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Original Article

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A Comparison of Serum Immunoglobulins Lvelels in Aggressive Periodontitis and Chronic Periodontitis

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Abstract

Background: Classification of 2017 of Periodontal and Peri-Implant disease and Conditions by AAP and EFP, Aggressive periodontitis and Chronic periodontitis was grouped under a single category. Unlike 1999workshop where Periodontitis was reclassified as chronic, aggressive (localised and generalised), necrotising and as a manifestation of systemic disease wherein Aggressive periodontitis and Chronic periodontitis were differentiated on the age of onset, rapid rate of progression, the composition of subgingival microflora, familial aggregation, racial influence and immuneresponse. In this study we aim to show a statistically significant difference in serum antibodies level of both Aggressive periodontitis and Chronic periodontitis, which could be suggestive of difference in implicated primary pathogen and frequency and tissue invasion of microorganism. **Subjects and Methods:** Two groups consisting of 10 patients each were formed. Group A consisted of patients with aggressive periodontitis and group B consisted of chronic periodontitis, blood samples were taken from both the groups and checked for IGG, IGA, IGM titres. **Results:** Both the groups were compared for the levels of immunoglobulin levels, group A showed a statistically significant raise in the levels in comparison to group B. While IGG levels were beyond the normal levels in group A. **Conclusion:** As treatment of aggressive periodontitis should differ from that of chronic periodontitis on the basis of immunological response. Hence clubbing Aggressive periodontitis and Chronic periodontitis in same category in The New Classification from the 2017 can not be justified.

Keywords: Aggressive periodontitis, Chronic periodontitis, immunoglobulins, immuneresponse.

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Introduction

The primary function of the Immune system is to eliminate infectious agents & minimize the damage they cause. It ensures that most infections in normal individuals are short-lived & leaves little permanent damage. Pathogens use many modes of transmission & reproduction, so the immune system has evolved many ways responding to them. Some pathogens evoke 'humoral immunity', like bacteria evade these formidable defenses by being intracellular pathogens & replicating within the host cells. 'B' cells are responsible for the humoral arm of the adaptive immune system. Immunoglobulins are an essential component of humoral immunity. They are present in serum, tissuefluid or on cell membranes. Amongst the five classes of Immunoglobulin namely IgG, IgA, IgM, IgD, IgE, the immunoglobulin IgG is the predominant immunoglobulin in normal human serum & IgA is predominant immunoglobulin in seromucous secretions.

Periodontitis is defined as an inflammatory disease of supporting tissues of teeth caused by specific microorganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with periodontal pocket formation, gingival recession or both.[1]

Periodontitis is a chronic multifactorial inflammatory disease associated with dysbiotic plaque biofilms,[2-5] and characterised by the progressive destruction of the tooth-supporting apparatus. Periodontitis is characterised by inflammation that results in the loss of periodontal attachment. While the formation of bacterial biofilm initiates gingival inflammation, the disease of periodontitis is characterised by three factors: • The loss of periodontal-tissue support, manifested through clinical attachment loss (CAL) and radiographically assessed alveolar bone loss; • The presence of periodontal pocketing; • Gingival bleeding

In the 1999 International Workshop for a Classification of

Periodontal Diseases and Conditions, the authors of the Consensus Report on Chronic Periodontitis stated that chronic periodontitis is "An infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment, and bone loss.[1] It is characterized by pocket formation and/or gingival recession."

In addition, the consensus report stated that periodontitis can be further characterized by extent and severity: "As a general guide, severity can be categorized on the basis of the amount of clinical attachment loss (CAL) as follows: Slight = 1 to 2 mm CAL, Moderate = 3 to 4 mm CAL, and Severe = ≥5 mm CAL." Numerous important studies since 1999 have used similar parameters to define periodontitis. For example, the recent epidemiologic studies outlining the prevalence of periodontitis in the United States used attachment loss parameters to define various severities of periodontitis.[6,7]

The 1999 Armitage review summarized the rationale for the introduction of the diagnostic terms "chronic periodontitis" and "aggressive periodontitis" as the recommended nomenclature for the two principal forms of destructive periodontal disease.[8-11]

Chronic Periodontitis[1]	**Aggressive Periodontitis[9]**
• Most prevalent in adults, but can occur in children and adolescents • Amount of destruction is consistent with the presence of local factors (i.e., primary and secondary etiologic factors) • Subgingival calculus is a frequent finding • Associated with a variable microbial pattern • Slow to moderate rate of progression, but may have periods of rapid destruction • Can be further classified on the basis of extent and severity • Can be associated with predisposing factors (e.g., tooth-related and iatrogenic factors) • May be modified by and/or associated with systemic diseases (e.g., diabetes mellitus, HIV infection) • Can be modified by factors other than systemic diseases such as cigarette smoking and emotional stress	• Except for the presence of periodontitis, patients are clinically (medically) healthy • Rapid attachment loss and bone destruction • Familial aggregation • Secondary features (generally but not universally present): ○ Amounts of microbial deposits (biofilm) are inconsistent with the severity of periodontal tissue destruction ○ Elevated proportions of Actinobacillus actinomycetemcomitans and in some populations Porphyromonas gingivalis ○ Phagocyte abnormalities ○ Hyper-responsive macrophage phenotype (elevated levels of PGE2 and IL-1β) ○ Progression of attachment loss and bone loss may be self-arresting • Discrimination of localized versus generalized forms of aggressive periodontitis as unique "subclassifications"

There of it was affirmed the use of the terms "chronic periodontitis" and "aggressive periodontitis" as separate, distinct clinical entities, both presenting with signs of periodontal destruction and inflammation.

In classification of periodontitis, published in 1999, shortcomings including substantial overlap and difficulties in implementation. The New Classification from the 2017 World Workshop on Periodontal and Periimplant Disease and Conditions ("the World Workshop") reviewed the scientific evidence and reached four main conclusions: 1. There is no evidence of a specific pathophysiology that enables the differentiation of cases as "aggressive" or "chronic" periodontitis or provides guidance for different kinds of intervention. 2. There is little consistent evidence that aggressive and chronic periodontitis are different diseases. Based on these findings, a new periodontitis classification clubs previously described as "chronic" and "aggressive" under the single category of "periodontitis".

If Aggressive periodontitis and Chronic periodontitis are a similar disease then the treatment modality will also be same. Hence the use of antibiotics should be negated. In this study we aim to defy that Aggressive periodontitis and Chronic periodontitis can be addressed as a single category as the immunologic response in both the disease vary. Thereof their treatments are also variable.

Subjects and Methods

Twenty systemically healthy patients from the O.P.D of Department of Periodontology were selected. A detailed medical and dental history was elicited from all the patients. The gingival index (Loe and Silness) values were determined and recorded. The Shick and Ash Plaque index was used to assess the plaque. Probing pocket depth (gingival margin to the base of the gingival sulcus or pocket) was measured to the nearest mm on 6 sites per tooth using a William's periodontal probe and recorded. Based on the clinical parameters assessed, the subjects were grouped as follows:

Group A comprised of 10 patients who fulfilled the diagnostic criteria of Aggressive periodontitis as stated in 1999 International Workshop for a Classification of Periodontal Diseases and Conditions. Ann Periodontol 1999.Group B comprised of 10 patients who fulfilled the diagnostic criteria of Chronic periodontitis.

Blood samples were taken from patients of both the groups (A and B) on the first day after establishing a definitive diagnosis. The blood samples were then sent to nearby Diagnostic Lab for evaluating the antibodies titre using Polyethlene glycol enhanced Immunoterbidimetric method.

Results

As shown in [Figure 1] there was a statistically significant difference between IGA titres, group A displayed higher levels than group B which is in accordance with the studies of Ebersole E.L et al.[12,13] Similar findings were observed in Graph 2 ie the IGM titres were statistically significantly raised in group A than group B. But it should be stated here that though the group A showed significantly higher titres of IGM & IGA the average was well within the normal range. As shown in Graph 3 IGG titres were raised in group A significantly higher than group B and above the normal range. this is in accordance with studies of Ebersole E.L ,et al.[12,13]

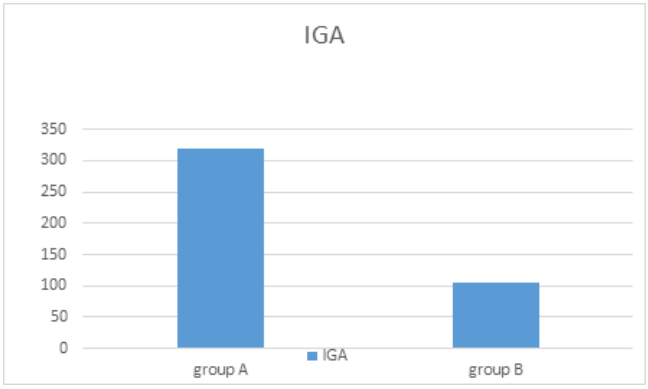

Figure 1: IGA comparison in group A &B.

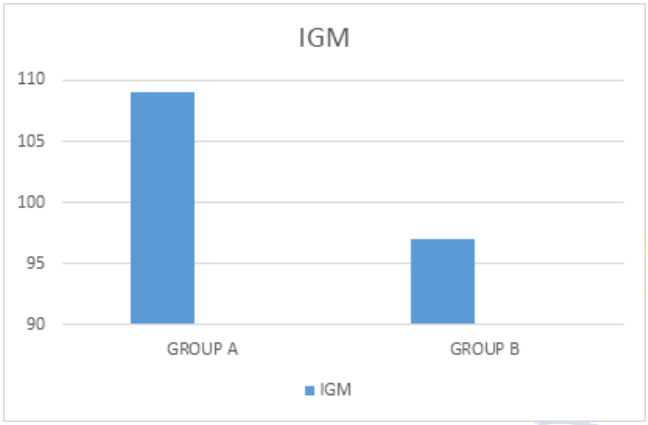

Figure 2: IGM comparison in group A &B.

As was observed that Group A which constituted the aggressive patients had marked increase in the serum antibodies level (IGG, IGM, IGA) which was almost double in some cases. Hence a definitive immunologic respose was seen in patients with aggressive periodontitis. In Group B that constituted the chronic periodontitis patients, the serum antibodies level (IGG, IGM, IGA) was almost unaltered. Some patients who showed an increase the numbers were only marginally raised. Hence there was no or very minimal immunologic response in chronic periodontitis patients.

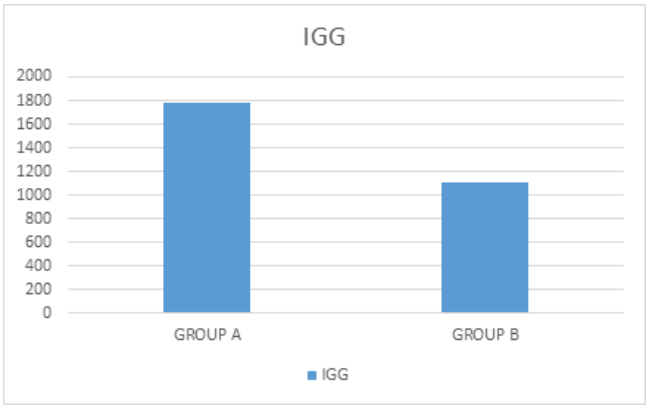

Figure 3: IGG comparison in group A &B.

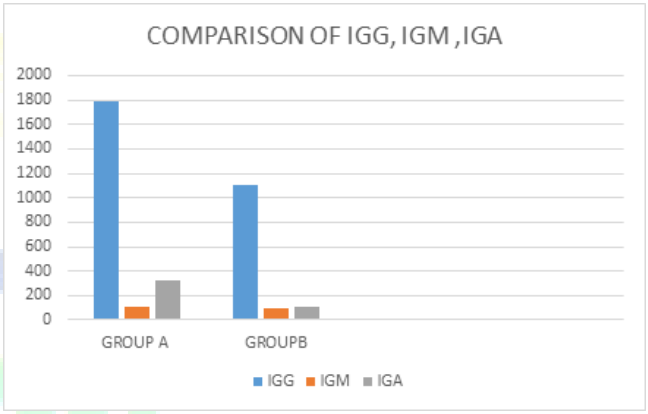

Figure 4: IGG IGM IGA comparison in group A &B.

Table 1: ?

Immunogloblins	Group A	Group B
IGG (average)	1784 mm/ dl	1108 mm/ dl
IGM (average)	109 mm/ dl	97 mm/ dl
IGA (average)	320 mm/ dl	105 mm/ dl

Normal Range of IGG: 650-1600 mm/ dl; IGM 50-300 mm/ dl ; IGA 40-350 mm/ dl

Discussion

Immune system has evolved to protect us from pathogens. Intracellular pathogens infect individual cells, whereas extracellular pathogens divide extracellularly within tissues or the body cavities. A highly discriminatory immune system is fundamental to survival. The Immune system has a powerful collection of defense mechanisms to protect against potential invaders that would otherwise take advantage of the rich source of nutrients provided by the host.

It has been suggested that the humoral immune response has a protective role in the pathogenesis of periodontal disease. Alterations in specific IgG and IgA responses both locally at inflamed sites and systemically have relevance in disease progression,[10,11] although the exact mechanisms are complex and insufficiently understood. GCF antibodies are both serum-derived,[12-15] and locally produced by the abundant plasma cells of the diseased periodontal tissue.[15-17]

As given in [Table 1] it was seen that there was statistically significant difference between the levels of IGG, IGA, IGM in group A & group B. IGG levels were markedly raised in group A above the normal rage , and were significantly higher than group B which was well with in the normal range .IGA and IGM were within the normal range but there was a statistically significant difference in the levels of both between two group group A being higher. A study conducted by Kaslick et al.[10] Revealed increased levels of serum IgA, IgG and IgM in Aggressive periodontitis patients. It has been reported that the protein and lipid carbohydrate antigens of Porphyromonas gingivalis induce two distinct patterns of

IgG and IgA subclass responses in inflamed gingiva .16 Therefore it is possible that the change in IgG and IgA obtained is due to the change of microbial antigens or possible other mitogens involved at various stages of periodontitis. B cell differentiation and the regulation of the switch to different immunoglobulin isotypes is cytokine-driven.[17,18] Transforming growth factor-beta 1 (TGF-β1) and/or IL-4 increase the level of a germ-line transcript, and then B cells are induced to switch from membrane IgM to membrane IgA.[19]

These antibodies may influence the oral microbiota by interfering with adherence or by inhibiting bacterial metabolism. Furthermore, the IgG antibodies may enhance phagocytosis and killing of oral microorganisms through activation of complement or opsonisation.It has been demonstrated that systemic immunization of animals with periodontopathogens may reduce the colonization of these bacteria in the gingival crevice and reduce periodontal destruction .IgG antibodies may enhance phagocytosis and killing of oral microorganisms through activation of complement or opsonisation .It has been demonstrated that systemic immunization of animals with periodontopathogens may reduce the colonization of these bacteria in the gingival crevice and reduce periodontal destruction. Results are suggestive of Aggressive periodontitis occurs because there is disturbance of immune system.

Aggressive periodontitis is closely related to the quality and the susceptibility of the host, caused by abnormal immunocompetent cells that have disturbance of immune response. As consequence, the susceptibility of patients towards aggressive periodontitis is increased.

Results are suggestive Aggressive periodontitis occurs because there is definitive immune response difference from chronic periodontitis. The immunoglobulin response in group A ie; the aggressive periodontitis is raised. Showing a definitive effect on immunologic parameters. Treatment of aggressive periodontitis is aimed at reducing chronically inflamed tissue and the number of microbes in the deep vertical periodontal pockets. As with other periodontal diseases, initial therapy begins with the establishment of good oral self-care. A regimen of scaling and root planing and the administration of systemic antibiotics has been successful in treating patients with high levels of A. actinomycetemcomitans. Surgical treatment in conjunction with systemic antibiotic therapy has been found effective in more advanced cases,[25] in the presence of A. actinomycetemcomitans and Bacteroides. Systemic tetracycline, minocycline, and metronidazole have been shown to be effective nonsurgical treatments as has the combination of amoxicillin and metronidazole.[23,24]

In addition to its antimicrobial effects, minocycline has been a useful treatment because of its inhibition of collagenase activity. In cases in which surgical intervention is indicated, periodontal regeneration has been shown to be effective. It is usually advisable to refer an adolescent patient with aggressive periodontitis to a periodontist for treatment. Therefore the modality of treatment of aggressive periodontitis should differ from that of chronic periodontitis. Hence clubbing Aggressive periodontitis and Chronic periodontitis in same category in The New Classification from the 2017 can not be justified.

Conclusion

As treatment of aggressive periodontitis should differ from that of chronic periodontitis on the basis of immunological response. Hence clubbing Aggressive periodontitis and Chronic periodontitis in same category in The New Classification from the 2017 can not be justified.

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