

The existence of *Entamoeba gingivalis* in the oral cavity with mild oral hygiene

Emma Rachmawati

Department of Oral Biology Faculty of Dentistry Universitas Padjadjaran

ABSTRACT

Entamoeba gingivalis is protozoa (non-pathogen amoeba) that lives in the oral cavity and act as opportunistic parasite. The parasite is most common found within oral cavity with advance periodontal disease. This study aimed to find the existence *Entamoeba gingivalis* within oral cavity with mild oral hygiene. The study was conducted by making preparation of plaque taken from patient's oral cavity with mild oral hygiene. The degree of oral hygiene was determined by using Silness and Loe Index and Ramfjord Periodontal Index. The number of *Entamoeba gingivalis* was gained by counting them from the preparation examined under microscope. The result demonstrates that mild oral hygiene has a high percentage of *Entamoeba gingivalis* is that 80 % either for various plaque index or ages.

Key words: *Entamoeba gingivalis*, mild oral hygiene

INTRODUCTION

Currently, there has been studied the part of *Entamoeba gingivalis* in causing of periodontal disease. This study has showed a significantly and positive-linearly relationship between the number of *Entamoeba gingivalis* and oral hygiene degree and has proven that this kind of oral parasite may enhance the severity of periodontal disease.^{1,2}

Entamoeba gingivalis could be found in saliva, plaque, and calculus. This species exists in view number in healthy oral cavity and will increase sharply as periodontal disease, caries, and abundant deposit of plaque and calculus present.³ Thus, it has been substantiated that there is a tight relationship between *Entamoeba gingivalis* and periodontal disease.

Talking about *Entamoeba gingivalis*, this parasite belongs to protozoa kingdom which

can adapt itself to oral cavity environment, consequently it is a pathogen and commensally parasite which lives in oral cavity. Unlike others *Entamoeba* sp. *Entamoeba gingivalis* does not posses cyst stage, its life cycle consists of simply trophozoit stage with 5 μ m to 15 μ m in diameter, so it is considered to be a nonpathogen, rather than the similar species that have a cyst stage.^{5,6} Its protoplasm comprise of the transparent ectoplasm and the granular endoplasm.

The amoeba is aggressively motile because the ectoplasm possesses a capability to act as pseudopod is that a long and lobular ectoplasm prolongation despite it is not rare to prolong as a short and blunt pseudopods.³ So, it is obvious that *Entamoeba gingivalis* will be able to move rapidly with pseudopods forming quickly in response to the conditions around the organism.

Still about the morphology of *Entamoeba*

gingivalis, its cytoplasm contains food vacuoles, epithelial cells, bacteria, spirilla, and often contains ingested leucocytes. The numbers of food vacuoles are predominantly higher than other content with the result that would exhibit soap bubble feature. The species has an oval to round nucleus with 3.5 μm to 4 μm in length and 2.5 μm to 3 μm in width. The nucleus possesses a clear nucleus membrane with a tight granular chromatin. *Entamoeba gingivalis* is the only species that ingests white blood cells. On the permanent stained smear, nuclear fragments of the white cells ingested can be seen within the food vacuoles, which are usually larger than the vacuoles seen in *Entamoeba histolytica*.⁴ Thus, this fact will help to differentiate the two.

Refer to its discovery history, *Entamoeba gingivalis* was the first parasitic amoeba of humans to be described. It was firstly discovered by Gross from soft teeth tartar between the teeth in the year of 1849 and the further examination was done by Prowazek in the year of 1904.^{4,6} Another name of this species were *Amoeba buccalis*, *Entamoeba buccalis*, *Endamoeba buccalis*.⁶ The species had also been recovered from the tonsillar crypts and could multiply in bronchial mucus, thus was appearing in sputum. Although these amoebas were most often recovered from patients with pyorrhea alveolaris, *Entamoeba gingivalis* was still considered to be nonpathogenic.

As mention above, there was a tight relationship between *Entamoeba gingivalis* existence and either oral hygiene degree or periodontal disease even though it might be found in healthy oral cavity. This had been established by Rosen and Willet⁷, who stated that *Entamoeba gingivalis* presented in healthy oral cavity 5-30% incidentally and 100% in periodontal disease. The present of this commensally parasite should still be avoided even though they are not harmful because the periodontal disease that appear due to low oral hygiene would be worsen by increasing the amount of this parasite. Therefore, the existence of *Entamoeba gingivalis* in oral cavity could indicate that oral hygiene must be raised.

On the other hand, *Entamoeba gingivalis* was considered as a causative organism of periodontitis but in fact, this species solely acted as opportunist organism that would use the condition to grow

and multiply. It had been proven by the data that the species was most common found from soft calculus, *Acute Necrotizing Ulcerative Gingivitis*, *Chronic Suppurative Periodontal lesion*, deep gingival and periodontal pocket, ulcer of buccal and tongue mucose, and tonsil inflammation. Yet the diseases mentioned above were inhabited by enormous amount of this species, there were no histological evident of species invasion into the periodontal tissue. Thus, the amoebas' inability to penetrate periodontal tissue exhibited that the species was apathogen but on the contrary, the species would produce proteolytic enzyme that might increase periodontitis pathogenicity.

The previous study had proven that a bad oral hygiene with a big plaque deposit and high periodontal index would increase the amount of *Entamoeba gingivalis* and on the other hand the increasing of the species might evoke the periodontal disease worse. According to this, there is a question, how is the number of *Entamoeba gingivalis* in mild periodontal index which mean in mild periodontal disease. Hence, this study will search for the answer of the question above.

MATERIALS AND METHODS

This study was conducted at the Clinic of Periodontology, Faculty of Dentistry Universitas Padjadjaran after being approved by Ethical Committee of the institution and that the subjects gave informed consent to the work. Data collecting included oral hygiene index (OHI), periodontal disease index (PDI), and *Entamoeba gingivalis* counting. OHI was determined by using Silness and Loe plaque index whereas PDI was performed by Ramfjord's Periodontal Disease Index.^{8,9}

The specimen were taken from plaque put in normal saline and subsequently wet preparations were made by using lugol. The preparations were examined under microscope afterward for counting the amoebas found. Eventually the datas collected were tabulated.¹⁰

RESULT AND DISCUSSION

The result of this study is served in 3 tables to gain a clear feature about the existence of *Entamoeba gingivalis* in mild oral hygiene.

Table 1. Distribution of patients with mild oral hygiene based on plaque index and gender.

Plaque index	Gender		Total	Percentage
	Man	Woman		
1.125-1.291	3	8	11	36.67
1.292-1.458	7	6	13	43.33
1.459-1.625	-	1	1	3.33
1.626-1.792	2	1	3	10
1.793-1.960	1	1	2	6.67
Total	13	17	30	100

Table 2. Distribution of patients with mild oral hygiene based on plaque index and existence of *Entamoeba gingivalis*.

Plaque index	<i>Entamoeba gingivalis</i> existency				Total
	+	++	+++	-	
1.125-1.291	7	-	1	3	11
1.292-1.458	10	-	-	3	13
1.459-1.625	-	1	-	-	1
1.626-1.792	2	1	-	-	3
1.793-1.960	2	-	-	-	2
Total	21	2	1	6	30
		24(80%)		(20%)	(100%)

Table 3. Distribution of patients with mild oral hygiene based on *Entamoeba gingivalis* existence and age.

Age	<i>Entamoeba gingivalis</i>		Total
	+	-	
20-24	7	-	7
25-29	8	3	11
30-34	8	3	11
35-39	1	-	1
Total	24	6	30
	(80%)	(20%)	(100%)

Table 1 shows that there are 11 patients (36.6%) consisting 3 men and 8 women has plaque index of 1.125-1.291, 13 patients (43.33%) consisting 7 men and 6 women has plaque index of 1.292-1.458. Plaque index of 1.459-1.625 belongs to 1 women only with percentage of 3.33% whereas plaque index of 1.626-1.972 belongs to 3 patients (10%) comprising 2 men and 1 woman. And the last plaque index is that 1.793-1.960 is possessed by 2 patients (6.67%) comprising 1 man and 1 woman.

Table 2 demonstrates the existence of *Entamoeba gingivalis* within oral cavity with mild oral hygiene. Group 1 (1.125-1.291) has 8 specimens

possessing *Entamoeba gingivalis*. The specimens of group 2 (1.292-1.458) that shows the amoeba is 10 specimens whereas group 3 (1.459-1.625) has 1. Group 4 (1.626-1.792) has 3 specimens and group 5 (1.793-1.960) has 2 specimens. So, the datas shows that *Entamoeba gingivalis* is almost always found in oral cavity with mild oral hygiene with percentage of 80% (24 specimens).

Table 4 exhibits the distribution of patients with mild oral hygiene based on *Entamoeba gingivalis* existence and patient's age. *Entamoeba gingivalis* is found from entire specimens of group age of 20-24 while group 2 (age of 25-29) has 8 specimens revealing the amoebas. The species is encountered from 8 specimens in Group 3 (age of 30-34) whereas Group 4 (age of 35-39) has barely 1 specimen displaying the organism. The datas show that *Entamoeba gingivalis* is presented within oral cavity with mild oral hygiene in all of kind of ages.

CONCLUSION

Entamoeba gingivalis has high percentage within oral cavity with mild oral hygiene (plaque index: 1.1-20) is that 80%. This happens because mild oral hygiene might still make inflammation present in oral cavity which will be a good environment for the amoebas to multiply by utilizing death bacteria and inflammation end product. So, as long as the plaque present and be considered as inflammation causative will provoke *Entamoebas gingivalis* to exist.

REFERENCES

1. Beaver PC, Jung RC, Cupp EW. Clinical parasitology. 9th ed. Philadelphia: Lea & Febiger; 1984.
2. Suhartina I. Hubungan antara banyaknya *Entamoeba gingivalis* dengan tingkat kebersihan mulut. J Kedokt Gigi 1993;V;1.
3. Cole MF, Arnold RR. Dental microbiology: Oral ecology and the normal flora of the mouth. Philadelphia: Harper & Row Publisher; 1982.
4. Garcia LS, Bruckner DA, Diagnostic medical parasitology. 3rd ed. Washington DC: ASM Press. 1997.
5. Ryan KJ. Medical microbiology an introduction to infectious diseases. Philadelphia: The C.V.

- Mosby Co.; 1994.
6. Faust EC, Russel PF. Clinical parasitology. 7th ed. Philadelphia: Lea & Febiger; 1964.
 7. Rosen S, Willet N, White R. Essential dental microbiology. London: Prentice Hall International Inc.; 1991.
 8. Lindhe. Clinical periodontology and implant dentistry. 44th ed. Blackwell. 2003.
 9. Newman, Takei, Carranza. Carranza's clinical periodontology. Philadelphia: W.B. Saunders Co.; 2002.
 10. Nolte WA. Oral microbiology with basic microbiology and immunology. 4th ed. St. Louis: The C.V. Mosby Co.; 1982.