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Analysis Of Aspartate Aminotransferase In Gingival Crevicular Fluid Before And After Phase I Periodontal Therapy In Gingivitis And Periodontitis Patients

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Abstract

Objective: Amount of Aspartate Amino Transferase (AST) is increased during periodontal inflammation.

The aim was planned to: Estimate the change in AST levels at selected sites after treatment and to correlate the AST levels with clinical periodontal parameters.

Method: 40 subjects were selected and included in the study, divided into two groups: Gingivitis group and Periodontitis group, each consisting of 20 subjects. One site for Gingival Crevicular Fluid (GCF) collection in each subject was selected and Aspartate Amino Transferase (AST) concentration was assessed by clinical parameters. Clinical parameters were Plaque Index (PI), Gingival Index (GI), Probing pocket depth (PPD) and clinical attachment level (CAL) in Periodontitis group at baseline and 3 months after Scaling and Root planing (SRP). The Aspartate Amino Transferase (AST) level was measured by semiautomated autoanalyser. It works on the principle given by Indian Federation of Clinical Chemistry (IFCC).

Results: A very significant correlation was found between Aspartate Amino Transferase (AST) and Gingival Index (GI) ($p<0.01$) in both the groups at baseline. In Periodontitis Group correlation between Probing Pocket Depth (PPD) and Aspartate Amino Transferase (AST) at baseline was statistically significant ($p<0.05$) and correlation between Clinical Attachment Loss (CAL) and Aspartate Amino Transferase (AST) was statistically significant ($p<0.01$) after treatment.

Conclusion: Aspartate Amino Transferase (AST) enzyme in Gingival Crevicular Fluid (GCF) might be an indicator of periodontal destruction.

Key Words
Gingival crevicular fluid, Aspartate Aminotransferase.

Introduction

The advance diagnostic measures have shown that pathogenesis of periodontal disease is episodic and does not occur in a continuous linear manner.[1] The microbial tooth deposits having pathogenic bacteria are one of the important risk factors.

To overcome this microbial insult, host immune response is activated which is a critical determinant of periodontal disease pathogenesis. The marginal gingival and sulcular area act as battle field where this interaction between bacteria and host takes place. Various components of GCF that have been studied including tissue degradation products e.g. hydroxyproline and glycosaminoglycans, interleukines (IL) and prostaglandin (PG), and proteases, lactate dehydrogenase and aspartate aminotransferase (AST also known as SGOT).[2],[3],[4]

AST is particularly important in the transport of reducing equivalents across the mitochondrial membrane via the malate aspartate shuttle and is a sensitive indicator of necrosis in a number of tissues.[5] In GCF it has been reported as a possible marker for distinguishing between active and inactive disease sites.[1]

During inflammation and cell death this enzyme is not utilized and gets liberated in the extracellular fluid such as GCF where it can be assessed. So, it is hypothesized that during gingival inflammation its concentration will be increased and after the scaling and root planing when the microbial load is reduced AST levels in gingivitis and periodontitis may also be decreased

The present study was thus carried out to analyze and compare the levels of AST in gingivitis and periodontitis patients as a biochemical marker before and after Scaling and Root planing (SRP).

Aims and objectives

1 To analyze the levels of AST in GCF before and after SRP.
2 To examine the cross sectional relationship between clinical parameters of periodontal disease and GCF AST levels

Materials & Method

Type of study: A prospective, interventional, comparative, bio-chemical study was planned. Research protocol was submitted to and approved by ethical committee of Kothiwal Dental College and Research Centre (KDCRC) Moradabad, before conducting the study. 20 subjects were selected in each group taking account of the following criteria-

Gingivitis group-
(a) Moderately inflamed gingiva with GI >2 (Loe and Silness 1967).
(b) More than 30% of teeth involved.
(c) No true pocket.
(d) No loss of attachment[6].

Periodontitis Group

(a) Moderately inflamed gingiva with GI (Loe and Silness 1967) >2 in involved sites.
(b) More than 30% of teeth involved.
(c) With pocket depth 5-8 mm in at least 5 teeth.
(d) Loss of attachment 3-4 mm in at least 5 sites[6].

Inclusion criteria:

The inclusion criteria were: subjects having a minimum of 20 natural teeth, definite clinical evidence of chronic gingivitis and chronic periodontitis, age group 16-40 years[4], serum glutamatic oxaloacitic transaminase (SGOT) values within normal limits i.e. 5-34 IU/L,

According to selection criteria, the subjects were placed into gingivitis or periodontitis group.

Exclusion Criteria[1]:

Presence of any systemic disease that could influence the course of periodontal disease AST/SGOT levels like leukaemia, febrile conditions, chronic respiratory, cardiac, liver, muscular or any other significant systemic disease[7], Current smokers or ex-smokers since at least six months, Pregnancy[7], inability of the persons to cooperate because of their physical or mental status or daily routine, intake of antibiotics or anti-inflammatory drugs in the last 6 months[8], full crown, orthodontic bands or denture clasps on teeth, disease severity requiring periodontal therapy other than SRP, periodontal therapy other than standard prophylaxis during the previous 6 months[8], an inadequate number of qualifying diseased and healthy sites.[8]

Selection of test site:

Test site was selected according to following criteria[9]

a. Easily approachable site such as anterior or premolar or first molar area.
b. Upper or lower, right or left quadrant tooth, anterior or posterior tooth i.e. tooth selection was done either from 16-26 or 36-46.
c. GI>2[9]
d. No loss of attachment and true pocket but if pseudo pocket was present then probing pocket depth should not be more than 3 mm in Gingivitis group.
e. Clinical Loss of attachment (CAL) of at least 3-4 mm and probing pocket depth (PPD) of 5-8 mm. for Periodontitis group.

Data collection:

Data collection was done using Plaque index (PI) (Silness P and Loe H 1967)[10], Gingival index (GI) (Loe H and Silness J 1967)[10], Probing pocket depth (PPD), Clinical attachment level (CAL) as parameters.

GCF Collection:

Patient was asked to rinse mouth with water. Prior to the collection of GCF, any supracrevicular plaque and soft deposits from the test sites were removed carefully without causing trauma to the gingival crevice. If any hemorrhage was evident after this procedure, no fluid was collected from that site. The area was then thoroughly irrigated with distilled water, isolated by cotton rolls and dried by a stream of air. About 15-20 minutes after isolation of teeth, a ring of clear GCF was seen at the gingival margin and interdental areas. Hirschmann micropipette **(Figure 4b)** was placed extra sulcularly parallel to tooth surface and GCF was collected up to first marking of micropipette corresponds to 1µl of GCF volume **(Figure 1)**.

GCF collection was done twice in each patient. Once it was done just before starting the treatment in the patients fulfilling the inclusion criteria another was done after completion of treatment (after 3 months) when all the signs and symptoms of gingivitis in gingivitis group and periodontitis in periodontitis group were eliminated and periodontium became healthy. During the treatment period of 3 months, patient was revaluated after every 1week and if required SRP was repeated until the periodontal tissue became healthy.

Test Procedure:

Estimation of enzymatic activity: The Erba SGOT (SGOT or AST) kit **(Figure 3)** was used for the in vitro quantitative estimation of AST activity. This kit is based on the reference method of the International Federation of Clinical Chemistry (IFCC).[4] AST activity is measured photometrically by measuring transamination of aspartic acid and oxaloacetic acid. AST activity was measured on semiautomated autoanalyzer **(Figure 2)**.

Working reagent (available in ERBA SGOT kit) was allowed to attain 37*C.

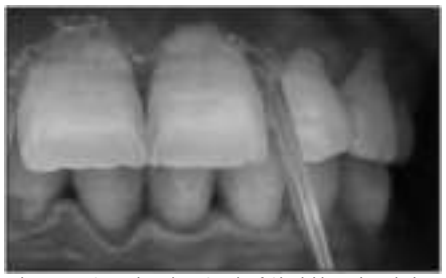


Fig 1 : Pre-Operative PhotoGraph of Gingivitis Patient during GCF collection

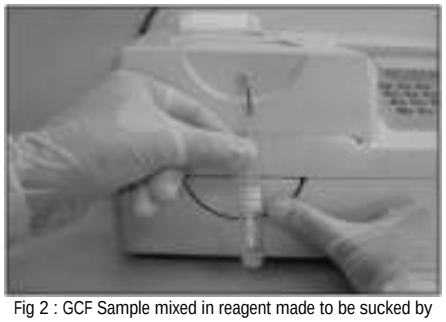


Fig 2 : GCF Sample mixed in reagent made to be sucked by autoanalyzer

Fig 3 : Reagent bottle and Aqua - 4 supplied in Erba AST Kit

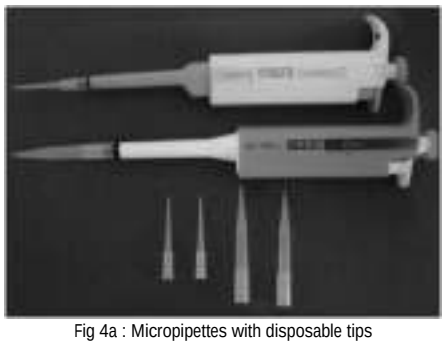


Fig 4a : Micropipettes with disposable tips

Fig 4b : Hirschmann Micropipette with caliberations in u1

GCF sample from Hirschmann micropipette was transferred into test tube having 100 µl normal saline for dilution of GCF sample. With the help of disposable tips of micropipette **(Figure 4a)** 500µl of AST (SGOT) regent mixed in the diluted 50µl GCF sample and shacked to get the uniform homogenous sample. This homogenous sample is made to be sucked by semi automated autoanalyzer.

Principle:

The principle used in this enzyme assay was,

AST
2-oxoglutarate + L-aspartate L-Glutamate+ Oxaloacetate
MDH
Oxaloacetate + NADH Malate + NAD
LDH
Sample pyruvate + NADH L-Lactate +NAD
MDH: Malate dehydrogenase
LDH: Lactate dehydrogenase

The rate of NADH consumption is measured photometrically and is directly proportional to AST activity in the sample. A standard curve of absorption of light by constituents of solution per unit time at 340 wavelengths in the GCF sample is obtained along with estimated AST value in the GCF through the printer attached with the autoanalyser. The data collected were recorded on the prepared proforma.

After completion of the study, summations and statistical calculations were performed and presented in tables.

TABLE 1: Test of significance (Paired't' test) of various parameters in Gingivitis group

Parameters	Mean ± SD		% Reduction	't' cal value ('t'tab (18,0.01) = 2.88)	'p' value
	Baseline	After 3 months			
PI	1.44 ± 0.3706	0.8625 ± 0.3845	40.1%	5.93	P* < 0.01
GI	2.09 ± 0.1468	1.13 ± 0.3193	45.93%	12.31	P* < 0.01
AST	1529.11 ± 364.16	494.7 ± 273.4	67.65%	12.47	P* < 0.01

p* < 0.01-Statistically very significant

Table 2: Coefficient Correlation Between Different Parameters In Gingivitis Group At Baseline (Karl Pearson's Correlation Coefficient Test)

S. No.	Parameters	'r' value	't' cal value	't'tab value	'p' value
1.	PI and GI	0.64	3.5	t(18, 0.01) = 2.88	p* < 0.01
2.	PI and AST	0.2967	1.32	t(18, 0.1) = 1.73	p** > 0.1
3.	GI and AST	0.7337	4.58	t(18, 0.01) = 2.88	p* < 0.01

p* < 0.01-Statistically very significant, p** > 0.1-Statistically insignificant

Table 3: Coefficient Correlation Between Different Parameters In Gingivitis Group After 3 Months (Karl Pearson's Correlation Coefficient Test)

S. No.	Parameter	'r' value	't' cal value	't'tab value	'p' value
1	PI and GI	0.25	1.73	t(18,0.05) = 2.1, t(18,0.1) = 1.73	P* > 0.05, p** = 0.1
2	PI and AST	0.002	1.06	t(18,0.1) = 1.73	P*** > 0.1
3	GI and AST	0.87	3.71	t(18, 0.01) = 2.88	p? < 0.01

p* > 0.05-statistically insignificant, p** = 0.1-Statistically weakly significant, p*** > 0.1-Statistically insignificant, p? < 0.01- Statistically very significant.

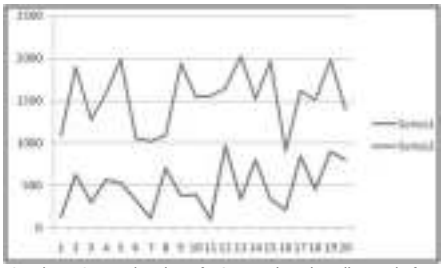
Table 4: Test Of Significance (Paired't' Test) Among Various Parameters In Periodontitis Group

Parameter	Mean ± SD		% Reduction	't'cal value ('t'tab(18,0.01) = 2.88)	'p' value
	Baseline	After 3 months			
PI	1.7 ± 0.4022	0.9625 ± 0.4388	43.38%	3.74	p* < 0.01
GI	2.1375 ± 0.2497	1.1 ± 0.3183	48.54%	12.65	p* < 0.01
PPD	3.76 ± 1.0338	2.75 ± 0.7988	28.26%	7.326	p* < 0.01
CAL	4.21 ± 0.9222	3.225 ± 0.7385	24.47%	6.7959	p* < 0.01
AST	3276.91 ± 1350.25	1658.22 ± 539.67	49.4%	6	p* < 0.01

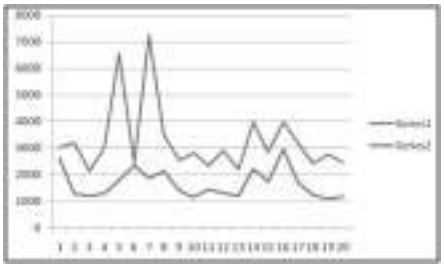
p* < 0.01-Statistically very significant

Results

Though this study was conducted on 40 patients (20 in gingivitis group and 20 in periodontitis group). There were statistically significant reductions in all the periodontal parameters as well as AST levels in both Gingivitis and Periodontitis groups (p<0.01) after SRP. Graphic representation of AST levels in 20 patients were compared at baseline and after 3 months of treatment in both gingivitis and periodontitis groups and a marked reduction in AST level was found in both groups **(Graph 1 and 2)**. On correlating the clinical parameters with AST levels, it was found that there was a statistically very significant correlation (p<0.01) between GI and AST levels before and after treatment in Gingivitis group. In Periodontitis group there was statistically very significant correlation (p<0.01) before treatment; but 3 months after SRP though a positive correlation was found, it was statistically insignificant (p>0.01).



Graph 1 : Curve showing of AST Levels at baseline and after 3 months in Gingivitis Patients
Series 1 - Baseline, Series 2 - After 3 Months



Graph 2 : Curve showing of AST Levels at baseline and after 3 months in Periodontitis Patients
Series 1 - Baseline, Series 2 - After 3 Months

In Periodontitis group correlation between PPD and AST at baseline was statistically significant (p<0.05) but 3 months after treatment a positive but weak correlation was found (p<0.1).

The correlation between CAL and AST though insignificant at baseline, was statistically significant (p<0.01) after treatment. Correlation between sex and AST was found insignificant (p>0.05), interpreting there was no effect of sex on AST concentration levels.

A higher mean AST level was observed in Periodontitis group than that of Gingivitis group and this difference was statistically significant (p<0.01) at baseline and after 3 months of SRP.

Discussion

Recent longitudinal studies have demonstrated that the progression of periodontal disease is episodic in nature. It is important for a clinician to be able to identify a stable or active site to evaluate the risk of further deterioration

Hence, the present study was carried out to correlate the AST level with periodontal parameters at selected test sites (groups) on two successive visits before and after 3 months of SRP.

To measure the effect of SRP the paired 't' test was applied and a statistically very significant reduction (p<0.01) was found suggesting that SRP and motivation for oral hygiene measures helped in improving the periodontal health of subjects **(Table 1, 4)**. This is established in our study also and is in agreement with several studies like those of Li R (1992)[11], Page Roy C (1992)[12].

On correlating AST levels with clinical parameters by Karl Pearson correlation coefficient test there was very significant correlation between AST level and GI (p<0.01) in gingivitis and periodontitis

Table 5: Coefficient Correlation Between Different Parameters In Periodontitis Group At Base Line (Karl Pearson's Correlation Coefficient Test)

S. No.	Parameter	'r' value	't' cal value	't'tab value	'p' value
1.	PI and GI	0.072	0.306	t(18,0.1) = 1.73	p* > 0.1
2.	PI and PPD	0.1757	0.75	t(18,0.1) = 1.73	p* > 0.1
3.	PI and CAL	0.24	1.05	t(18,0.1) = 1.73	p* > 0.1
4.	PI and AST	0.0163	0.069	t(18,0.1) = 1.73	p* > 0.1
5.	GI and PPD	0.541	2.71	t 18,0.05 = 2.1 t18 0.01 = 2.88	p** < 0.05, p*** > 0.01
6.	GI and CAL	0.37	1.69	t(18,0.1) = 1.73	p* > 0.1
7.	GI and AST	0.908	9.28	t(18,0.01) = 2.88	p? < 0.01
8.	PPD and CAL	0.75	4.79	t(18,0.01) = 2.88	p? < 0.01
9.	PPD and AST	0.53	2.65	t(18, 0.05) = 2.1 t(18 0.1) = 1.73	p** < 0.05, p*** > 0.01
10.	CAL and AST	0.33	1.48	t(18,0.1) = 1.73	P*** > 0.1

p* > 0.1-Statistically insignificant, p** < 0.05-Statistically significant, p*** > 0.01-Statistically insignificant, p? < 0.01- Statistically very significant

Table 6: Coefficient Correlation Between Different Parameters In Periodontitis Group After 3 Months (Karl Person's Correlation Coefficient Test)

S. No.	Parameters	'r' value	't' cal. value	't' tab value	'p' value
1.	PI and GI	0.240	1.57	t(18,0.1) = 1.73	p* > 0.1
2.	PI and PPD	0.375	1.69	t(18,0.1) = 1.73	p* > 0.1
3.	PI and CAL	0.048	2.038	t(18,0.1) = 1.73	p* > 0.1
4.	PI and AST	0.005	0.02	t(18,0.1) = 1.73	p* > 0.1
5.	GI and PPD	0.37	1.7967	t(18,0.1) = 1.73 t(18, 0.05) = 2.1	p** < 0.1, p*** > 0.05
6.	GI and CAL	0.16	0.68	t(18,0.1) = 1.73	p* > 0.1
7.	GI and AST	0.306	1.36	t(18,0.1) = 1.73	p* > 0.1
8.	PPD and CAL	0.323	1.44	t(18,0.1) = 1.73	p* > 0.1
9.	PPD and AST	0.382	1.75	t(18,0.1) = 1.73 t(18, 0.05) = 2.1	p** < 0.1, p*** > 0.05
10.	CAL and AST	0.83	6.32	t(18,0.01) = 2.88	p** < 0.01

p* > 0.1-Statistically insignificant, p** < 0.1-Statistically weakly significant, p*** > 0.05-Statistically insignificant

sites **(Table 2, 3, 5)** before treatment suggesting that AST level is correlated with severity of gingival inflammation. This observation is in agreement with the findings of the studies of Kolte Rajashri (2003)[4].

Though a positive correlation was present in Periodontitis group after treatment between AST and GI **(Table 6)** but it was statistically insignificant (p>0.1). This might be due to presence of inflammation in deep pockets. SRP reduced the inflammation but might be unable to completely eliminate it in deep pockets. Presence of inflammation is believed to be related to the penetration of antigenic substances via gingival sulcus and junctional epithelium. Even in clinically healthy gingiva with GI scores '0' small foci of inflammatory cells are found in the connective tissue near the base of the sulcus. According to study performed by Page and Schroder (1975), histologically a classical vasculitis of vessels subjacent to JE and gingival sulcus, extracellular presence of serum protein and alteration of most coronal portion of JE could be seen[4]. Since, deep periodontal pocket having larger surface area are exposed to external environment, a higher concentration of AST is expected. This finding also supports that only SRP may not be a sufficient treatment modality for complete debridement of tooth surface and inflammed periodontal tissue; an additional surgical approach may be required for elimination of pocket.

A positive correlation was found between PI and AST in pre and post treatment gingivitis and periodontitis sites, but was statistically insignificant (p>0.1) **(Table 2, 3, 5, 6)**. This can be explained on the basis of the pathogenicity of plaque supporting the specific plaque hypothesis (Loesche WJ et al. 1976). Specific pathogenic microorganisms in the plaque cause tissue destruction which are responsible for the release of AST in the GCF. A study performed by Kuru B. and Yilmaz S. et al. (1999)[13] concludes that P. gingivalis, Actinobacillus actinomycetemcomitance and Provetella intermedia were detected in AST positives (AST>800µIU) while Actinobacillus actinomycetemcomitance species were found in AST negative sites (AST<800 µIU). An another study of Kuru B. et al. in (1999) shows that C. rectus, E. corrodence, F. nucleatum, Capnocytophaga species, P. intermedia and P. gingivalis were significantly higher in positive than negative sites. Additionally PI measures the amount and extent of plaque on the tooth surface supragingivally only. This may be the reason for insignificant correlation between AST and PI. In their studies Silva Emilio Barbosae et al.(2003)[14] have also found no statistical correlation between AST levels and PI.

Karl Pearson correlation coefficient test was applied to find the correlation between PPD and AST levels in pre-and post treatment Periodontitis sites **(Table 5, 6)**. A positive and statistically significant association (p<0.05) was found at pretreatment sites. This finding suggest that increased periodontal destruction resulted by microbial activity leads to increased AST concentration. Study performed by Persson et al. (1995)[8] also supports this statement. Post treatment PPD values were found to be weakly correlated (p<0.1, p>0.05) with AST values. After SRP when microbial load was reduced, periodontal destruction was also reduced which resulted in the decreased AST concentrations in GCF but reduction was not much significant. This might be due to residual inflammation and ulcerations in the tissues even after SRP the deep pockets. SRP is not sufficient to remove the inner lining epithelium of the pocket. In this study there is only a maximum of 1.01 mm. reduction in pocket depth, and remaining pocket depth is difficult to be maintained by the patient. AJ Smith et al. (1998) have also found no statistically significant correlation between PPD and AST level.[15]

Karl Pearson correlation coefficient test was applied in Periodontitis sites to correlate AST with CAL at baseline, a positive correlation was found though it was not statistically significant, but a statistically very significant correlation was found at post treatment stage between CAL and AST (p<0.01). This finding suggests that resolution of inflammation at diseased site along with clinical attachment gain is strongly associated with decreased AST levels. Chambers DA et al. (1991)[16] in their study have also found a positive correlation between AST and CAL. The weak correlation at the baseline may be due to presence of more inactive sites at the time of GCF collection. Loss of attachment is the resultant of cumulative effect of periodontal diseases in the past. The present CAL may or may not be associated with active disease status. A longitudinal study is required to truly correlate CAL with AST.

However the detection of the disease at the earliest stage is not possible with the clinical parameters. It requires a very sensitive and specific test. In this aspect AST has got very important role to detect the, quiescent, active and vulnerable sites for periodontal destruction.

Conclusion

A reduction in clinical parameter scores and enzymatic level following periodontal therapy is an indicator of a positive correlation between AST levels

and periodontal disease. Effective control of inflammatory process by the reduction of pathogenic bacteria can be provided by early diagnosis at subclinical level with the help of AST estimation. This can be used in subjects to increase awareness and motivation which can arrest the disease process at an early stage, before it becomes irreversible.

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