ORIGINAL ARTICLE

A study on clinical attachment loss and gingival inflammation as etiologic factors in pathologic tooth migration

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Abstract

Background: Several etiologic factors have been listed for pathologic migration of periodontally involved teeth based mainly on clinical observations with scarce scientific evidence. Present study was carried out to find out relationship of clinical attachment loss and gingival inflammation with pathologic tooth migration.

Materials and Methods: A total of 37 patients having 50 pairs of migrated and non-migrated contralateral teeth were taken into consideration.

Results: Mean total attachment loss per tooth in migrated and non migrated tooth is 13.32 ± 0.74 S.E. and 8.34 ± 0.58 S.E., respectively ($P < 0.001$), which reveals a positive correlation. There seems to be an association between frequency of migration and severity of attachment loss since highest percentage of migrations were seen in maximum total attachment loss group. Relationship could not be established between severity of attachment loss and severity of migration for which more data may be required. Also, it was seen that gingival index was significantly higher in migrated group.

Conclusion: Findings suggest that there exists a direct relationship between pathologic migration and clinical attachment loss as well as gingival inflammation.

Clinical relevance: Results emphasize the importance of early treatment of periodontitis to curb inflammation, which seems to be more important since it is completely reversible, and attachment loss also in order to prevent unaesthetic complications. Moreover bleeding along with recent change in position of teeth should be considered as important sign of active, moderate to severe periodontal disease by general dentists and hygienists so that they can refer for specialist consultation.

Key words: Clinical attachment loss, pathologic tooth migration, periodontal disease

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Introduction

Pathologic tooth migration is defined as the movement of a tooth out of its natural position usually as a result of periodontal disease.[1] Two major factors play a role in maintaining the normal tooth position, health and normal height of periodontium, and the forces exerted on teeth that include forces of occlusion and pressure from lips, cheeks and tongue.[2] With the presence of periodontal disease there is reduction in the surface area of attachment apparatus supporting and maintaining normal position of tooth. Furthermore, inflammatory alterations affect the quality of remaining supporting structure thereby making the tooth less adaptive to tensile forces and promote pathologic migration.

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The prevalence of pathologic migration is not known precisely but has been reported to be between 30.03% and 55.8%[3-6] in various studies. It may manifest in the form of incisor flaring, diastema formation, rotation, extrusion, tipping into edentulous spaces or a combination of any of these.

The etiology of pathologic tooth migration is multifactorial as well as complex[3] that includes periodontal attachment loss,[7,8] inflamed tissues,[9,10] bone loss,[5-7] occlusal factors,[11,12] loss of teeth without replacement,[5,13] labial frenum, and iatrogenic dentistry. Specific behaviors associated with it include bruxism, tongue thrusting, lip and finger sucking habits and playing of wind instruments.[3]

The movement of teeth without periodontal destruction is called drifting. Hence, attachment loss is the mandatory prerequisite for pathologic migration to occur either in isolation or in association with some other listed etiologic factor. Is there some threshold value of attachment loss after which migration starts? Is attachment loss in itself sufficient to initiate migration or some other factors like severity of attachment loss, number of root surfaces involved, tooth type, amount of inflammation, type of occlusion, type of habit, etc are more important? There is paucity of scientific data on the relationship of each of these etiologic factors independently with pathologic migration and also on the determination of relative importance of each.

The aim of the present study was to investigate the relationship of pathologic migration with gingival inflammation and clinical attachment loss, which are supposed to be the most important etiologic factors.

Materials and Methods

Study population

A total of 45 subjects suffering from localized or generalized chronic periodontitis with a history of recently formed diastema in upper or lower anterior segment or noticeable increase in already existing diastema were selected from the patients attending the Department of Periodontics, Govt. Dental College, Rohtak. Out of these 37 patients consisting of 30 females and 7 males fulfilling the following criteria were taken into consideration for the present study

- Presence of pathologic migration and attachment loss of >4 mm on at least one surface.
- Presence of homologous tooth with attachment loss of >4 mm but without pathologic migration to be used as control [Figure 1].

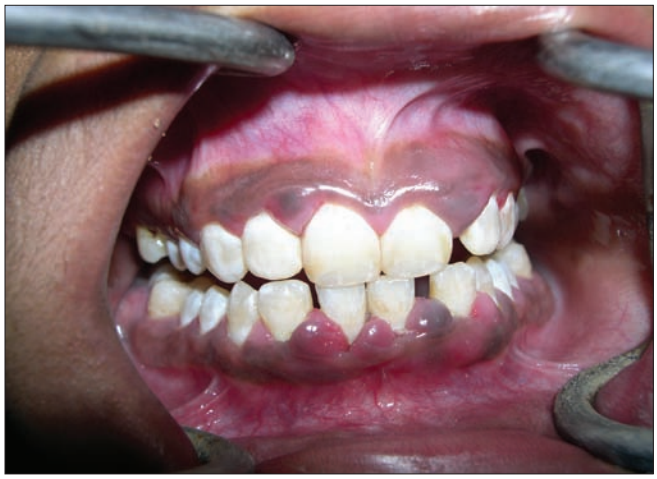

Figure 1: Non-migrated right maxillary lateral incisor and migrated contra lateral tooth

Exclusion criteria

Pathologic migration in the presence of habits like bruxism, tongue thrusting, lip biting, finger sucking and conditions like high frenal attachment were not included in the study as these habits are responsible for migration of tooth to a varying degree. Teeth with periapical pathology were also excluded since that may also be an additive factor in pathologic migration. Non replacement of an extracted tooth may cause occlusal disturbance, which may lead to migration and drifting of teeth. Hence, patients with a history of extraction were also not a part of the present study.

Comprehensive oral examination including periodontal charting was performed for all enrolled subjects. Plaque index by Sillness and Loe, gingival index by Loe and Sillness, clinical attachment loss, probing pocket depth were recorded.

The 50 diastema sites (migrated teeth) of these 37 patients were divided into the following groups on the first visit, based on the severity of pathologic migration which was measured by standardized cellotape gauges.[2]

- Group I (Gp I) sites measuring 0.1−1 mm
- Group II (Gp II) sites measuring 1.1−2 mm
- Group III (Gp III) sites measuring 2.1−3 mm

Fifty pairs of migrated and contra-lateral non-migrated teeth (100 teeth) were grouped according to the total attachment loss on all the four surfaces (sites) to study relationship between severity of attachment loss and pathologic migration.

Gp A : Total attachment loss of 4−10 mm
Gp B : Total attachment loss of 11−17 mm
Gp C : Total attachment loss of 18−25 mm

Probing depth and attachment level measurements using William's periodontal probe were taken at four sites (i.e. mesial, distal, mid buccal, and mid palatal) around each migrated and non-migrated contralateral tooth.

Interproximal measurements were taken with probe angulations as close as possible to the long axis of tooth.

Probing reliability

Clinical attachment loss measurements were taken with a William's periodontal probe by two examiners simultaneously who were blinded to each other's results. Total attachment loss around each pair of migrated and non-migrated contralateral tooth was taken. Results showed a standard deviation of 0.674 ± 0.067SE.

Results

Fifty pairs of migrated and non-migrated teeth in a subset of 37 patients were compared for attachment levels. Relationship of mean of total attachment loss in migrated and non-migrated teeth has been evaluated in Table 1. Mean total attachment loss in migrated teeth is 13.32 + 0.74 S.E., which is significantly higher than an mean total attachment loss of 8.34 ± 0.58 SE in non-migrated teeth as shown in Figure 2 ($P < 0.001$, paired t test). This reveals a positive correlation between attachment loss and migration.

There was a non-significant correlation between severity of attachment loss and severity of migration with correlation coefficient of 0.149 (pearson product moment correlation coefficient). Table 2 compared the mean of total attachment loss in GpI (12.1176 ± 1.17 SE) and GpII (14.5 ± 1.36 SE), which shows a statistically non-significant difference between the two. Similarly, Tables 3 and 4 compare mean of total attachment loss between Gp II and Gp III and Gp I and III, respectively, with results showing a non-significant difference in both the comparisons. Thus a positive correlation could not be established between *severity of attachment loss and severity of migration*.

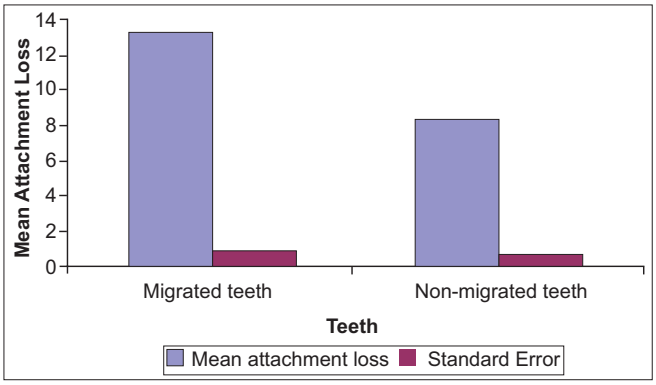

Figure 2: Mean of total attachment loss and standard error in migrated and non-migrated teeth investigated

Table 1: Comparison of mean total attachment loss in migrated and non-migrated teeth

Variable	Migrated teeth (n = 50)	Non-migrated teeth (n = 50)	t	P
Mean attachment loss	13.32	8.34	5.262	****
Standard deviation	5.235	4.168		
Standard error	0.7403	0.5894		

****Significant at 0.001

Relationship of migrated and non-migrated teeth with severity of attachment loss has been compared in Table 5. Since the calculated value of χ^2 i.e. 11.87 (Chi square test) is higher than the table value at 1% level of significance, $P < 0.01$ and it can be concluded that there is an association between frequency of migration and severity of attachment loss.

Gingival inflammation and pressure from granulation tissue in pocket as an etiologic factor has also been stressed in the literature. Table 6 compares mean gingival index of both the groups, which show a significant difference between both the groups. This implies that mean gingival index was significantly higher in migrated teeth.

Discussion

Results reveal that teeth affected by migration had significantly more attachment loss than contra lateral teeth without migration [Table 1], which is in accordance with earlier study by Towfighi *et al.*,[3] that studied 75 pairs of migrated and contra lateral non migrated teeth. The mean attachment loss of migrated teeth (4.79 ± 0.28 mm) was significantly greater than control teeth (3.21 ± 0.18 mm) in that study which is quite comparable to our results. Since the etiology of migration is multifactorial, using the same patient as a control may be more accurate means of studying these factors, as the factors which influence tooth position would remain relatively comparable within an individual. It may here be deduced that pathologic migration initiates after clinical attachment loss exceeds a threshold value.

Literature demonstrates that tooth drifts in a direction diametrically opposite to the site exhibiting most severe destruction[3,6] except in cases of facial pockets where direction of movement is same. This study included patients with recently formed diastema or increase in existing diastema, the precise reason for formation of which is unknown and may be rotation, flaring, extrusion, tipping and most commonly a combination of all. So the exact direction of migration cannot be determined objectively. The decision of adding attachment loss at four sites and calculating a mean attachment loss thus seemed to be most prudent.

Total clinical attachment loss on migrated teeth versus that on contralateral non-migrated teeth showed a mean difference of 4.98 mm in our study. This difference was

calculated to be average 1.245 mm (13.32 mm – 8.34 mm/4) on a single surface comparable to mean attachment loss difference of 1.58 mm in Towfighi[2] study.

Relationship of mean attachment loss per tooth and severity of migration could not be established since the difference between mean attachment loss in GpI, GpII, and GpIII was not statistically significant [Tables 2-4]. Further investigation needs to be done on this point with a greater sample size.

Relationship between severity of attachment loss and frequency of migration was compared [Table 5]. It can be clearly seen that out of 50 non-migrated teeth, a major fraction i.e. 35 teeth fall in the group of least severe attachment loss, i.e. GpA. On the other hand out of 50 migrated teeth almost half fall in the group of moderately severe attachment loss, i.e., GpB and 7 in the GpC. Results are statistically significant at level of $P < 0.01$. Results are in accordance with the general notion that frequency of pathologic migration increases after clinical attachment loss exceeds a threshold value. In Gp A 35.1%,Gp B 63.1%, and Gp C 87.5% showed migration which authenticates the fact that more is the total attachment loss more are the chances of migration of tooth keeping all other patient factors constant by taking same patient as control also.

Table 2: Comparison of mean total attachment loss in GpI and GpII

Variable	GpI (n = 17)	GpII (n = 18)	t	P
Mean attachment loss	12.1176	14.5	-1.317	NS
Standard deviation	4.8332	5.793		
Standard error	1.1722	1.3654		

NS- Not significant

Table 3: Comparison of mean total attachment loss in GpII and GpIII

Variable	GpII (n = 18)	GpIII (n = 14)	t	P
Mean total attachment loss	14.500	13.500	0.510	NS
Standard deviation	5.793	5.0952		
Standard error	1.3654	1.3618		

NS- Not significant

Table 4: Comparison of mean total attachment loss in GpI and GpIII

Variable	GI (n = 17)	GIII (n = 14)	t	P
Mean total attachment loss	12.1176	13.500	0.773	NS
Standard deviation	4.8332	5.0952		
Standard error	1.1722	1.3618		

NS- Not significant

Table 6 which compares mean gingival indices in both groups shows significantly more inflammation in migrated teeth. But it is difficult to say whether inflammation is cause of migration or consequence of migration due to opening of contact.

Similarly in a study by Martinez Canut P *et al.*,[6] the relationship of pathologic tooth migration with factors like bone loss, tooth loss and gingival inflammation was seen. The odds ratio indicated that probability of pathologic tooth migration increased between 2.95 to 7.97 times as bone loss increased.

Another study by Costa[5] also shows similar results in which it was observed that anterior teeth with pathologic migration presented greater attachment loss and bone loss than non-migrated teeth. Although patient factor was kept constant but tooth type (and hence tooth size and position and root surface area) was not constant as against our study in which tooth type was also kept stable by including contralateral non migrated teeth as done by Towfighi.[3]

One of the limitations of our study was subjective nature of determining whether the control teeth had any type of displacement or not. For this purpose an ideal arch form was estimated within patient's arch and if any type of displacements were noted in control teeth, the patients were not included in this study. Moreover manual probing was used for measurements whose error is often regarded as ±1.00 mm.[14] Probing depth, probing force, probe angulation, state of tissue health, and tooth types and surfaces affect variability in repeated measurements.[2] Having two examiners to ensure probing reproducibility showed a very low standard deviation of 0.674 mm, which is quite less then error limit for manual probing.

Table 5: Comparison of number of migrated and non migrated teeth in Gp A, Gp B and Gp C

Variable	Migrated teeth	Non-migrated teeth	χ^2	P
GpA	19	35	11.87	**
GpB	24	14		
GpC	7	1		
Total	50	50		

**Significant at 0.01

Table 6: Mean and standard deviation of Gingival index (Loe and Silness) in migrated and non-migrated teeth

Variable	Migrated teeth		Non-migrated teeth		A	P
	Mean	S.D	Mean	S.D		
GI	2.02	1.858	1.25	0.010	0.4982	0.0036

Further research work can be done on analyzing relationship of pathologic migration with tooth type, specific surface involvement, number of root surfaces involved etc since the literature is deficient in this.

Periodontitis when associated with pathologic migration can devastate a patient's self-esteem and self-confidence sometimes even resulting in severe psychological problems. The best to way treat it is to rather prevent it. But lack of objective information about relative importance of all stated etiologic factors is a great deterrent in preventive treatment. Bleeding along with recent change in position of teeth should be considered as important sign of active, moderate to severe periodontal disease by general dentists and hygienists (who are not performing periodontal check-up during regular dental check-up) so that they can refer for specialist consultation. Since treatment of pathologic tooth migration in its advanced stage is complex, expensive, time consuming, requiring inter-disciplinary approach and sometimes not even possible due to time and financial constraints, its prevention needs more attention in periodontal research which can be done only when relative importance of all etiologic factors is known.

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