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# A Survey on Relationship between Post Surgery Infection of Latent Mandible's Wisdom Tooth and Smoking.

# Sirous Risbaf Fakour<sup>1</sup>\*, Fariba Shahri<sup>2</sup>, and Somaye Ansari Moghadam<sup>3</sup>.

<sup>1</sup>Department of Oral Maxillofacial Surgery, Oral and Dental Disease Research Center, Zahedan University of Medical Sciences, Zahedan, Iran.

<sup>2</sup>Department of Orthodontics, Oral and Dental Disease Research Center, Zahedan University of Medical Sciences, Zahedan, Iran.

<sup>3</sup>Department of Periodontology, Oral and Dental Disease Research Center, Zahedan University of Medical Sciences, Zahedan, Iran.

# ABSTRACT

One of the most important and most common oral operations is removing latent teeth, especially mandible's wisdom tooth. An important factor in stopping enough perfusion to and healing of injury due to surgery which is also effective in post-operation infection is smoking. The current study is designed to examine the effect of smoking on after surgery mandible's wisdom tooth's situation. The current study was cross-sectional and people were in 17-40 age range, the entire group had at least one latent wisdom tooth in their mandible. There was also two smoker and non-smoker group in the study. The operations were done in standard procedure and patients visited to check their situation including Inflammation and infection at mouth and the wound due to the surgery. It was found that 8 and 2 patients of smoker and non-smoker group suffered from post-operation infection. There was no significant difference between the groups in terms of gender and infection as well as the operation's durance (p=0.128, p=0.373 and0.091, respectively). As the role of smoking is just postponement in healing of the wound tissue due the operation and also bleeding it; so, if the person has suitable health situation, and also, has not trauma, smoking by itself cannot make infection. **Keywords:** mandible's latent wisdom tooth, post-operation infection, smoking



\*Corresponding author

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#### INTRODUCTION

Latent tooth is a type which cannot develop at expected time [20]. Latent tooth is associated with pathological changes such as inflammation of surrounded gum, roots' dystrophy, gum and alveolar bone illnesses, injury to next tooth, cysts, and tumors development [15]. Operation of latent wisdom tooth is one of the most common ones which are done by maxillofacial surgeons, daily [1]. There are so many complaints from patients in effect of surgery [23]. However, the most common complaints are about pain, inflammation as well as trismus which are affected by different factors and variables [21-4].

Affective factors on post-operation infection can be listed as poor health, topical systemic illnesses, and smoking which is one of the most dangerous as well as exaggerating factors in this regard [9]. There is some evidence of exaggerating mouth-related illnesses due to tobacco abuse [17]. Surgeons count smoking as an important factor of making disruption in healing process of post-operation wound, and ischemia is the most pathological factor in this regard [10-22].

Three main components of cigarette which intervene with improvement of the injury are nicotine, carbon monoxide, and cyanide hydrogen. Nicotine is absorbed from lungs and enters in blood cycle; the factor can make effects such as reduction in erythrocytes proliferation, fibroblasts and macrophages [24]. Cigarette also reduces oxygen and nutrients in gum's lamina propria and increases negative gram bacteria which in turn makes the person susceptible to periodontitis from one side and postpone his/her wound, healing due to lack of oxygen in tissue as well as effect on immune system [26]. Cigarette also plays an important role in suffering from mouth candidasis. All, the above mentioned factors are important in formation of infection and post pone of the injury's healing. In other word, cigarette is a predisposition factor in accumulation of infectious elements at jaws operation position [25]. Cigarette also suppresses leucocyte activities and reduction of the leucocyte and immunoglobolins, then, leads to candidiasis colonization in mouth. Another effect of cigarette can be cited as temporary reduction of mouth's PH which in turn plays an important role in bacteria's colonization in the smoker person [12].

According to the above cited evidences, cigarette is an important factor in delaying of wound's healing and perfusion to the wound tissues increasing probability of post-operation surgery infection.

Thus, the current study is designed to assess the relationship between after surgery infection of mandible's latent wisdom tooth and smoking.

## **Methodology of Research**

The current study was cross- sectional and designed to examine the effect of smoking on making post -operation infection of removing mandible's latent wisdom tooth, the study endured for 12 months from April to march (2012-2013) and sample selected among clients of dentistry faculty of Zahedan. There were two criteria to be included in the sample: being in the age range of 17-40, and having at least one latent mandible. The people were classified as smokers and non-smokers groups.

Criteria to be excluded from the study were having allergy to especial medicine and inflammation at the surgery place. Other groups which excluded from the study were deficient immunization, malignant cancers, systemic diseases and people who take corticosteroids. Also, a group who their operations endured more than 20 minutes was excluded. Other variable which included in the study was literacy level; so, all people in the study were bachelor to control the variable. All participants were trained to know the personal health tips of after surgery and also risks of violation of health rules describe to them.

Surgery was done in a standard mode and the patients visited for three times at two, four and seven days of operation and their probable inflammation and infection at the surgical position were checked. The collected data was analyzed by 16<sup>th</sup> version of SPSS software.



## **Data Analysis**

The collected data was analyzed with applying descriptive indexes such as frequency, percentage, mean, and standard deviation. The applied statistics, for inferential statistics were chi-square and t.test and significant level was 0.05.

## RESULTS

Patients were examined in five age group: 7 person%5.3 percent of all selected people in 17-20, 42 person%21 percent of all selected people in 21-25, 53 person %26.5percent of all selected people in 26-30, 50 person %25 percent of all selected people in 31-35, and 48 person %24 percent of all selected peoplein 36-40 age ranges as shown in Table.1.

#### Table 1: Age distribution of participants according to smoking

	Age groups											
Groups	17-20		21-25		26-30		31-35		36-40		Total	
	F	Р	F	Р	F	Р	F	Р	F	Р	F	Р
Smoker	0	0	15	5.7	25	5.12	34	17	26	13	100	50
Non- smoker	7	5.3	27	5.13	28	14	16	8	22	11	100	50
Total	7	5.3	42	21	53	5.26	50	25	48	24	200	100

Note: F shows Frequency and P shows percentage.

Majority of patients in smoker group were in the 31-35 age range (%34) and Majority of non-smokers were in 26-30 age range (%28). Also, a significant relationship was found between the groups and the age (p=0.002).

According to Table.2, around %28 (56 patients) in smoker group were male and 44 person (%22) were female, for the other group the values were 46(%23) and 54(%27) respectively. As the significant level was p= 0.128, it can be said that no significant relationship was existed between gender and the group.

Groups	M	ale Fem		ale	Total		
	F	Р	F	Р	F	Р	
Smoker	44	22	56	28	100	50	
Non- smoker	54	27	46	23	100	50	
Total	64	32	136	68	200	100	

#### Table 2: Gender distribution of smoker and non-smoker groups

Note: F shows Frequency and P shows percentage.

According to Table.3, among the 200 participants including smoker or non-smoker, 12 people (%6) had post-operation infection. Of the group, 8 person (%14) were smoker and 4 person (%2) were non-smoker. There was no significant difference between the groups, in terms of Fisher Accuracy Test (P=0.373). From the infected people (smoker group), one case was in 26-30 age range and others were in 31-35. For the other group (non-smoker) all four infected cases were in 31-35 age range.

#### Table 3: Infection distribution of smoker and non-smoker groups

		Infe	Total			
Groups	Yes					No
-	F	Р	F	Р	F	Р
Smoker	8	4	92	46	100	50
Non- smoker	4	2	96	48	100	50
Total					20	100

Note: F shows Frequency and P shows percentage.

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The mean time of operation durance was 14.68 ( $\pm$ ) 3.29 minutes and 14.62 ( $\pm$ ) 4.39 for smoker and non-smoker respectively as shown in Table.4. (P=0.911).

Groups	Mean	SD	Maximum	Minimum	Frequency
Smoker	68.14	29.3	20	5	100
Non-smoker	62.14	39.4	19	5	100

Table 4: Mean and SD of operation time of smoker and non-smoker groups

#### DISCUSSION AND CONCLUSION

Smoking has systemic effect on heart, central nervous system, and endocrine glands, reduces the capacity of lungs, and more condensation in environmental vessels [15]. Moreover, improvement of wounds in smokers is slower than non-ones [14]. Also, Leucoplasia, an illness which has relationship with candidasis infection is more common among smokers [18]. Cigarette can be an important factor in suffering from mouth candidasis and predisposes situation for accumulating infectious factors at surgical position [25]. There are several pathogens which make periodontal diseases among smokers, among them can be referred to Aggregate bacteractino mycetencomitans 'Prevotella intermedia 'Porphyromonasgingivalis 'Bacteroidesforsythus andFusobacterium nucleatum [16].

According to the achieved results, 8 patients in smoker group had post-operation infectious; however, the frequency for other group was only 4. Although, the infection was twice among smoker group, but the difference was not significant. There are several intervening factor to make infectious after operation, so cigarette cannot counted as the only factor in this regard. Smoking just has a role in delaying of healing and perfusion to the position [10-22]. Thus, if the person has suitable health situation and no trauma was existed, cigarette cannot make infection.

In the U.S.A, half of people with periodontal diseases are smokers. Also, according to the achieved results, smokers are high-risk for the periodontal diseases four times more than non-smokers. The mechanism of relationship between smoking and periodontal diseases has been explained in different studies; however, the mechanism of relationship between cigarette and pathogeneses which are existed in periodontal infections has not been completely known yet [16]. Lung and colleagues found out that smoking makes a suitable situation for developing periodontal pathogeneses which in effect leads to increasing of the illnesses [13]. Shine et al. compared 180 smokers with the same group of non-smokers and reported a partial significant relationship and explained that cigarette affects on salvia and mouth's microbial flora which the effects need more examination [3]. Carriches and colleagues assessed effects of low third molar tooth's operation among smokers. Of the group two cases were associated with infection. Although, no difference between the groups in terms of pain; but, trismus was more among the smokers; however, no effect was observed in terms of situation and appearance of the wound due to smoking [7]. Ra'ed and colleagues also assessed pain, inflammation and trismus after latent third molar tooth between two smoker and non-smoker groups. Their results showed no relationship between severity of pain, inflammation and trismus from one side and smoking on the other side [22].

As mouth health has drastic effect on the wound's healing, it was trained to all smokers and nonsmokers patients. However, Andrews and colleagues' study showed that smokers have poor mouth health than non-smokers [2]. Campbell et al. Found positive effects among %60 of patients who were suggested to stop smoking, they also had better mouth health [6]. Thus, although, smoking can postponing wound intention as well as perfusion [14, 19]; but, mouth and dental health of the group should be counted.

Among the 8 patients with infection in the smoker group, one case was in 26-30 and 7 were in 31-35 age ranges. Also, 4 patients of non-smoker group with infection were in 31-35 age range.

Regardless of the groups, the findings showed more infection in higher age ranges and, as the smokers were in higher age ranges, it can be said age in addition to smoking are effective factors in increasing infection of patients after operation.



Kaye and colleagues showed a significant relationship between age and infection at surgical place after operation [11]. Delgado-Rodriguez and colleagues [8], in an examination of risk factors of post operation to infection, introduced age as a risk factor. Buren et al. also showed post- operation infection is increased with age. Thus, patients with more than 50 should stay at hospitals for longer time after an operation than younger [5].

Hence, according to the achieved results, cigarette is an effective factor in immunization reduction due making infection as well as a predisposition for post-operation infection; however, this is not an effective factor in making post- operation infection of mandible's latent wisdom tooth. But, it is suggested to smokers to stop smoking for a definite time to prevent from the probable effects.

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# REFERENCES

- [1] Adeyemo W, Ogunlewe M, Ladeinde A, Abib G, Gbotolorun O, Olojede O, et al. Afr J Med MedSci 2006; 35(4):479-83.
- [2] Andrews JA, Severson HH, Lichtenstein E. JADA 1998; 129:313-20.
- [3] Babanov S. The epidemiological characteristics of tobacco smoking. Vestnik Rossiiskoiakademiimeditsinskikhnauk/Rossiiskaiaakademiiameditsinskikhnauk. 2005:(8);27-9.
- [4] Bello SA, Adeyemo WL, Bamgbose BO, Obi EV, Adeyinka AA. Head Face Med 2011;7(8).
- [5] Byrne D, Lynch W, Napier A, Davey P, Malek M, Cuschieri A. J Hosp Infect 1994; 26(1):37-43.
- [6] Campbell H, Sletten M, Petty T. J Am Dent Assoc 1999;130(2):219-26.
- [7] Carriches CL, Font RG, Martínez-González JM, Rodríguez MD. Med Oral Patol Oral Cir Bucal 2006;11:E56-60.
- [8] Delgado-Rodríguez M, Gómez-Ortega A, Sillero-Arenas M, Llorca J. Infect Control Hosp Epidemiol 2001;22(1):24-30.
- [9] Haenle MM, Brockmann SO, Kron M, Bertling U, Mason RA, Steinbach G, et al. BMC Public Health 2006; 6(1):233.
- [10] Heng CK, Badner VM, Clemens DL, Mercer LT, Mercer DW. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104(6):757-62.
- [11] Kaye KS, Schmit K, Pieper C, Sloane R, Caughlan KF, Sexton DJ, et al. J Infect Disease 2005;191(7):1056-62.
- [12] Krogh P, Hald B, Holmstrup P. Carcinogenesis 1987;8(10):1543-8.
- [13] Lung Z, Kelleher M, Porter R, Gonzalez J, Lung R. Br Dent J 2005;199(11):731-7.
- [14] Meechan J, Macgregor I, Rogers S, Hobson R, Bate J, Dennison M. Br J Oral Maxillofac Surg 1988;26(5):402-9.
- [15] Mettes T, Nienhuijs M, Van der Sanden W, Verdonschot E, Plasschaert A. Cochrane Database Syst Rev 2005; 2.
- [16] Neto C, Batista J, Rosa EF, Pannuti CM, Romito GA. Smoking and periodontal tissues: a review. Braz Oral Res 2012;26(SPE1):25-31.
- [17] Ojima M, Hanioka T, Shimada K, Haresaku S, Yamamoto M, Tanaka K. Toba Induc Dis 2013;11(1):13.
- [18] Oliver A, Helfrick JF, Gard D. J oral maxillofac Surg 1996;54(8):949-54.
- [19] Pabst MJ, Pabst KM, Collier JA, Coleman TC, Lemons-Prince ML, Godat MS, et al. J Periodontol 1995;66(12):1047-55.
- [20] Peterson LJ, Ellis E, Hupp JR, Tucker MR. Contemporary oral and maxillofacial surgery. Mosby St Louis Mo 2008; 170-1.
- [21] Pitekova L, Satko I, Novotnakova D. Bratisl Med J 2010;111: 296-8.
- [22] Ra'ed M. J ClinExp Dent 2013;5(3): 117.
- [23] Ruta D, Bissias E, Ogston S, Ogden G. Br J Oral Maxillofac Surg 2000;38(5):480-7.
- [24] Silverstein P. American J Med 1992;93(1):S22-S4.
- [25] Soysa N, Ellepola A. Oral Dis 2005;11(5):268-73.
- [26] Warnakulasuriya S, Sutherland G, Scully C. Oral Oncol 2005; 41(3):244-60.

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