Review article

Does smoking affect your skin?

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Abstract

Smoking has a negative influence on human beings. Carcinogens detected in smoke can increase the risk of developing chronic disorders, cancer and premature death. Nicotine can also affect dermatological diseases such as psoriasis, hidradenitis suppurativa, chronic dermatoses, alopecia, lupus erythematosus, polymorphous light eruption, skin cancer and tobacco-associated oral lesions. Advanced education at a doctor's surgery in various medical occupations can change the bad habits and protect people from the consequences.

Key words: smoking, nicotine, tobacco, skin.

Introduction

The World Health Organization (WHO) reports that nearly half of addicted people are killed by smoking. More than eight million people are killed due to smoking each year. According to WHO data, in 2025 there will be still around 1.1 billion smokers worldwide. A relatively easy addiction to smoking is due to a chemical characteristic of the alkaloid contained in tobacco called nicotine that is easily absorbed into the respiratory tract, skin and intestinal mucosa. It is metabolised through the liver to active metabolites [1, 2]. Nicotine can react with different subtypes of nicotinic acetylcholine receptors which are located not only in the nervous system, adrenal medulla, but also in skin keratinocytes and inflammatory cells (monocytes and dendritic cells). Nicotine passes into the nicotinic acetylcholine receptors and transitionally activates ganglionic transmission which is followed by a lasting depression (biphasic effect), initially causes release of catecholamines from the adrenal medulla and postganglionic sympathetic neurons. Nicotine can have an influence on the skin, increase keratinocyte adhesion and upward migration in the epidermis. After all, it provokes the immunomodulatory system [3]. We observe the dysfunction of some structures due to extended exposure to benzo(a)pyrene such as decreased mass of lymphoid tissue and incorrect work of the neutrophils [4]. After meta-analysis resolution, we differentiate the relationship between gender and the result of smoking. Moreover, smoking women have increased morbidity and mortality compared to smoking men [5]. Nicotine is a very insidious factor, which can pass into the human body through smoke inhalation, ingestion, intranasal spray, transdermal patch, topical cream, or enema. Furthermore, nicotine can also be located naturally in small quantities in some vegetables such as tomatoes, potatoes, and aubergines. This chemical bound is absorbed through the oral cavity, lung, bladder, gastrointestinal tract and skin. There was a detailed study of gene expression showing that nicotine contains 14 miscellaneous genes involved in the xenobiotic metabolism, oxidative stress and stress response [6]. Activation of a few pathways releasing cytokines stimulate T lymphocytes leading to the initiation of a chronic inflammatory process [7]. Apart from genetic factors, exposure to nicotine in untimely infancy is related to diseases such as psoriasis [8]. Nicotine is not a carcinogen, but it is a highly addictive chemical compound. Despite this, it is a medically approved medicine as a replacement therapy, used to help with quitting smoking or stopping chewing tobacco [9].

Eye detected characteristics of smokers

Smokers can be easily identified by their bad habit. The characteristics are yellow discolouration of light-coloured moustaches, problems with normal nail growth and discoloured fingernails – Harlequin or quitter's nails. About one third of tobacco users have oral pigmentation that is visible to the naked eye [10]. The special case is that gingival pigmentation can be also visible in children of parents with sublingual nicotine use or exposed

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to passive smoking. Parts of the oral cavity such as the tongue or the palate might also be affected. Most common problems are black hairy tongue, hyperplasia of the papillae with black pigmentation on the dorsal surface of the tongue, leukokeratosis, nicotine glossy and inflammation of salivary glands [10].

Smoking effects

Premature ageing

Smoking is an undeniable risk factor for wrinkling and premature skin ageing. This negative influence on the skin was discovered more than 150 years ago. Pathogenesis of smoke-induced ageing is multifactorial. Smoking can affect reticular dermis by increasing the number of elastic fibres resulting in changes similar to solar elastosis [11]. Smoking can lead to the activation of metalloproteinase which can cause degradation of collagen, elastic fibres and proteoglycans [12]. Smoking can also reduce the level of active form of the transforming growth factor β1 (TGF-β1) and down-regulate their receptors causing dysfunctions of tissue remodelling and repairing. An *in vivo* study showed that mice treated with intracutaneous or topical tobacco smoke can have a decreased level of type I collagen [13, 14]. "Smoker's face" presents some characteristic features such as prominent wrinkles, prominence of underlying bony contours and grey-coloured, atrophic complexion of the skin [10]. Ageing is correlated with pack-years of smoking and number of cigarettes smoked per day [15]. Studies confirmed the association especially with more than 40 packs smoked per year [16, 17].

Wound healing

Smoking is a risk factor for delayed wound healing and other complications. It can affect cutaneous blood flow causing microcirculation and tissue oxygenation disorders [10]. Moreover, smoking can decrease activity of fibroblasts, reducing their migration into the wound [18–20]. Smoking can lead to wound infections [21–23], dehiscence of the wound [24, 25], graft and flap necrosis [26, 27]. Surgeons suggest patients to quit smoking before operations. Some surgeons hesitate to perform operations on smokers if they refuse to quit [28].

Psoriasis

Smoking is a factor that can aggravate the clinical symptoms of psoriasis [29]. Smokers compared to non-smokers have an increased possibility to develop psoriasis [30]. Children who are in very early years exposed to the cigarette smoke are in a group with a higher risk of psoriasis development [8]. There are a few pathways to induce the psoriasis such as genetic, inflammatory or oxidative mechanisms [31]. Chronic inflammation which is a trigger to psoriasis is incited by released cytokines, which activates T lymphocytes [32]. Both smoking and obesity can cause an elevated serum concentration of tumour necrosis factor α (TNF-α) [33]. A protracted inflammatory process increases the risk of cardiovascular incidents [34]. A number of pack years correlates to the level of TNF-α [35]. Also, another inflammatory indicator such as C-reactive protein is increased at a higher age and in a group of smokers [36]. According to the research, smoking can decrease the response of medical treatment. For example, better response to treatment with etanercept, fumaric acid, retinoids (after 3, 6, 12 months of treatment), cyclosporine A (after 6, 12 months of treatment) is noticed in groups of non-smokers compared to smokers [37]. A prospective pharmacovigilance study (BADBIR) revealed that even one episode of smoking can be connected with reducing treatment response in a group of patients on biological therapy (n = 3,079 at 6 months and n = 3,110 at 12 months).

Chronic dermatoses and smoking

Smoking can be a risk factor for developing allergic contact dermatitis. There are a lot of different allergens in the filter, paper and tobacco. The most common are menthol, formaldehyde, cocoa, liquorice, and colophony [38]. Smoking can be a factor inducing a positive patch reaction to nickel [39]. Smoking can increase the serum total IgE level [40]. Cigarette use is also a well-known risk factor for hand eczema [41].

Hidradenitis suppurativa

Many studies revealed a correlation between smoking and hidradenitis suppurativa. Almost 98% of patients with this disease are addicted to nicotine [42–45]. Moreover, smokers have been proved to have more severe symptoms [46]. Nicotine is a factor that can cause increased activity of sweat glands. Furthermore, it can affect chemotaxis of granulocytes and macrophages [43, 44]. *In vitro* studies showed that nicotine can promote hyperplasia of epidermis and follicular plugging [47, 48].

Acne

Correlation between acne and smoking cigarettes is subject of many studies. However, results of those studies are conflicting. There are some studies that showed a positive effect [49, 50] and those which showed a negative impact of smoking [51].

Alopecia

Smoking is a well-known risk factor for androgenic alopecia among men. Studies showed that smoking over 20 cigarettes per day can be associated with developing moderate to severe androgenic alopecia [52]. Nicotine can cause microcirculation dysfunctions among hair papilla. Cigarette smoke is a source of substances which can cause DNA mutation. Studies revealed that smok-

ing can affect the balance between proteases and antiproteases. Moreover, it can increase pro-inflammatory cytokine concentration in anagen hair which can lead to follicular microinflammation and fibrosis [53]. Nicotine can decrease the estrogenic level due to stimulation of estradiol hydroxylation and inhibition of aromatase [54].

Lupus erythematosus

It has been documented that smoking and lupus erythematosus are connected. Some studies proved that smokers have an increased risk of developing SLE [55, 56]. On the other hand, some of the observational studies demonstrated no association [57, 58]. Smoking can also increase activity of the disease. In addition, it can be a risk factor for developing cutaneous lupus erythematosus including subacute cutaneous lupus erythematous and discoid lupus erythematosus [59–61]. Smokers with discoid lupus erythematosus (DLE) can have a more severe onset of the disease. Nicotine can also interfere with antimalarial therapy by decreasing treatment efficiency [62–64].

Polymorphous light eruption

A retrospective case-control study including 74 patients with PMLE and 102 controls was conducted. It has shown that people who smoke more than 15 cigarettes daily have an increased risk of developing polymorphous light eruptions [65].

Skin cancer

The most detailed list of harmful and potentially harmful constituents in tobacco smoke includes about 80 carcinogens (FDA HPHC List) [66]. Despite the fact that carcinogens are present in tobacco smoke, it is controversial whether smoking is a risk factor for the development of skin cancer. Some studies showed that smokers usually spend most of the time indoors because they are less active than non-smokers. A positive aspect of this model of life is reduced sun exposure [67, 68].

Squamous cell carcinoma

Some studies have proven that smoking is a risk factor for developing squamous cell carcinoma (SCC). On the other hand, some researchers believe that there is no correlation between nicotine and SCC [69, 70]. It has been documented that keratoacanthoma is connected to smoking [71]. A case-control study including 78 patients with keratoacanthoma and 199 controls was conducted. It showed that 69.2% of patients with keratoacanthoma are smokers compared with 21.6% of controls [71].

Squamous cell carcinoma in the oral cavity and smoking

Oral SCC is frequently detected among people who are habitual smokers. The localisation is in the oral cavity, pharynx and larynx. Five-year survival rate is lower than 50% [72]. Tobacco is one of the selected factors which increases the risk of developing that disease [73]. Initially it develops as a small lesion which does not look alarming. After that it can transform into a white or red spot. Then the medical intervention is very desirable. Waiting might have negative consequences such as a higher rate of morbidity and mortality [74].

Basal cell carcinoma

It has been documented that aetiology of basal cell carcinoma (BCC) is complex. Excessive exposure to UV radiation is a well-known risk factor for BCC development [75]. On the other hand, results of studies regarding BCC and smoking cigarettes are conflicting. There is a prevalence of smokers among women with BCC [76, 77]. Moreover, a retrospective medical record review of 220 patients with Mohs surgery shows that smoking is connected with developing of BCC larger than 1.0 cm in diameter [78]. However some studies showed no association between smoking and developing of BCC [79, 80].

Melanoma

Smoking is not a risk factor for developing cutaneous malignant melanoma. There are some studies on the protective role of smoking cigarettes [81–84]. It may be connected with the immunosuppressive effect of nicotine which can protect melanocytes from ultraviolet-induced inflammation.

Anogenital cancer

Smoking is a risk factor for developing anogenital skin cancers [85]. Moreover, the number of cigarettes smoked is directly connected with the estimated risk. A group of 903 patients with anogenital cancers was investigated in a population-based study. The study has demonstrated that 40% of people with vaginal and cervical cancer and 60% of those with vulvar and anal cancers were smokers compared to 25% of controls (OR = 1.3–14.6) [86].

Tobacco alternatives

Study results indicate that effectiveness of different forms of nicotine addiction is low. The tobacco industry started to display special, very graphic pictures and warnings on the packs of cigarettes to show the negative consequences of smoking [87]. But the number of smokers still remains high. Consequently, heavy smoking patient management should also include tobacco harm reduction strategy. Alternatives like electronic cigarettes (e-cigarettes) and tobacco heating devices (heat-not-burn products – HnB) induce the perception of smoking, but without tobacco burning. Eliminating the tobacco burning process substantially reduced the number and levels of toxins in aerosol in comparison with combustible cigarette smoke. E-cigarettes consist of an atomizer,

battery and special liquids (cartridge) – full of vegetable glycerine (VG) and/or propylene glycol (PG), nicotine, and flavours. There is a lack of standardization among a few thousand models of these products; the benefits and the health risks of e-cigarettes are debatable [88, 89]. More importantly, some of them can come to the market from unknown sources. On the other hand, a raising number of studies show that heat-not-burn devices offer smokers a substantial reduction of harmful and potentially harmful chemical compounds in comparison to smoking continuation [90, 91]. In 2019, the first heat-not-burn device was authorised by the FDA in the US to be sold to smokers in the US market as an appropriate alternative for the protection of the public health. Some clinical experts recommend HnB devices as a harm reduction strategy after pharmacotherapy failure for heavy smokers [92].

Conclusions

No doubt that even short episodes of smoking negatively affect human health. A lot of chemical substances are produced during the process of smoking, which also pollutes our environment. People close to smokers also suffer from the effects of tobacco smoke. Smoking can cause the aggravation of many chronic diseases including dermatological ones. Moreover, usage of some medicines during smoking can decrease or increase their concentration in the blood leading to activity disorders. It is important for any doctor to promote awareness of the consequences of smoking. Despite the availability of many methods of counteracting tobacco addiction, their effectiveness is still insufficient. An alternative to smoking cigarettes after pharmacotherapy failure could be tobacco products based on heating tobacco ('heat-not-burn'), the use of which may be associated with a lower cardiovascular risk.

Conflict of interest

The authors declare no conflict of interest.

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