, as experienced in UK in the 1950s and 1960s, are now being experienced in the developing countries where cotton production is increasing. Prevalence rates of byssinosis of about 30% in Indonesia[8], 37% in Sudan[9], 40% in Etiopia[10] and up to 50% in India[11, 12] have been experienced. Jaroš in Slovakia stated the byssinosis rate of 5% in the factories processing flax[13]. In our country Navrátil detected clinical symptoms of byssinosis in 1.7% of cotton spinners in 1966[14]. During the thorough examination in 118 spinners – women in 1977 he did not find any case of byssinosis[15]. Although in all the studies the same standards for diagnosis of byssinosis have not been used, it is possible to say that while in some countries the occurence of the disease is decreasing owing to lower dust exposure and recession in textile industry, in other countries the disease is becoming very frequent. Generally, many studies confirm that the prevalence of byssinosis is increasing with longer exposure and with dust concentration at the workplace[2, 16, 17, 18, 19]. According to Barnes, also microclimatic conditions in working climate assert. In Australia he found lower prevalence of byssinosis than in England, while the dust concentration in the Australian mill was eight times higher than at the workplace in England[20]. The fibres of jute and sisal have lower capability in comparison with flax, cotton and hemp to provoke byssinosis[21].

PATHOLOGICAL ANATOMY

The pathological features of byssinosis have not been clearly defined yet. In the most studies the authors acknowledge the hyperplasia of mucous glands and mild smooth muscle hypertrophy of bronchi and neutrophil recruitment, but these changes are not specific for byssinosis[22]. In mortal cases, among pathological findings the symptoms of chronic bronchitis, emphysema and chronic cor pulmonale can be found in the foreground. No studies have been performed so far in which bronchial biopsy or samples of BAL, both in satisfactory number, in individuals with a well substantiated diagnosis of byssinosis have been investigated.

AETIOPATHOGENESIS

Aetiological mechanism of the disease is still not cleared. It is sure that byssinosis is not a simple reaction to inert dust. Studies have shown that washed cotton, even though in comparable dust concentration, has a very low capability to evoke lung changes[23]. Although pure cotton contains some histamine, its concentration is too low to cause bronchoconstriction in man[24]. The airy environment in spinning mills is a milieu of biochemical and microbiologic agents capable of provoking pulmonary reactions.

Among immunological mechanism an early reaction caused by IgE antibodies, alergic immunocomplex reaction and activation of complement are often mentioned in aetiopathogenesis. Even when alergic IgE mechanism is plausible as to highly antigen composition of cotton dust and its contaminants, a number of studies have not proved a relationship between atopy and disease[25]. Precipitating IgG antibodies are present in cotton workers. There are the highest titres in workers with byssinosis and they are the greatest at the beginning of the working week. Nevertheless, the tests with identified compound of dust failed to provoke any pulmonary changes which could support these theories[26]. Activation of complement has been described. But even this mechanism does not give any satisfactory explanation[26].

One of suspicious aetiological agents are endotoxins. By studying microbiological contaminant of textile dust and its influence on the origin of the disease, a relationship between Gram positive and Gram negative bacteria and byssinosis has been found. The relationship is the closest in the case of Gram negative bacteria. Endotoxins are a heterogenous group of lipopolysacharids. Inhalation of endotoxins causes both bronchoconstriction and an inflamatory response including reproducing of polymorphonuclears[27]. In people experimentally exposed to cotton dust, the levels of endotoxins were measured and these correlated most closely to bronchoconstriction[28]. Nevertheless, the exposure to endotoxin occures in many workplaces where organic dust is present, for example swine confinement buildings and poultry farms, but workers in these industries do not suffer from byssinosis-like syndrome[6, 26].

In the cotton dust also tannins are present. They are capable to provoke mild changes in epitel and in muscle of bronchi[29, 30]. Till now, no epidemiological study has included measurement of tannins in vivo and the role of tannins has not been cleared yet.

SYMPTOMS

The danger of professional exposure in textile workers has been known more than 300 years. In 1705 Romazini described respiratory symptoms in workers with hemp. Then Kay described respiratory disease in workers working with cotton (1831). He reported work-related cough and chest tightness and later an unusual periodicity of the disease. The English author Proust, who was the first to use the term byssinosis (1877), pointed out the shortness of breath which occured in older workers in textile industry on Monday, while at the end of the working week it abated. The name byssinosis originates from the Greek word byssos, which means cotton in Greek. Schilling, who described and classified specific features of the disease called byssinosis, carried out the first substantial epidemiological study in the workers with cotton nearly one hundred years later[1].

Classical form of byssinosis is characterized by chest tightness and shortness of breath, which the worker experiences mostly on the first day after the day off. The symptoms can occure at the end of the shift, sometimes they may become worse during the evening. Less complaints may last to the next day. In the following course of the disease the troubles do not abate, but they last on following working days and lessen only on the days off. Unless the worker is not displaced to another working place, the disease continues and the shortness of breath and chest tightness outlast on more working days. In this period the symptoms of bronchitis and emphysema usually associate to byssinosis. Byssinosis cannot be distinguished from the mentioned diseases then. The symptoms are experienced after many years of exposure, rarely in workers exposed for less than 10 years. It is reported that the disease progresses in dependence on the exposure. Nevertheless, it was found out that the symptoms can temporarly decline in spite of continuing exposure[17]. However, also other symptoms than those described by Shilling have been published. Some workers complain about cough and wheezing in brest that are most severe on the first working day, but they do not have chest tightness. Maybe it is the same disease, but expressed in a different way[26].

CHANGES IN LUNG FUNCTIONS

Changes in lung function are objective findings supporting influencing of textile dust on respiratory system in exposed workers. For byssinosis an obstructive disorder in lung function is characteristic, on the one hand acute across the shift, on the other hand chronic. The decline of lung functions in course of the first working day was proved by a great number of studies[13, 31, 32]. It is possible that the severity of symptoms depends on the size of lung functional change combined with the adaptation to the stimulation. Most of the studies focused on FEV1. A few studies have shown changes in the calibre of small airways, which have led to the suggestion that the disease process starts in the peripheral airways[31, 32]. A decline in FEV1 across the shift is presented as an unfavourable factor for long-term decline of lung function in cotton workers[33]. It was demonstrated that workers working with cotton, flax and hamp have decreased lung function[5, 26, 32]. In the advanced period of the disease a combination of obstructive and restrictive changes can be developed.

In addition to changes in lung function in cotton workers also changes in reactivity of airways proceed. Fishwick and Pickering demonstrated that byssinosis was associated with increased bronchial reactivity in 78% workers with byssinosis and in 37% workers who did not have any specific symptoms connected with work. In the control group of workers who were not exposed to textile dust, bronchial reactivity was identified in only 17%[34].

CLASSIFICATION AND DIAGNOSIS

Schilling's original classification of byssinosis was updated later. The WHO experts proposed classification of byssinosis in 1983 which should be used henceforth (Table)[16]. This classification respects respiratory symptoms, acute and chronic disorders in lung ventilation. It also enables the classification of nonspecific respiratory tract irritation by dust which can iniciate byssinosis as well. The value of FEV1 can be declined also in workers not having byssinosis; on the contrary, byssinosis cannot even be excluded in persons, in whom the decline is not expressed[16]. What is the most decisive is the anamnesis. In the differential diagnosis it is important to distinguish other chronic nonspecific diseases of airways, especially chronic bronchitis and asthma bronchiale.

TREATMENT AND PREVENTION

Besides displacing from further exposure, inhaled steroids, antihistaminics and bronchodilatatory drugs are recomended. In case of development of chronic bronchitis with emphysema the treatment is the same as at these diseases, then.

Table 1. WHO grading system for byssinosis

Classification	Symptoms
Grade 0	No symptoms
Byssinosis:	
Grade B1	Chest tightness and/or shortness of breath on most of first days back at work
Grade B2	Chest tighness and/or shortness of breath on the first and other days of the working week
Respiratory tract irritation:	
Grade RTI 1	
Grade RTI 2	Cough associated with dust exposure
Grade RTI 3	Persistent phlegm (i.e. on most days during 3 months of the year) initiated or excerbated by dust exposure Persistent phlegm initiated or made worse by dust exposure either with exacerbacions of chest illness or persisting for 2 years or more
Lung function:	
Acute changes	
No effect	A consistent[a] decline in FEV1 of less than 5% or increase in FEV1 during the work shift
Mild effect	A consistent[a] decline of 5-10% in FEV1 during the work shift
Moderate effect	A consistent[a] decline of 10-20% in FEV1 during the work shift
Severe effect	A decline of 20% or more in FEV1 during the work shift
Chronic changes	
No effect	FEV1[b] 80% of predicted value[c]
Mild to moderate effect	FEV1[b] 60-79% of predicted value[c]
Severe effect	FEV1[b] less than 60% of predicted value[c]

[a] A decline occuring in at least three consecutive tests made after an absence from dust exposure of two days or more.
[b] Predicted values should be based on data obtained from local populations or similar ethnic and social class groups.
[c] By a preshift test after an absence from dust exposure of two days or more.

Technical prevention is very important, which the situation in the USA proves. The technical and technological improvement has practically brought to reduction and finally to disappearance of the disease there. There are different highest admited concentrations of textile dust in different countries. In the USA the National Institute for Occupational Safety and Health (NIOSH) recommends time-weighted averages (TWA) for cotton dust bellow 0.2 mg/m^3. Occupational Safety and Health Administration (OISHA) recommends TWA from 0.1 to 1 mg/m^3 according to performed operation, personal exposure limit (PEL) 1 mg/m^3. In Germany TWA makes 1.5 mg/m^3, in Sweden and the UK it is 0.5 mg/m^3, in Switzerland 0.3 mg/m^3 [35]. In our country hygienic regulations specify PEL for cotton dust of 2 mg/m^3, for flax and hamp 4 mg/m^3 and for jute and sisal 6 mg/m^3 [36]. The proposals that are being prepared suggest a reduction of PEL for flax and hamp to 2 mg/m^3.

In the programme of medical prevention there is necessary to realize preventive inspection including the inspection of lung functions. The persons with chronic diseases of airways, especially with asthma bronchiale, chronic bronchitis and chronic rhinitis should not work at the workplace with hazard of textile dust[37]. At periodical inspections, the lung functions should be examined on the days after non working days, before the shift and after the shift[31, 37]. When byssinosis is detected, the workers should be replaced from the risk milieu to prevent further development of the disease. Byssinosis can be registered as an occupational disease in our country since 1985 . In the year 1989 Jaroš stated two cases in Slovakia[37]. In our country byssinosis has not been notified till now. Only two cases of endangering by this occupational disease were reported. More precise standards for reporting byssinosis as the notifiable occupational disease have not been defined until now.

CONCLUSION

Long term exposure to dust of cotton, flax, jute, hemp, and sisal is connected with classical form of byssinosis. The most specific feature is an unusual periodicity of symptoms. The disease is characterized by a decline of lung function and increased bronchial reactivity. Aetiopathogenesis has not been cleared, but probably it is a secondary inflamation reaction to an immunological or endotoxin induced process. Although byssinosis is a rare disease in the Czech Republic, it will have worldwide significance for many years in the future.

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