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Mini Review

Smoking and Cancer

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Abstract

Tobacco and its products are severe threats to the personal and environmental health. Smoking cause 5,4 million death annually worldwide. Some reports from USA reveal that 35% of smoking related deaths are due to cancer and among these 80% are lung cancer. Smoking and lung cancer link is known since 1912. The carcinogenic material in cigarette smoke (especially polycyclic aromatic hydrocarbons and nitrosamine ketons) causes genetic mutation in cellular DNA of human. Inhibition of tumour suppressor genes and activation of oncogenes, causes lung cancer development in 20% of smokers. As the link of smoking and lung cancer has been clearly proved, annual screening with low dose thoracal computed tomography is advised in selected high risk groups by Lung Cancer Diagnosis and Treatment Guidelines. Early detection of lung cancer is very important because first stage nonsmall cell lung cancer has a five year survival of 77%, while stage four lung cancer has 2 - 22%. Smoking cessation in every stage of lung cancer, is as much important as early detection and treatment of lung cancer. Smoking cessation in lung cancer patients whether at the time of diagnosis or treatment increases the survival. In conclusion, every doctor should explain to their smoker patients that they have an increased risk of lung cancer. If the doctor is dealing with a cancer patient he/she should insistently advise smoking cessation.

Keywords: *Smoking; Lung Cancer; Smoking Cessation*

Tobacco and its products are highly consumed worldwide with severe threats to the personal and environmental health. According to 2015 reports, 942 million male and 175 million female over 15 years of age, are smoker worldwide [1]. Also smoking rates among young population (below 15) is increasing annualy. Reports from England reveal that more than 200 000 children start smoking every year [2]. Consumption of cigarette and alternative forms of tobacco also increases by time and smokeless tobacco products (snuff, snus…) are largely consumed in South Asia and North European countries [2,3]. Another form of tobacco, water pipe (hookah, narghile) which is traditionally used in Middle Eastern countries for decades (more than 10% of the population) is being used increasingly among American youth [2,3].

Recently electronic-cigarette (e-cigarette) and warmed forms of tobacco is started to be used widely. E-cigarette consumption among Europeans in 2012 is 20,3% and among these only 3.7% is used for smoking cessation. On the other hand there is a consumption of e-cigarette among the youth who never think of smoking [2].

There are more than 5300 chemicals in cigarette smoke and more than 70 carcinogenic substance has been detected among these (polycyclic aromatic hydrocarbons, N Nitrosamines, aromatic amines, aldehyts, fenoles, volatyl hydrocarbons,other organic and inorganic compounds…) [3]. These carcinogenic compounds are present in higher concentrations in cigar and water pipe than cigarette while to some extent in lower concentrations in smokeless tobacco products [3]. There are important proof about the presence of chemicals that cause DNA damage and mutation in e-cigarettes [4].

Smoking cause 5,4 million death annually worldwide. 7,1 million smoking related death (5.1 million male and 2 million female) has been reported only in 2016 and 6,3 million of this is related to active smoking while 884,000 is related to passive smoking [1]. USA has reported that every one out of 5 death is related to smoking [5]. According to 2010 data 25% of male and more than 15% of female death is related to tobacco consumption among the World population. In USA effective tobacco control policy and measures prevented 8 million early deaths from the year 1964 to 2014 [2]. Reports in 1995-99 from USA reveal that 35% of smoking related deaths are due to cancer and among these 80% are lung cancer [6]. Among the preventable cancers like head and neck cancers 80-90% are related to tobacco consumption. Risk of lung cancer is 20 times more among smokers who smoke 10 or less cigarettes per day and it is 5 times more among people who smoke less than 4 cigarette per day [2]. There are definite proof that secondhand smoke causes lung cancer in adults and there are strong proof that it causes upper airway cancer [1,2,7]. Among children causative relationship of secondhand smoke was shown mostly in hematologic malignancies (leukemia, lymphoma), and some central nervous system tumours and hepatoblastoma [7].

It has been known since 1985 that cigarette smoking causes sino-nazal, oropharyngeal, nasopharengeal, hypopharengeal, larengeal, eusophageal, bladder and renal cancers [2,3,8]. The relation of cervical cancers with smoking has also long been known and it is still accepted even though the causative effect of Human papilloma virüs has been detected in 1995 [8]. Smoking doubles the risk of stomach and colon cancers. Hepatitis B virüs is the main causative factor in Hepatic cancer but there are some reports that it is more frequent among smokers [8]. It has long been known that benzen in cigarette smoke causes acute myeloid leukemia [2,3,8].

Smoking and lung cancer link is known since 1912. Adler was the first scientist who mentioned that smoking causes lung cancer. In 1939 Müller performed the first clinical research that showed the causative relation of heavy smoking and lung cancer. Death from lung cancer has been strikingly increased in England and USA between the years 1928 - 1945. In 1941 Ochsner and DeBakey reported the parallel increase in lung cancer incidence and cigarette sale. In 1950 Schrek reported that smoking has a very important role in respiratory tract cancers [9]. Wynder who was a medical student at USA in 1950, together with his lecturer Graham, reported the first controlled study that shows the relation of smoking and lung cancer (9,10). In this study 684 lung cancer patients and 780 patients other than lung cancer were inquired about their smoking habits and occupation, the two group were compared. In conclusion, heavy smoking (more than 15 cigarette/day) was found the most important causative factor in bronchogenic carcinoma and increased risk of lung cancer continued more than 10 years after the cessation of smoking. Lung cancer risk was much less among people who were non smokers or smoked less than 10 cigarette/day [9,10]. Doll and Hill in England, performed a similar study in these years and concluded that people over 45 years of age who smoke more than 25 cigarette/day have 50 times increased risk of lung cancer [10,11]. After these initial reports, a lot of research showed the relation of smoking and lung cancer, so there is definite proof in this subject today [2,3,5,8]. An extra one cigarette added to your daily consumption increases the risk of lung cancer 7% [3]. It has long been known that passive smoking also increases the risk of lung cancer [2,7]. In 2013 China has reported that passive smoking caused more lung cancer death among women than men (40000 women - 12000 men) [1].

The carcinogenic material in cigarette smoke (especially polycyclic aromatic hydrocarbons and nitrosamine ketons) causes genetic mutation in cellular DNA of human. These carcinogenes are metabolised in the liver mainly by cythochrome P-450 and some by lipoxygenase, cyclooxygenase, myeloperoxydases and monoamine oxydase enzymes. They are converted to the last metabolites by glutathiones, sulfatases or uridine-5'-diphosphate-glucuronosyltransferase (U5'DPGT). Metabolites that are produced like PAH-benzopyrenes and nicotine derived nitrosamine ketone (NNK or NNAL) react with cellular DNA. Genetic mutations in K-Ras, p53, p16 (inhibition of tumour suppressor genes and activation of oncogenes) leads to lung cancer development in 20% of smokers [12,13]. Recently 20 - 24 nucleotides that are called microRNAs, have been detected to have a role in cigarette smoke induced carcinogenesis. They have been found to be effective in cell cycle, angiogenesis, metabolism, apoptosis and metastasis. Data from various studies have shown that MicroRNAs may have a role as tumour suppressor, oncogenic, diagnostic and prognostic biomarker in lung cancer [14].

First reports have shown that especially squamous cell and small cell lung cancer is increased among smokers but there has been an important increase in adenocarcinoma after 1970's. The reason for this situation is: 1- After 1950's filtered cigarettes with low tar content has been manufactured and blended tobacco in these cigarettes contain more carcinogens than pure tobacco; 2- New configuration in cigarettes caused much easy consumption and diffusion of carcinogens to the lung periphery [15].

As the link of smoking and lung cancer has been clearly proved, annual screening with low dose thoracal computed tomography is advised in selected high risk groups by Lung Cancer Diagnosis and Treatment Guidelines. These high risk groups are defined as:

1. 55 - 74 age group who smoked 30 pack-year or more, who is active smoker or who quitted less than 15 years ago.
2. 50 years of age or more who smoked 20 pack-years of cigarette with an additional risk factor (History of previous cancer, fam ily history of lung cancer, occupational exposure, having COPD or Pulmonary Fibrosis) [16].

In a recent review the use of circulatory MicroRNAs in screening and early detection of lung cancer is suggested as they are detected in body fluids and sputum before the clinical symptoms but there are still some discrepancies in this subject [14].

Early detection of lung cancer is very important because first stage nonsmall cell lung cancer has a five year survival of 77%, while stage four lung cancer has 2 - 22% [17].

Smoking cessation in every stage of lung cancer, is as much important as early detection and treatment of lung cancer [18]. Peto and coworkers have performed long controlled surveys and have found that lung cancer risk decreases significantly after cessation of smoking even in middle aged group. People who quit before the middle ages, have a decreased lung cancer risk of 90% or more and smoking cessation decreases 50% of the lung cancer mortality [19].

Smoking cessation in lung cancer patients whether at the time of diagnosis or treatment increases the survival. Median survival has been shown to be longer among the patients who quit after the diagnosis [20,21]. When a lung cancer patient stops smoking, acute reactions like recovering the sense of taste and smell, decreased blood pressure, increased immune response, increased cognitive functions, activity, performance and life quality occur as in all people [21]. Policyclic aromatic hydrocarbons in cigarette smoke decreases the effectivity of chemotherapotic drugs and increases the risk of secondary cancer and radiation pneumonitis among the patients who get radiotherapy. In other words smoking cessation decreases the risk of syncrone/metachrone cancer and radiation pneumonitis, increases the effectivity of chemotherapy [21]. Postoperative complication risk in early stage lung cancer patient is found as 23% among the active smokers while it is 8% in the ones who quit. Smoking cessation significantly decreases postoperative complication, morbidity and mortality [21].

In International Lung Cancer Diagnosis and Treatment guide, tobacco dependence treatment for lung cancer patients is strictly advised and physiological, behavioral and pharmacological therapies are described in detail [22,23].

Conclusion

In conclusion, every doctor should explain to their smoker patients that they have an increased risk of lung cancer. If the doctor is dealing with a cancer patient, he /she should inform the patient that smoking cessation will increase his quality of life, treatment response and life expectancy. So he/ she should insistently advise smoking cessation.

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