International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor (2018): 7.426

Clinicopathological Study of Premalignant Lesions of Oral Cavity

Dr. G. Sanjay Sagar, Dr. Rajesh Kumar Padhy, Dr. Sudeepta Ashe

Abstract: ***Background: Oral cancer is one of the leading Cancer today. Significant number of these cases would present initially with a precursor lesions that are further classified as precancerous lesions. Early detection of disease course will reduce the morbidity and mortality. Aim of study is to evaluate clinical presentation and various histopathological types of premalignant lesions. Methods: This is descriptive, prospective study of 2 year duration. The detail clinical presentation, relevant investigation and histopathological study on biopsy tissue was done and data is analysed. Results: During study period,92 cases having premalignant oral lesions were observed. Among these, the most common age group affected was between 51-60 years. Male predominance was noted. Male to Female ratio was 4:1. Buccal mucosa was affected in 54.5% of cases, Tongue in 16.86% cases, Retromolar trigone in 14.6% cases. Gingiva and alveolus in 3.63% of cases each. Common etiological factor observed was tobacco chewing. On Histopathological study, Leucoplakia is seen in 59.78% of cases, Oral submucosal fibrosis in 19.56%, Erythroplakia in 10.56%, Oral lichen planus in 5.43% and Actinic cheilitis in 4.34%. Conclusion: In This study, Various premalignant lesions were observed. Clinical and histopathological evaluation was done. Oral Leucoplakia was the most common lesion in the present study. Buccal mucosa was the most common site in oral cavity. Tobacco chewing habit was the most common risk factor observed. Early detection of Premalignant lesion is of utmost importance to prevent further morbidity as these lesions further progress to Oral Cancer.***

Keywords: Premalignant lesions

1. Introduction

In India, oral cancer is one of the leading cancer today. Its incidence is 12.6 per 1,00,000 population.[1,2] The premalignant lesion is a disease or syndrome if left untreated have significantly increased risk to develop cancer.

However, in 2005 WHO workshop, it was decided to use the term “Potentially Malignant Disorders,” on it conveys that not all disorders defined under this term may transform into cancer.[3]

Local oral examination, application of toluidine blue to suspected lesion, cytological study, and tissue biopsy are used to investigate these cases. Oral lesion biopsy is usually indicated to rule out causes of white patches. It also helps to determine the detailed histologic examination to grade the presence of any epithelial dysplasia. The sites of a leukoplakia lesion that are preferentially biopsied are the areas that show in duration, redness, erosive or ulcerated areas. These areas are more likely to show any dysplasia than homogenous white areas.

2. Methods

This is descriptive, observational, analytical type of study of five-year duration from june 2016 to sept 2018. During study period patient from Surgical OPD in our hospital having oral lesions were included. The cases of oral lesions were clinically suspected was studied as per WHO criteria for potentially malignant diseases. The classification of oral leukoplakia was done according to size of oral lesion, its clinical presentation and histopathological features. The detail clinical history was taken. The location of oral lesion, size, gross examination etc. were done. All relevant investigations were done. Biopsy were taken, detailed histopathological features were noted. The tissue sections were stained with H and E stain. The statistical data was analysed by using SPSS-10 version. The results were analysed.

3. Results

According to the data collected, 173 patients having oral lesions visited the diagnostic centre of our institute were included in the study. Among these 92 (53.17%) cases were of premalignant lesion and 81 (46.82%) were of oral cancers. The premalignant lesions were distributed as per age and sex of patients (Table 1), which showed most common age was in between 50-59 years. The mean age was 54.5 year. The earlier age in the present study was detected in a boy of 15 years who was addicted to tobacco and gutka use. The male:female ratio was 4:1. The various premalignant lesions (Table 2) of 92 cases, on histopathological features were leucoplakia 55 cases (59.78%), oral lichen planus 5 cases (5.43%) , oral submucous fibrosis 18 cases (19.56%), actinic cheilitis (squamous dysplasia related with immunodeficiency or oral hygiene) 4 case (4.34 %) and erythroplakia 10 case(10.56%). The leucoplakia was the commonest lesion (55 cases) 59.78% was observed in the present study. As for risk factors, patients reported being using tobacco, smokers, drinking alcohol, working under direct sunlight etc. were studied. The tobacco chewing was most common habit noted in our cases. The various habit of cases was given in (Table 3).

Table 1: Age and sex distribution of the oral pre-malignant cases

AGE GROUPS	MALE	FEMALE	TOTAL	Point E
10 -19	2	0	2	2.17%
20-29	17	1	18	19.5%
30-39	12	2	14	15.21%
40-49	11	7	18	17.30%
50-59	25	4	29	31%
60-69	3	2	5	5.40%
>70	4	2	6	6.50%
TOTAL	74	18	92	100%

Table 2: Histopathological patterns of pre-malignant lesions

TYPE OF LESION	NO. OF CASES	PERCENTAGE%
LEUKOPLAKIA	55	59.78%
ORAL SUBMUCOSAL FIBROSIS	18	19.56%
ERYTHROPLAKIA	10	10.56%
ORAL LICHENPLANUS	05	5.43%
ACTINIC CHELITIS	04	4.34%

Table 3: Tobacco habits in oral pre-malignant cases

ADDICTION TO	MALE	FEMALE	TOTAL	PERCENTAGE
BETEL	43	27	70	76.08%
TOBACCO CHEWING	58	11	69	75%
SMOKING + TOBACCO CHEWING	21	0	21	22.82%
ALOCHOL + TOBACCO CHEWING	17	0	17	18.47%
NO ADDICTION	3	1	4	4.34%

Table 4: Site wise distribution of oral pre-malignant cases

SITE OF LESION	NO OF CASES	PERCENTAGE
BUCCAL MUCOSA	50	54.26%
TONGUE	9	16.36%
GINGIVA	2	3.63%
ALVEOLUS	2	3.63%
LIP	1	1.81%
RETROMOLAR TRIGONE	8	14.54%
FLOOR OF MOUTH	1	1.82%
PALATE	2	3.63%
Total	**92**	**100%**

The site wise distribution of oral premalignant lesions was shown in Table 4. The most common site was buccal mucosa 55.26%, followed by tongue, gingival, alveolus and lip.

Various grades of squamous epithelial dysplasia were done as per the WHO criteria. Grades of epithelial dysplasia observed in the present study were given in Table 5. The most common grade of squamous epithelial dysplasia was mild and noted in 44 cases (48.48%), moderate in 14 cases (15.15%) and severe in 34 cases (36.36%). All oral dysplasias were followed up regularly, even if the lesion has been completely excised. Also, whether the patient has stopped using tobacco products were observed.

Table 5: Grades of epithelial dysplasia observed in pre-malignant lesions

Grades of epithelial dysplasia	No. of cases	percentage
Mild	44	48.48%
Moderate	14	15.15%
Severe	34	36.36%
Total	92	100%

4. Discussion

The oral premalignant disorders consist of a group of diseases were named in literature as precancer, intraepithelial neoplasia, premalignant lesions etc. To avoid confusion WHO in 2005 termed various lesions and conditions of the oral mucosa which may undergo malignant transformation are defined as “Potentially malignant disorders” (PMD). There are 5 most common lesions described as PMD are, leucoplakia, erythroplakia, oral submucous fibrosis, actinic cheilitis and oral lichen planus. Along with these other less common conditions like inherited diseases, xeroderma pigmentosa, immunodeficiency, Fanconi’s anemia, chronic discoid lupus erythematosus have been described.[4]

In the present study the age distribution of oral premalignant cases was maximum in age group of 50-59 year (31%) with male predominance (male:female 4:1). The mean age of presentation was 54.5 year. The study by Mehrotra R et al, in 2006 showed maximum number of cases were in 6th decade.[5] The male predominance was noted by Dietrich T, et al.[6] Present study showed male predominance with 80.78% while in female 19.21%.

Leucoplakia defined by the WHO working group as keratotic white patch or plaque that cannot be scrubbed off and cannot be characterized clinically or pathologically as any other disease. The leucoplakia remains the most common premalignant lesion having prevalence of 2.6% globally.7 The striking variation is noted in different areas. In India, its prevalence is maximum in Gujarat (11.7%) while 0.2% in Bihar.8 The various etiological factors implicated are tobacco, alcohol, chronic irritation, human papilloma virus infection, ultraviolet radiation, hot spicy foods etc.[6-9] It has the strongest association with the use of tobacco in various forms like chewing tobacco (as in paan, paan masala, gutka, zarda), heavy smokers etc.10 There is risk factor leads to hyperplastic or dysplastic squamous epithelial lesions which progress to carcinoma in situ to invasive squamous cell carcinoma.

On clinical examination, various types of leucoplakia were described as homogenous and non-homogenous. They appear as flat, thin, nodular, proliferative verrucose types. Lesions are mostly unifocal but can be multifocal. These lesions can be found in any part of oral mucosa with most frequent site is buccal mucosa.[9,12] In the present study buccal mucosa was common site constituting 54.26% followed by tongue (16.31%), gingiva (3.63%), alveolus, lip etc. Various differential diagnosis for of oral lesions include chemical injury, oral hairy leucoplakia, leucoderma, smoker’s palate, papillomas, lichenoid reaction, frictional lesions etc.13 In leucoplakia, the rate of malignant transformation ranges from 0.13% to 2.2% per year.[7,14]

Oral lichen planus (OLP)

It is a chronic, autoimmune, inflammatory disease. Several etiological factors are responsible for it which includes genetic, drugs, infectious agents (mostly viral), dental material, autoimmune, stress, diabetes, immunodeficiency

etc.[15] It affects skin, oral mucosa, genitals, nails, scalp etc. Any part of oral mucosa may be affected but dorsum of tongue is most common site. Patient presents with symmetrical, bilateral lesion. Surface is white with fine white striations, "Wickham's Striae". Various forms like reticular, plaque, atrophic, papular, erosive, bullous were noted.[16] For confirmatory diagnosis biopsy is required. On histopathology shows atrophy of surface epithelium, liquefactive degeneration of basal layer, subepithelial band of lymphocytic infiltration. Malignant transformation is usually noted in <1% of cases.[17] In the present study 3 cases showed plaque lesion and 2 cases show reticular lesion. In the present study, no change of malignant transformation was noted.

Oral submucous fibrosis (OSF)

It is a chronic disorder characterized by fibrosis of the mucosa of the upper GIT. OSF is more commonly seen in south east Asia especially India where people having habit of Areca nut chewing with betal quid.[18] Other etiological agents like ingestion of chilies, genetic, immune, nutritional deficiency have been noted. Clinical manifestation is related to fibrosis of mucosa of cheek giving rise to trismus, difficulty in eating, burning sensation etc. Histological features of OSF are mucosal atrophy, pigmentation, granulation, fibrosis, chronic inflammation, dysplasia and carcinoma.In India 0.2-1.2% of population is affected by OSF,with annual malignant transformation is about 0.5-7.6%.[19-20] In the present study one patient had severe problem in mouth opening (<10mm) having extensive faucial, buccal and labial fibrotic bands. He was addicted to use gutka and tobacco chewing.

In the present study 4 cases related to actinic cheilitis having diffuse thickening of epithelium of lip with squamous hyperplasia, mild to moderate dysplasia, thickening of keratin layer. The etiology observed was related to ill-fitting denture, tobacco use, poor oral hygiene and exposure to solar radiation. It has been reported that malignant transformation has been reported in 1.4-36% of cases over 1 to 3 decades of period.[21]

We have 10 cases of oral erythroplakia. Erythroplakia is red or erythematous patch of oral mucosa and is associated with higher rate to develop dysplasia, carcinoma in situ and invasive carcinoma than leucoplakia.[22]

Diagnosis of premalignant status of oral lesions include clinical examination, topical application of toluidine blue on suspicious area and confirmation is done by biopsy of lesion. Evaluation of every lesion in oral cavity is essential. It is important to re-access on each follow up visit. First order of treatment must include stabilization of the mouth: treat for dryness, and fungal infection. Insist on the importance of good oral hygiene, and good nutrition. The various treatment modalities include removal of etiological agent, observation only, chemo-prevention, medical treatment, CO2 laser surgery, surgical excision etc.

5. Conclusion

Significant number of oral cancer cases present initially with precursor lesion. We observed various premalignant oral lesions in the present study which include leukoplakia, oral submucous fibrosis, lichen planus, actinic cheilitis and erythroplakia. Oral leukoplakia was most common lesion in the present study. Buccal mucosa was the most common site. Tobacco chewing habit was common risk factor observed. Early detection of premalignant oral lesion is of utmost importance to prevent further progress to oral cancer.

References

[1] Elango JK, Gangadharan P, Sumithra S, Kuriakose MA. Trends of head and neck cancers in urban and rural India. Asian Pacific J Cancer Prevention. 2006;7(1):108-12.
[2] Sankaranarayanan R, Ramadas K, Thomas G, Muwonge R, Thara S, Mathew B, et al. Effect of screening on oral cancer mortality in Kerala, India: a cluster randomized controlled trial. Lancet. 2005;365(9475):1927-33.
[3] Warnakulasuriya S, Johnson NW, Waal VDI. Nomenclature and classification of potentially malignant disorders of the oral mucosa. J Oral Pathol Med. 2007;36:575-80.
[4] Liu W, Shen ZY, Wang LJ, Hu YH, Shen XM, Zhou ZT, et al. Malignant potential of oral and labial chronic discoid lupus erythematosus: a clinicopathological study of 87 cases. Histopathol. 2011;59:292-8.
[5] Mehrotra R, Gupta A, Singh M, Ibrahim R. Application of cytology and molecular biology in diagnosing premalignant and malignant oral lesions. Molecular Cancer. 2006;5(11):476-98.
[6] Dietrich T, Reichart PA, Scheifelea C. Clinical risk factors of oral leucoplakia in a representative sample of the US population. Oral Oncol. 2004;40:158-63.
[7] Mishra M, Mohanty J, SenguptaS, Tripathy S. Epidemiological and clinicopathological study of oral leucoplakia. Ind J Dermatol Venerol Leprosy. 2005;71(3):161-5.
[8] Sharma P, Saxena S, Aggarwal P. Trends in the epidemiology of oral squamous cell carcinoma in western UP: an institutional study. Ind J Dent Res. 2010;21(3):316-9.
[9] Mehta FS, Pindborg JJ, Gupta PC, Daftary DK. Epidemiologic and histologic study of oral cancer and leucoplakia among 50,915 villagers in India. Cancer. 1969;24:832-49.
[10] Khandekar SP, Bagdey PS, Tiwari RR. Oral cancer and some epidemiological factors: A hospital based study. Indian J Community Med. 2006;31:157-9.
[11] Fisher MA, Bouquot JE, Shelton BJ. Assessment of risk factors for oral leucoplakia in West Virginia. Community Dent Oral Epidemiol. 2005;33(1):45-52.
[12] Axell T. Occurrence of leukoplakia and some other oral white lesions among 20,333 adult Swedish people. Community Dent Oral Epidemiol. 1987;15:46-51.

[13] Terézhalmy GT, Huber MA, Jones AC, Sankar V, Noujeim M. Physical Evaluation in dental practice, Ed: 1st. Ames, Iowa: Wiley-Blackwell; 2009:170-171.
[14] Kawabe R, Fujita K. Survival analysis: application to evaluation of risk factors for cancer development in oral leucoplakia. Japanese J Oral Mucosa. 2001;7:45-58.
[15] Scully C, Carrozzo M. Oral mucosal disease: Lichen planus. Br J Oral Maxillofac Surg. 2008;46:15
[16] Farhi D, Dupin N. Pathophysiology, etiologic factors, and clinical management of oral lichen planus, part I: facts and controversies. Clin Dermatol. 2010;28:100-8.
[17] van der Meij EH, Mast H, van der Waal I. The possible premalignant character of oral lichen planus and oral lichenoid lesions: a prospective five-year follow-up study of 192 patients. Oral Oncol. 2007;43:742-8.
[18] Tilakaratne WM, Klinikowski MF, Saku T, Peters TJ, Warnakulasuriya S. Oral submucus fibrosis: review on aetiology and pathogenesis. Oral Oncol. 2006;42:561-8.
[19] Pindborg JJ, Mehta FS, Gupta PC, Daftary DK. Prevalence of oral submucus fibrosis among 50,915 Indian Villagers. Br J Cancer. 1968;22:646-54.
[20] Murti PR, Bhonsle RB, Pindborg JJ, Daftary DK, Gupta PC, Mehta FS. Malignant transformation rate in oral submucous fibrosis over a 17-year period. Community Dent Oral Epidemiol. 1985;13:340
[21] Lee JJ, Hong WK, Hittelman WN, Mao L, Lotan R, Shin DM, et al. Predicting cancer development in oral leukoplakia: ten years of translational research. Clin Cancer Res. 2000;6:1702-10.
[22] Reichart PA, Philipsen HP. Oral erythroplakia: a review. Oral Oncol. 2005;41:551-61