Short Commentary

Treatment of Asthma in Tobacco Smokers

Puri S and Mohanty B

Department of Biotechnology, Faculty of Science and Humanities, SRM University, Chennai, Tamil Nadu, India
Corresponding author: Puri S, Department of Biotechnology, Faculty of Science and Humanities, SRM University, Chennai, Tamil Nadu, India, Tel: +919882668649; Email: sarkinapuri07@gmail.com

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Short Commentary

Smoking cigarette particularly affects persons with asthma. Asthma is the most common respiratory disease, occurs due to allergic inflammations in the bronchial tubes of lungs. Since prevalence of smoking is increasing, constant inhalation of tobacco stimulates the mucous glands in the bronchial tubes which gives access to the mucus and leads to symptoms like mucus production, dyspnea (shortness of breath), wheezing, cough and phlegm [1,2].

In asthma, contraction of muscles and swelling around the bronchial tubes creates breathing problems. Most cigarette smokers with the disease develop an irreversible narrowing in the bronchial passage causing them to face permanent difficulty in breathing, smoke-induced lung disease, emphysma and chronic bronchitis. On average, adults who experience asthmatic problems after age 50 mostly have a past history of smoking cigarettes. In most developed countries nearly 25% adults with respiratory illness are cigarette smokers. Active cigarette smokers with asthma might have severe symptoms, major reduction in lung function, and increase in corticosteroids sensitivity.

Smoking cigarettes might reconstruct inflammation associated with asthma, though there is little data about airway obstruction in smokers but compared to non-smokers a combination of both heightened and suppressed inflammatory responses are found in smokers.

Chronic bronchitis (CB) and chronic obstructive pulmonary disease (COPD) are the common type of chronic airway diseases. While asthma the airway disorder mainly starts in childhood, smoking is not directly responsible for its cause. Recent research studies have found enough evidences, which proves there is an adverse effect of smoking on symptoms of asthma [3-6]. Both active and passive (exposure to environmental tobacco smoke) tobacco smoking have shown to increase exacerbation of asthma.

Onset of asthma in smokers

Some studies have shown that active smoking can be involved in the development of asthma [7-12]. The development of asthma-like symptoms in asymptomatic teenagers (over a 6 years period) has been associated with tobacco smoking (active), along with few cases of bronchial hyper responsiveness to methacholine and atopy [8]. In a study involving population size of 15813 adults, examined under respiratory questionnaire resulted in presence of maximum number of tobacco smokers (95%) as compared to non- smokers [10]. Strong association of smoking with the development of asthma was found to be common amongst individuals [7]. This study also proves that smoking can be a major risk factor for onset of asthma in older adults [9]. The arginine-14 genotype and β2-adrenergic receptor gene polymorphism, shows an inclination in the risk of asthma among active smokers compared with non- smokers [13]. In another experiment the risk factors associated with asthma among cigarette smokers have indicated that asthma which develops before starting smoking is associated with atopy and asthma which develops after initiating smoking is connected with lowering of forced expiratory volume [14].

Asthma: Effects and control

Cigarette smokers face high rate of morbidity and mortality due to development of asthma as compared with never-smokers. Asthma developed in smokers had more severity of symptoms as compared to non-smokers and need instant medictaions [15-18]. Major broncho constriction is developed due to smoking a cigarette, although smoking does not act as an acute allergen in all patients [19]. Inhalation of cigarette is directly related to the immediate response of forced expiratory volume (FEV), which indicates that asthmatic smokers with poor lung function can be vulnerable to the acute effect of tobacco smoke. Smokers with atopic asthma are likely to be less responsive towards inhaled adenosine compared to non-smokers which shows the differences in airway inflammation [20,21].

Treatment of asthma in smokers

For cessation of smoking, different and alternative treatments of inhaled corticosteroids are required for the patients who have persistent symptoms. Non-corticosteroid therapy in the treatment of asthma in smokers does not have much evidences, but there are many recently developed drugs, along with several under developed drugs which may produce efficient results in patients (active smokers) suffering from asthma.

β2 receptor agonist along with corticosteroid (inhaled) have shown efficient effects in the treatment of asthma, also this combination have shown effective results in the treatment of COPD [22,23]. The above mentioned combination of drugs suppresses the inflammation associated with COPD and asthma [24,25]. The possibly the combination of these drugs may show efficient results in the treatment of asthma in smokers. Activation of HDAC due to theophylline (low dose) suppresses the inflammation (theophylline is recruited by corticosteroids [26]. In vitro experiments have shown that theophylline can reverse the restraining effect of smoking on HDAC and hence can regenerate sensitivity of corticosteroid in asthmatic smokers [27]. Cilomilast and roflumilast are phosphodiesterase-4 inhibitors which are effective drugs that can treat COPD (Table 1). Inhibitory activity of the phosphodiesterase-4 inhibitors can be the effective compounds that can prove to be a potent drug for treatment of asthma and smokers [28].
Table 1: Available drugs for the treatment of smoking.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Dosage</th>
<th>Common adverse effects (CSE)</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nicotine patch</td>
<td>If&lt;10 cig/day-21 mg</td>
<td>Skin irritation and insomnia</td>
<td>Easy to use and steady nicotine level</td>
<td>Slow release and not to be used during cravings</td>
</tr>
<tr>
<td></td>
<td>If&lt;10 cig/day-14 mg</td>
<td></td>
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<tr>
<td>Varenicline</td>
<td>3 days-0.5 mg/day</td>
<td>Nausea, insomnia and abnormal dreams</td>
<td>Dual action-Relieves withdrawal and blocks reward from smoking</td>
<td>Psychiatric effects</td>
</tr>
<tr>
<td></td>
<td>4 days-1 mg/day and then 2 mg/day</td>
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<tr>
<td>Nicotine lozenge</td>
<td>First Cigarette&gt;30 minutes after waking-2 mg/h</td>
<td>Hiccups and heartburn</td>
<td>User controls nicotine dose</td>
<td>No food or drink 30 minutes before use</td>
</tr>
<tr>
<td></td>
<td>First Cigarette&lt;30 minutes after waking-4 mg/h</td>
<td></td>
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<tr>
<td>Nicotine nasal spray</td>
<td>1-2 hours: Once/nostril</td>
<td>Nasal irritation, cough, sneezing, teary eyes</td>
<td>Most rapid nicotine delivery</td>
<td>Local irritation</td>
</tr>
<tr>
<td>Nicotine inhaler</td>
<td>Inhal as needed: 6–16 cartridges/day (10 mg/cartridges)</td>
<td>Mouth, throat irritation</td>
<td>User controls nicotine dose; cigarettes oral substitute</td>
<td>Frequent puffing required</td>
</tr>
<tr>
<td>Bupropion SR</td>
<td>3 days-150 mg/day and then 300 mg/day</td>
<td>Insomnia and dry mouth</td>
<td>Oral agent and reduce weight gain</td>
<td>Seizures risk and psychiatric effects</td>
</tr>
<tr>
<td>Nicotine gum</td>
<td>If&lt;25 cig/day-2 mg/h</td>
<td>Mouth irritation and heartburn</td>
<td>Cigarette substitute</td>
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References


