DOI: https://doi.org/10.2298/SARH170508191S
UDC: 616.24-002.5:613.84

ORIGINAL ARTICLE / ОРИГИНАЛНИ РАД

The impact of smoking on clinical characteristics and treatment outcome of patients with pulmonary tuberculosis

Sonja Smiljić
University of Priština, Faculty of Medicine, Department of Physiology, Kosovska Mitrovica, Serbia

SUMMARY
Intoroduction/Objective The objectives of our study were to determine the impact of smoking on clinical characteristics, the scope of radiological severity, and treatment outcome of patients with pulmonary tuberculosis (PTB).
Method This prospective study included patients suffering from PTB treated at the Pulmonology Department of the Clinical Hospital Center in Kosovska Mitrovica in the period between 2010 and 2016.
Results Among smokers suffering from PTB there were significantly more males ($p = 0.05$) between 30 and 49 years of age ($p < 0.001$). There was significantly more alcohol consumption present in smokers ($p < 0.001$), whose social factor for developing PTB ($p = 0.002$) was more expressed. A more severe form of PTB with cavitation was more common in smokers (38.8%), while a milder, parenchymatous, unilateral PTB was present in non-smokers (31.8%). Extensive X-ray changes were more common in smokers ($p = 0.002$). Relapse of the disease was more prevalent in smokers ($p = 0.05$). In multivariate logistic regression, the risks of being a smoker included years of age: 30–39 [odds ratio (OD) = 18.11], 40–49 (OR = 19.66), and 50–59 (OR = 9.06), and alcohol consumption habits (OR = 9.32).
Conclusion Smokers were more often afflicted with sputum-positive PTB, had extensive radiological changes, and the relapse of the disease was more common. Smokers were mostly middle-aged, with alcohol consumption habits, and constructed a group of patients whose habits were a critical factor for the eradication of tuberculosis.
Keywords: pulmonary tuberculosis; smoking; alcohol; X-ray abnormality

INTRODUCTION

Smoking and tuberculosis (TB) are the two biggest public health problems in the world. Smoking is one of the leading causes of premature death, causing six million deaths annually. Today, about 33% of the world's population smokes, mainly in countries with a high prevalence of TB. In 2015, pulmonary tuberculosis (PTB) affected 10.4 million people and one fifth of TB cases can be blamed on tobacco smoking [1]. The 2014 United States Surgeon General's Report implicates smoking as a cause of TB disease among those latently infected with *Mycobacterium tuberculosis* [2].

While the link between smoking and PTB was recognized almost a century ago, the impact of smoking on the development of TB has mostly been explained in the last few decades [3]. Active and passive exposures to tobacco smoke are independent risk factors for TB infection, the progression of TB infection to an active disease, severe clinical profile, and an increased risk of relapse and mortality. Thus, smokers are at a higher risk of developing TB than non-smokers. In smokers, the latent form of TB often turns into an active one, and the determining factor in this is their general health status. Smoking affects health and modifies the immune response, which favors the development of TB [4, 5, 6].

A recent study based on mathematical modeling estimated that, between 2010 and 2050, smoking can increase the number of TB patients in the world by 18 million, and cause a significant increase in mortality if the current trend in tobacco consumption continues unchecked [7]. Smoking prevalence among TB patients is higher than among the general population in many countries. However, the data are different, from 48% in Catalonia, 54.6% in China, to 81.5% in rural India [8, 9, 10]. Patients who are smokers often spread the disease to other family members [11]. The World Health Organisation estimates that about one third of people in the world are infected with *Mycobacterium tuberculosis* and that 90% of them exhibit no symptoms, making it a latent TB infection [1]. Smoking, alcohol consumption, and malnutrition can influence the transition from the latent to the active form of TB.

Possible mechanisms of the impact of smoking include reduced clearance of secretions on tracheobronchial mucosal surface, reduced phagocytic function of alveolar macrophages, reduced alveolar macrophage production of tumor necrosis factor (TNF), and increased macrophage hemochromatosis [3, 12]. Smoking reduces the effectiveness of the alveolar macrophages in developing effective immune response by altering the expression of cell

Received • Примљено: May 8, 2017
Revised • Ревизија: October 23, 2017
Accepted • Прихваћено: October 24, 2017
Online first: October 31, 2017

Correspondence to:
Sonja SMILJIĆ
Faculty of Medicine, University of Priština
Anri Dinana bb
38220 Kosovska Mitrovica
Serbia
sonja.smiljic@med.pr.ac.rs

proinflammatory cytokines [13]. In addition to nicotine, which modulates the activity of macrophages, about 5,000 more substances present in tobacco smoke modulate the activity of inflammatory cells. The correlation of TB and smoking can be a result of the inhibitory effect of nicotine in the production of TNF-alpha and anti-inflammatory cytokines CKSCL8 that make patients susceptible to the progression of the latent form of the infection into the active one. Smoking disrupts the phagocytic function of alveolar macrophages and induces apoptosis in macrophages. Chronic exposure to cigarette smoke reduces the expression of surface proteins related to antigen presentation by macrophages. Monocyte macrophage system in TB patients has a reduced phagocytic capacity, which is further reduced in patients with TB who smoke [3, 14].

Chronic alcohol use can reduce the response of macrophages and the immune system [15]. Body mass index value under 18.5 kg/m^2 is seen as a marker for malnutrition, and malnutrition can reduce immune response either through the interaction of monocyte-macrophages and T-lymphocytes and their cytokines or the secondary immune deficiency that increases the susceptibility of the host to infection [16]. Alcohol consumption significantly worsens the clinical manifestation of the disease and the outcome of the treatment, which may be the result of the adverse reactions to medication used in the treatment of PTB. People that abuse alcohol are 1.32 times more likely to exhibit an adverse reaction to tuberculostatics [10, 17]. The relation between the manifestation of adverse drug effects during treatment and unsuccessful treatment plays an important role in controlling the spread of TB.

The social and clinical reasons for the failure of antituberculosis therapy can be seen as a result of smoking and alcoholism. Smoking and alcohol are related to other pathological conditions that can be among the causes of treatment failure, and often of the premature discontinuation of therapy.

Unsuccessful treatment outcome is an adverse health condition for both the patients and for the public health, because it increases the duration of infectiousness; thus, individual and public health concerns should be considered together in planning effective control strategies. A large proportion of cases in which treatment will be unsuccessful could be predicted at entry through screening for age group, smoking, and alcoholism, and specially targeted measures could be taken in such cases.

The aim of our study was to determine the impact of smoking on clinical characteristics, the scope of radiological severity, and the treatment outcome of patients with PTB.

METHODS

The survey was conducted in accordance with the ethical principles and was approved by the Ethics Committee of the Priština Faculty of Medicine, temporarily seated in Kosovska Mitrovica.

This prospective study was conducted at the Department of Pulmonology of the Clinical-Health Center in Kosovska Mitrovica, the reference hospital for TB treatment. The study included patients suffering from TB, a total of 104 subjects treated during the period between 2010 and 2016.

The inclusion criteria for the selection of patients in this study were as follows: 1) older than 20 years of age; 2) typical symptoms of PTB (cough, sputum production, fever, night sweats, and weight loss); 3) typical fibrocavitary pulmonary infiltrates on chest radiographs standard; 4) at least one smear-positive sputum, with the subsequent positive culture of *Mycobacterium tuberculosis*, and 5) all study patients could already have been on the antituberculosis treatment (processed with all necessary radiological, microbiological, and laboratory and spirometric examinations, before starting the antituberculosis treatment).

On admission, the patients' data regarding demographics, age, sex, residence, marital status, education, occupation and possible contact with people suffering from TB were gathered. Special attention was paid to risk factors for developing PTB: smoking, alcohol consumption, drug use, and social status. In regard to comorbidities, we processed instances of diabetes mellitus.

All the patients were divided into two groups – smokers and non-smokers. The subjects were considered smokers if they reported that they smoked one or more cigarettes per day during the year preceding the diagnosis of TB, or non-smokers if they consumed less than 100 cigarettes in their life [8, 18]. Alcohol consumption was considered significant if male subjects consumed more than 280 g of alcohol per week, and female subjects over 168 g per week. None of our subjects used drugs (intravenous heroin and/or cocaine, or other).

We processed the following initial symptoms and signs in our patients: cough, sputum production, hemoptysis, chest pain, fever, night sweats, fatigue, and weight loss. In regard to the laboratory parameters, we processed the parameters for anemia (hemoglobin, hematocrit, hematological indices) and sedimentation rate. Sputum samples were taken from all the patients for a direct microscopy of the preparations stained according to the Ziehl–Neelsen method. Also, cultivation of the bacillus on the Lowenstein–Jansen medium was performed for all samples. The sputum was collected in the morning, before eating, after a spontaneous expectoration. Each sputum positive for direct microscopy was verified by the culture on the Lowenstein–Jansen medium. PTB was bacteriologically confirmed if the two sputum findings confirmed bacillus and/or in the case of positive sputum cultivation.

Chest X-ray results were categorized according to the scale of changes, their localization and their morphological structure. The interpretation of chest X-ray abnormalities was performed by a radiologist. Chest X-rays were focused on pulmonary parenchyma and caverns. The interpretation of abnormalities in the pulmonary parenchyma included unilateral or bilateral changes, the location of changes (in the lower, medium and upper fields) and the scale of changes (minimum/moderate and extensive).The final diagnosis of PTB was made based on the positive findings pertaining to acid-resistant bacilli in sputum and/or chest X-rays.

Statistical analysis

The data were analyzed by descriptive statistical methods and presented as frequencies and relative numbers. For the analysis of frequency differences between the groups, χ^2 test was used. Binary logistic regression was the technique used to analyze the dependencies between activities. The multiple logistic regression model included all the predictors that had statistical significance at 0.05 in a single logistic regression model. The criterion for statistical significance was $p < 0.05$.

For statistical data analysis, we used the IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY, USA).

RESULTS

The study involved patients with PTB treated at the Department of Pulmonology in the period between 2010 and 2016. During these six years, we treated 104 patients with TB, who were predominantly male (67%). Out of the total number of patients with PTB, 60 were smokers (57.7%) and were 44 non-smokers (42.3%). Among smokers suffering from TB there were significantly more males ($p = 0.05$), between 30 and 49 years of age ($p < 0.001$). The majority of non-smokers who became ill (19) with TB are among women of the age between 20 and 29 years.

There was no significant difference in relation to the place of residence, family status, and education level between smokers and non-smokers. However, smokers suffering from TB were more likely to be manual laborers (40%). Smokers consumed alcohol significantly more often than non-smokers did ($p = 0.001$). The social determinant was significant in smokers with PTB ($p = 0.002$). A possible contact with the PTB affected people and diabetes mellitus were equally present in smokers and non-smokers (Table 1).

Respiratory symptoms were typical for both groups of patients, and did not differ significantly. Laboratory parameters for anemia and erythrocyte sedimentation rate did not differ significantly in smokers and non-smokers. A direct sputum bacilloscopy was more frequently positive in smokers than in non-smokers (Table 2).

There were some significant differences in radiographic severity. Smokers were often diagnosed with TB with cavitation, while in non-smokers the more common form was unilateral parenchymal PTB. Upper lung fields were significantly more affected by the changes in smokers (68.3%). The extent of radiological changes was significantly higher in smokers ($p = 0.002$). The incidence of relapse in smokers was 23.3% and 9.1% in non-smokers. The relapse of the disease was significantly more common in smokers ($p = 0.05$) (Table 3). Multiple logistic regression of the variables related to socio-demographic characteristics and risk factors associated with smoking included age [30–39 (OR = 11.18), 40–49 (OR = 19.66), and 50–59 (OR = 9.06)] and the habit of alcohol consumption (OR = 9.32) (Table 4).

Table 1. Sociodemographic characteristics and risk factors for pulmonary tuberculosis in smokers and non-smokers (n = 104)

Baseline patient characteristics	Population examined		p
	Smoker n (%)	Non-smoker n (%)	
	60 (57.7)	44 (42.3)	
Age, years			
20–29	3 (5)	17 (38.6)	< 0.001
30–39	11 (18.3)	3 (6.8)	
40–49	13 (21.7)	2 (4.5)	
50–59	21 (35.0)	10 (22.7)	
> 60	12 (20)	12 (27.3)	
Sex			
Male	45 (75)	25 (56.8)	0.050
Female	15 (25)	19 (43.2)	
Residence			
Rural	45 (75.0)	32 (72.7)	0.794
Urban	15 (25.0)	12 (27.3)	
Marital status			
Single	25 (41.7)	25 (56.8)	0.127
Married	35 (58.3)	19 (43.2)	
Education			
Primary	36 (60)	23 (52.3)	0.432
Secondary	22 (36.7)	21 (47.7)	
High	2 (3.3)	0 (0)	
Employment status			
Unemployed	13 (21.7)	11 (25)	0.185
Manual laborer	24 (40)	9 (20.5)	
Office worker	9 (15)	8 (18.2)	
Pensioner	14 (23.3)	16 (36.4)	
Alcohol use			
Yes	19 (31.7)	1 (2.3)	
No	41 (68.3)	43 (97.7)	< 0.001
Social determinants			
Yes	30 (50)	9 (20.5)	
No	30 (50)	35 (79.5)	0.002
Tuberculosis contact			
Yes	6 (10)	4 (9.1)	
No	54 (90)	40 (90.9)	0.877
Tuberculosis history			
Yes	9 (15)	7 (16.3)	
No	51 (85)	37 (83.7)	0.860
Diabetes mellitus			
Yes	9 (15)	5 (11.4)	
No	51 (85)	39 (88.6)	0.591

DISCUSSION

The World Health Organisation estimates that, during 2015, 9.4 million people were affected by TB, with a fatal outcome in 1.4 million of the treated patients. The main reasons for maintaining this high number of patients in the past two decades are the large number of HIV-infected patients and multidrug-resistant TB [19, 20]. Another very important risk factor whose effects on morbidity related to TB have been explained in the past several years is smoking [21]. In European countries, 16% of fatalities among adults older than 30 years were caused by tobacco consumption [21]. Other risk factors for developing TB include alcohol, drugs and associated diseases, especially diabetes, contact

Table 2. Symptoms and clinical signs between smokers and non-smokers with pulmonary tuberculosis (n = 104)

Symptoms	Population examined		p
	Smokers n (%)	Non-smokers n (%)	
	60 (57.7)	44 (42.3)	
Cough			
Yes	53 (88.3)	34 (77.3)	0.132
No	13 (11.7)	11 (22.7)	
Productive cough			
Yes	36 (60)	21 (47.7)	0.214
No	24 (40)	23 (52.3)	
Hemoptysis			
Yes	5 (8.3)	7 (15.9)	0.232
No	55 (91.7)	37 (84.1)	
Fever			
Yes	41 (68.3)	24 (54.5)	0.151
No	19 (31.7)	20 (45.5)	
Night sweats			
Yes	34 (58.6)	25 (56.8)	0.935
No	26 (41.4)	19 (43.2)	
Asthenia			
Yes	37 (61.7)	30 (68.2)	0.493
No	23 (38.3)	14 (31.8)	
Weight loss			
Yes	27 (45)	25 (56.8)	0.234
No	33 (55)	19 (43.2)	
Anemia			
Yes	19 (31.7)	11 (25)	0.458
No	41 (68.3)	33 (75)	
Sedimentation rate			
Yes	42 (70)	29 (65.9)	0.658
No	18 (30)	15 (34.1)	
Sputum			
Negative	17 (28.3)	20 (45.5)	0.072
Positive	43 (71.7)	24 (54.5)	

Table 3. Comparison of severity of pulmonary tuberculosis and treatment outcome between smokers and non-smokers

Clinical characteristics	Population examined		p
	Smokers n (%)	Non-smokers n (%)	
	60 (57.7)	44 (42.3)	
Case type			
New	46 (76.7)	40 (90)	0.050
Retreatment	14 (23.3)	4 (10)	
Diagnosis			
PTB unilateral	14 (23.3)	14 (31.8)	0.273
PTB bilateral	18 (30)	17 (38.6)	
PTB with multiple cavities	23 (38.3)	9 (20.5)	
Pleural effusion	5 (8.3)	4 (9.1)	
Location of CXR abnormality			
Upper field	41 (68.3)	27 (61.4)	0.390
Medium field	11 (18.3)	7 (15.9)	
Lower field	8 (13.4)	10 (22.7)	
Radiological severity			
Initial	21 (48.1)	29 (65.9)	0.002
Advanced tuberculosis	39 (51.9)	15 (34.1)	
Outcomes			
Cure	46 (44.2)	39 (97.5)	0.475
Relapse	11 (10.6)	4 (3.8)	
Death	3 (2.9)	1 (1)	
Intra hospital therapy (days)			
< 30	11 (18)	9 (20.5)	0.786
> 30	49 (81.7)	35 (79.5)	

CXR – chest X-ray; PTB – pulmonary tuberculosis

Table 4. Multivariate logistic analysis of the association of demographic characteristics and risk factors with smoking

Factors	OR (CI 95%)	p
Age (years)		
20–29 (reference)	-	-
30–39	11.18 (1.75–71.52)	0.011
40–49	19.66 (2.67–144.94)	0.003
50–59	9.06 (2.10–39.01)	0.003
> 60	5.29 (1.21–23.04)	0.027
Sex	0.8 (0.28–2.25)	0.615
Alcohol use	9.32 (1.12–77.67)	0.039
Social determinants	2.01 (0.72–5.66)	0.172

OR – odds ratio; CI – confidence interval

with infected persons, as well as poor living conditions such as overcrowding and poor ventilation. There are no detailed data on the global prevalence of risk factors, so it is assumed that the prevalence of risk factors is the same in all segments of the adult population.

Smokers suffering from TB had a more severe clinical and radiological presentation of the disease, a more common sputum-positive TB in the beginning, as well as two months after the treatment, a lower therapy success rate, and a higher risk of relapse [13]. Patients treated for TB who were included in our study were mostly smokers (57.7%). This is a higher percentage of smokers than in studies conducted in different parts of the world, including countries with high PTB prevalence [9, 10, 15]. A similar number of smokers suffering from TB were recorded in Russia (49%) and Spain (48%). Also, the number of smokers among our patients suffering from PTB was higher than in the general population, where it amounted to 41.2% in people between the ages of 18 and 64 years [8, 22].

Socio-demographic characteristics of smokers and non-smokers suffering from PTB included in our study did not notably differ. A significant difference was detected in relation to patients' sex. Smokers suffering from TB were mostly middle-aged men, between 30 and 49 years of age, who were manual laborers [23]. Among the non-smokers, ill with TB were mainly females between 20 and 29 years of age, from the rural environment. The traditional way of behavior among unemployed women from the countryside continues to be present in this region, so that women are less likely to be smokers. Place of residence, family status, and education level did not differ significantly, which is in contrast with data from other regions, where there the number of affected smokers in the rural areas was greater [24]. The results of the 2013 Health Survey in Serbia show that the percentage of smokers in the cities is statistically significantly higher compared to inhabitants of rural areas. It also showed that there are fewer females among smokers.

Risk factors considered important in the occurrence of PTB were a possible contact with an affected person and a positive family history. However, these were equally present in non-smokers and smokers. We obtained the same results concerning comorbidities. Our study was not able to confirm the existing evidence that the association of smoking and diabetes increases the risk of developing TB, probably because among our patients, smokers suffering from TB were mostly middle-aged persons who did not suffer from diabetes mellitus [23, 25]. Older patients suffering from TB who were treated for diabetes mellitus were rarely smokers.

The risk factors are more common in men than in women, which was concluded in the study that covered 14 countries with the highest rate of TB incidence. In addition to cigarette smoking, alcohol consumption and the social determinant were the most important predictors for developing PTB among our subjects. Men who drank alcohol and smoked cigarettes were more susceptible to TB. Among the general population, 4.7% were daily alcohol consumers, while the percentage was several times higher among our TB patients (31.7%). There were a significant number of smokers who were also alcohol consumers [18]. Among the affected women, there were fewer smokers and they rarely drank alcohol [19]. Fewer smokers and alcohol consumers among our female patients treated for PTB can be explained by the fact that these habits are not socially acceptable in their social settings, where men drink six times more than women.

Important symptoms in the diagnosis of TB are the following: a cough present for at least two weeks, sputum, fever, night sweats, weight loss, asthenia, and hemoptysis [18]. A single symptom can be present, or there can be a combination of several TB-sensitive symptoms. In patients involved in our study, the symptoms of the disease occurred earlier in smokers than in non-smokers, but there were no significant differences present in the manifestations of symptoms of the disease, which was not in line with other studies. We confirmed that the sputum-positive PTB was more common in smokers. Smokers who suffer from TB are likely to have a greater ability to spread germs and a greater risk to infect their family members. It is possible to protect the family by changing their habits and quitting smoking. In patients with PTB, we detected laboratory abnormalities such as anemia and accelerated erythrocyte sedimentation rate, which did not significantly differentiate between smokers and non-smokers. Chronic infections, including TB, cause anemia, which is explained by the suppression of erythropoiesis of inflammatory mediators. On the other hand, the disruption of iron homeostasis develops with an increased absorption and retention of iron in the reticuloendothelial system during a chronic infection, such as TB [26].

Cigarette smoking is associated with an increased risk of advanced and more severe forms of the disease, such as cavitation, a positive sputum culture, and subsequent conversion of sputum culture after starting the treatment. Smoking has adverse effects on the completion of the treatment and relapse [27, 28]. In our study, smokers with PTB were more likely to experience bilateral changes on lung parenchyma or caverns. An Indian study showed that among non-smokers there were more people with minimal changes, while extensive changes were more common among smokers. Cavitation is more common in smokers [29].

In our patients, in addition to severe clinical manifestations of the disease, poorer treatment outcomes and relapse were significantly more frequent. This is consistent with other studies, where relapse of TB was recorded in 10.4% of patients [30]. For example, in a survey conducted in Georgia, smokers had a 70% poorer outcome than non-smokers [22]. Patients experiencing extensive changes on the lung parenchyma often suffer from relapses and are at increased risk of mortality [29, 30]. Among our patients, there were more death outcomes in smokers suffering from TB. We could not statistically confirm this fact due to a relatively small sample and because the treatment of PTB in the Northern Kosovo region is mostly successful. The effect of smoking on clinical parameters (lung cavitation and positive sputum culture) and a slower sputum conversion rate after the start of treatment has a serious impact on the prevention of disease transmission. Even in patients who are sensitive to treatment with tuberculostatics, the success rate of the treatment is lower than the desired 85%, which is the objective set by the World Health Organisation.

Several lifestyle factors are associated with an increased risk of PTB, including smoking and alcohol abuse [15, 16]. Smoking and excessive alcohol consumption are major health risks globally and are targets for interventions to reduce the global burden of disease. Ensuring that patients make appropriate lifestyle changes would help reduce the overall burden of PTB.

The impact on the social determinants that are significant predictors for developing PTB must take place through a number of actions at the social level in order to minimize poverty and promote better education on prevention measures. Integrated public health programs are needed, which can help reduce the number of patients with diabetes mellitus, smoking, and excessive alcohol use [31].

CONCLUSION

Smokers suffering from TB were more often middle-aged males that consumed alcohol and lived in poor social conditions. They had more severe clinical manifestations of TB with extensive X-ray changes in the lungs, often with caverns. Smokers who suffered from TB had a higher risk of relapse. The risk of death was higher in smokers than in non-smokers.

A detailed understanding of the diffusion of smoking in our environment, as well as the socio-demographic and clinical factors associated with smoking among patients with PTB, is the first step towards the formation of effective strategies for early diagnosis, control, and monitoring, in order to reduce the number of patients and improve treatment outcomes. Smoking is a risk factor for more frequent incidence of PTB with severe clinical forms and poorer treatment outcome. A part of the strategy for eradicating TB needs to be directed towards the campaign against smoking.

REFERENCES

1. World Health Organization. Global tuberculosis report 2016. Geneva: World Health Organization. 2016 (WHO/HTM/TB/2016).
2. ASPA. The Health Consequences of Smoking–50 Years of Progress: A Report of the Surgeon General, 2014. Available from: http://www.surgeongeneral.gov/library/reports/50- years-of progress/ index.html
3. Aryanpur M, Mortaz E, Masjedi MR, Tabarsi P, Garssen J, Adcock IM, et al. Reduced Phagocytic Capacity of Blood Monocyte/ Macrophages in Tuberculosis Patients Is Further Reduced by Smoking. Iran J Allergy Asthma Immunol. 2016; 15(3):174–82.
4. Aryanpur M, Masjedi MR, Hosseini M, Mortaz E, Tabarsi P, Soori H, et al. Cigarette smoking in patients newly diagnosed with pulmonary tuberculosis in Iran. Int J Tuberc Lung Dis. 2016; 20(5):679–84.
5. Altet-Gômez MN, Alcaide J, Godoy P, Romero MA, Hernández del Rey I. Clinical and epidemiological aspects of smoking and tuberculosis: a study of 13038 cases. Int J Tuberc Lung Dis. 2005; 9(4):430–6.
6. Zellweger JP, Cattamanchi A, Sotgiu G. Tobacco and tuberculosis: could we improve tuberculosis outcome by helping patients to stop smoking? Eur Respir J. 2015; 45(3):583–5.
7. Basu S, Stuckler D, Bitton A. Projected effects of tobacco smoking on worldwide tuberculosis control: mathematical modelling analysis. BMJ. 2011; 4(343):2–11.
8. Hernáiz CR, Giardín JM, Clotet CN, Lebrato CJ. Tuberculosis and immigration in an area of southwest Madrid. Int J Tuberc Lung Dis. 2016; 20(4):530–5.
9. Wang J, Shen H. Review of cigarette smoking and tuberculosis in China: intervention in needed for smoking cessation among tuberculosis patients. BMC Pub Health. 2009; 9:292.
10. Gajalakshmi V, Peto R. Smoking, drinking and incident tuberculosis in rural India: population-based case–control study. Int J Epidemiol. 2009; 38(4):1018–145.
11. Huang CC, Tchetgen E, Becerra MC, Cohen T, Galea J, Calderon R, et al. Cigarette smoking among tuberculosis patients increases risk of transmission to child contacts. Int J Tuberc Lung Dis. 2014; 18(11):1285–91.
12. van Zyl-Smit RN, Binder A, Meldau R, Semple PL, Evans A, Smith P, et al. Cigarette smoke impairs cytokine responses and BCG containment in alveolar macrophages. Thorax. 2014; 69(4):363–70.
13. Gegia M, Magee MJ, Kempker RR, Kalandadze I, Chakhaia T, Golub JE, et al. Tobacco smoking and tuberculosis treatment outcomes: a prospective cohort study in Georgia. Bull World Health Organ. 2015; 93(6):390–9.
14. Feng Y, Kong Y, Barnes PF, Huang FF, Klucar P, Wang X, et al. Exposure to cigarette smoke inhibits the pulmonary T-cell response to influenza virus and *Mycobacterium tuberculosis*. Infect Immun. 2011; 79(1):229–37.
15. Davis A, Terlikbayeva A, Aifah A, Hermosilla S, Zhumadilov Z, Berikova E, et al. Risks for tuberculosis in Kazakhstan: implications for prevention. Int J Tuberc Lung Dis. 2017; 21(1):86–92.
16. Patra J, Jha P, Rehm J, Suraweera W. Tobacco smoking, alcohol drinking, diabetes, low body mass index and the risk of self-reported symptoms of active tuberculosis: individual participant data (IPD) meta-analyses of 72,684 individuals in 14 high tuberculosis burden countries. PLoS One. 2014; 9(5):e96433.
17. Lin HH, Ezzati M, Murray M. Tobacco smoke, indoor air pollution and tuberculosis: a systematic review and meta-analysis. PLoS Med. 2007; 4(1):e20.
18. Jiménez-Fuentes MA, RodrigoT, Altet MN, Jiménez-Ruiz AC, Casals M, Antón Penas A. Factors associated with smoking among tuberculosis patients in Spain. BMC Infect Dis. 2016; 16:486.
19. Glaziou P, Falzon D, Floyd K, Raviqilione M. Global epidemiology of tuberculosis. Semin Respir Crit Care Med. 2013; 34(1):3–16.
20. Ferrara G, Murray M, Winthrop K, Centis R, Sotgiu G, Migliori GB, et al. Risk factors associated with pulmonary tuberculosis: smoking, diabetes and anti-TNFα drugs. Curr Opin Pulm Med. 2012; 18(3):233–40.
21. WHO global report: mortality attributable to tobacco. Geneva: World Health Organization 2012; (Accessed 9 Septmber 2014). Available from: http://www.who.int/tobacco/publications/ surveillance/ rep_mortality_attributable/en/
22. WHO Report on the Global Tobacco Epidemic 2013. World health organization. Available from: http://www.who.int/gho/countries/en/
23. Bai KJ, Lee JJ, Chien ST, Suk CW, Chiang CY. The influence of smoking on pulmonary tuberculosis in diabetic and non-diabetic patients. PLoS One. 2016; 11(6):e0156677.
24. Kolappan C, Gopi PG. Tobacco smoking and pulmonary tuberculosis. Thorax. 2002; 57(11):964–6.
25. Reed GW, Choi H, Lee SY, Lee M, Kim Y, Park H, et al. Impact of diabetes and smoking on mortality in tuberculosis. PLoS One. 2013; 8(2):e58044.
26. Pednekar MS, Hakama M, Gupta PC. Tobacco use or body mass – do they predict tuberculosis mortality in Mumbai, India? Results from a population-based cohort study. Plos One. 2012; 7:e39443.
27. Leung CC, Yew WW, Chan CK, Chan KC, Law WS, Lee SN, et al. Smoking adversely affects treatment response, outcome and relapse in tuberculosis. Eur Respir J. 2015; 45(3):738–45.
28. Lin HH, Murray M, Cohen T, Colijn C, Ezzati M. Effects of smoking and solid-fuel use on COPD, lung cancer, and tuberculosis in China: a time-based, multiple risk factor, modelling study. Lancet. 2008; 372(9648):1473–83.
29. Waitt CJ, Squire SB. A systematic review of risk factors for death in adults during and after tuberculosis treatment. Int J Tuberc Lung Dis. 2011; 15(7):871–5.
30. Mahishale V, Patil b, LollyM, Eti A, Khan S. Prevalence of smoking and its impact on treatment outcomes in newly diagnosed pulmonary tuberculosis patients: a hospital-based prospective study. Chonnam Med J. 2015; 51(2):86–90.
31. Lönnroth K, Castro KG, Chakaya JM, Chauhan LS, Floyd K, Glaziou P, et al. Tuberculosis control and elimination 2010–50: cure, care, and social development. Lancet. 2010; 375(9728):1814–29.

Утицај пушења на клиничке карактеристике, радиолошке промене и исход лечења оболелих од туберкулозе плућа

Соња Смиљић

Универзитет у Приштини, Медицински факултет, Институт за физиологију, Косовска Митровица, Србија

САЖЕТАК

Увод/Циљ Циљ нашег истраживања био је да утврдимо утицај пушења на клиничке карактеристике, обим радиолошких промена и исход лечења оболелих од туберкулозе плућа.

Методологија Проспективном студијом обухватили смо све оболеле од туберкулозе плућа (ТБП) лечене на Одељењу пулмологије Клиничко-болничког центра у Косовској Митровици у периоду од 2010. до 2016. године.

Резултати Међу пушачима оболелим од ТБП било је значајно више особа мушког пола ($p = 0,05$), старости између 30 и 49 година ($p < 0,001$). Пушачи су значајно више конзумирали алкохол ($p < 0,001$) и имали су изражен социјални фактор за обољевање од ТБП ($p = 0,002$). Тежа клиничка форма ТБП са кавернама била је чешћа код пушача (38,8%), а код непушача лакша, паренхиматозна, једнострана плућна туберкулоза (31,8%). Обимне радиолошке промене су биле израженије код пушача ($p = 0,002$). Рецидив болести се чешће јављао код пушача ($p = 0,05$). У мултиваријантној логистичкој регресији ризик да неко буде пушач јесу године живота 30–39 ($OR = 11,18$), 40–49 ($OR = 19,66$) и 50–59 ($OR = 9,06$) и навика конзумације алкохола ($OR = 9,32$).

Закључак Пушачи су чешће боловали од спутум позитивне туберкулозе плућа, имали су обимније радилошке промене и рецидив болести је био чешћи. У највећем броју били су средњих година живота са навиком конзумирања алкохола и чинили су групу оболелих чије навике представљају главни разлог за искорењивање туберкулозе.

Кључне речи: туберкулоза плућа; пушење; алкохол; РТГ абнормалности