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Smoking and Coronavirus: Possible Evidence from Our Work

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ABSTRACT

Smoking and coronavirus is a controversial issue because different studies give different opinions. The objective of this study was to give a possible evidence from our previous research. We demonstrated from our previous studies that smoking has negative impacts by the induction of liver toxicity and tissue dysfunction such as trachea. Accordingly, it is obvious that smoking should be quitted particularly in areas of pandemic, and wherever passive smoking exists.

KEYWORDS: Smoking; Coronavirus; Liver; Trachea; Passive smoking

INTRODUCTION

(Vardavas and Nikitara, 2020) [5] summarized the literature of smoking and coronavirus. Because the pandemic is still progressing, there is a scarcity of information about the clinical characteristics of patients as well as their prognostic factors (Khot and Nadkar, 2020) [8]. To date, smoking has been thought to be possibly associated with poor illness prognosis, owing to significant evidence highlighting the deleterious impact of tobacco use on lung health and its causative relationship with a variety of respiratory diseases (Tonnesen et al., 2019) [11]. Smoking also harms the immune system and its ability to respond to infections, making smokers more susceptible to infections (Zhou et al., 2016). Previous research has found that smokers are twice as likely as non-smokers to get influenza and experience more severe symptoms, as well as having higher mortality rates during the previous MERSCoV outbreak (Park et al., 2018) [10]. Given the data gap, we undertook a systematic analysis of COVID-19 studies that included information on patients' smoking status to assess the relationship between smoking and COVID-19 outcomes such as illness severity, the need for mechanical ventila-

tion, ICU stay, and death. Zhou and colleagues (2020) investigated 191 people for their epidemiological characteristics. Individuals infected with COVID-19, without, however, reporting in more detail the mortality risk factors and the clinical outcomes of the disease. Among the 191 patients, there were 54 deaths, while 137 survived. Among those that died, 9 percent were current smokers compared to 4 percent among those that survived, with no statistically significant difference between the smoking rates of survivors and non-survivors (p=0.21) regarding mortality from COVID-19. Similarly, Zhang et al (2020) described the clinical features of 140 COVID-19 patients. The findings revealed that 3.4 percent of severe patients (n=58) were current smokers and 6.9 percent were previous smokers, compared to 0 percent of non-severe patients (n=82) who were current smokers and 3.7 percent of former smokers, resulting in an OR of 2.23 (95 percent CI: 0.65–7.63; p=0.2). In a study of 41 patients, Huang et al (2020) looked at the epidemiological characteristics of COVID-19. None of those who required to be admitted to an ICU (n=13) in this study were current smokers. Three patients in the non-ICU group, on the other hand, were current smokers, with no statistically significant difference between the two groups of patients (p=0.31), despite the study's small sample size. Guan et al (2020) presented the biggest study population of 1099 COVID-19 patients from diverse locations of mainland China. For the 1099 patients, descriptive results on their smoking status were reported, with 173 having severe symptoms and 926 having non-severe symptoms. Patients with severe symptoms were 16.9% current smokers and 5.2 percent past smokers, compared to 11.8 percent current smokers and 1.3 percent former smokers among patients with non-severe symptoms. Furthermore, 25.5 percent of patients who required mechanical ventilation, admission to an ICU, or died were current smokers, while 7.6 percent were past smokers. Only 11.8 percent of current smokers and 1.6 percent of past smokers were found in the group of patients who did not have these negative consequences. In that study, no statistical analysis was done to see if there was a link between the severity of the disease result and smoking status. Finally, Liu et al (2020) discovered that among their 78 COVID-19 patients, the bad outcome group had a substantially greater proportion of patients with a smoking history (27.3%) than the group that improved or stabilized (3.0%), with this difference statistically significant at the p=0.018 level. Smoking history was found to be a risk factor for illness development in their multivariate logistic regression study (OR=14.28; 95 percent CI: 1.58–25.00; p=0.018).

POSSIBLE EVIDENCE FROM OUR SMOKING STUDIES

We have conducted several studies on smoking utilizing human clinical studies and basic studies using animal smoking models.

HUMAN STUDIES

Shotar et al (2015) [4] conducted a study to to show how carbon monoxide and its equivalent carboxyhaemoglobin in the blood changed kinetically during smoking simulations. This is a descriptive cross-sectional experimental study. A carbon monoxide detector (piCO+TM Smokerlyzer) was used in this investigation, which was conducted in Irbid cafes. Personal and smoking-related data were collected using a previously designed questionnaire. In this study, 437 people were separated into four groups: passive smokers, cigarette smokers, waterpipe smokers, and waterpipe and cigarette smokers. Age, sex, employment, nationality, smoking type, and smoking inhalation frequency were all found to be substantially linked with carbon monoxide and carboxyhaemoglobin readings ($p<0.05$). The findings also revealed that after one hour of smoking, all smokers had dangerously high levels of carbon monoxide and carboxyhaemoglobin in their blood. When compared to smokers, passive smokers exhibited significant carbon monoxide effects in all circumstances. The findings revealed that smoking causes breath carbon monoxide and blood carboxyhaemoglobin levels to rise to harmful levels, and that cigarettes cause higher levels of carbon monoxide and blood carboxyhaemoglobin than from waterpipe smoking.

Alkhatib et al (2014) [3] conducted a study to look at carbon monoxide and carboxyhemoglobin levels in a group of Jordanian students in cafes in Irbid City, Jordan. Carbon monoxide and carboxyhemoglobin levels were tested before and after 1 hour of smoking. There were 236 individuals in the study, with 102 passive smokers and 134 cigarette smokers. Carbon monoxide and carboxyhaemoglobin were detected in breathing using a device called the carbon monoxide monitor (piCO+TM Smokerlyzer), which was designed to measure both carbon monoxide and carboxyhaemoglobin. Passive smokers had CO levels of 2.8 ppm and carboxyhemoglobin of 2.9 percent before starting to smoke, while cigarette smokers had CO levels of 35.4 ppm and carboxyhemoglobin of 35.7 percent. Passive smokers had higher levels of carbon monoxide (10.1 ppm) and carboxyhemoglobin (9.6%) after 1 hour of smoking. Carbon monoxide levels were 38.2 ppm and carboxyhemoglobin levels were 39 percent in cigarette smokers after 1 hour of smoking. Taken together, the findings revealed that carbon monoxide and blood carboxyhaemoglobin levels caused by smoking are dangerously high. Passive smokers are more harmed by ambient smoking exposure than cigarette smokers and are exposed to real smoking dangers.

ANIMAL STUDIES

The major goals of this study were to look at the effects of waterpipe smoking on liver injury as measured by the liver function tests ALT, AST, and LDH on one hand, and to investigate the usage of Ammi visnaga to treat the liver injury on the other. The procedure entailed creating an animal smoking model in which rats were exposed to waterpipe smoking using a digital smoking

machine on a regular basis. The animals were divided into three groups at random: control (N=8), waterpipe smoking (N=8), and waterpipe smoking plus Ammi visnaga (N=8). Liver damage was determined using the ALT, AST, and LDH liver function assays. The researchers discovered that waterpipe smoking increased the levels of ALT (from 513.2 U/L in the control group to 761.8 U/L in the smoking group, p=0.000), AST (from 2227.5 U/L in the control group to 3149.3 U/L in the smoking group, p=0.000), and LDH (from 1861.2 U/L in the control group to 49817.4 U/L in the smoking group, p=0.000). The usage of Ammi visnaga, on the other hand, significantly decreased the levels of AST (184.68 U/L, p=0.03), ALT (40.38 U/L, p=0.04), and LDH (247.42 U/L, p=0.00) (Alkhatib and Ababneh, 2021) [1].

In another study, Alkhatib and Ababneh (2021)[2] investigated the histological changes in the rat's trachea caused by cigarette and waterpipe smoking, as well as to look into the benefits of Ammi visnaga in reversing the negative consequences of smoking. Male rats were randomly assigned to separate groups (N=8) to develop an animal smoking model. The participants were divided into four groups: control, cigarette smoking, waterpipe smoking, cigarette smoking treated with Ammi visnaga, and waterpipe smoking treated with Ammi visnaga. For one month, a no-smoking strategy was followed. Animals were sacrificed at the end of the experiment, the trachea was separated and fixed in formalin (10%), and tissue slices were produced using Hematoxylin and Eosin stain. The findings revealed that smoking a cigarette or a waterpipe had unfavorable effects on tracheal tissue in terms of cilia disruption or amalgamation. There was a lot of lymphocyte infiltration. The application of Ammi visnaga resulted in a reduction in lymphocyte infiltration, partial healing of epithelial cells, and reversal of cilia damage. Taken together, this work may be the first to demonstrate the value of Ammi visnaga in reducing the histological alterations generated by smoking models in smoking rats' tracheal tissues.

From these studies, it is plausible to say that smoking generates a toxic status that is reflected on other body organs such as respiratory system tissues including trachea and lung as well as liver. Furthermore, our studies highlighted the importance of passive smoking in which exposing to it transfers persons to severe levels of carbon monoxide and carboxyhemoglobin. If a person who is smoking is exposed to coronavirus, he is likely to be affected by the disease because pathologic conditions favor the occurrence of the infection. These findings were further confirmed from animal studies in which smoking significantly increased the levels of liver function tests, particularly the LDH level. On histologic level, the trachea, the tube or canal in which pathogens are entered, is expected to filtrate the pathogens by their cilia. Trachea under the effect of smoking loses their cilia, or they are amalgamated which reduce their efficacy in impeding the entrance of pathogens including coronavirus. Smoking was also found to make trachea inflamed as indicated by congestion and infiltration of polymorph nuclear leukocytes.

CONCLUSIONS

This study discussed a controversial issue in which smoking may increase or not the pathogenicity of coronavirus. Although studies did not give clear picture on this issue, we demonstrated from our previous studies that smoking has negative impacts by the induction of liver toxicity and tissue dysfunction such as trachea.

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