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CORRELATION BETWEEN DYSFUNCTIONAL OCCLUSION AND PERIODONTAL BACTERIAL PROFILE

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The aim of this study is to compare the evolution in bacterial profile at evident periodontitis sites following two types of treatment - oral hygiene procedures alone (Group 1) and oral hygiene plus occlusal adjustment through selective grinding (Group 2). The presence of periodontal disease was ascertained by clinical examination (redness, oedema, probe depth, bleeding-on-probing). Bacterial profiling was carried out via phase contrast microscopy on plaque samples taken from periodontitis sites in both patient groups. Bacterial populations were characterized in terms of coccus content before (T0) and at monthly intervals after treatment (T1-6) over a period of six months. Static and dynamic occlusion was evaluated only in Group 2 patients. Whereas the poor pre-treatment bacterial profile was re-established progressively over the evaluation period in Group 1 patients, coccus populations flourished in Group 2 patients, reaching healthy levels (>70%) two months after occlusal adjustment, and clinical examination confirmed an absence of periodontal inflammation in these patients. Occlusal adjustment can lead to a marked, stable improvement in periodontal health in terms of bacterial profile and clinical appearance, presumably by obviating tissue distress caused by occlusal dysfunction, thereby providing unfavourable conditions for bacterial growth. Bacterial profiling is an effective indicator of periodontal health.

Microbiological studies in patients suffering from periodontal disease have led to the identification of several hundred bacterial species and sub-species with pathogenic potential, although there is still some discussion as to the significance of these results. Despite the controversy, however, it remains true that no single species meets all of Koch's postulates and therefore none has been identified as the sole aetiological agent behind the disease (1-4).

In fact, the typical clinical profile of periodontitis appears to be brought about by the action of opportunistic pathogens, which are present as saprophytes in healthy subjects. This hints at a further, underlying cause of the disease, able to compromise periodontal tissue and thereby provide fertile ground for this bacterial transformation to take place.

Although the mechanism by which the saprophytic bacterial flora invade the healthy epithelial barrier has not been incontrovertibly and scientifically demonstrated (4), it appears that these organisms are unable to perforate an intact mucogingival seal (1, 4-7). In fact, the numerous studies aimed at identifying the presence of harmful bacteria in the healthy human chorion have failed to demonstrate this beyond doubt (1, 3, 4).

Furthermore, periodontal disease is often characterized by a relatively low presence of the so-called pathogenic bacteria (8), and the

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incidence of biomechanical anomalies acting on the stomatognathic system (especially malocclusion and tongue thrust) in these patients is, epidemiologically speaking, far higher (30). In fact, anomalous stress factors such as malocclusion, parafunctions, and lingual dysfunction have been described to be present in the vast majority of chronic gingival and periodontal inflammation cases (9, 10).

In this context, clinical evidence of a correlation between malocclusion-related tissue trauma and chronic periodontal disease has long been the subject of debate in the scientific community, and researchers are still seeking to define and interpret the cause and effect relationships involved.

Nonetheless, this hypothesis could explain not only the apparent site-specificity of periodontal lesions (11, 12), there is a clear and constant clinical correlation between periodontal lesion site and the topography of the direct or indirect occlusal disturbance, at least in terms of chronic dysfunctional biomechanical stress factors, but also the clinical profile of the disease, which features localized gingival oedema, stasis and vasal congestion (13-17).

The decisive role of chronic occlusal trauma in provoking tissue distress is widely recognized, and could be the condition that facilitates bacterial infection of the periodontium and transformation of the saprophytic flora to opportunistic pathogen (8, 9, 18-24).

Moreover, the striking effects that arise from functional mechanical stimulation of bony tissue, in terms of both remodelling and dystrophy, are well known. In particular, compression forces, especially if biomechanically incongruous and repeated often over time, induce significant modifications via global tensegretive pathways, accompanied by the development of piezoelectric effects and consequent neural, vascular, cellular, nuclear and metabolic responses, thereby leading, in some cases, to dystrophy and/or atrophy of the entire periodontium (8, 9, 12, 25-30). In implant and prosthetics patients, this situation is exacerbated by the lack of functional organs of mediation to modulate the load distribution. In this respect the periodontal ligament plays a fundamental role, preventing direct contact between the tooth root and the alveolar bone. Unfortunately, however, this optimal biomechanical condition is impossible to achieve in cases of implant–bone ankylosis.

In other patients, however, treatment involving occlusal equilibration by selective grinding generates enormous changes in the dental and periodontal districts in terms of microcirculation, leading to improvements in tissue trophism and, consequently, bacterial dynamics (8, 31, 32). In fact, functional treatment by occlusal adjustment and neutralization of repeated anomalous forces (parafunctions, tongue thrust, etc.) almost invariably results in a good prognosis in terms of healing and stability of results (8-10).

In order to confirm these findings and to investigate the possibility of using bacterial profiling to monitor treatment outcomes, the aim of the present study was to use this technique to ascertain any benefit from treating periodontitis patients via combined occlusal adjustment and oral hygiene procedures with respect to oral hygiene procedures alone.

As suggested in the literature, a ratio of 70% spherical bacteria (coccus) to 30% other forms of bacteria (rod-shaped, fusiform, filamentous and spiral-shaped) was taken to indicate periodontal health, whereas a prevalence of the latter types was taken as indicative of periodontal disease.

MATERIALS AND METHODS

Two hundred and sixty cases of overt periodontitis, confirmed by clinical examination (redness, oedema, probe depth, bleeding-on-probing) were allocated to one of two groups on the basis of the treatment to be performed: Group 1, comprising 81 subjects, to be treated by means of oral hygiene procedures alone (root planing, scale and polish), and Group 2, comprising 179 subjects, scheduled for treatment by means of occlusal adjustment (selective grinding) as well as the above oral hygiene procedures. Informed consent was obtained from all patients.

Plaque samples were removed with the aid of a sterile curette from periodontal pockets exhibiting acute inflammation, taking care to observe a certain degree of homogeneity in terms of sample number (3 per patient), sampling site (incisor, premolar and molar) and degree of inflammation present (probe depth, bleeding-on-probing, mobility) (Fig. 1).

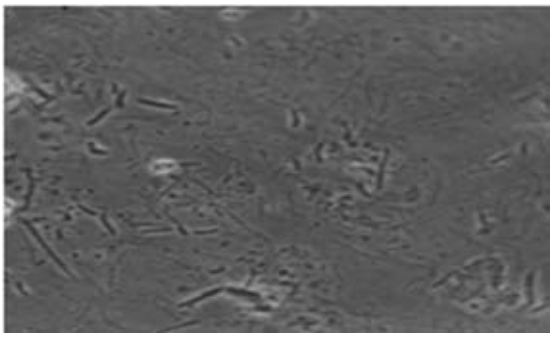

Fig. 1. *Phase contrast image of plaque with various bacterial morphologies (coccus, rod-shaped, fusiform, filamentous and spiral-shaped).*

Fig. 2. *The surface Burker's chamber for counting of bacterial morphologies.*

The subgingival plaque samples taken were suspended in 2 ml sterile aqueous solution containing 0.9% NaCl (weight/volume), and examined fresh via phase-contrast microscopy (Leica DMR microscope, Leica GmbH, Wetzlar, Germany) at 400x magnification to reveal the bacterial morphology and calculate the percentage coccus content in the first 200 bacteria counted (Fig. 2) (7).

Patients from both groups then underwent standard oral hygiene procedures (root planing, scale and polish), following which Group 1 patients were dismissed.

Occlusal load testing was then performed on Group 2 patients alone, with the patients in a standardized position seated against an upright backrest, facing forward with the eyes fixed on a point in the far distance. Contact points and interference were revealed on 40-μm-thick articulating paper (Bausch-Articulating Paper, Inc, Nashua, USA), blue for static and red for dynamic occlusion.

Static occlusion was evaluated with the patient in their habitual position of maximum intercuspidation. Dynamic occlusion was then measured as the maximum excursion in all directions (forwards, backwards and sideways).

Premature contact points were then removed through selective grinding (occlusal adjustment) to improve the occlusion.

The bacterial monitoring protocol described above was repeated in both patient groups at one-monthly intervals over a period of six months in order to highlight any variation in the bacterial profile in terms of percentage trend in coccus population in respect to the other bacterial types (rod-shaped, fusiform, filamentous, spiral-shaped).

Statistical analysis of the resulting findings was performed by means of Student's *t*-test.

RESULTS

The results are reported in Table I, which shows that at even at the first recall, one month following treatment, the bacterial flora of Group 2 patients, treated by means of occlusal adjustment and dental cleaning procedures, featured a significantly larger coccus content than their Group 1 counterparts, treated by means of dental cleaning alone. This difference reached an extremely high level of significance ($p<0.001$) at three months after treatment and remained at this level until the end of the monitoring period.

The change in coccus content over time in the two Groups is illustrated in Fig. 3, which reveals that the Group 1 bacterial profile tended towards pre-treatment values over the course of the monitoring period.

DISCUSSION

In periodontics, sampling of bacterial plaque in the dentogingival area is a vital element in all preventative, diagnostic and treatment protocols, as it contains that which is considered to be the prime cause of periodontal disease, i.e. bacteria (18, 33-36). Nonetheless, certain aspects of clinical management of periodontal disease still require further study,

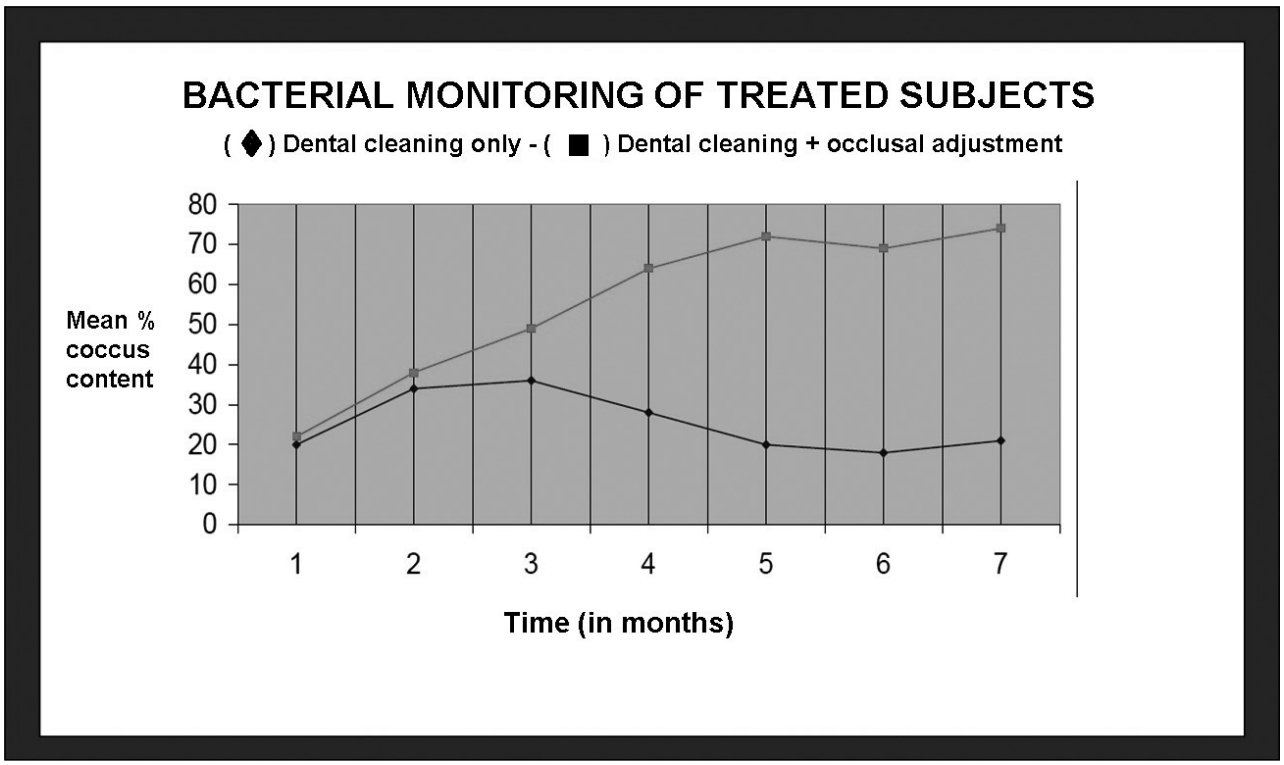

Fig. 3. *The trend in mean percentage coccus content over the course of the monitoring period; results pertaining to patients treated by means of dental cleaning alone (Group 1) are shown with ♦ line, while those pertaining to patients treated by means of dental cleaning and occlusal adjustment (Group 2) are shown with ■ line.*

particularly as regards follow-up planning, as the evolution of the disease profile is often unpredictable.

It is our belief, however, that this uncertain prognosis is mainly the consequence of over-relying on an approach limited to treating the bacterial plaque response alone, via interminable follow-ups, root planing and frequent pharmacological treatments, which aside from being wearing for the patient, generally fail to lead to definitive stabilization.

In fact, it is particularly evident in this study that the classic clinical signs and bacterial dynamics of the disease, represented by a re-establishment of a profile rich in pathogenic forms such as filamentous, rod-shaped, spiral-shaped and fusiform bacteria, tended to re-establish themselves in periodontitis cases treated via dental cleaning procedures alone, thereby indicating that removal of bacterial plaque alone is not sufficient to remove the real cause of the gingival and periodontal inflammation.

In contrast, the health status of periodontal sites treated by means of normalization of biomechanical function was found to be stable in terms of bacterial profile, which remained saprophytic in bacterial species with a clear prevalence of spherical forms.

Stabilization of a mainly saprophytic bacterial profile at sites of periodontitis treated via occlusal adjustment indicates that this approach leads to the establishment of an ecosystem characterized by a trophic condition. Indeed, clinically evidence suggests the return of normal microcirculation, with stable reduction of oedema, lack of bleeding and perhaps even reduction of dental mobility. This agrees with the finding that a trophic site, i.e. in occlusal equilibrium, fails to develop an aggressive bacterial profile, while a site of occlusal trauma tends to re-establish a profile typical of a periodontitis patient, despite assiduous hygiene follow-up (8, 35), and confirms the view periodontopathy should not be interpreted as merely an infectious disease (2, 3) but a syndromic condition of multifactorial origin, not forgetting its psychogenic aspect (37, 38).

Indeed, in our opinion, to ignore the

Table I. *Mean percentage coccus content in plaque samples taken from sites of periodontal lesion immediately after treatment (T0) and then at monthly intervals (T1-T6) over a 6-month monitoring period. A strongly significant difference between the two treatment Groups is revealed from the second month onwards.*

Treatment Group	N° of cases	Mean % *coccus* content at monthly post-treatment intervals						
		T0	T1	T2	T3	T4	T5	T6
1: dental cleaning alone	81	34	53	48	31	27	32	26
2: dental cleaning + occlusal adjustment	179	28	62	74	76	71	70	77
Significance level	*n/a*	p<0.06	p<0.01	p<0.001	p<0.001	p<0.001	p<0.001	p<0.001

biomechanical dysfunction component in the genesis of periodontal damage, a pathology that is, in essence, dystrophic/atrophic in nature, and to focus merely on the consequent infection would be incomplete, misleading and scientifically unproductive and therefore a grave epistemiological mistake.

Instead, it is fundamental to distinguish the diagnostic profile of chronic dystrophic/atrophic parodontopathy, tissue damage resulting from biomechanical dysfunction, from that of periodontitis, the consequent bacterial infection. This change in thinking also necessarily entails a change in diagnostic and therapeutic approach, which must be first and foremost functional in nature, the latter being aimed at re-establishing the occlusal and therefore biomechanical equilibrium.

Likewise, prevention strategies must be reassessed, as concentrating on hygiene alone does not remove the cause of periodontal disease, merely providing a temporary "quick-fix" solution.

In this context, however, the bacterial content of the dentogingival plaque does have a fundamental role to play. Indeed, it is the morphological (and intrinsically metabolic) quality of the bacterial profile that indicates the trophic or dystrophic conditions of the periodontal niche, as well as the microbiotic state of the oral ecosystem as a whole, as certain morphological types of anaerobic bacteria (rod-shaped, fusiform, filamentous and spiral-shaped) thrive in dystrophic conditions, at the expense of the predominantly aerobic coccus (7, 8, 39). Thus, bacterial profiling, in terms of the percentages of bacterial forms present (7, 39), is an indicator of periodontic health.

The periodontal index proposed herein, i.e. the percentage composition of spherical bacteria in the periodontal flora, counted fresh under phase-contrast microscopy, has proved its validity as a diagnostic, and especially prognostic, aid, permitting early evaluation of any tendency towards periodontic or peri-implant infection, before later signs (inflammation, bleeding, mobility) become apparent. Not only that, but the test is highly sensitive and of greater significance than the usual indicators (probe depth, bleeding-on-probing, mobility and plaque index), not to mention simple and rapid to perform. Moreover, the equipment necessary to evaluate the proposed periodontal index, i.e. a phase-contrast microscope, is readily available and easy to use, without the need for special training, and no special materials are required, merely physiological solution, sterile tips and cover slips.

Thus, it is our opinion that monitoring of the conventional periodontal parameters (probe depth, bleeding-on-probing, mobility) should be supplemented with combined bacterial profiling and

assessment of the biomechanics of the stomatognathic apparatus and, particularly, the occlusal relationship. This approach consents characterization of the clinical and bacterial evolutionary trend in relation to the functional occlusal/factorial profile in all its static and dynamic aspects (8), and may shed light on the intimate pathogenic mechanism behind the disease, allowing us to better plan the follow-up of periodontopathic patients.

In conclusion, more attention should be paid to the fact that periodontal disease is a biomechanical dysfunction-based syndrome in which the damage is, in essence, dystrophic/atrophic, and the resulting infection merely a septic evolution. Thus, occlusal adjustment to normalize the load distribution, leading to re-establishment of microcirculation in the periodontal tissues and providing an environment hostile to the opportunistic bacteria that cause the classic symptoms of the disease, should be considered a useful adjunct to dental cleaning.

Furthermore, due to its capacity to detect the presence of unhealthy microorganism populations, likely to result in periodontal disease, bacterial profiling should be considered as an indicator of periodontal health, not only for diagnostic purposes, but also in monitoring the success of treatment strategies and in aiding the unveiling of the complex mechanisms behind the disease.

REFERENCES

1. Listgarten, MA. The role of dental plaque in gingivitis and periodontitis. J Clin Periodontol 1988; 15(8):485-7.
2. Socransky, SS. Criteria for the infectious agents in dental caries and periodontal disease. J Clin Periodontol 1979; 6(7):16-21.
3. Socransky, SS and Haffajee, AD. Periodontal microbial ecology. Periodontol 2000 2005; 38(135-87.
4. Newman, MG. Current concepts of the pathogenesis of periodontal disease. Microbiology emphasis. J Periodontol 1985; 56(12):734-9.
5. Allaker, RP and Dimock, D. Proceedings of the 8th European Oral Microbiology Workshop. in *Adv Dent Res* Vol. 18 27 (2005).
6. Wolff, LF, Liljemark, WF, Pihlstrom, BL, Schaffer, EM, Aeppli, DM and Bandt, CL. Dark-pigmented Bacteroides species in subgingival plaque of adult patients on a rigorous recall program. J Periodontal Res 1988; 23(3):170-4.
7. Gandolfo, S, Meynardi, F, Corrente, G and Nelken, A. Microbiological analysis in fresh dental gingival plaque. Rivista Italiana di Stomatologia 1994; 5(275-86.
8. Meynardi, F and Biancotti, PP. Etiopathogenetic correlations between periodontal disease and occlusal trauma. IAPNOR-International Academy of Posture and Neuromuscular Occlusion Research 2009; 13(
9. Pasqualini, U. Le patologie occlusali. Place: Masson Ed. ,1994.
10. Bogren, A, Teles, RP, Torresyap, G, Haffajee, AD, Socransky, SS and Wennstrom, JL. Locally delivered doxycycline during supportive periodontal therapy: a 3-year study. J Periodontol 2008; 79(5):827-35.
11. Harrell, JC and Stein, SH. Prostaglandin E2 regulates gingival mononuclear cell immunoglobulin production. J Periodontol 1995; 66(3):222-7.
12. Mendieta, CF, Reeve, CM and Romero, JC. Biosynthesis of prostaglandins in gingiva of patients with chronic periodontitis. J Periodontol 1985; 56(1):44-7.
13. Beerstecher, E, Jr. and Bell, RW. Some aspects of the biochemical dynamics in the periodontal ligament and alveolar bone resulting from traumatic occlusion. J Prosthet Dent 1974; 32(6):646-50.
14. Palcanis, KG. Effect of occlusal trauma on interstitial pressure in the periodontal ligament. J Dent Res 1973; 52(5):903-10.
15. Chasens, A. The effect of traumatic occlusion on the periodontium and the associated structures and treatment by selective grinding of the natural dentition. Dent Clin North Am 1962; 6(
16. Stallard, RE. Occlusion: a factor in periodontal disease. Int Dent J 1968; 18(1):121-32.
17. Ash, MM. Wheeler's Dental Anatomy, physiology and occlusion. Place: W. B. Saunders.,1984.
18. Genco, RJ, Goldman, HM and Cohen, DW. Contemporary Periodontics. Place: C. V. Mosby,1990.
19. Socransky, SS, Smith, C and Haffajee, AD. Subgingival microbial profiles in refractory

periodontal disease. J Clin Periodontol 2002; 29(3):260-8.
20. Glickman, I. Occlusion and the periodontium. J Dent Res 1967; 46(1):53-9.
21. Karolyi, M. Beobachtungen ueber Pyorrhea alveolaris. Oest Viertel Ischr Z 1901; 17.
22. Stillman, PR. The management of pyorrhea. Dent Cosmo 1917; 59.
23. Box, HK. Experimental occlusal trauma. Oral Health 1935; 25.
24. Tones, HH. An experimental investigation into the association of traumatic occlusion with periodontal disease. Proc Soc Med 1938; 31(5):479-95.
25. McNeill, C. Occlusion: what it is and what it is not. J Calif Dent Assoc 2000; 28(10):748-58.
26. Ingber, DE. Mechanobiology and diseases of mechanotransduction. Ann Med 2003; 35(8):564-77.
27. Poiate, IA, de Vasconcellos, AB, de Santana, RB and Poiate, E. Three-dimensional stress distribution in the human periodontal ligament in masticatory, parafunctional, and trauma loads: finite element analysis. J Periodontol 2009; 80(11):1859-67.
28. Pasqualini, U and Pasqualini, M. [Uses and advantages of the dry self-molding varnish, Red Indicator, in selective impressions]. Dent Cadmos 1982; 50(10):49-63.
29. Ingber, D. Integrins as mechanochemical transducers. Curr Opin Cell Biol 1991; 3(5):841-8.
30. Jones, DB, Nolte, H, Scholubbers, JG, Turner, E and Veltel, D. Biochemical signal transduction of mechanical strain in osteoblast-like cells. Biomaterials 1991; 12(2):101-10.
31. Motohira, H, Hayashi, J, Tatsumi, J, Tajima, M, Sakagami, H and Shin, K. Hypoxia and reoxygenation augment bone-resorbing factor production from human periodontal ligament cells. J Periodontol 2007; 78(9):1803-9.
32. Jankelson, R. Neuromuscular dental diagnosis and treatment. Place: Ishiyaku Euroamerica,1990.
33. Kenney, EB. A histopathologic study of incisal dysfunction and gingival inflammation in the Rhesus monkey. J Periodontol 1971; 42(1):3-7.
34. Lindhe, J. Parodontologia clinica e odontoiatria implantare. Place: Edizioni Martina,2006.
35. Listgarten, MA. Structure of the microbial flora associated with periodontal health and disease in man. A light and electron microscopic study. J Periodontol 1976; 47(1):1-18.
36. Socransky, SS and Haffajee, AD. The nature of periodontal diseases. Ann Periodontol 1997; 2(1):3-10.
37. Saito, M, Saito, S, Ngan, PW, Shanfeld, J and Davidovitch, Z. Interleukin 1 beta and prostaglandin E are involved in the response of periodontal cells to mechanical stress in vivo and in vitro. Am J Orthod Dentofacial Orthop 1991; 99(3):226-40.
38. Gelb, H. Clinical management of Head, Neck and TMJ. Pain and Dysfunction. Place: W.B. Saunders 1977.
39. Meynardi, F, Rossi, F, Grivet Brancot, L and Pasqualini, ME. Not only bacteria but occlusal trauma to the origin of periodontal disease. Dental Tribune 2013; 9(1):19-20.

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