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Antibody response after single-visit full-mouth ultrasonic debridement *versus* quadrant-wise therapy

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Abstract

Introduction: The aim of this study was to compare serum antibody responses to periodontal pathogens after single-visit full-mouth ultrasonic debridement and quadrant-wise therapy.

Material and Methods: Thirty-six subjects with chronic periodontitis were randomized into three groups: quadrant-wise debridement in four visits, one-visit full-mouth debridement with water and with povidone iodine. Blood samples were collected before and immediately after treatment and 1, 3 and 6 months post-therapy. Serum antibody titres and avidity to *Porphyromonas gingivalis*, *Actinobacillus actinomycetemcomitans*, *Prevotella intermedia* and *Treponema denticola* were determined by enzyme-linked immunosorbent assay (ELISA) and thiocyanate ELISA, respectively.

Results: IgG titres to *P. gingivalis* significantly decreased at 1, 3 and 6 months in full-mouth debridement with water group, while significant reductions were seen only at 3 and 6 months after quadrant-wise debridement. Both full-mouth groups showed significant reduction in IgG titres to *A. actinomycetemcomitans* at 3 and 6 months. Significant increases in antibody avidity to *P. gingivalis* and *A. actinomycetemcomitans* were noted 3 months following full-mouth debridement with povidone.

Conclusion: Both full-mouth and quadrant treatments generally resulted in a decrease in antibody titres and increase in antibody avidity. Full-mouth debridement induced an earlier reduction of IgG titre to *P. gingivalis* and *A. actinomycetemcomitans*, than quadrant-wise therapy.

Key words: antibody avidity; antibody titres; chronic periodontitis; full-mouth disinfection; periodontal pathogens; povidone iodine; ultrasonic debridement

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Periodontitis is a multifactorial infection elicited by a complex of bacterial species and host immune responses. Several bacterial species, such as *Porphyromonas gingivalis*, *Actinobacillus actinomycetemcomitans*, *Prevotella intermedia* and *Treponema denticola*, are implied in chronic periodontitis (AAP 1996). Previous host response studies regarding antibody production generally reported higher levels of reactive antibody to periodontal pathogen in diseased patients (Mouton et al. 1981, Ebersole et al. 1982, Naito et al. 1985, Ishikawa et al. 1988, Mooney & Kinane 1994).

Non-surgical mechanical therapy constitutes the initial step in management of periodontal diseases, conventionally performed in a quadrant- or sextant-wise manner with 1–2 week intervals between the sessions. Mechanical therapy, either hand instrumentation or ultrasonic debridement, is the most common therapy for periodontitis and has well-documented success. However, the reports on the effect of scaling and root planing (SRP) on humoral responses are controversial. Some reported that antibody titres to *P. gingivalis*, *A. actinomycetemcomitans* (Ebersole et al. 1985, Mooney et al. 1995) and *P. intermedia* (Ebersole et al. 1985) increased post-therapy. Others showed that the mean antibody titres to *P. gingivalis* and *P. intermedia* significantly decreased after SRP with clinical

improvement (Tolo et al. 1982, Horibe et al. 1995).

Antibody avidity is a measure of the net binding strength between multivalent antigen and polyclonal antibodies. It reflects the functional activity of the antibodies, and increased relative avidity often occurs as a consequence of maturation of humoral response. There are inconsistent reports on the antibody avidity to *P. gingivalis*. Lopatin et al. (1992) found elevated IgG antibody avidity to *P. gingivalis* in chronic periodontitis patients compared with healthy controls. On the other hand, Chen et al. (1991) reported that generalized aggressive periodontitis patients had lower antibody avidity, which increased following non-surgical therapy. A study by Holbrook et al. (1996) noted low antibody avidity to *P. gingivalis* in chronic periodontitis, which did not change with therapy including a combination antibiotic regimen.

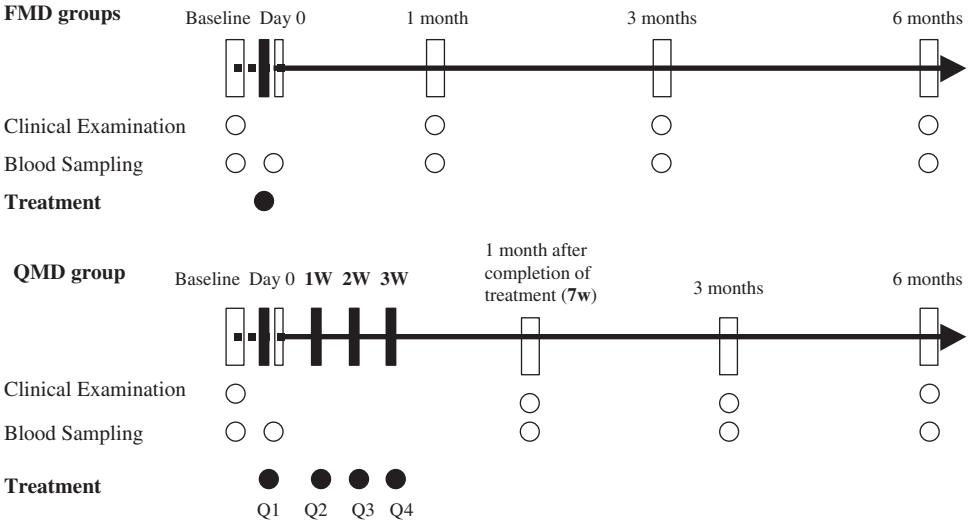
Quirynen and his co-investigators demonstrated that one-stage full-mouth disinfection (two, 2-h sessions within 24 h) resulted in significantly greater clinical and microbiological improvements than conventional quadrant-wise therapy at two-weekly intervals (Quirynen et al. 1995, 1998, 1999, 2000, Mongardini et al. 1999). The role of chlorhexidine in the one-stage full-mouth disinfection treatment was investigated by the same research group (Quirynen et al. 2000). The major effect of the one-stage full-mouth disinfection seemed to be related with the SRP of all four quadrants within 24 h, rather than to the adjunctive chlorhexidine regimen (Quirynen et al. 2000). In their study, both full-mouth treatments provided within 24 h resulted in superior improvements, than the conventional quadrant-wise therapy. However, contradictory results were reported in recently published papers (Apatzidou & Kinane 2004a, Apatzidou et al. 2004, Koshy et al. 2005, Wennström et al. 2005, Jervoe-Storm et al. 2006). Apatzidou & Kinane (2004a) compared the effect of a same-day full-mouth root planing excluding antiseptics with that of quadrant SRP at two-weekly intervals in 40 chronic periodontitis patients up to 6 months. They failed to find any difference between the two treatment protocols with respect to pocket depth reduction and gain in relative attachment level. Lately, Wennström et al. (2005) indicated that a single 1-h session of full-mouth ultrasonic debridement resulted in similar clinical improvements as quadrant SRP at weekly intervals. More recently, Jervoe-Storm et al. (2006) also demonstrated equally favourable clinical results following full-mouth root planing in two sessions within 24 h and quadrant root planing at intervals of 1 week. The discrepancy is explained by major differences in experiment design, especially in a risk control for microbial cross-contamination (Quirynen et al. 2006). Until now, above-mentioned clinical and microbiological investigations have suggested similar or superior results of full-mouth disinfection compared with conventional quadrant-wise therapy. However, few studies have noticed immunological response after full-mouth mechanical debridement. The purpose of the present study was to evaluate the humoral immune response after single-visit full-mouth ultrasonic debridement with or without additional antiseptics compared with conventional quadrant-wise debridement.

Material and Methods

Study population

The study population included 36 untreated chronic periodontitis patients, who attended the Periodontics Clinic at Tokyo Medical & Dental University. Informed consent was obtained from each subject. Each subject had at least five teeth and two pocket sites with probing depth $\geq$ 5 mm in each quadrant and radiographic evidence of bone loss. All subjects were generally healthy, non-smoking and had not received any periodontal treatment and/or systemic antibiotic administration during the previous 6 months.

The demographic details, clinical study design and treatment protocol are described in our earlier published report (Koshy et al. 2005). The trial design and the timing of clinical interventions and sampling are summarized in Fig. 1. Briefly, the present study was a randomized clinical trial comparing conventional quadrant-wise ultrasonic debridement (QMD) with full-mouth



Day 0 = immediately after the first treatment,
FMD = full mouth debridement, QMD = quadrant-wise mechanical debridement

Fig. 1. Treatment schedule.

ultrasonic debridement with water in a single visit (FMD+water) or full-mouth ultrasonic debridement with povidone iodine in a single visit (FMD+povidone). All subjects were recalled every month for re-inforcement of oral hygiene instructions.

All subjects were examined at baseline (before treatment), 1, 3 and 6 months after completion of treatment by a single calibrated and blinded examiner. Moreover, subgingival plaque and saliva samples were collected at baseline (before and immediately after treatment), and 1, 3, 6 months after for the detection of four putative periodontal pathogens: *P. gingivalis*, *A. actinomycetemcomitans*, *P. intermedia* and *T. denticola* by polymerase chain reaction as described before (Koshy et al. 2005). Simultaneously, blood samples were also obtained from each subject to determine the serum IgG antibody responses to the homologous organism.

Serum sample

Peripheral blood samples were collected from all subjects. Each blood sample was centrifuged at 1500 × *g* for 20 min. The serum was filtered through a sterile 0.45 mm diameter filter (Millex, Millipore Japan Ltd, Tokyo, Japan) and stored at −20°C until analysis.

Preparation of microorganisms

A. actinomycetemcomitans ATCC 43719, *P. gingivalis* 381, *P. intermedia* ATCC 25611 and *T. denticola* ATCC 33520 were employed in this study. *A. actinomycetemcomitans* ATCC 43719 was grown in brain heart infusion (BHI) broth (Difco, Sparks, MD, USA) supplemented with 1% (w/v) yeast extract at 37°C for 3 days in a 5% CO_2 atmosphere. *P. gingivalis* 381 and *P. intermedia* ATCC 25611 were grown in BHI broth (Difco) supplemented with vitamin K and haemin under anaerobic conditions (10% H_2, 5% CO_2 and 85% N_2) and harvested after 5 days. *T. denticola* ATCC 33520 was grown on New Oral Spirochete medium (NOS) under an atmosphere of 10% H_2, 10% CO_2 and 80% N_2. The microorganisms were harvested by centrifugation, washed three times with distilled water and lyophilized.

Sonicated whole cell extracts of periodontopathogens were prepared according to the method of Naito et al. (1984). Cells were suspended in saline (200 mg/ml, wet weight) and disrupted by sonication at 30 s intervals for a total sonication time of 3 min. at maximum output on ice. The sonicated cell suspensions were centrifuged and the supernatants were lyophilized.

Serum antibody titre measurement

Specific serum IgG titres were measured by ELISA as described previously (Ishikawa et al. 1988) using sonicated whole cell extracts of each periodontopathogens. Briefly, 96-well microtitre plates (EIA plate, Costar, Cambridge, MA, USA) were coated with sonicated extracts (10 μg/ml) in carbonate buffer and incubated 2 h at 37°C. After blocking with 2% bovine serum albumin (BSA) in carbonate buffer, plates were washed three times with phosphate-buffered saline (PBS)-T (1 × PBS, 0.05% Tween 20, pH 7.2). Serially diluted reference positive control serum (2^5–2^{14}, 100 μl per well) and single diluted (2^{10} for *P. gingivalis* and *A. actinomycetemcomitans*; 2^8 for *P. intermedia* and *T. denticola*) patient serum were added into each well in duplicate and the plates were incubated for 2 h at 37°C. Following incubation, the plates were washed again three times. Subsequently, 100 μl per well of alkaline phosphatase-conjugated goat antihuman IgG (Sigma, St. Louis, MO, USA) was added. Following incubation, plates were washed three times and developed with phosphate substrate (Sigma 104, St. Louis, MO, USA). The optical density was read using a Microplate reader (SOFT Max™, Sunnyvale, CA, USA) at 405 nm with a 650 nm reference wavelength. All serum samples collected from the same patient before and after treatment were assayed in duplicate and on the same plate. Calculation for the antibody titre was done according to the method of Ishikawa et al. (1988) and described more fully in Wang et al. (2005).

Antibody avidity measurement

Antibody avidity was determined by the dissociation of antigen–antibody binding in a way that is similar to the ELISA for the serum analysis described above. Patient serum was added in duplicate at a dilution of 2^7. After incubation with sera, the wells were treated with increasing concentrations of ammonium thiocyanate (0–8.0 M). The plates were incubated at 37°C for 60 min., washed three times and then the ELISA continued as before. Percentage of antibody binding was calculated for each concentration [(OD of thiocyanate treated well/OD of control well) × 100] of thiocyanate. The avidity was expressed by the calculation of thiocyanate concentration required to inhibit 50% of the bound antibody (ID50) (Pullen et al. 1986; Macdonald et al. 1988). All the samples from the same patient were tested together on the same plate.

Statistical analysis

The data was analysed with the subject as unit and reported as median and interquartile ranges. Non-parametric statistical comparisons were applied. All statistical comparisons between visits within group were performed with the Friedman ANOVA and the Wilcoxon signed rank-sum test for post hoc comparisons. Analysis of covariance was used for comparison of the changes in IgG titre and avidity between the three treatment groups adjusting for the potential difference in the baseline data. The comparisons of the changes of antibody responses (titre and avidity) post-treatment among the three treatment groups were assessed by Kruskal–Wallis ANOVA test. The α value was set at 0.05. All statistical analysis was carried out with the aid of statistical software (StatView® for Windows, Version 5.0, SAS Institute Inc., Cary, NC, USA).

Results

Effect of treatment on IgG antibody titres

The effects of treatments on serum IgG antibody titres are shown in Table 1. Clear significant changes in the IgG titres to *P. gingivalis* were observed between baseline and 1, 3 and 6 months after FMD+water ($p = 0.021$, Friedman ANOVA). The IgG titres to *P. gingivalis* significantly decreased from baseline to 1 month ($p < 0.01$, Wilcoxon's rank-sum test) and remained reduced at 3 and 6 months ($p < 0.01$, $p < 0.05$, respectively, Wilcoxon's rank-sum test). In FMD+povidone group, despite the IgG titre to *P. gingivalis* decreased slightly, no significant changes were seen. FMD+povidone group showed significant variations in the median IgG titres to *A. actinomycetemcomitans* from baseline to 3 and 6 months ($p = 0.04$, Friedman ANOVA). The IgG titres to *A. actinomycetemcomitans* significantly reduced at 3 and 6 months ($p < 0.05$, $P < 0.05$ respectively, Wilcoxon's

Table 1. Serum IgG antibody titers before and after treatment within each group

$N_{FMD+water} = 12$ $N_{FMD+povidone} = 12$ $N_{QMD} = 12$	Baseline	Times after periodontal treatment				
		Day 0	1 month	3 months	6 months	$p^{\dagger}$
P. gingivalis						
FMD+water	10.61 (9.95, 12.15)	10.95 (9.76, 11.59)	10.38 (9.29, 12.03)**	10.3 (9.06, 11.03)*	10.28 (9.4, 11.26)*	0.021
FMD+povidone	11.61 (10.82, 13.27)	11.46 (10.55, 13.45)	11.29 (10.56, 13.24)	11.68 (10.53, 12.83)	11.06 (10.49, 13.05)	NS
QMD	9.27 (8.95, 11.45)	9.66 (8.7, 10.79)	8.94 (8.65, 11.18)	9.08 (8.11, 10.46)**	8.92 (7.89, 10.98)*	0.089
A. actinomycetemcomitants						
FMD+water	7.37 (6.44, 10.79)	7.38 (6.19, 10.17)	7.49 (6.21, 10.44)	7.28 (6.01, 10.25)**	7.49 (6.02, 10.32)*	0.059
FMD+povidone	6.85 (6.12, 9.26)	6.73 (6.06, 8.95)	6.82 (6.07, 9.13)	6.49 (5.71, 9.11)*	6.42 (5.98, 8.07)*	0.04
QMD	6.94 (6.17, 8.96)	7.09 (6.51, 8.92)	7.02 (6.5, 8.87)	7.17 (6.12, 8.54)	6.89 (6.28, 8.33)	NS
P intermedia						
FMD+water	7.68 (7.14, 8.65)	7.8 (7.19, 8.77)	7.53 (7.12, 8.04)	7.68 (7.05, 8.06)	7.72 (6.82, 8.64)	NS
FMD+povidone	8.27 (7.67, 8.94)	8.14 (7.69, 8.9)	7.93 (7.68, 8.75)	8.26 (7.72, 8.79)	8.13 (7.72, 8.5)	NS
QMD	8.43 (7.4, 8.79)	8.62 (7.59, 8.82)	8.44 (7.41, 8.82)	8.41 (7.27, 8.8)	8.04 (7.25, 8.82)	NS
T. denticola						
FMD+water	7.44 (6.46, 8.79)	7.74 (6.73, 9.28)	7.53 (6.68, 8.74)	7.32 (6.88, 8.53)	7.52 (6.81, 9.02)	NS
FMD+povidone	7.54 (6.81, 7.98)	7.56 (6.85, 8.22)	7.39 (7.02, 7.95)	7.21 (6.91, 7.91)	6.96 (6.57, 8.2)	NS
QMD	7.9 (7.28, 8.52)	7.92 (7.35, 8.56)	7.87 (7.55, 8.64)	7.59 (7.18, 8.18)	7.53 (6.61, 8.16)*	0.048

Median (interquartile range).
†Friedman ANOVA.
*$p < 0.05$.
**$p < 0.01$ Wilcoxon's signed-rank test *versus* baseline.
FMD+water, full-mouth debridement with water; FMD+povidone, full-mouth debridement with povidone; QMD, quadrant-wise mechanical debridement; Day 0, immediately after the first treatment.
Bacteria tested: *Porphyromonas gingivalis*; *Actinobacillus actinomycetemcomitans*; *Prevotella intermedia*; *Treponema denticola*.

signed-rank test) in both FMD+water and FMD+povidone groups. For *T. denticola* and *P. intermedia*, the median IgG titres decreased at 1, 3 and 6 months in both FMD+water and FMD+povidone groups. However, no statistical significance was found.

In QMD group, a significant alteration in the IgG titre to *T. denticola* at 3 and 6 months after treatment was noted ($p = 0{,}048$, Friedman ANOVA). The IgG titres to *T. denticola* declined significantly at 3 and 6 months ($p < 0.05$, respectively, Wilcoxon's signed-rank test) compared with baseline. QMD group also showed a significant reduction in IgG titres of *P. gingivalis* at 3 and 6 months ($p < 0.01$, $p < 0.05$, respectively, Wilcoxon's signed-rank test) compared with those at baseline. Given the IgG titres to *A. actinomycetemcomitans* and *P. intermedia*, no significant changes were detected.

The comparisons of the reductions of IgG titres to the four periodontal pathogens among three treatment groups are displayed in Fig. 2. FMD+water group exhibited a significantly greater reduction in serum IgG titres to *P. gingivalis* at 1 month than those in FMD+povidone ($p < 0.05$). Furthermore, the reduction in IgG titres to *A. actinomycetemcomitans* was significantly greater ($p < 0.05$) at 3 months in FMD+water group than that in QMD group. On the other hand, the changes of IgG titres to *T. denticola* at 6 months after treatment were significantly greater in QMD than those in FMD+water group ($p < 0.05$). No significant difference was found in the IgG titre to *P. intermedia*.

Effect of treatment on IgG antibody avidity

The effects of treatment on serum IgG antibody avidity are shown in Table 2. On the whole, IgG antibody avidity increased following therapy. FMD+water group showed a significant increase in the avidity of *A. actinomycetemcomitans* and *P. intermedia* at 1 month ($p < 0.05$, respectively, Wilcoxon's signed-rank test). In FMD+povidone group, the antibody avidity of *A. actinomycetemcomitans* ($p < 0.05$) and *P. gingivalis* ($p < 0.05$) significantly increased after 3 months. A similar trend was also found in the QMD group. However, the changes failed to reach statistical significance.

There were no significant differences in the changes of antibody avidity among the three treatments groups.

Discussion

Effect of treatment on humoral response

P. gingivalis infection characterized patients with chronic periodontitis as well as aggressive periodontitis, and the sera of these patients exhibit high IgG titres to this bacterium (Mouton et al. 1981; Naito et al. 1984; Ishikawa et al. 1988). *P. gingivalis* (92%) was highly detected at baseline in the current subjects (Koshy et al. 2005). Both FMD and QMD treatments resulted in a significant reduction in the median IgG titres to *P. gingivalis*. This agrees with data reported by other investigators (Tolo et al. 1982; Naito et al. 1985; Aukhil et al. 1988; Horibe et al. 1995). However, a wide variation in the subjects' antibody response existed. Nevertheless, the present data are contradictory to those of Ebersole et al. (1985), who showed marked increases for *P. gingivalis*, *P. intermedia* and *A. actinomycetemcomitans* antibody titres at 2–4 months after SRP. In the current study, no significant increase was detected in IgG titres to tested microorganisms after both FMD and QMD treatments. Reports by Chen et al. (1991) and Mooney et al. (1995) pointed out that the effect of therapy on antibody titres may differ in subjects who are seropositive or seronegative before treatment, and that treatment induces a reduction in seropositive patients but an increase in the seronegative patients.

Antibody avidity, the net binding strength between multivalent antigen and polyclonal antibodies has been investigated in a number of infectious

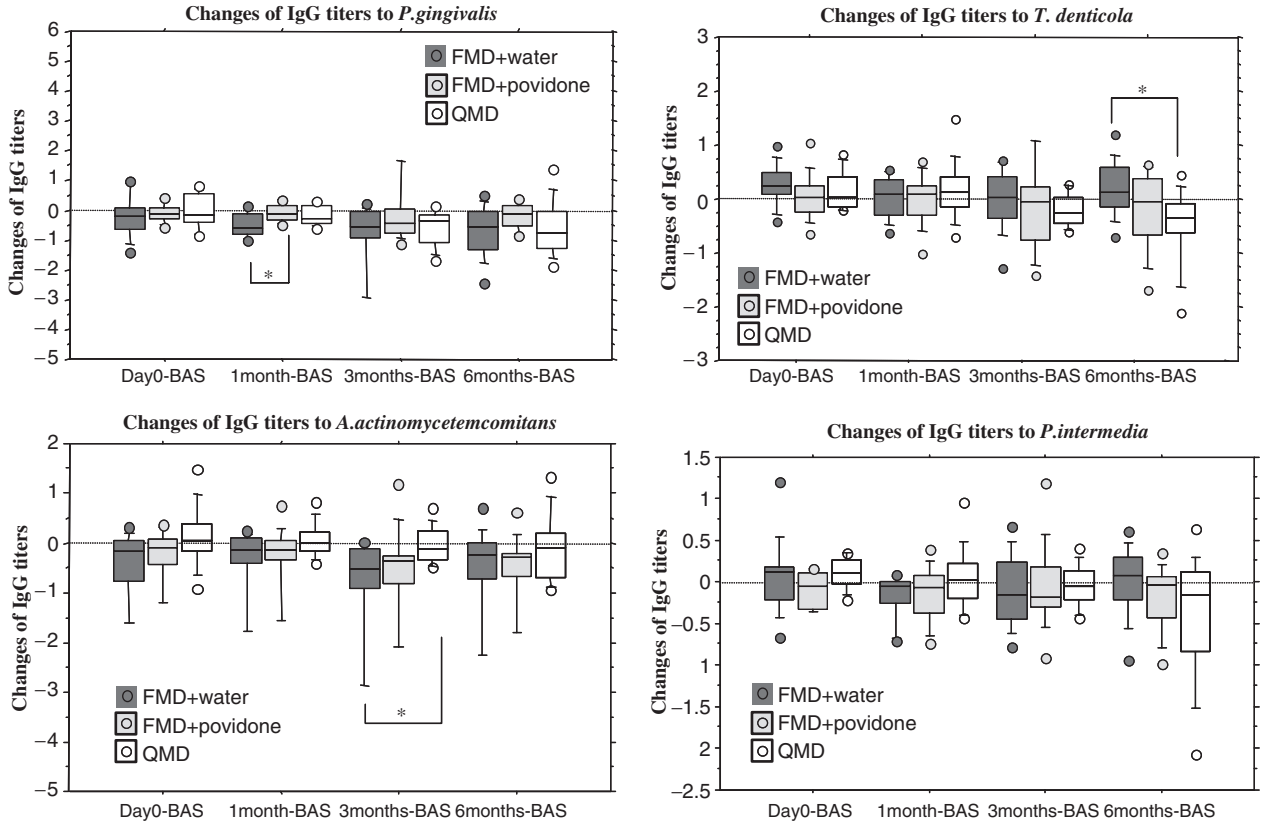


Fig. 2. Comparison of changes in IgG titres to periodontal pathogens among the three treatment groups. Box and whiskers plots show changes in IgG titres at each visit after treatment compared with baseline (BAS). The box refers to the 25th (bottom) and 75th (up) percentiles and the median is the horizontal line inside. Black circles represent outliers whereas asterisks stand for extreme observations. $^{*}p<0.05$ (Wilcoxon's signed-rank test).

diseases, both in relation to antibody titres and in terms of disease susceptibility and progression (Grzybowski et al. 1989, Meurman et al. 1992, Johnson et al. 1993). Successful treatment results in the elimination of the etiological agents and maturation of the immune system to produce antibodies with high avidity (Chen et al. 1991). Mooney et al. (1995) also found a significant increase in *P. gingivalis* IgG antibody avidity in chronic periodontitis patients, but no significant change in *A. actinomycetemcomitans* avidity. Antibody avidity generally tended to increase after treatment in our study, but wide variation existed among subjects. Antibody avidity to *A. actinomycetemcomitans* increased significantly after treatment in both FMD groups. A significant increase of antibody avidity to *A. actinomycetemcomitans* was reported at 6 months after initial treatment of full-mouth or quadrant SRP in both non-smoking and smoking chronic periodontitis patients (Apatzidou et al. 2005). In addition, the current report demonstrated a significant increase in the antibody avidity to *P. intermedia* at 1 month following FMD+water therapy. The findings from present study suggest full-mouth disinfection may effectively enhance antibody affinity maturation against periodontal pathogens.

Comparison of immunological effect of FMD and QMD treatments

There are few studies which have addressed the effect of full-mouth mechanical debridement on the immune responses to periodontal pathogens compared with the conventional quadrant-wise approach. A recent report by Apatzidou et al. (2004b) showed that there was a significantly greater reduction in IgG antibody levels to *P. gingivalis* and *T. denticola* in FM-SRP group than the Q-SRP group, whereas no significant differences in IgG avidity were found between the two groups. In the current study, FMD+water group completed in single visit exhibited significantly decreased IgG titres of *P. gingivalis* at 1, 3 and 6 months after treatment (Fig. 2). On the other hand, QMD completed within 4 weeks showed no significant changes at 1 month after completion of treatment (7 weeks from baseline). The significant reductions on IgG titres of *P. gingivalis* were noted at 3 and 6 months after (Fig. 2). In addition, FMD treatments induced a significantly greater reduction in IgG titres to *A. actinomycetemcomitans* at 3 months from baseline when compared with QMD (Fig. 2). On the other hand, QMD group exhibited greater changes in the IgG antibody titres to *T. denticola* at 6 months than those in FMD+water group (Fig. 2). These findings imply that full-mouth treatment seems to induce an earlier effect on humoral responses to *P. gingivalis* and *A. actinomycetemcomitans* compared with quadrant-wise debridement.

It was of interest to note that antibody avidity increased significantly to *A. actinomycetemcomitans* and *P. gingivalis* at 3 months in FMD+povidone group, indicating that povidone iodine may have a potentially beneficial effect on the humoral immune response.

On the basis of these results, it is concluded that both full-mouth and

Table 2. Comparison of IgG antibody avidity before and after FMD+water, FMD+povidone and QMD

$N_{FMD+water} = 12$ $N_{FMD+povidone} = 12$ $N_{QMD} = 12$	Baseline (BAS)	Day 0	1 month	3 months	6 months	Change			
						Day 0–BAS	1 month–BAS	3 months–BAS	6 months–BAS
P. gingivalis									
FMD+water	2.01 (1.4, 2.46)	1.83 (1.3, 2.42)	1.48 (1.28, 2.51)	1.5 (1.13, 2.29)	1.79 (1.1, 2.28)	0.03 (−0.1, 0.19)	−0.06 (−0.27, 0.04)	−0.11 (−0.37, 0.14)	−0.1 (−0.34, 0.02)
FMD+povidone	2.28 (1.57, 2.57)	2.27 (1.63, 2.6)	2.24 (1.58, 2.75)	2.41 (2.15, 2.77)*	2.12 (1.6, 2.68)	0.06 (−0.02, 0.15)	0.01 (−0.04, 0.04)	0.14 (0.04, 0.42)	0.02 (−0.19, 0.17)
QMD	1.27 (0.74, 1.77)	1.24 (0.77, 1.89)	1.32 (0.72, 1.84)	1.29 (0.77, 1.73)	1.12 (0.55, 1.67)	0.03 (−0.06, 0.18)	0.06 (0.01, 0.08)	0.03 (−0.07, 0.16)	−0.05 (−0.21, 0.1)
A. actinomycetemcomitants									
FMD+water	0.95 (0.51, 2.76)	1.24 (0.53, 3.08)	0.98 (0.64, 2.8)*	0.95 (0.57, 3.25)	1.13 (0.64, 2.74)	0.04 (−0.1, 0.64)	0.08 (0.02, 0.22)	0.1 (−0.1, 0.51)	−0.03 (−0.17, 0.53)
FMD+povidone	0.66 (0.56, 2.69)	0.69 (0.58, 3.02)	0.72 (0.59, 2.9)	0.80 (0.59, 3.22)**	0.66 (0.53, 2.69)	0.09 (−0.02, 0.26)	0.06 (0, 0.21)	0.21 (0.02, 0.53)	0.01 (−0.05, 0.14)
QMD	0.63 (0.46, 1.67)	0.60 (0.43, 2.4)	0.74 (0.51, 3.9)	0.61 (0.39, 2.26)	0.62 (0.4, 2.53)	−0. 04 (−0.14, 0.04)	0.03 (−0.06, 0.32)	−0.02 (−0.13, 0.05)	0.04 (−0.13, 0.11)
P intermedia									
FMD+water	0.69 (0.6, 1.01)	0.87 (0.74, 0.98)*	0.81 (0.61, 1.04)*	0.87 (0.63, 0.99)	0.68 (0.54, 0.88)	0.09 (−0.03, 0.12)	0.04 (0, 0.11)	0.03 (−0.11, 0.2)	−0.08 (−0.15, −0.01)
FMD+povidone	0.77 (0.57, 1.02)	0.77 (0.66, 1.0)	0.80 (0.56, 1.09)	0.80 (0.56, 1.07)	0.73 (0.59, 0.96)	0.03 (−0.09, 0.08)	0.03 (−0.03, 0.1)	−0.02 (−0.08, 0.05)	−0.01 (−0.13, 0.03)
QMD	0.74 (0.53, 1.09)	0.81 (0.51, 1.02)	0.81 (0.55, 1.04)	0.77 (0.64, 1.04)	0.67 (0.45, 0.93)	0.05 (−0.01, 0.1)	0.03 (−0.01, 0.06)	0.06 (−0.03, 0.19)	−0.1 (−0.18, 0.04)
T. denticola									
FMD+water	0.38 (0.2, 0.52)	0.47 (0.29, 0.55)	0.45 (0.27, 0.56)	0.48 (0.31, 0.55)	0.36 (0.23, 0.49)	0.03 (−0.03, 0.09)	0.02 (−0.01, 0.08)	0.04 (−0.01, 0.11)	0.02 (−0.1, 0.02)
FMD+povidone	0.35 (0.28, 0.49)	0.42 (0.34, 0.55)	0.38 (0.29, 0.46)	0.45 (0.34, 0.54)	0.32 (0.27, 0.38)	0.06 (−0.03, 0.16)	−0.01 (−0.06, 0.03)	0.04 (−0.02, 0.12)	−0.05 (−0.14, 0.01)
QMD	0.36 (0.3, 0.44)	0.4 (0.3, 0.41)	0.39 (0.32, 0.44)	0.41 (0.34, 0.45)	0.31 (0.24, 0.41)	0.03 (−0.04, 0.07)	0.01 (0, 0.05)	0.03 (−0.03, 0.08)	−0.01 (−0.09, 0.06)

Median (interquartile range).

*$p < 0.05$.

**$p < 0.01$, which represent changes from baseline within each treatment group.

FMD+water, full-mouth debridement with water; FMD+povidone, full-mouth debridement with povidone; QMD, quadrant-wise mechanical debridement; Day 0, immediately after the first treatment.

Bacteria tested: *Porphyromonas gingivalis*; *Actinobacillus actinomycetemcomitans*; *Prevotella intermedia*; *Treponema denticola*.

quadrant ultrasonic treatment generally resulted in a decrease in antibody titres and an increase in antibody avidity, although not all the changes were statistically significant. Full-mouth debridement seems to induce an earlier reduction of IgG titre to *P. gingivalis* and *A. actinomycetemcomitans* than quadrant-wise debridement, and help to enhance antibody affinity maturation.

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Clinical Relevance

Scientific rationale: A series of clinical and microbiological investigations have suggested similar or superior results of full-mouth disinfection compared with conventional quadrant-wise therapy. However, few studies have addressed immune response after full-mouth mechanical debridement.

Principle findings: An earlier reduction of IgG titre to *P. gingivalis* and *A. actinomycetemcomitans* was observed after full-mouth debridement compared with quadrant-wise therapy. Full-mouth debridement with povidone resulted in a significant decrease in IgG titres and an increase in antibody avidity to *A. actinomycetemcomitans* at 3 months.

Practical implications: Full-mouth ultrasonic debridement may have a potentially beneficial effect on the humoral immune response.