Original Article

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Periodontal Infection Modifying Radiation Mucositis in Patients Receiving Radiation Therapy

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Abstract

Background: The severity of mucositis varies and depends on factors such as the dose of irradiation, anatomic site of irradiation, and other factors which are attributed as the patient sensitivity to the therapy. Investigators are in constant pursuit of exploring regarding the factors responsible for onset, variability of distribution and severity of mucositis in the oral cavity during irradiation. This variable distribution of mucositic lesions led investigators to evaluate other contributing factors for the initiation and aggravation of radiation-induced mucositis.

Materials and Methods: The present clinical study evaluates the correlation of the influence of periodontitis in modifying mucositis during radiation therapy of oropharyngeal carcinoma patients. The aim of the study was to evaluate the definitive influence of periodontal status in modifying the progress of mucositis during radiation therapy and also to compare the efficacy of povidone iodine (5% w/v) and chlorohexidine gluconate 0.2% for controlling radiation-induced mucositis in patients with oropharyngeal cancer.

Results: In the 2nd, 3rd, 4th, and 5th weeks, the values of Pearson co-efficient of co-relation were 0.62 and 0.73, 0.31, and 0.07, respectively, indicating a positive co-relation between periodontal index and mucositis index.

Conclusions: The periodontal disease is one, among the various contributing factors responsible for the initiation and degree of severity of radiation mucositis during treatment of oropharyngeal cancer.

Key words: Irradiation, Mucositis, Oropharyngeal carcinoma, Periodontal status, Radiation

INTRODUCTION

Radiation mucositis is defined as an inflammatory like the process of the oropharyngeal mucosa following therapeutic irradiation of patients who have head and neck cancer. The patients undergoing radiation treatment for malignant neoplasms of the oral cavity suffer a great deal of discomfort in speech, mastication, deglutition, and salivation because of these lesions.[1] Oral complications are painful, diminish the quality of life and may lead to significant compliance problems, often discouraging the patients from continuing treatment.[2] One contributing factor to the development of fatal infection is also described to be radiation-induced mucositis.[3] Previous studies have observed that it is not possible to account for the onset, variability of distribution and severity of mucositis in the oral cavity during irradiation.[4] This variable distribution of mucositic lesions from the data obtained in the studies of various authors led investigators to evaluate other contributing factors to the initiation and aggravation of radiation-induced mucositis. Oral flora is thought to contribute to irradiation mucositis.[5] Moreover, negligence of oral hygiene may also contribute to mucositis. Even if oral hygiene measures are being instituted in the presence of oral diseases, there are greater chances of increase in the number and imbalance of the oral flora.[5] In previous literatures, a positive correlation between the presence of mucositis and radiation therapy

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is present but there is a lack of correlation between the various factors inducing and aggravating it. Here, in this study, it has been attempted to enquire whether any relationship exists between pre-existing periodontal disease and initiation or aggravation of mucositic lesions during radiation therapy, where the oral cavity is not free from infection and inflammation. Keeping all the above factors in mind, present clinical study was designed with the following aims and objectives:

Aims and Objectives

1. To establish and correlate the definitive influence of periodontal status in modifying the progress of mucositis in patients receiving radiation therapy of head and neck region
2. To correlate the distribution of initial lesions of mucositis to different causative factors
3. To compare the effects of different antimicrobial agents for the elimination of pathogenic microbial flora to control mucositis during radiation therapy
4. To evaluate the need of periodontal treatment in the pre-treatment program before radiation therapy.

MATERIALS AND METHODS

The sample for the present study comprised of 80 patients of oropharyngeal carcinoma selected for irradiation, attending AHRCC, Cuttack. The patients were selected irrespective of age, sex, and stage of the disease. The sample size was selected after consulting the statistician.

Inclusion criteria of these patients were as follows:

i. Oropharyngeal carcinoma patient selected for radiation therapy
ii. All patients were treated by Co-60
iii. All patients received the same provisional total dose of radiation; i.e., 60Gy
iv. All patients were treated by same fractionation daily dose of 200 cGy, for 5 days a week
v. All patients were having a provisional overall time of 6 weeks radiation protocol treatment
vi. None of the patients was completely edentulous
vii. Irradiation portals include teeth, oral mucosa, and salivary glands.

The treatment outlined by the oncology team was based on the type of radiation treatment to be instituted, differing from each other according to different stages of the disease.

The neoplasm included, were from all the stages.
Stage I: $T_1 N_0 M_0$,
Stage II: $T_2 N_0 M_0$,
Stage III: $T_3 N_0 M_0$, $T_{1\text{-}2\text{-}3}/N_1/M_0$,
Stage IV: $T_4 N_0 M_0$, $T_{any} N_{any} M_{any}$

All patients received a thorough dental examination before instituting radiotherapy. All patients were given oral prophylaxis. Extraction of mobile cariously exposed and grossly decayed teeth were done 1 week prior to instituting radiotherapy.

During radiation, only fluoride gel therapy was instituted for all.

Artificial saliva (carboxy methyl cellulose preparations) was given to those patients who complained about the dryness of the mouth.

The patients were then divided into two groups:

1. Study group: Included those patients, who were suffering from periodontitis
2. Control group: Included those patients, who were not suffering from any kind of periodontal diseases.

Study Group

Only the hopeless, mobile, grossly decayed, and cariously exposed teeth were extracted. All patients were given oral prophylaxis.

No attention was given to observe the resolution of periodontal inflammation before irradiation. Smoothening of sharp cusps was also done in some patients, whenever required. During radiation therapy, partially edentulous patients were advised not to wear their removable prosthesis.

This group was again sub-divided into two subgroups:

- S_1: Using chlorohexidine gluconate 0.2%.
- S_2: using povidone iodine 5% w/v mouth rinse.

Each subgroup consisted of 20 patients.

Control Group

Oral prophylaxis and root planning were done for these patients.

It was strictly observed that periodontally involved teeth were present in the oral cavity and that periodontal inflammation was completely resolved before initiation of radiation therapy.

Furthermore, it was strictly observed that no carious exposed or periapically diseased teeth existed in the oral cavity.

Orthopantomograph was taken for all these patients. It was observed that all teeth present were apparently free

from inflammation and depth of their gingival sulci was not more than 2 mm.

Other Measures

1. Patients using removable partial denture were not allowed to wear the denture.
2. Smoothening of sharp restoration and cusps was done.

Variables used are: The scores of mucositis index, Russell's periodontal Index, which were recorded at weekly interval.[6,7]

RESULTS

Mucositis Index

Table 1 shows the profile baseline characteristics of the patients. The average mucositis index scores for both control and study groups are illustrated in Table 2. This Table 2 shows the average mucositis of all the groups at each week interval. Table 3 elucidates the average mucositis index values of study Subgroup 1 and control group along with their standard deviations for comparison of mucositis index in both the groups. The difference show, however, was statistically significant at 5% level of significance $P < 0.05$, indicating that the degree of mucositis was greater in the study Subgroup 1, despite the use of chlorohexidinegluconate mouth rinse. Table 4 shows average mucositis index of study Subgroup 2 and control group along with their standard deviation and the difference between these two groups. Variables were assessed by Student's *t*-test. Here also the difference was significant.

Table 5 compared mucositis index between study Subgroups 1 and 2, and it was found to be not significant.

Correlation between Mucositis Index and Periodontal Index

Table 6 showed the average value of periodontal index and mucositis index of study Subgroup 1. Table 7 showed the average values at each week interval, the standard deviation along with the Pearson co-efficient of co-relation. During the1st week, mucositis index was found to be 0. Thus it could not be correlated with the periodontal index. In the 2nd and 3rd weeks, the values of Pearson co-efficient of co-relation were 0.62 and 0.73, respectively, indicating a positive co-relation between periodontal index and mucositis index. During the 4th and 5th week, the values of co-efficient of co-relation were 0.31 and 0.07, respectively, which also indicates a positive correlation between periodontal index and mucositis index. However, the correlation is of better degree during the 2nd and 3rd week than during the 4th and 5th weeks. Table 8 showed average value of periodontal index and mucositis index of study subgroup-2. Table 9 showed the co-relation between the periodontal index and mucositis index for the study Subgroup 2. In the 2nd and 3rd week, the co-efficient of co-relation were found to be 0.6 and 0.38, respectively, indicating a positive co-relation between periodontal index and mucositis index. During the 4th and 5th week, these values were found to be 0.327 and 0.09, respectively, which also indicates a positive co-relation between the two indices.

Table 1: Profile of baseline characteristics

Characteristics	Numbers
Number of patient enrolled	40
Number of patients treated	40; 20 (S)+20 (C)
Number of patients followed up	40
Sex (*n*=40)	
Female	5
Male	35
Age	
Range (years)	
18-30	2
35-70	38

S: Study, C: Control

DISCUSSIONS

The concept that "Radiation Mucositis" is the direct effect of radiation.[8] The statement was reinforced later by Rosenthal and Wilkie,[9] Baker[10] and Tikriti *et al.*

The variability of occurrence and distribution led others to think about the contributing factors. It is found that vascular changes occurring during radiation therapy decrease the blood supply of the tissue, which reduces the ability of the tissue to withstand trauma and infection.[11] It is again confirmed the fact that minimal trauma within the field of irradiation could cause ulcerations which might take a month to heal and often lead to exposure of bone.[12] New evidence supports the view that oral mucositis is a complex process involving all the tissues and cellular elements of the mucosa. Other findings suggest that some aspects of mucositis risk may be determined genetically. GI pro-apoptotic and anti-apoptotic gene levels change along the GI tract, perhaps explaining differences in the frequency with which mucositis occurs at different sites. Spijkervet *et al.*[13] concluded that oral hygiene may also contribute to mucositis. In 1991, he reported about the role of gram negative bacilli or endotoxin in the pathogenesis of mucositis during irradiation. Although direct cell damage from radiation therapy initiates the process, evidence suggests that the pathogenesis of mucositis is more a complex phenomenon.[14] The five stage model that has been proposed, includes (1) reactive oxygen species, (2) second messengers, (3) proinflammatory cytokines, (4) pathways evading host defense, and (5) metabolic by products of colonizing microorganisms. This model is believed to play a role in amplifying tissue injury.[15]

Table 2: Average Spijkervet's MI patient wise at different phases

Study group						Control group					
Patient number	Week					Patient number	Week				
	I	II	III	IV	V		I	II	III	IV	V
Subgroup 1						Control					
1	0	2.01	2.05	4.06	4.88	1	0	0.501	0.602	0.701	0.86
2	0	1.53	1.57	2.7	2.88	2	0	0.620	0.701	0.803	0.9
3	0	1.63	2.7	3.95	4.13	3	0	0	0	0	0
4	0	1.63	1.67	3.95	4.13	4	0	0.701	0.803	0.901	1.05
5	0	1.41	1.45	2.7	2.88	5	0	0.401	0.504	0.603	0.75
6	0	1.43	1.47	3.45	3.63	6	0	0	0	0	0
7	0	1.01	1.65	3.2	2.88	7	0	1.18	1.202	1.301	1.40
8	0	1.71	1.75	3.7	3.88	8	0	0	0	0	0
9	0	1.14	1.15	3.7	3.88	9	0	0.01	1.01	1.101	1.25
10	0	1.01	1.05	4.1	4.38	10	0	0.751	0.85	0.9501	1.10
Subgroup 2						11	0	1.201	1.301	1.301	1.60
1	0	2.101	2.201	4.701	4.75	12	0	0	0	0	0
2	0	1.621	1.721	2.601	2.75	13	0	1.011	1.101	1.101	1.35
3	0	1.721	1.821	3.851	4.02	14	0	1.001	1.101	1.101	1.35
4	0	1.721	1.821	3.851	4.01	15	0	0	0	0	0
5	0	1.501	1.601	2.601	2.75	16	0	0.251	0.351	0.452	0.601
6	0	1.623	1.623	3.351	3.59	17	0	0.351	0.451	0.551	0.701
7	0	1.101	1.201	3.101	3.25	18	0	0	0	0	0
8	0	1.801	1.901	3.601	3.75	19	0	0.401	0.501	0.601	0.751
9	0	1.201	1.301	3.601	3.75	20	0	0.501	0.601	0.701	0.851
10	0	1.101	1.201	4.101	4.25						

MI: Mucositis index

Table 3: Comparison of MI between study subgroup 1 and control group

Week	Study subgroup 1	Control group
II		
Average	1.484	0.44
SD	0.309	0.402
t-value	26.84	
Statistical significance		S
III		
Average	1.48	0.55
SD	0.307	0.403
t-value	9.84	
Statistical significance		S
IV		
Average	3.7	0.62
SD	0.317	0.408
t-value	32.42	
Statistical significance		S
V		
Average	3.8	0.77
SD	0.311	0.41
t-value	31.56	
Statistical significance		S

SD: Standard deviation, S: Significance, MI: Mucositis index

Table 4: Comparison of MI between study subgroup 2 and control group

Week	Study subgroup 2	Control group
I		
Average		
SD	0	0
t-value		
Statistical significance		
II		
Average	1.53	0.44
SD	0.298	0.402
t-value	7.17	
Statistical significance		S
III		
Average	1.63	0.55
SD	0.308	0.403
t-value	7.6	
Statistical significance		S
IV		
Average	3.52	0.62
SD	0.311	0.402
t-value	12.07	
Statistical significance		S
V		
Average	3.67	0.77
SD	0.307	0.41
t-value	19.59	
Statistical significance		S

SD: Standard deviation, S: Significance, MI: Mucositis index

A variety of interventions has been assessed for preventing oral mucositis or reducing the severity of mucositis and its sequelae. These include meticulous pre radiation on going mouth care, calcium phosphate solution, near-infrared light and lower-energy laser treatment, interleukin-11, sucralfate, oral glutamine, granulocyte-macrophage colony-stimulating factor rinse, tretinoin, and keratinocyte growth factor.[16] Particularly, promising results have been observed with the use of the cytoprotectant/radioprotectant agent amifostine.[16] To the best of our knowledge, palifermin (keratinocyte growth factor-1) is the only agent that has been approved as a drug by the United States Food and

Table 5: Comparison of MI between study subgroup 1 and study subgroup 2

Week	Subgroup 1	Subgroup 2
I		
Average		
SD	0	0
t-value		
Statistical significance		
II		
Average	1.44	1.53
SD	0.3089	0.298
t-value	0.608	
Statistical significance		NS
III		
Average	1.48	1.63
SD	0.307	0.308
t-value	0.258	
Statistical significance		NS
IV		
Average	3.7	3.52
SD	0.317	0.311
t-value	0.72	
Statistical significance		NS
V		
Average	3.8	0.77
SD	0.311	0.41
t-value	0.706	
Statistical significance		NS

SD: Standard deviation, S: Significance, NS: Not significance, MI: Mucositis index

Table 6: Average values of PI and MI of study subgroup 1

Patient number	Week									
	I		II		III		IV		V	
	MI	PI	MI	PI	MI	PI	MI	PI	MI	PI
1	0	5.1	2.01	5.1	2.05	5.2	4.6	5.8	4.88	5.88
2	0	4.8	1.53	4.8	1.57	4.9	2.7	5.55	2.88	6.63
3	0	5.3	1.63	5.3	1.67	5.4	3.95	6.05	4.13	6.13
4	0	4.1	1.63	4.1	1.67	4.2	3.95	4.8	4.13	4.88
5	0	8.9	1.41	3.9	1.45	4.0	2.7	4.65	2.88	4.63
6	0	4.1	1.43	4.1	1.47	4.2	3.95	4.8	3.63	4.88
7	0	4.3	1.01	4.3	1.65	4.4	3.2	5.0	2.88	5.13
8	0	3.6	1.71	3.6	1.75	3.7	3.7	4.3	3.88	4.38
9	0	3.3	1.14	3.3	1.15	3.4	3.7	4.05	3.88	4.13
10	0	3.1	1.01	3.1	1.05	3.2	4.1	3.8	4.38	3.88

MI: Mucositis index, PI: Periodontal index

Drug Administration and the European Medicines Agency for oral mucositis.[17] There was adequate positive evidence to support a suggestion in favor of using oral care protocols for the prevention of oral mucositis. The evidence also supported the use of chlorohexidine mouthwash for the prevention of oral mucositis in patients receiving radiotherapy.[18] Other agents used are; antimicrobials, coating agents, anesthetics, anti-inflammatory-analgesics, natural miscellaneous agents like zinc supplements. Laser, light therapy, and cryotherapy are also used for management of mucositis. With this back ground of information, the present investigation was conducted to correlate clinically the initiation and progress of irradiation oral mucositis with pre-existing periodontal infection.

Table 7: Correlation between periodontal index and mucositis index of study subgroup I

Week	PI	MI
I		
Average	4.1	
SD	7.25	0
II		
Average	4.1	1.44
SD	0.725	0.309
r=0.62		
III		
Average	4.87	1.48
SD	0.74	0.307
r=0.73		
IV		
Average	4.87	3.7
SD	0.76	0.317
r=0.317		
V		
Average	4.95	3.8
SD	0.75	0.311
r=0.07		

MI: Mucositis index, PI: Periodontal index, SD: Standard deviation

Table 8: Average values of PI and MI of study subgroup 2

Patient number	Week							
	I		II		III		IV	
	PI	MI	PI	MI	PI	MI	PI	MI
1	5.3	0	5.3	2.1	5.4	2.2	5.6	4.7
2	5.0	0	5.0	1.620	5.15	1.72	5.35	2.6
3	5.5	0	5.5	1.720	5.65	1.82	5.85	3.85
4	4.3	0	4.3	1.720	4.4	1.82	4.6	3.851
5	4.05	0	4.05	1.502	4.15	1.601	4.35	2.601
6	4.3	0	4.3	1.52	4.4	1.62	4.6	3.35
7	4.5	0	4.5	1.101	4.65	1.2	4.85	3.1
8	3.8	0	3.8	1.8	3.9	1.9	4.1	3.6
9	3.5	0	3.5	1.2	3.65	1.3	3.85	3.6
10	3.3	0	3.3	1.1	3.4	1.2	3.6	4.1

MI: Mucositis index, PI: Periodontal index

Mucositis index in 1st week could not be correlated to periodontal index as no mucositis appeared during the 1st week of radiation in this study. Toward the end of the 2nd week mucositis index was significantly correlated with periodontal index, as the area of distribution showed that mucositis started developing around the teeth with greater degree of periodontal disease.

In the 3rd week, the lesions started spreading to other surrounding areas while some lesions started in the area more susceptible to trauma. In this week, also a significant correlation was found.

Table 9: Correlation between PI and MI of study subgroup 2

Week	PI	MI
I		
Average	4.35	
SD	0.698	0
r - could not be correlated		
II		
Average	4.34	1.53
SD	0.709	0.298
r=0.6		
III		
Average	4.47	1.63
SD	0.706	0.308
r=0.38		
IV		
Average	4.67	3.52
SD	0.698	0.311
r=0.327		
V		
Average	4.82	3.67
SD	0.712	0.308
r=0.09		

MI: Mucositis index, PI: Periodontal index, SD: Standard deviation, *r*: Pearson co-efficient of correlation

During the 4th week, the lesions were spread to other areas, increasing the severity of mucositis index. However, here, the correlation between periodontal index and mucositis index declined which could have been due to other contributing factors such as poor oral hygiene and trauma. In the 5th week also the correlation further declined between mucositis index and periodontal index, which could also have been due to the other contribution factors as described before. However, the co-relation is of better degree during 2nd and 3rd week than during the 4th and 5th weeks.

It can thus be suggested that though radiation mucositis occurs during irradiation, its initiation and severity cannot be solely attributed to the dose of radiation. As per the present observation, periodontal infection in the form of periodontitis can be considered as an initiating factor in the development of mucositis. Neither of the antimicrobial agents *viz.*, chlorohexidinegluconate norpovidone-iodine (5% w/v) are effective in controlling the severity of mucositis during radiation therapy in patients suffering from oropharyngeal cancer.

The stages of the pathogenesis of periodontal disease involve the process of Colonization–Invasion–Destruction.

Irradiation reduces the vascularity of tissue thereby compromising the local defense mechanism inherent in the healthy mucosa. This factor along with the decrease turnover of mucosa; could make it easier for the invasion of the adjacent mucosa around periodontally involved teeth by the periodontopathic micro-organism. This postulation could possibly be considered as a reason for the development of mucositis around the periodontally compromised teeth.

CONCLUSIONS

The periodontal disease is one of the contributing factors responsible for the initiation and degree of severity of radiation mucositis during treatment of oropharyngeal cancer.

The radiation-induced mucositis lesions were found to develop around periodontally involved teeth especially during the 2nd and 3rd weeks of the irradiation period indicating a positive correlation between the degree of mucositis and periodontal status during this period. As the oral hygiene maintenance level of the patient falls, and the periodontitis develops, there is an increase in the progress and severity of the mucositis lesions. Neither of the antimicrobial agents *viz.*, chlorohexidine gluconatenor povidone-iodine (5% w/v) are effective in the control of the severity of mucositis during radiation therapy in patients suffering from oropharyngeal cancer. It can thus be stated that there exists a definitive influence of the infection and inflammation associated with periodontitis in modifying the initiation and progress of radiation-induced mucositis in patients suffering from oropharyngeal cancer.

Hence, it may be advised that in consultations with the oncology team, treatment procedure aimed at improvement of the overall periodontal health and oral hygiene status of patients suffering from oropharyngeal cancer should be instituted and completed satisfactorily prior to the radiation protocol period to prevent the initiation and progression of radiation - induced mucositis in these patients.

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