Fusobacteria Bacteremia Post Full Mouth Disinfection Therapy: A Case Report

Parth, Purwar¹, Vaibhav Sheel¹, Manisha Dixit¹, Jaya Dixit¹

¹ Department of Periodontology, Faculty of Dental Sciences, King George's Medical University, Lucknow, Uttar Pradesh, India.

Abstract: Oral bacteria under certain circumstances can enter the systemic circulation and can lead to adverse systemic effects. Fusobacteria species are numerically dominant species in dental plaque biofilms and are also associated with negative systemic outcomes. In the present case report, full mouth disinfection (FMD) was performed in a systemically healthy chronic periodontitis patient and the incidence of fusobateria species bacteremia in peripheral blood was evaluated before, during and after FMD. The results showed a significant increase in fusobacterium sp. bacteremia post FMD and the levels remained higher even after 30 minutes. In the light of the results it can be proposed that single visit FMD may result in transient bacteraemia.

Keywords: Chronic Periodontitis, Non surgical periodontal therapy, Fusobacterium species, Full mouth disinfection Therapy

I. Introduction

After scaling and root planing (SRP), bacteremia has been analyzed predominately in aerobic and gram-positive bacteria. Fusobaterium is a potential periopathogen which upon migration to extra-oral sites may provide a significant and persistent gram negative challenge to the host and may enhance the risk of adverse cardiovascular and pregnancy complications [1]. To the authors knowledge this is a seminal case report which gauges the occurrence and magnitude of fusobactrium sp. bacteremia after FMD.

II. Case Presentation

A 32 year old female patient, with blackish complexion visited the Out Patient Department of Periodontology with a chief complaint of bleeding from gums for 3 months [Fig-1]. The medical history was non contributory in nature. On extra oral examination, no abnormalities were detected. Upon intra oral examination, the gingiva was moderately swollen and appeared magenta colored. Loss of stippling was also observed with rolled out gingival margins and flattened papillae. Multiple sites showed either spontaneous or provocative bleeding with abundant supra gingival and sub gingival plaque accumulation. The probing depth (PD) was measured as the distance between the gingival margin and the base of the pocket while clinical attachment loss (CAL) was measured as the distance from cemento enamel junction to the base of the pocket with the help of a sterile PCP UNC- 15 periodontal probe. The clinical findings of gingival inflammation, clinical attachment loss (CA loss) in excess of 5mm, probing depth (PD) \geq 4mm at 3-4 sites in more than 4 teeth in each quadrant and radiographic evidence of bone loss was observed in the dentition. The propositor had plaque index (PI) [2] and gingival index (GI) [3] of 1.73±0.10 & 1.81±0.21 (mean±SD) respectively indicating poor oral hygiene status. Bleeding on probing (BOP) was checked by running the probe in the sulcus area and 65.6% of the sites were found to be BOP+. Additionally, > 30 % of the sites had similar clinical and radiographic characteristics. The propositor was diagnosed as having generalized chronic periodontitis (CP) according to the Armitage criteria [4]. The tailored treatment included induction of full mouth disinfection (FMD) and re-evaluation after 4 weeks for reassessment of periodontal parameters. A written informed consent was taken from the propositor before the initiation of the periodontal therapy. The patient was included in the study at her own accord.

To evaluate peripheral fusobacterium sp. bacteremia three blood samples were drawn from the propositor at the following intervals: Pre-treatment (P1), immediately after FMD (P2), and at 30 minutes after completion of FMD (P3). Prior to each sampling, the site was wiped with isopropyl alcohol to minimize the number of potential skin contaminants, cannulation of the cubital vein was performed, and 2 ml of blood was drawn at different times as indicated above. The blood samples were sent in the laboratory for estimation and identification of pure bacterial cultures using standard methods. Fusobacteria species were cultured on selective media (5% sheep blood with 5 mg/ml hemin and 1.0 mg/l vitamin K1) with antibiotics (0.001% w/v nalidixic acid, 2.5 mg/l vancomycin containing Brain–Heart-Infusion (BHI) (Wilkens–Chalgren-Agar) and incubated for 96 h at 37°C in an anaerobic atmosphere (Gaspak-Plus-Systems, BD). The identification of the microorganisms was followed on the basis of their typical colony and bacterial morphology, as well as spot test, a biochemical

identification system (REMEL) and their oxygen tolerance. The total number of the colony-forming units (CFU) was determined on the basis of serial dilution from 10^{-1} to 10^{-3} on selective media.

III. Investigations

A full mouth X-ray series (FMX) was advised which showed multiple sites having horizontal bone loss thus affirming the diagnosis of generalized chronic periodontitis [Fig-2].

IV. Differential Diagnosis

Other forms of periodontitis include aggressive periodontitis and periodontitis as a manifestation of systemic diseases. The propositor was found to be systemically healthy along with abundant plaque and calculus deposits which are found to be relatively scanty in cases of aggressive periodontitis. Based on clinical and radiological findings the propositor was diagnosed as generalized chronic periodontitis.

V. Periodontal Intervention

Initially oral hygiene instructions were given and once patient got acquitted to it the patient was scheduled for active periodontal treatment. We planned full mouth disinfection (FMD) in the present case so as to prevent cross contamination between the periodontally healthy and diseased sites. The entire oral cavity was cleaned by an ultrasonic scaler (P5 booster, Suprasson, Satelec) and hand curettes (Hu-Freidy scalers and curettes, USA) by an experienced clinician in a single session. Subgingival irrigation of the periodontal pockets was carried out with 0.2% chlorhexidine using an insulin syringe in order to remove all remaining bacteria. Additionally, an antiseptic agent (0.2% chlorhexidine) was used to brush the dorsum of the tongue to decrease the bacterial load in the area; an antiseptic mouthwash (0.2% chlorhexidine) was also used to reduce the bacterial load in the saliva and on the tonsils. FMD in CP patient has been one of the established treatment modality. The blood samples were drawn before FMD to evaluate pre therapy levels of fusobacteria sp while during and after FMD for evaluation of post disinfection bacteremia. The patient has been kept under stringent follow up thereafter.

VI. Outcome and Follow Up

During first recall visit after 4 weeks of FMD comprehensive oral hygiene instructions were reinforced. The patient was instructed to use mechanical methods i.e. toothbrushes and interdental cleaning aids along with mouthwashes whereas antibiotics were not prescribed. At recall visit the PI, GI, CA Loss, % of sites with BOP & sites with PD>4 mm decreased significantly from baseline showing the effectiveness of FMD in resolving periodontal inflammation [Fig-3]. The analysis of P1 showed that fusobacterium sp. count was 10^2 /ml of peripheral blood [Fig-4]whereas in P2 sample the count of fusobacterium sp. increased to 10^4 /ml of blood [Fig-5]which remained up to a level of 5000 species/ml of blood even after 30 min of completion of FMD[Fig-6]. Our finding suggests that single visit FMD therapy may induce gram negative bacteremia transiently and may represent significant risk of developing systemic complications.

VII. Discussion

Numerous studies have determined the frequency of passage of microorganisms into the bloodstream after treatment procedures which ranges from extraction [5], scaling [6], scaling and root planning [7], periodontal probing [8], after periodontal surgery [9], suture removal [10], orthodontic treatment [11], restorative dentistry [12] to root canal treatment [13]. Additionally, chewing [14], subgingival irrigation [15] and oral hygiene procedures such as tooth brushing [16] and flossing [17] have also been reported to cause peripheral bacteremia. The results in this report have shown considerable variability due to the techniques used, timing of blood sample collection, and periodontal status and identification methods for the isolation of microorganisms. However, there is limited information on bacteremia induced by single visit FMD in the same patient. The aim of this case report was to evaluate fusobacterium sp. bacteremia in peripheral blood after single visit FMD in a CP patient. Periodontal therapy can be performed as a single visit or in a quadrant wise manner. In this case FMD was performed in a single visit to avoid cross-contamination between the treated and untreated regions between the treatment sessions and main aim was rapid elimination or at least suppression of all periodontal pathogens from the oropharyngeal areas(periodontal pockets, saliva, oral mucosa and tonsils) until the appropriate healing of periodontal pockets. The microorganisms of the oral microbiome can migrate away from the oral cavity leading to infection and inflammation at the extra oral sites. Outside the oral cavity, fusobacterium sp. becomes a bona fide pathogen and has been implicated in plethora of adverse systemic outcomes [18]. Among fusobacteria sp., fusobacerium nucleatum is a prominent component of dental plaque quantitatively and acts as a central species involved in physical interactions between gram positive and gram negative species that are likely to play a cruicial role in biofilm colonization, and contributing to the reducing conditions necessary for the emergence of oxygen-intolerant anaerobes. It is also considered as an intermediate colonizer bridging the attachment of commensals that colonizes the tooth and epithelial surface with true pathogens. Fusobacterium nucleatum may be involved in the pathogenesis of periodontal disease by activating multiple cell signalling systems that lead to stimulation of collagenase 3 expression and increased migration and survival of infected epithelial cells [19]. Cultivation methods are considered as the golden standard, although limitations with respect to detecting non-viable bacteria, the inability of some species to grow reliably on selective media as well as high costs narrow the use in periodontal microbiological diagnostics [20]. However, anaerobic cultivation used in this study was not able to distinguish between different species of Fusobacterium. A molecular biological method such as PCR is required for a differentiation between such bacteria [21].

The periodontal procedures such as SRP performed in CP patients can lead to the translocation and release of the microorganisms from the oral cavity into the bloodstream which may play a significant role in the pathogenesis of atherosclerosis, thrombus formation, leading to cerebral and myocardial infarction by different mechanisms. There is ever increasing evidence that components of fusobacteria sp. particularly the lipopolysaccharide, may promote atherosclerosis, affect blood coagulation, the function of platelets and prostaglandin synthesis systemically[22].

Generally, the microorganisms in systemic circulation are eliminated by the reticuloendothelial system within a few minutes. Chronic low-level bacteremia and systemic inflammatory response have been suggested as a pathogenic link between periodontal disease and atherosclerosis. This study evaluated the presence of bacteria in peripheral blood before, immediately after SRP and 30 min after SRP. The highest count of fusobateria sp. was observed immediately after treatment and decreased after 30 min of treatment although remained higher as compared to the counts before initiation of SRP. The capability of neutralizing the microorganism in blood varies among patients and may represent an additional risk factor for developing remote infections. Moreover, degradation of gram-negative bacteria by the immune system can promote the expression of lipopolysaccharides in the peripheral blood initializing cell activation and the subsequent production of proinflammatory cytokines [23]. SRP can also elevate significantly the levels of pro-inflammatory cytokines in a short time [24, 25]. Although blood cultures are considered as the gold standard, evaluation of bacteremia by this method can lead to false-negative results and cannot detect bacteria degraded by the immune system. This study supports the evidence that bacteremia is associated with periodontopathic microorganisms after SRP in patients with chronic periodontitis. It also supports the relationship between periodontal disease associated bacteria and its distant effects in the human body in general, and the interaction between periodontal disease and cardiovascular disease, in particular.

VIII. Conclusion

In this case, full-mouth FMD provided in a signle session resulted in an increase in fusobacterium sp. bacteremia. Larger sample size studies with longitudinal design should be performed to investigate the issue further.

Acknowledgement

The authors extend their regards to the participant who gave her consent for the study.

References

- Han YW, Houcken W, Loos BG et al. Periodontal disease, atherosclerosis, adverse pregnancy outcomes, and head-and-neck cancer. Adv Dent Res. 2014; 26: 47-55.
- Silness J, Loe H. Periodontal disease in pregnancy. Correlation between oral hygiene and periodontal condition. Acta Odontal. Scand1964; 22:121-135.
- [3]. Loe H, Silness J. Periodontal disease in pregnancy. Prevalence and severity. Acta Odontol Scand 1963; 21:533-551.
- [4]. Armitage GC. Periodontal diagnoses and classification of periodontal diseases. Periodontol 2000. 2004;34:9-21.
- [5]. Heimdahl A, Hall G, Hedberg M et al. Detection and quantification by lysis filtration of bacteria after different oral surgical procedures. J Clin Microbiol. 1990; 28: 2205–2209.
- [6]. Conner HD, Haberman S, Collings CK et al. Bacteremias following periodontal scaling in patients with healthy appearing gingiva. J Periodontol. 1967; 68:466–472.
- [7]. Lazansky JP, Robinson L, Rodofsky L. Factors influencing the incidence of bacteraemia following surgical procedures in the oral cavity. J Dent Res. 1949; 28: 533–543.
- [8]. Daly C, Mitchell D, Grossberg D et al. Bacteremia caused by periodontal probing. Aust Dent J. 1997; 42: 77–80.
- [9]. Lockhart PB. An analysis of bacteraemia during dental extraction. A double-blind, placebo-controlled study of chlorhexidine. Arch Int Med. 1996; 156: 513–520.
- [10]. King RC, Crawford JJ, Small EW. Bacteraemia following intraoral suture removal. Oral Surg Oral Med Oral Pathol. 1988; 65: 23– 28.
- [11]. Erverdi N, Kadar T, Ozkan H et al. Investigation of bacteremia after orthodontic banding. Am J Orthod Dentofacial Orthop. 1999; 116: 687–90.
- [12]. LaPorte DM, Waldman BJ, Mont MA et al. Infections associated with dental procedures in total hip arthroplasty. J Bone Joint Surg. 1999; 81:56–59.
- [13]. Debelian GJ, Olsen I, Tronstad L. Bacteraemia in conjunction with endodontic therapy. Endod Dent Traumatol. 1995;11: 142–149.
- [14]. Cobe HM. Transitory bacteraemia. Oral Surg Oral Med Oral Pathol. 1954; 7: 609–615.

- [15]. Waki MY, Jolkovsky DL, Otomo-Corgel et al. Effects of sub gingival irrigation on bacteraemia following scaling and root planning. J Periodontol. 1990; 61:405–411.
- [16]. Roberts GJ. Dentists are innocent! "Everyday" bacteraemia is the real culprit: A review and assessment of the evidence that dental surgical procedures are a principal cause of bacterial endocarditis in children. Pediatr Cardiol. 1999; 20: 317–325.
- [17]. Lineberger LT, De Marco TJ. Evaluation of transient bacteraemia following routine periodontal procedures. J Periodontol. 1973; 44: 757–762.
- [18]. Han Y W ,Wang X . The mobile oral mirobiome : oral bacteria in extra-oral infection and inflammation. J Dent Res 92: 485-491.
- [19]. Kolenbrander PE. Oral microbial communities: biofilms, interactions, and genetic systems. Annu Rev Microbiol. 2000; 54:413-437.
- [20]. Loomer, P. M. (2004) Microbiological diagnostic testing in the treatment of periodontal diseases. Periodontology 2000 34, 49-56.
- [21]. Bolstad, A. I. & Jensen, H. B. (1993) Polymerase chain reaction-amplified nonradioactive probes for identification of Fusobacterium nucleatum. Journal of Clinical Microbiology 31, 528–532.
- [22]. Beck JD, Garcia R, Heiss G et al. Periodontal disease and cardiovascular disease. J Periodontol. 1996; 67: 1123–1137.
- [23]. Dzink JL, Socransky SS, Haffajee AD. The predominant cultivable microbiotaof active and inactive lesions of destructive periodontal disease. J Clin Periodontol.1988; 15:161–168.
- [24]. D'Aiuto F, Nibali L, Mohamed-Ali V et al. Periodontal therapy: A novel non-drug-induced experimental model to study human inflammation. J Dent Res. 2004; 39: 294–299.
- [25]. D'Aiuto F, Parkar M, Tonetti MS. Periodontal therapy: A novel acute inflammatory model. Inflamm Res. 2005; 54: 412-414.



Fig-1: Pre operative intra oral photograph showing abundant plaque and calculus deposits With slight fluorosis in upper anterior tooth region.

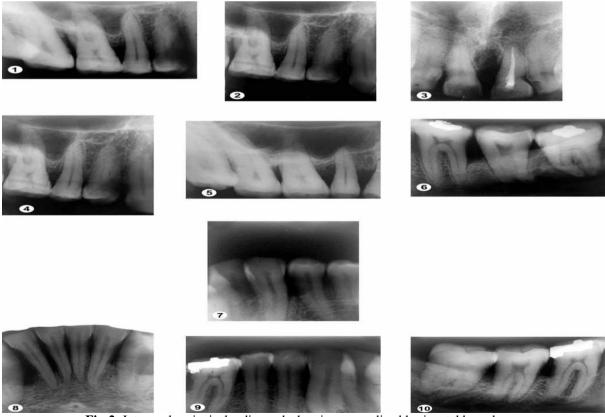


Fig-2: Intra oral periapical radiograph showing generalized horizontal bone loss.



Fig-3: Post operative photograph showing excellent oral hygiene status after 4 weeks follow up.



Fig-4: Blood agar plate (P1) showing growth of fusobacteria species after 10 days of inoculation period.



Fig-5: Blood agar plate (P2) showing growth of fusobacteria species after 10 days of inoculation period.



Fig-6: Blood agar plate (P3) showing growth of fusobacteria species after 10 days of inoculation period.