ORIGINAL RESEARCH

Comparison of the Antimicrobial Activity of *Aloevera* Mouthwash with Chlorhexidine Mouthwash in Fixed Orthodontic Patients

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Abstract

Aim: Aim of the present research was to compare the antimicrobial activity of *Aloevera* mouthwash with chlorhexidine mouthwash in fixed orthodontic patients.

Materials and methods: A sample of 90 fixed Orthodontic patients participated in this study. Full-mouth oral prophylaxis was performed for every patient at the start of the study. Patients were advised to brush twice a day with the modified bass technique and rinse with respective mouthwashes for 20 days. Once the patients with fixed orthodontic appliances were accepted to participate in the study, they received dental prophylaxis which includes the removal of plaque, calculus, and stains from the teeth by scaling and polishing. Then they were randomly divided into the following three groups: chlorhexidine (group I), *Aloevera* (group II), and control (group III). A washout period of 8–10 days (baseline) was awaited post-oral prophylaxis and then the following clinical parameters were recorded: Plaque index (PI) and gingival index (GI). The data included clinical examination, inspection, and microscopic observation techniques.

Results: The mean reduction of the PI score on the 20th day of group II was 0.03 ± 0.18, group I was 0.43 ± 0.49, and the control group was 1.65 ± 0.88. The mean reduction of GI score on the 20th day of group II was 0.83 ± 0.40, group I was 0.93 ± 0.55, and group III was 1.85 ± 0.77. Student's *t*-test had been used to evaluate within each group between day 1 and day 20, group I and group II had shown higher differences compared to control.

Conclusion: In conclusion, both chlorhexidine mouthwash and *Aloevera* mouthwash are important chemical adjuncts in controlling gingival inflammation, gingival bleeding, and plaque accumulation in orthodontic patients. *Aloevera* could be an alternative to chlorhexidine in patients who are seeking a chemical-free, indigenous, and patient-friendly oral hygiene aid.

Clinical significance: Chlorhexidine is known to produce temporary tooth discoloration, allergic responses, dry mouth, burning in the mouth, and transient bad taste, which deter patients from using this mouthwash. The hunt for plant extract-based antimicrobial medicines has been prompted by the emergence of medication resistance and the unfavorable side effects of several antibiotics. These natural remedies can be a valuable substitute for creating a comparable effect.

Keywords: *Aloevera* mouthwash, Antimicrobial, Chlorhexidine mouthwash, Fixed orthodontics.

The Journal of Contemporary Dental Practice (2022): 10.5005/jp-journals-10024-3369

Introduction

The presence of fixed appliances in the oral cavity is frequently linked to the growth of microorganisms that cause oral health issues that result in progressive enamel mineral loss and inflammatory changes in the gingiva. By lowering microbial plaque, mouthwash improves oral hygiene. Chlorhexidine has proven to be one of the most potent mouthwashes for reducing tooth plaque and pathogenic germs since chlorhexidine is regarded as a gold standard.[1]

Chlorhexidine is known to produce temporary tooth discoloration, allergic responses, dry mouth, burning in the mouth, and transient bad taste, which deter patients from using this mouthwash. The hunt for plant extract-based antimicrobial medicines has been prompted by the emergence of medication resistance and the unfavorable side effects of several antibiotics.[2] These herbal remedies can be a good substitute for synthetic ones in producing the same results.

Vitamins, minerals, enzymes, sugars, phenolic compounds, lignin, saponins, sterols, and amino acids are just a few of the nutrients found in the *Aloevera* plant.[3] Saponins, soap-like compounds found in *Aloevera*, are general cleaners with antibacterial and anticarcinogenic qualities. *Aloevera* contains free anthraquinones

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How to cite this article: Ayesha S, Bhargava A, Philip AK, *et al*. Comparison of the Antimicrobial Activity of *Aloevera* Mouthwash with Chlorhexidine Mouthwash in Fixed Orthodontic Patients. J Contemp Dent Pract 2022;23(7):743–748.

Source of support: Nil

Conflict of interest: None

(a phenolic molecule) as well as anthrone-C-glycosides, chromones, isobarbaloin, and barbaloin-IO-aloe emodin-9-anthrone. These substances have strong analgesic and purgative effects in addition to being potent antibacterial agents.[4]

Commercially available mouth rinses often contain alcohol in high percentages along with additional substances such as detergents, artificial sweeteners, emulsifiers, organic acids, and colors.[5] This has led to increased use of herbal mouthwashes without any adverse effects. Hence, this study was conducted to assess the antimicrobial activity of *Aloevera* mouthwash with chlorhexidine mouthwash in fixed orthodontic patients.

Materials and Methods

A total of 90 fixed orthodontic patients participated in this clinical study. Ethical approval was obtained from the institutional review board. The sample was collected from patients undergoing fixed orthodontic treatment in the Department of Orthodontics and Dentofacial Orthopaedics, Sri Siddhartha Dental College and Hospital, Tumakuru, Karnataka, India. Absence of systemic disease, a minimum of 20 fixed orthodontic attachments, no supragingival calculus, moderate gingival inflammation during the study, a mean PI score below 3, and all the subjects must be voluntarily willing to participate in the study were included in the study. Prior use of any antibiotic or mouthwash for 10 consecutive days in the last 3 months, a history of sensitivity to any mouthwashes, the use of corticosteroid in the last 3 months, Severe gingival inflammation during the study, poor co-operation reported by parents of subjects were excluded from the study.

Method of Preparation of Mouthwashes (Chlorhexidine, *Aloevera*, and Normal Saline)

According to the manufacturer's instructions, chlorhexidine is used at a 0.2% concentration. In this study, mouthwash made from commercially available *Aloevera* juice is used in the same concentration as mouthwash without being diluted. For group III, sterile normal saline with a 0.085% sodium chloride concentration is used.

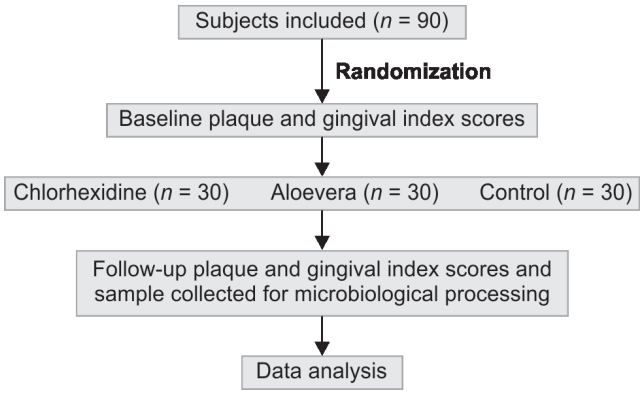
Method of Oral Hygiene Maintenance

Each subject received a full mouth oral prophylactic at the beginning of the research. The patient was instructed to use the "modified bass" technique to brush twice daily and to rinse with the appropriate mouthwash for 20 days. Once the patients with fixed orthodontic appliances were approved to take part in the study, they underwent dental prophylaxis, which involves scaling and cleaning the teeth to remove plaque, calculus, and stains. After that, they were split into the following three groups using a random number method: Chlorhexidine group (group I), *Aloevera* group (group II), and control group (group III) (Flowchart 1). After an oral prophylaxis washout period of 8–10 days (baseline), the PI and GI were recorded as clinical measures (Fig. 1). The information covered clinical inspection, investigation, and microscopic observation methods. For microscopic observation techniques, sampling was carried out three times. Before the elastomeric rings around the brackets of the upper left canine and premolar teeth were removed and submerged in thioglycolate media for the first sample T1, 5 mL of ordinary saline was administered to rinse their mouths.

For the second sample T2, 5 mL of chlorhexidine mouthwash, *Aloevera* mouthwash, or normal saline rinse were given to groups I–III, respectively to rinse their mouths for 1 minute. Then, the elastomeric module on the right side was also immediately removed and submerged in thioglycolate media. To preserve the bacterial cell shape, normal saline with a concentration of 0.085% NaCl is needed. Because it contains additional pollutants and germs, plain water was not employed as the study's control.

Twenty days after the first sample, T1, and after using chlorhexidine/*Aloevera*/normal saline mouthwash, the third sample, T3, was collected. Right canine and premolar elastomeric rings were removed for the sample and similarly submerged in the same medium. The patient received their specific mouthwash to rinse with twice a day between T2 and T3. At T3, any dental discoloration, taste or color change, or burning sensation was noted in the presence or absence.

Flowchart 1: Flow chart depicting the methods and steps used in this study



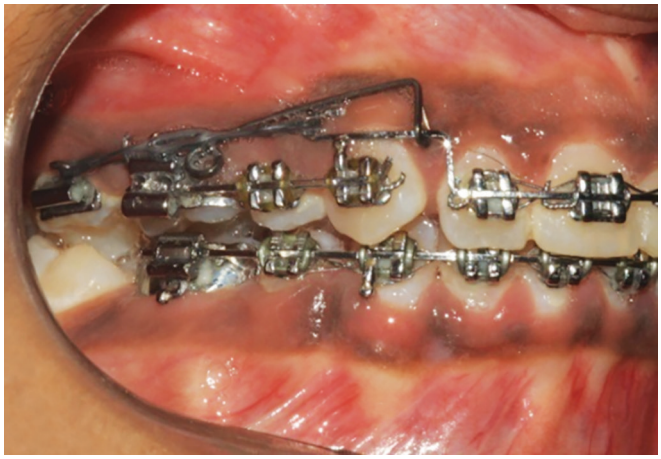


Fig. 1: Site used for plaque collection

Microbiological Processing

After the sample collection, it was delivered right away to the microbiological laboratory within 2 hours; in the event of a delay, it might be refrigerated for 4 hours and transported in thioglycolate broth. For the isolation and cultivation of the organism, samples were grown on two distinct mediums, namely, blood agar and Mac Conkey's agar. In order to accommodate two cases per plate for T1 and T2 samples and four cases for T3 samples, each culture plate is divided into four quadrants. The inoculums were transported using a metal loop with an interior diameter of 2 mm. Prior to inoculation, plates were made sure to be dry. A Bunsen burner flame was used to

Figs 2A to C: Blood agar culture plates of (A) *Aloevera*; (B) Chlorhexidine; (C) Control group showing growth of organisms

inoculate the plates. The inoculums were streaked over the relevant quadrant of each vial after the metal loop.

Both plates were then incubated for 18–24 hours at 37°C using blood agar in a candle extinction jar with 5–10% CO_2 and Mac Conkey's agar directly in an incubator. Jars were then opened and the plates were inspected. To determine the sorts of organisms, observations of cultural traits and colony morphologies of the organisms were connected. On the culture media, the aerobic bacterial growth was assessed as sparse (Fig. 2A), moderate (Fig. 2B), and heavy (Fig. 2C) growth.[6]

Statistical Analysis

Descriptive and inferential statistical analyses have been carried out in this study. Results on continuous measurements are presented on mean ± SD (Minimum–Maximum) and results on categorical measurements are presented in number (%). Analysis of variance (ANOVA) test has been used to compare the three groups. Student's *t*-test (two-tailed, dependent) has been used to find the significance of study parameters on a continuous scale between two intergroup analyses is on metric parameters. Significance is assessed at a 5% level of significance.

Results

To compare the mean PI in three groups on days 1 and 20, ANOVA was used, and was shown significant in three groups; the mean reduction of PI score on day 20 of group II was 0.03 ± 0.18, group I was 0.43 ± 0.49, and group III was 1.65 ± 0.88. Student's *t*-test has been used to evaluate each group between days 1 and 20, group I and II showed a higher difference compared to group III (Table 1).

To compare the mean GI in three groups on day 1 and 20, ANOVA test was used, and was shown significant in three groups; the mean reduction of GI score on day 20 of group II was 0.83 ± 0.40, group I was 0.93 ± 0.55, and group III was 1.85 ± 0.77. Student's *t*-test has been used to evaluate within each group between day 1 and 20; groups I and II showed higher difference compared to control (Table 2).

Tables 3 to 5 show the intragroup comparison of groups I–III. This study showed that at two-time intervals *Aloevera* showed statistically significant inhibition of growth of *Streptococcus viridians*, *Neisseria*, Coagulase-negative staphylococci, β-hemolytic streptococci, *Escherichia coli*, *Lactobacillus*, *Staphylococcus aureus*, *Klebseilla* and has a less inhibitory effect on *Candida* and least on *Pseudomonas*. Group I showed significant inhibition of growth of all the organisms, whereas group III did not have any significant effect on microorganisms.

Table 1: Intergroup comparative evaluation of PI

PI	*Day 1*	*Day 20*	*Difference*	*t-value*	*p-value*
Group I	2.27 ± 0.69	0.43 ± 0.49	1.833	14.903	<0.001**
Group II	1.53 ± 0.92	0.03 ± 0.18	1.500	8.939	<0.001**
Group III	2.02 ± 0.79	1.65 ± 0.88	0.367	3.958	<0.001**
p-value	0.003**	<0.001**	–	–	–

**Highly significant

Table 2: Intergroup comparative evaluation of GI

GI	*Day 1*	*Day 20*	*Difference*	*t-value*	*p-value*
Group I	2.38 ± 0.58	0.93 ± 0.55	1.450	13.750	<0.001**
Group II	1.93 ± 0.84	0.83 ± 0.40	1.100	8.462	<0.001**
Group III	2.20 ± 0.69	1.85 ± 0.77	0.350	3.252	0.003**
p-value	0.003**	<0.001**	–	–	–

**Highly significant

Discussion

It is generally known that the microbial flora of the oral cavity plays a role in the majority of infectious oral disorders, including dental caries and periodontal disease. Acidogenic bacteria, primarily *Lactobacillus* and *Streptococcus mutans*, are what cause dental caries. Dental caries is caused by oral bacterial species that break down sucrose into lactic acid and other organic acids in dental plaque that forms on the surface of teeth and dissolve calcium phosphate in the enamel.[7]

According to Maret et al.[8] fixed orthodontic appliances have been found to cause specific changes in the oral environment, including increased plaque accumulation, raised *S. mutans* colonization, and increased *Lactobacillus* species, all of which are closely related to dental caries.

These results demonstrated that gram-negative periodontal pathogenic microorganisms of the orange and red complexes can colonize orthodontic brackets. Since brackets are frequently positioned in close proximity to the gingival sulcus and these bacterial species are highly associated with the presence of gingival

Table 3: Intragroup comparison of group I

	T1 (n = 30)				*T2 (n = 30)*				*T3 (n = 30)*			
Group I	*NG*	*Scanty*	*Medium*	*Heavy*	*NG*	*Scanty*	*Medium*	*Heavy*	*NG*	*Scanty*	*Medium*	*Heavy*
1. *S. viridians*	0 (0%)	2 (6.7%)	2 (6.7%)	20 (66.7%)	23 (76.7%)	1 (3.3%)	0 (0%)	0 (0%)	24 (80%)	0 (0%)	0 (0%)	0 (0%)
2. *Neisseria*	0 (0%)	2 (6.7%)	3 (10%)	4 (13.3%)	9 (30%)	0 (0%)	0 (0%)	0 (0%)	9 (30%)	0 (0%)	0 (0%)	0 (0%)
3. *Candida*	0 (0%)	1 (3.3%)	0 (0%)	3 (10%)	0 (0%)	4 (13.3%)	0 (0%)	0 (0%)	4 (13.3%)	0 (0%)	0 (0%)	0 (0%)
4. β-Hemolytic streptococci	0 (0%)	0 (0%)	0 (0%)	2 (6.7%)	1 (3.3%)	0 (0%)	1 (3.3%)	0 (0%)	2 (6.7%)	0 (0%)	0 (0%)	0 (0%)
5. Coagulase-negative staphylococci	0 (0%)	3 (10%)	3 (10%)	5 (16.7%)	10 (33.3%)	0 (0%)	1 (3.3%)	0 (0%)	10 (33.3%)	1 (3.3%)	0 (0%)	0 (0%)
6. *Lactobacillus*	0 (0%)	0 (0%)	0 (0%)	2 (6.7%)	2 (6.7%)	0 (0%)	0 (0%)	0 (0%)	2 (6.7%)	0 (0%)	0 (0%)	0 (0%)
7. *E. coli*	0 (0%)	3 (10%)	0 (0%)	0 (0%)	3 (10%)	0 (0%)	0 (0%)	0 (0%)	3 (10%)	0 (0%)	0 (0%)	0 (0%)
8. *Pseudomonas*	0 (0%)	2 (6.7%)	0 (0%)	1 (3.3%)	2 (6.7%)	1 (3.3%)	0 (0%)	0 (0%)	3 (10%)	0 (0%)	0 (0%)	0 (0%)
9. *S. aureus*	0 (0%)	1 (3.3%)	0 (0%)	1 (3.3%)	2 (6.7%)	0 (0%)	0 (0%)	0 (0%)	2 (6.7%)	0 (0%)	0 (0%)	0 (0%)
10. *Klebseilla*	0 (0%)	0 (0%)	2 (6.7%)	0 (0%)	0 (0%)	2 (6.7%)	0 (0%)	0 (0%)	2 (6.7%)	0 (0%)	0 (0%)	0 (0%)

Table 4: Intragroup comparison of group II

	T1 (n = 30)				*T2 (n = 30)*				*T3 (n = 30)*			
Group II	*NG*	*Scanty*	*Medium*	*Heavy*	*NG*	*Scanty*	*Medium*	*Heavy*	*NG*	*Scanty*	*Medium*	*Heavy*
S. viridians	0 (0%)	1 (3.3%)	3 (10%)	18 (60%)	13 (43.3%)	3 (10%)	2 (6.7%)	4 (13.3%)	15 (50%)	4 (13.3%)	2 (6.7%)	1 (3.3%)
Neisseria	0 (0%)	2 (6.7%)	1 (3.3%)	9 (30%)	6 (20%)	2 (6.7%)	2 (6.7%)	2 (6.7%)	8 (26.7%)	2 (6.7%)	2 (6.7%)	0 (0%)
Candida	0 (0%)	0 (0%)	1 (3.3%)	3 (10%)	0 (0%)	0 (0%)	1 (3.3%)	3 (10%)	0 (0%)	1 (3.3%)	0 (0%)	3 (10%)
β-Hemolytic streptococci	0 (0%)	0 (0%)	2 (6.7%)	1 (3.3%)	2 (6.7%)	1 (3.3%)	0 (0%)	0 (0%)	3 (10%)	1 (3.3%)	0 (0%)	0 (0%)
Coagulase-negative staphylococci	0 (0%)	3 (10%)	0 (0%)	6 (20%)	3 (10%)	2 (6.7%)	4 (13.3%)	0 (0%)	8 (26.7%)	1 (3.3%)	0 (0%)	0 (0%)
Lactobacillus	0 (0%)	0 (0%)	0 (0%)	2 (6.7%)	0 (0%)	2 (6.7%)	0 (0%)	0 (0%)	1 (3.3%)	1 (3.3%)	0 (0%)	0 (0%)
E. coli	0 (0%)	1 (3.3%)	0 (0%)	2 (6.7%)	3 (10%)	0 (0%)	0 (0%)	0 (0%)	3 (10%)	0 (0%)	1 (3.3%)	0 (0%)
Pseudomonas	0 (0%)	2 (6.7%)	0 (0%)	3 (10%)	0 (0%)	2 (6.7%)	0 (0%)	3 (10%)	0 (0%)	2 (6.7%)	0 (0%)	3 (10%)
S. aureus	0 (0%)	0 (0%)	1 (3.3%)	1 (3.3%)	0 (0%)	2 (6.7%)	0 (0%)	0 (0%)	2 (6.7%)	0 (0%)	0 (0%)	0 (0%)
Klebseilla	0 (0%)	0 (0%)	1 (3.3%)	2 (6.7%)	0 (0%)	2 (6.7%)	1 (3.3%)	0 (0%)	2 (6.7%)	1 (3.3%)	0 (0%)	0 (0%)

Table 5: Intragroup comparison of group III

	T1 (n = 30)				*T2 (n = 30)*				*T3 (n = 30)*			
Group III	*NG*	*Scanty*	*Medium*	*Heavy*	*NG*	*Scanty*	*Medium*	*Heavy*	*NG*	*Scanty*	*Medium*	*Heavy*
S. viridians	0 (0%)	3 (10%)	5 (16.7%)	14 (46.7%)	0 (0%)	3 (10%)	5 (16.7%)	14 (46.7%)	1 (3.3%)	5 (16.7%)	5 (16.7%)	11 (36.7%)
Neisseria	0 (0%)	3 (10%)	8 (26.7%)	9 (30%)	0 (0%)	3 (10%)	8 (26.7%)	9 (30%)	1 (3.3%)	3 (10%)	8 (26.7%)	9 (30%)
Candida	0 (0%)	1 (3.3%)	3 (10%)	3 (10%)	0 (0%)	1 (3.3%)	3 (10%)	3 (10%)	0 (0%)	2 (6.7%)	3 (10%)	3 (10%)
β-Hemolytic streptococci	0 (0%)	0 (0%)	3 (10%)	0 (0%)	0 (0%)	0 (0%)	3 (10%)	0 (0%)	0 (0%)	1 (3.3%)	2 (6.7%)	0 (0%)
Coagulase-negative staphylococci	0 (0%)	2 (6.7%)	2 (6.7%)	1 (3.3%)	0 (0%)	2 (6.7%)	2 (6.7%)	1 (3.3%)	1 (3.3%)	1 (3.3%)	2 (6.7%)	1 (3.3%)
Lactobacillus	0 (0%)	1 (3.3%)	0 (0%)	1 (3.3%)	0 (0%)	1 (3.3%)	0 (0%)	1 (3.3%)	0 (0%)	1 (3.3%)	1 (3.3%)	0 (0%)
E. coli	0 (0%)	1 (3.3%)	1 (3.3%)	1 (3.3%)	0 (0%)	1 (3.3%)	1 (3.3%)	1 (3.3%)	0 (0%)	1 (3.3%)	1 (3.3%)	1 (3.3%)
Pseudomonas	0 (0%)	1 (3.3%)	1 (3.3%)	1 (3.3%)	0 (0%)	1 (3.3%)	1 (3.3%)	1 (3.3%)	0 (0%)	1 (3.3%)	2 (6.7%)	1 (3.3%)
S. aureus	0 (0%)	1 (3.3%)	1 (3.3%)	1 (3.3%)	0 (0%)	1 (3.3%)	1 (3.3%)	1 (3.3%)	0 (0%)	2 (6.7%)	1 (3.3%)	0 (0%)
Klebseilla	0 (0%)	1 (3.3%)	0 (0%)	1 (3.3%)	0 (0%)	1 (3.3%)	0 (0%)	1 (3.3%)	0 (0%)	1 (3.3%)	0 (0%)	1 (3.3%)

inflammation and chronic periodontal disease, their presence in brackets may facilitate gingival inflammation or even periodontal breakdown.[9]

However, it is advised to administer antibiotics before invasive dental treatments in order to prevent bacteremia, and endocarditis caused by *S. mutans* and other oral bacterial species. As mouthwashes and irrigating agents, chlorhexidine, sodium hypochlorite, cetylpyridinium chloride, and amine fluoride are frequently used because they can stop the growth of oral germs that could be harmful. These antimicrobial medicines are routinely used, however, there have been reports of acute hypersensitivity reactions, toxicity, tooth discoloration, and other side effects. Additionally, chlorhexidine and sodium hypochlorite have been shown to be toxic to human periodontal ligament cells, limit protein synthesis, and impair mitochondrial activity, all of which have an adverse impact on important tissues.[10]

There has been a demand for various types of medicines with improved antimicrobial activity and less toxicity because of the potential emergence of oral bacteria that are multidrug-resistant and the adverse effects of current antibacterial treatments. Synthetic medications have been seen as inferior to the natural phytochemicals derived from medicinal plants utilized in traditional therapy. For the prevention and treatment of oral infections, many medicinal plants and their products are used widely. Among these, *Aloevera* is of particular interest and has been used therapeutically for a very long period.[7]

Both groups I and II showed a reduction in plaque and gingival scores significantly, compared to group III. The above findings are similar to a study done by Parkar and Janu[11] who concluded that *Aloevera* mouthwash was as effective as two commercially popular mouthwashes in controlling plaque and gingivitis.

The presence of the following chemicals in *Aloevera*, including pyrocatechol, cinnamic acid, p-Coumaric acid, and ascorbic acid, may be the cause of its antibacterial effect. A hydroxylated phenol known as pyrocatechol is harmful to microorganisms. The location, amount of hydroxyl groups on the phenol group, and an increase in hydroxylation that further increases toxicity are thought to be related to how poisonous they are to microorganisms. Additionally, phenolics damage cell membranes and denature proteins in order to work. They have antibacterial and tuberculocidal properties, are effective in the presence of organic material, and continue to work on the surface for a very long time after application. *Aloevera* contains cinnamic acid, which prevents bacteria's resting cells from absorbing glucose and producing adenosine tri-phosphate (ATP). p-Coumaric acid has the ability to both lengthen the lag phase and inhibit the enzymatic activity of the microorganisms. The enzymatic or genetic activity of microorganisms may be inhibited by ascorbic acid.[12]

During the experimental study, group II did not experience any instances of tooth discoloration, a burning sensation, or a change in taste; nevertheless, long-term studies are required to determine the mouthwash's sustained effects.

In an *in vitro* investigation by Lee et al.,[13] the antibacterial action of *Aloevera* was shown. It was found that dentifrice containing *Aloevera* prevented the development of a variety of oral microorganisms, including *Streptococcus sanguis*, *S. mutans*, *Actinomyces viscous*, and *Candida albicans*.

Siegrist et al.[14] found staining in both chlorhexidine and placebo groups. Dietary factors are a probable reason that staining is observed in the placebo group. Tannic acid, specifically tea, has been found to be causative factor in the increased degrees of stain in chlorhexidine users.

According to Diamati-Kipioti et al.,[15] placing orthodontic bands on children will encourage the creation of pseudopockets. Furthermore, according to the author, this circumstance would favor an increase in the total amount of cultivable bacteria as well as a change in its composition to a more anaerobic flora. Therefore, the antibacterial effect of mouthwashes on anaerobic flora was primarily examined in the current study.

According to a study by Alemdar and Agaoglu[16] gram-positive bacteria such as *S. aureus* and *Enterococcus faecalis* are the main targets of plant juice's antibacterial activity. With the exception of the gram-negative bacterium, *Klebsiella pneumoniae*, *Aloevera* juice had no inhibitory effect on the growth of gram-negative bacteria. These discrepancies may be explained by the fact that gram-positive bacteria have a single layer of the cell wall, but gram-negative bacteria have a multilayered and extremely complex cell wall. However, *Aloevera* juice did not inhibit *Pseudomonas aeruginosa*.

The primary limitation of the current study is its brief study duration. In order to assess the anti-plaque and anti-gingivitis efficacy of these mouthwashes, additional suggestions call for the inclusion of well-conducted randomized controlled trials with acceptable sample sizes integrating "crossover design" with sufficient wash-out interval.

Conclusion

This study concluded that both chlorhexidine mouthwash and *Aloevera* mouthwash are important chemical adjuncts in controlling gingival inflammation, gingival bleeding, and plaque accumulation in orthodontic patients. *Aloevera* could be an alternative to chlorhexidine in patients who are seeking a chemical-free, indigenous, and patient-friendly oral hygiene aid. Although chlorhexidine has demonstrated clear benefits and effectiveness, its long-term usage is frequently discouraged by adverse effects including tooth discoloration and impaired taste perception. Therefore, *Aloevera* has the potential to be an efficient antiplaque agent and, with the right taste and shelf-life refinements, an economical herbal alternative to chlorhexidine.

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