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Association Between Smoking and Oral Lichen Planus in Males - A Retrospective study

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Abstract: Oral lichen planus affects one to two percent of the general adult population and is the most common non-infectious oral mucosal disease. Tobacco smoking increases the risk of OLP malignant transformation as cigarette smoke contains substances that induce chronic inflammation at mucosal surfaces. The aim of this study was to assess the association between smoking and oral lichen planus in males. A retrospective study was conducted using the case records of patients visiting a private dental college in Chennai from June 2019 - March 2020. The study population included case records of male patients with oral lichen planus, selected by non-probability purposive sampling. Data regarding their smoking habit were collected. Descriptive and inferential statistics were done using SPSS software. Among the study population, 43.59% of patients were below 40 years and 56.41% were above 40 years of age. About 53.8% of male patients with oral lichen planus had a smoking habit. Erosive lichen planus was the most common variant followed by the reticular type.Within the limits of the study, there was a significant association between smoking and oral lichen planus in males, with an increased incidence of erosive lichen planus among smokers.

Keywords: Innovation; Oral lesions; Oral lichen planus; Periodontitis; Smoking; Tobacco

INTRODUCTION

Oral lichen planus (OLP) is a T-cell-mediated chronic inflammatory oral mucosal disease of unknown etiology. OLP presents as white striations, white papules, white plaques, erythema, erosions, or blisters affecting predominantly the buccal mucosa, tongue and gingiva (Axéll, 1976).

Oral lichen planus affects one to two percent of the general adult population and is the most common non-infectious oral mucosal disease(Silverman et al., 1991). OLP affects women more than men at a ratio of approximately 1.4:1 and occurs predominantly in adults over 40, although younger adults and children may be affected(Pindborg et al., 1968). There may be co-incident skin lesions that present typically as flat-topped violaceous papules affecting the wrists, ankles and genitalia. Nail involvement results in pitting, pterygium formation and permanent nail loss. Scalp involvement results in scarring alopecia(Markopoulos et al., 1997).

The oral lesions in lichen planus were described as white lines and white spots on the buccal mucosa and symmetric plaques on the sides of the tongue in several cases(Scully and Carrozzo, 2008). A clear and detailed description of the peculiar striae and dots found on the surface of a lichen planus papule was given by Louis Frederic Wickham in 1895 which are referred to as "Wickham‟s Striae"(Liu et al., 2010).

In the oral cavity, the buccal mucosa, tongue and gingiva are commonly affected by oral lichen planus. It presents as a symmetrical and bilateral lesion or multiple lesions(Dietrich, Reichart and Scheifele, 2004). It occurs in six clinical variants as reticular, papular, plaque-like, erosive, atrophic and bullous(Thongprasom et al., 2003). It is considered as a premalignant condition with malignant transformation rate of 0–2%(McCreary and McCartan, 1999).

Microscopically the disease is characterized by dense subepithelial lymphohistiocytic infiltrate, increased numbers of intra-epithelial lymphocytes and degeneration of basal keratinocytes. Epithelial basement membrane changes are common in OLP and include breaks, branches and duplications. Degeneration of the basement membrane causes weaknesses at the epithelial-connective tissue interface which may result in histological cleft formation (Max-Joseph space) and, rarely, clinical blistering of the oral mucosa (bullous lichen planus). Parakeratosis, acanthosis and 'saw-tooth' rete peg formation may be seen(Mollaoglu, 2000).

The etiology and pathogenesis of OLP is not completely understood. Several factors have been proposed for the etiology including genetic background, dental materials, drugs, infectious agents – bacterial and viral infections, autoimmunity – associated with other autoimmune diseases, immunodeficiency, food allergies, stress, habits, trauma, diabetes and hypertension, malignant neoplasms and bowel disease(Carrozzo and Gandolfo, 2008).
Lichen planus is believed to result from an abnormal T-cell-mediated immune response in which basal epithelial cells are recognized as foreign because of changes in the antigenicity of their cell surface. The cause of this immune-mediated basal cell damage is unknown. Likewise, it is unknown if lichen planus represents a single disease process or several closely related entities with similar clinical presentations(Rodríguez-Núñez et al., 2001).
Smoking habits are a serious risk factor for many diseases. The determination of smoking as a harmful habit among people is very important(Locker and Slade, 1994). Tobacco smoking is linked with many serious illnesses, such as cancer, cardiopulmonary diseases, low birthweight, as well as with many health problems. It is also linked to a detrimental impact on oral health, such as increasing risk of periodontal (gum) diseases(Meraw, 1998). In addition, dental implant failure is more common among smokers than among non-smokers, and peri-implantitis among smokers is also more prevalent(High, 1989). Tobacco can be consumed through the mouth in a variety of forms, varied from smoking to smokeless tobacco chewing on itself or combined with betel nut. These may induce a variety of oral manifestations of diseases. These lesions most likely result from the many irritants, toxins, and carcinogens found in the smoke emitted from burning tobacco, but they may also arise from drying of the mucosa by the high intra-oral temperature, pH change, alteration in immune response, or altered resistance to fungal or viral infections. Other effects include halitosis, staining of teeth and composite restorations, decreased ability to taste and smell, and nicotinic stomatitis and keratosis(Şimşek et al., 2019). Most of these problems are reversible after cessation of tobacco use(Madani, Bhaduri and Dikshit, 2012). Smoking is also associated with potentially malignant disorders of the oral mucosa. Tobacco smoking increases the risk of OLP malignant transformation as cigarette smoke contains substances that induce chronic inflammation at mucosal surfaces(Brown et al., 1993). It is possible that the discomfort associated with symptomatic OLP may play a role in the smoking patient's decision to stop smoking(Agnihotri and Gaur, 2014).
Previously our team had conducted various studies on treatment modalities for periodontal diseases and periodontal procedures(Panda et al., 2014),(Thamaraiselvan et al., 2015),(Varghese et al., 2015),(Mootha et al., 2016),(Ramesh, Varghese and Jayakumar, 2016),(Ramesh, Ravi and Kaarthikeyan, 2017),(Ravi et al., 2017),(Ramamurthy, 2018),(Ramesh et al., 2019), studies correlating various diseases and factors related to periodontal diseases(Ramesh et al., 2016),(Khalid et al., 2016),(Priyanka et al., 2017) and in-vitro & radiological studies(Khalid et al., 2017),(Avinash, Malaippan and Dooraiswamy, 2017),(Kavarthapu and Thamaraiselvan, 2018) over the past five years. Now we are focussing on retrospective studies. And our department is passionate about research we have published numerous high quality articles in this domain over the past years ((Kavitha et al., 2014) , (Praveen et al., 2001),(Devi and Gnanavel, 2014), (Putchala et al., 2013), (Vijayakumar et al., 2010), (Lekha et al., 2014a, 2014b) (Danda, 2010) (Danda, 2010) (Parthasarathy et al., 2016) (Gopalakannan, Senthilvelan and Ranganathan, 2012), (Rajendran et al., 2019), (Govindaraju, Neelakantan and Gutmann, 2017), (P. Neelakantan et al., 2015), (PradeepKumar et al., 2016), (Sajan et al., 2011), (Lekha et al., 2014a), (Neelakantan, Grotra and Sharma, 2013), (Patil et al., 2017), (Jeevanandan and Govindaraju, 2018), (Abdul Wahab et al., 2017), (Eapen, Baig and Avinash, 2017), (Menon et al., 2018), (Wahab et al., 2018), (Vishnu Prasad et al., 2018), (Uthrakumar et al., 2010), (Ashok, Ajith and Sivanesan, 2017), (Prasanna Neelakantan et al., 2015).
The aim of the present study was to assess the association between smoking and oral lichen planus in males.

MATERIALS AND METHODS

Study design and setting

This retrospective study examined the records of patients who underwent treatment at a private dental college in Chennai from June 2019 - March 2020. Ethical approval was obtained from the Institutional Ethics Committee of the University (SDC/SIHEC/2020/DIASDATA/0619-03200. The study population included case records of male patients with oral lichen planus, selected by non-probability purposive sampling. Female patients, patients with smoking habits less than a year were excluded from the study.

Data collection

Case records of 86000 patients were reviewed and analysed for the presence of oral lichen planus. Relevant data such as patient age, history of smoking, clinical variants of lichen planus were recorded. Repeated patient records and incomplete records were excluded. The final dataset consisted of 39 male patients with oral lichen planus and their smoking habit was assessed. Data was verified by an external reviewer.

Statistical analysis

Data was recorded in Microsoft Excel 2016 (Microsoft office 10) and later exported to the Statistical Package for Social Science (SPSS IBM version 20.0) and subjected to statistical analysis. Descriptive statistics and chi square test were employed with a level of significance set at $p<0.05$.

RESULTS AND DISCUSSION

The data for this retrospective study was based on patients seeking treatment at a private dental college. Currently there are very few studies investigating the association between smoking and oral lichen planus in males among the South Indian population. The results of the present study show that the prevalence of oral lichen planus among male patients below 40 years of age was 43.6% and 56.4% in patients above 40 years of age [Figure 1].

About 53.8% of male patients with oral lichen planus had a smoking habit. [Figure 2].

The prevalence of various clinical variants of oral lichen planus were as follows: annular (2.6%), bullous (5.1%), erosive (46.2%), lichenoid reaction (15.4%), papular (2.6%), reticular (25.6%), ulcerative (2.6%) [Figure 3, Table 1].

There was a statistically significant association between smoking and the prevalence of oral lichen planus among males ($p<0.05$), with an increased incidence of erosive lichen planus among smokers [Figure 4, Table 2].

In this study the number of oral lichen planus is higher in the age group above forty. These results indicate the coincidence of our research with Pakfetrat et al, where oral lichen planus incidence was found mostly in patients above 40 years of age(Pakfetrat et al., 2009). Axel et al concluded that patients above 55 years had a higher prevalence of OLP when compared to other age groups(Axell and Rundquist, 1987).

Many clinical, epidemiological and biological studies have demonstrated that not only active smoking but also exposure to other people's cigarette smoke (secondhand smoking, also called involuntary smoking or environmental tobacco smoking) are associated with detrimental health effects such as asthma, lung cancer and cardiovascular diseases(Behera, Xian and Balasubramanian, 2018),(Öberg et al., 2011). Smoking is also one of the major risk factors for oral diseases such as periodontal disease and tooth loss, and many studies have found that active smoking is closely associated with the prevalence or severity of periodontal disease and fewer remaining teeth(Johnson and Hill, 2004),(Johannsen, Susin and Gustafsson, 2014). Further, increasing evidence shows that secondhand smoking may aggravate periodontal disease in non-smokers(Ojima et al., 2006). Smoking was a common finding among the patients in our study since 53.8% of the group were smokers which is similar to the results reported in other studies as well (Shen et al., 2012),(Lavanya et al., 2011),(Bermejo-Fenoll et al., 2009). These findings confirm that oral lichen planus have an increased prevalence among smokers compared to the general population(Eisen, 2002). In a study by Neumann-Jenson et al, it was found that there was a significant association between smoking and oral lichen planus(Neumann-Jensen, Holmstrup and Pindborg, 1977). This is also in agreement with a study by Klosek et al, which concluded that cigarette smoking habit has a direct impact on the oral lichen planus course(Kłosek et al., 2011). Most of the oral lichen planus lesions were more prevalent among smokers than non-smokers in a study by Murti et al(Murti et al., 1986)

Erosive type of Oral lichen planus was most common (46.2%), followed by reticular type (25.6%) , lichenoid reaction (15.4%) , bullous (51%), erythematous and annular (2.6%). This was similar to a study by Thorn et al, where the erosive type of OLP was the most prevalent followed by papular OLP(Thorn et al., 1988). In contrast to our study, Vice Budimir et al concluded that, where the reticular type was found to be the most common (64.8%) followed by erosive type (22.9%) in Croatian population(Budimir et al., 2014).

The association of tobacco smoking with OLP is not clearly understood. It is hypothesized that the heat and irritation of smoking may aggravate symptomatic OLP lesions, and the risk of malignant transformation associated with tobacco use may play a role in patients stopping tobacco use(Alrashdan, 2014). Smoking OLP patients showed significantly greater mean area percent values for TLR-2 immunoexpression in the epithelium compared with non-smoker OLP patients in a study by Amin et al(Amin, Yussif and Ahmed, 2020). This explained that tissues exposed to tobacco carcinogens responded by expressing elevated levels of cytokines as part of response to injury. Therefore, we could speculate that smoking resulted in enhanced cytokine release which led to activated TLR-2 inflammatory signaling(Shearston et al., 2019). It was concluded that smoking enhanced TLR-2 and CD34 expression in OLP which are considered as inflammatory mediators and are contributing factors in the pathogenesis of OLP(Guida et al., 2019). Smoking enhances angiogenesis in OLP as confirmed by enhanced CD34 immunoexpression in OLP patients. A significant increase in blood vessel density stained by CD34 was noted in smoking OLP patients compared to non-smoker patients(S, Anandan and Prasanthi, 2013). The results were related to the effect of smoking on enhancing the release of pro-inflammatory cytokines(Meij, van der Meij and van der Waal, 2003).

Since the etiology of oral lichen planus is unknown there's no etiological treatment of the disease(Nico, Fernandes and Lourenço, 2011). The aim of the treatment is to relieve the symptoms and minimise the functional impact of the disease(Sehgal et al., 2017).

Oral lichen planus is a chronic mucosal disease affecting middle aged men with smoking habits. The lesions are symptom free and oral lichen planus can transform into malignant forms(Siponen et al., 2012). Hence this study emphasises the need for regular monitoring of all patients. This study sheds additional light on the epidemiological and clinical features of oral lichen planus in patients from South India.

CONCLUSION

Within the limits of the study, there was a significant association between smoking and oral lichen planus in males, with an increased incidence of erosive lichen planus among smokers. Further studies on the influence of smoking behavior on the characteristics of symptomatic Oral lichen planus and on the risk of malignant transformation of OLP are needed for further understanding.

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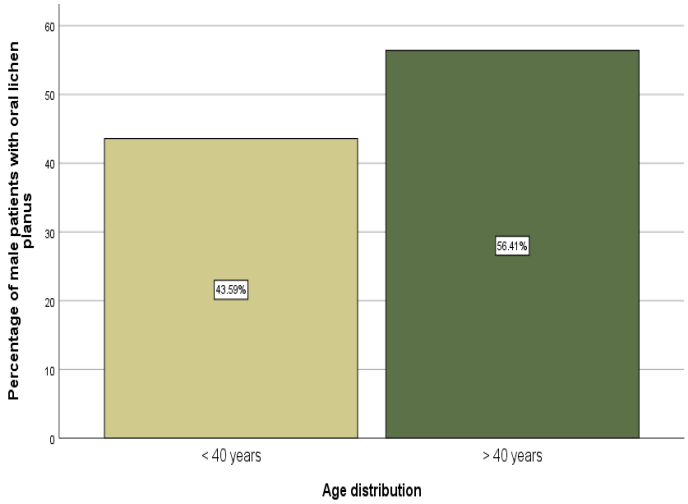

Fig.1: Bar graph representing the age distribution among male patients with oral lichen planus. X axis represents the age distribution and Y axis represents the percentage of male patients with oral lichen planus. Among the study population, 43.59% of patients were below 40 years and 56.41% were above 40 years of age.

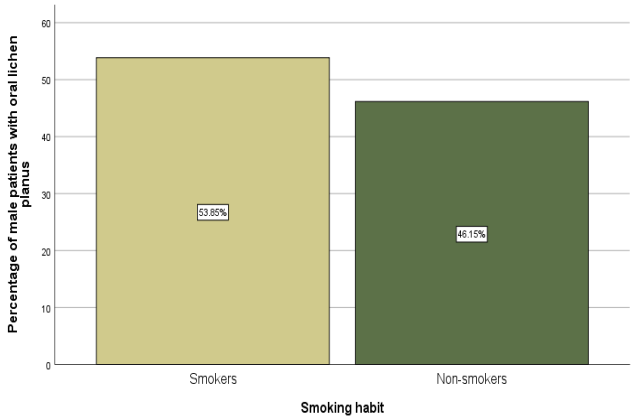

Fig.2: Bar graph representing the history of smoking among male patients with oral lichen planus. X axis represents the smoking habit and Y axis represents the percentage of male patients with oral lichen planus. About 53.8% of male patients with oral lichen planus had a smoking habit and 46.15% were non-smokers.

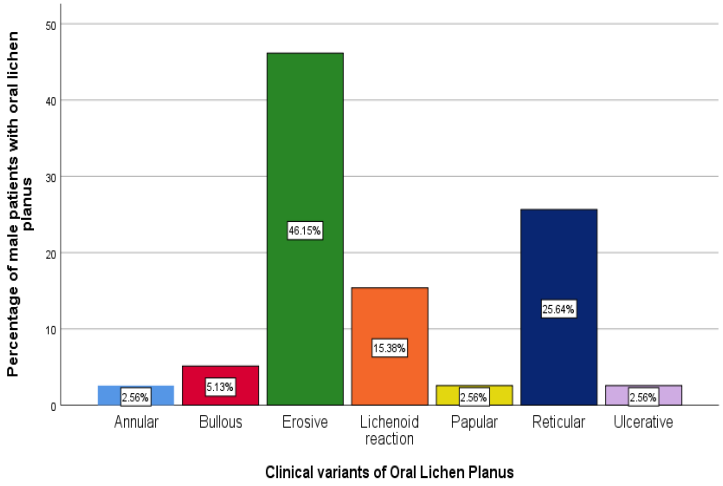

Fig.3: Bar graph representing the different clinical variants of oral lichen planus among male patients. X axis represents the history of smoking and Y axis represents the percentage of male patients with oral lichen planus. Erosive lichen planus was the most common variant followed by the reticular type.

Table 1: represents the frequency distribution of the different clinical variants of oral lichen planus among male patients.

Clinical variant of oral lichen planus	Frequency	Percentage
Annular	1	2.56%
Bullous	2	5.13%
Erosive	18	46.15%
Lichenoid reaction	6	15.38%
Papular	1	2.56%
Reticular	10	25.84%
Ulcerative	1	2.56%

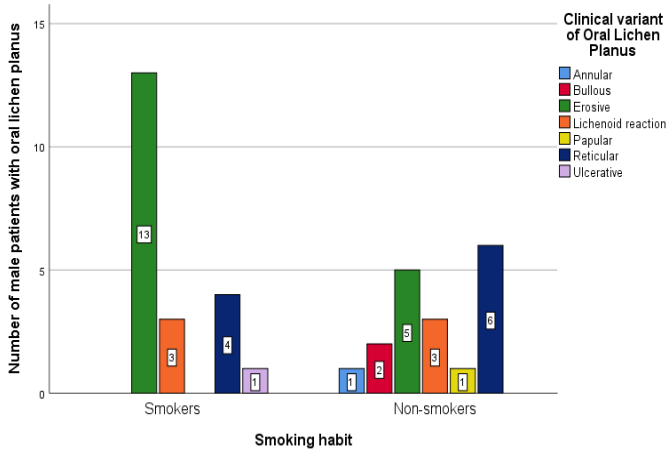

Fig.4 : Bar graph representing the association between smoking and oral lichen planus among male patients. X axis represents the smoking habit and Y axis represents the number of male patients with oral lichen planus. There was a higher prevalence of oral lichen planus, particularly

erosive type among smokers when compared to non-smokers. Chi square test showed that there was a statistically significant association. Pearson chi square value = 8.777; p-value = 0.007 (<0.05).

Table 2: represents the frequency distribution of the different clinical variants of lichen planus among smokers and non-smokers. Chi square test showed that there was a significant association between smoking and oral lichen planus (p<0.05).

	Clinical variants of oral lichen planus							Pearson Chi square value	p-value
Smoking habit	Annular	Bullous	Erosive	Lichenoid reaction	Papular	Reticular	Ulcerative		
Smokers	0	0	13	3	0	4	1	8.777	0.007 (<0.05)
Non-smokers	1	2	5	3	1	6	0		