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Psoriasis and cigarette smoking

Cigarette smoking is one of the most commonly widespread addictions believed to influence the health of general population. It can unfavourably affect the course of many diseases of various origin, including psoriasis. Psoriasis seems to be of interest because the interaction between genetic and environmental factors plays an essential role in provoking both the beginning and consecutive relapses of this chronic disease (1, 7). Cigarette smoke is a complex mixture of more than 4,000 substances, many of which are toxins, carcinogens, and poisons (7, 10). The most completely studied of these compounds is nicotine, mainly because of its known pharmacological activities. Nicotine is not only a highly addictive drug but it also plays a role in the poor wound healing and increased skin ageing observed in smokers, possibly due to cutaneous vasoconstriction (6, 10). Smoke, an acknowledged etiologic factor in various vascular disorders also plays a role in the evolution of some inflammatory or neoplasmatic bronchopulmonary diseases (including chronic bronchitis, asthma or lung cancer) (6, 7). Among other systemic diseases that are linked with smoking in general population are cardiovascular diseases, Crohn disease, renal neoplasms (7). The harmful effects of long-term smoking on the vascular system are well known (6). Both large and medium blood vessels in many organs can be affected (6). The acute decrease in the retinal blood flow and coronary circulation reported are findings of the utmost biological significance (6). On the other hand, cigarette smoking appears to protect against some inflammatory diseases, such as ulcerative colitis, possibly due to its constricting effect on vessels in the area of inflammation (5). It is related probably to the vasoconstrictive effect of nicotine mediated through the sympathetic system (6). It is believed that the sympathetic activation induced by smoking may be connected with an increased release or a reduced clearance of catecholamines at the neuronal junctions (6). What is interesting, smoking can also inhibit central sympathetic activity, probably through its influence on a baroreceptor stimulation (6).

Among various gaseous compounds being present in the cigarette smoke there are numerous reactive agents (4, 7). These agents including nitrogen oxides, organic peroxides and hydroperoxides, carbon monooxide, free radicals, and polycyclic aromatic hydrocarbons are capable to strongly affect various physiologic functions in humans (3, 4, 7). The oxidative damage can result not only from oxidants present in cigarette smoke but also from the activation of phagocytic cells that generate reactive oxigen species (3, 7). There are conflicting data about the influence of smoking on serum activity of some enzymes and microelements (4). The serum concentration of selenium (an important structural element of many enzymes), among others, has been found to be lower in smokers compared to non-smokers, but this phenomenon can be also related to the differences in dietary intake because smokers have often undesired diet habits (4). Glutathione peroxidase is selenoenzyme, that together with catalase and superoxide dismutase prevents oxidative damage to cells by breaking down hydrogen peroxide and other reactive oxigen species (4). Acording to some literature data, smokers require a 40% higher daily intake of vitamins and some micronutrients than non-smokers to maintain comparable

serum levels (4). Lower selenium level in serum of the smokers can influence defence not only against the oxidative stress but also against the heavy metal cations found in cigarette smoke (4). Selenite can act as a detoxifying agent against some heavy metal cations (including the cadm ions) being found in cigarette smoke (4). Thus, smoking may be a risk factor for insufficient selenium level in the peripheral blood (4). Futhermore, lowered selenium concentration should be regarded as an undesired side-effect of smoking, especialy considering relations reported between low serum selenium and the increased risk of many pathological events (4).

Smoking and nicotine seems to strongly influence inflammatory processes in the skin (7). Reported by many authors functional and morphological alterations of polymorphonuclear leucocytes induced by smoking may also cause an exaggerated release of chemotactic factors in skin (3, 7, 8). Particularly, high level of arachidonic acid metabolites, being the potent inflammatory mediators, have been documented in psoriatic lesions (7). Peroxidation of arachidonic acid by smoke may lead to the formation of isoprostanes, which are found increased dramatically in smokers as well as in the experiments with animal models of oxidant injury (7). What is more, nicotinic cholinergic receptors have been demonstrated on keratinocytes that can result in stimulating calcium influx and acceleration of cell differentiation (7). Constant activation of these receptors may influence keratinocyte adhesion and inflammatory cell migration in the epidermis (7). So, in psoriatic patients smoking can induce the skin inflammation via its influence both upon the recruitment of leukocytes and release of chemotactic factors. What is specially interesting, cigarette smoking and nicotine can affect the inflammation differently. Due to its multidirectional influence on many vital physiological processes, smoking seems to be associated with a decreased occurence of some inflammatory skin disorders and an increased prevalence of several others (10). Monfrecola et al. have studied an influence of smoking on the cutaneous circulation in habitual smokers and non-smokers (6). They demonstrated that not only large vessels but also the cutaneous microcirculation can be injured by greatly reducing the blood flow in non-smoker subjects as well as in smokers (6). Thus, not surprisingly, it has ben found that smoking significantly depresses the inflammatory response in the skin following application of irritants and rubefacients, possibly due to its vasoconstricting effect on cutaneous vessels (5, 10). So logically, as vessel dilatation is an essential feature of inflammation, the suppressing effect of smoking on the development of skin inflammation is only to be expected. This, however, is not observed in psoriasis. Psoriasis is among the skin diseases that are associated with a harmful effect of cigarette smoking, and although the mechanism is not entirely elucidated yet, it may be related to altered immune system function resulting from this chronic addiction (5). Literature data indicate that the clinical severity of psoriasis (expressed by the PASI score) is increased among smokers compared to non-smokers (3). The results of many epidemiological studies clearly indicate that the cigarette smoking is not only an exacerbating factor in psoriasis but sometimes even provoking the first relapse of this disease (3). Of the thousands of toxic components in cigarette smoke, the detrimental influence of nicotine is attributed to the provoking effect on the inflammatory process observed in psoriasis (5,10). Moreover, it is believed that there is an abnormal response to nicotine in psoriatic patients, which could result in cutaneous inflammation (10).

Influence of various environmental factors on the clinical course and severity of psoriasis has been considered and studied from about 20 years. First, in 1985 the strong corelation between smoking habit and palmoplantar psoriasis has been found and further studies have evidenced the influence of smoking on the epidemiology of this disease (3, 7, 11). The data of epidemiological studies support the earlier oppinion that smoking is connected with the prevalence of psoriasis (3, 7). Smoking has been found to be more frequent among psoriasis patients than among healthy population, both prior to the onset of the disease and after the onset (8). From the other side, this disease has proved to be more commonly observed among the cigarette addicts than among non-smokers (1, 3). At least a twofold

risk of psoriasis in smokers is quoted in most studies (11). It has appeared, that the psoriatic patients used to smoke much more cigarettes and more frequently than the healthy people (3, 7). What is more, there is evidence of a dose-dependent association between smoking habit and psoriasis (1, 7, 8), it means that the risk of the disease increases with the duration of this addiction. Futhermore, the risk of psoriasis is established to be higher in ex-smokers and in current smokers than in patients who never smoked (6). The epidemiological studies concerning the influence of environmental factors affecting psoriasis have showed that the risk varies according to sex, with smoking being more strongly associated with psoriasis among women and alcohol consumption among men (3, 7, 9, 11). The strongest relationship has been established between the pustular palmoplantar form of psoriasis in women smokers (7, 9). The results of other studies have suggested that smoking might be an important risk factor for chronic plaque psoriasis as well (11). Anecdotically, apart from the well-known vascular, systemic and cutaneous damage caused by cigarette smoking, F e d e r et al. have reported an additional hazard factor for patients treated with easily flammable antipsoriatic preparations (2). The patient with psoriasis whose cloth was stained with flammable tar solution was bursting into fire immediately after he had carelessly lit a cigarette (2).

It seems that not only the harmful effect of cigarette smoking in psoriasis is of interest but also the increased prevalence of smokers among the psoriatic patients as well (3, 8). The epidemiological studies support the clinical observations that smoking is unfavourably connected with psoriasis and the psoriatic patients smoke much more cigarettes than healthy people (3, 8). Higher prevalence of smokers among the psoriatics cannot be explained basing only upon the biological/somatic factors. Psoriasis, a chronic skin condition, although it is not life-threatening, can be regarded as uncomfortable and disfiguring. It is worth to stress that skin manifestation of some clinical forms (pustular psoriasis, psoriatic erythrodermia) can be severe, accompanied by intensive pain (arthropathic psoriasis) and considerable limitations of physical efficiency finally resulting in disability. Consciousness of suffering from chronic, incurable disease with visible skin lesions usually leads to emotional problems (3, 8). Lowered quality of life caused by numerous difficulties and troubles in private, social and professional life can easily encourage falling into some undesired addictions, including cigarette smoking, alcohol abuse, dietary habits (3, 8). Some authors indicate that these habits tend to be associated and themselves may increase the risk of developing psoriasis, or worsen pre-existing desease (3, 8). Up till now, however, there are not sufficiently documented data available to conclude whether stopping of the cigarette smoking could make any difference to the course of established psoriasis. Recognition of the environmental factors affecting the clinical course of psoriasis suggests some possibilities of the disease alleviating mostly through influence on the style of life. Influence on the known environmental risk factors such as smoking and emotional stress by promoting the healthy style of life is possible, at least in some patients (3, 9, 11). It is suggested that that some of the psoriatic patients might be more willing to change their habits because of present and socially debilitating skin disorder than because of long-term risks of cancer or ischemic heart disease (11). Prevention of smoking habit among patients with psoriasis could alleviate their disease and improve their quality of life (9).

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SUMMARY

Psoriasis is a chronic skin disease, whose prevalence in population is connected with interaction between genetic and environmental factors. Clinical observations indicate that cigarette smoking is an important environmental factor exacerbating the course of the disease and provoking its consecutive relapses. The harmful influence of smoking is connected with inducing of inflammatory mediators taking part in pathogenic phenomena in the skin of psoriatic patients. Psoriasis is observed more frequently in smokers than in general population. Vast spread of the smoking habit among the psoriatic patients can be related to the lowered quality of life due to the emotional problems and difficulties in the family and social life caused by the chronic disease.

Łuszczyca i palenie papierosów

Łuszczyca jest przewlekłą chorobą skóry, której występowanie w populacji związane jest z wzajemnym oddziaływaniem czynników genetycznych i środowiskowych. Zarówno obserwacje kliniczne, jak i wyniki badań epidemiologicznych wskazują na to, że palenie papierosów jest czynnikiem zaostrzającym przebieg choroby i prowokującym jej kolejne wysiewy. Jest to związane z pobudzaniem przez składniki dymu tytoniowego mediatorów zapalnych uczestniczących w zjawiskach patogenetycznych, które zachodzą w skórze chorych na łuszczycę. Łuszczyca jest stwierdzana częściej wśród osób palących niż w ogólnej populacji. Duże rozpowszechnienie nałogu palenia wśród chorych na łuszczycę jest prawdopodobnie związane z obniżoną jakością życia z powodu przewlekłej choroby, powodującej trudności w życiu rodzinnym i społecznym.