Periodontitis is a group of inflammatory diseases that affect the connective tissue attachment and supporting bone around the teeth. The initiation and the progression of periodontitis are dependent on the presence of virulent microorganisms capable of causing disease. (Socransky et al. 1984) A number of possible pathogens have been detected on the basis of their association with disease progression and also because of their possession of virulence factors which can damage the tissues. The main bacteria associated with periodontal disease are Aggregatibacter actinomycetemcomitans, Prevotella intermedia, Porphyromonas gingivalis and Tannerella forsythia. (Slots, Bragd, and Wikstrom 1986) These bacteria tend to be present in increased numbers at active disease sites and produces products which are capable of damaging the tissue either directly or indirectly. However, they may also be present in healthy and inactive sites and the composition of all these sites may vary between patients or even in the same patient at different time intervals. Attempts to relate microbiological data to clinical events have proved difficult due to the variability and unreliability of clinical diagnostic methods. Another factor, which complicates the quantitative assessments of the subgingival flora, is the technical problems associated with the sampling and culturing processes. (Socransky et al., 1991) Though bacteria are considered as the initiating factor, periodontitis is not a classic infectious disease. Majority of the causative organisms are present in the healthy mouth, and the host response modified by environmental and behavioral factors play a key role in disease development, progression and in the maintenance of the treatment result. (Socransky and Haftljee, 2005) Despite the etiology or type of periodontitis, the products of host response and tissue destruction are similar. Markers of inflammatory process for periodontal tissues migrate into the periodontal pockets within a serum originating fluid, Gingival Crevicular Fluid (GCF), and further from gingival crevices/pockets into the oral cavity where GCF associates with saliva. The more inflamed the periodontal tissues and deeper the periodontal pockets are, more GCF is excreted and it contains more inflammatory markers. (Uitto et al., 2003) This also means that the inflammatory burden of periodontium may be reflected in saliva. (Uitto et al., 1990) Unlike in medicine, biochemical testing in dentistry has not yet been taken into everyday clinical practice despite the advantages in supplementation of traditional diagnostics. GCF has been used for site specific diagnostic studies of periodontal disease status and diagnostic tests for host derived markers have been developed. Inflammatory markers from GCF analyzed individually or in combination may be valuable in the identification of individual...
“Periodontal diagnosis” is an important tag that a clinician ties on the periodontal disease condition of the patient, capturing all his past experience with the condition in question. The entire constellation of signs and symptoms, along with a detailed history, is elicited, documented, and interpreted to reach at a diagnosis. Most often an accurate diagnosis is the very first concrete step towards the planning and execution of an appropriate individualized treatment plan, contributing significantly towards the success of the therapy (Armitage, 2004). Clinical diagnostic parameters that were introduced more than half a century ago continue to function as the basic model for periodontal diagnosis in current clinical practice as well. A periodontal diagnostic tool, in general, provides pertinent information for differential diagnosis, localization of disease, and severity of infection. They include various disease characteristics such as probing pocket depths, bleeding on probing, clinical attachment levels, plaque index, and radiographs quantifying alveolar bone levels (Giannobile et al., 2009; Armitage, 2004). Although there have been significant advances in the understanding of the etiopathogenesis of periodontal disease over the past 4-5 decades, the traditional methods by which clinicians diagnose periodontal disease have remained virtually unchanged (Wolf and Lamster, 2011). These diagnostics were called in to question during the early 1980s, when longitudinal clinical studies demonstrated that long-held concepts concerning the natural history of periodontal disease required modification (Lamster and Gribic, 1995). More recent paradigms for periodontal disease diagnosis include the possibility of several disease types, based primarily on the rate of disease progression, the distribution of the disease within the mouth, and the chronological age of the patient as well as active and inactive stages of the disease.

Current Clinical Diagnostic Methods

Since no consistent microbial pattern or host responses are unique to a given type of periodontitis, accurate detection and prediction of disease activity are elusive goals. Currently the clinical parameters that are used to diagnose active periodontitis exhibit poor accuracy and reliability. Modern in-office diagnostic methods are clinical parameters of destructive periodontal disease such as longitudinal assessment of changes in pocket depth or attachment level, radiographic bone loss, bleeding on probing, suppuration, plaque scores, tooth mobility and patient reports of pain to detect disease. Although an increase in probing depth over time continues to be valuable in predicting future attachment loss, these measurements are influenced by the degree of tissue inflammation, the type of probe used, the angulation and the force exerted on the probe. These factors impact on the reproducibility of measurements. Alternatively, radiographic assessment often requires minimum of 30% bone demineralization before detection on radiographs, and radiographic change of bone loss is not always detected in sites exhibiting clinical attachment change. Other clinical signs of inflammation, such as mobility, suppuration and bleeding upon probing are very subjective measures and are poor predictors of attachment loss. In a clinical setting, the findings from these diagnostic procedures are generally considered collectively and are the foundation upon which periodontal diagnoses are made, despite several significant weaknesses. Notably, clinical signs of inflammation do not predict disease activity before it has caused significant destruction: physical assessment of periodontitis can only measure damage on the basis of past episodes of destruction. Currently, there is no practical clinical test to determine if disease is active. Moreover, traditional methods of diagnosis are unable to distinguish between successful treatment outcomes and refractory cases of periodontitis that may require further periodontal therapy. In view of these data there is a need in periodontology for the development of efficient, accurate and sensitive tests that will increase the probability of accurate early diagnoses and hopefully lead to improved prevention and treatment of periodontal attachment loss. This review is an attempt to summarize the current status of chair side diagnostic tests in the field of periodontology.

Chair side diagnosis

The microbial-enzymatic N-benzoyl-DL-arginine-2-naphthylamide (BANA) test is one of the modern alternatives to bacterial cultures. Dhalla et al conducted an invivo study for the detection of BANA micro-organisms in 20 adult periodontitis patients before and after scaling and root planing by BANA-Enzymatic™ test kit. It detects the presence of three periodontal pathogens in the subgingival plaque (Porphyromonas gingivalis, Treponemadenticola and Tannerella forsythia). Four test sites (permanent molar from each quadrant) were selected from each patient and assessed for plaque index, bleeding index and pocket depth before and after scaling and root planing. BANA test was used for the detection and prevalence of the “red complex” bacteria in plaque samples. There was a significant correlation between the BANA test results and the quantity of bacterial plaque, the test being influenced by the composition of bacterial plaque. This study encourages the use of such chair-side tests for a proper diagnosis of periodontal disease and for a good evaluation of the treatment results. (Nipun Dhalla et al., 2015) Halimeter is the most used apparatus in halitosis research. Hossam et al used halimeter for chair side diagnosis of halitosis in 60 chronic periodontitis patients. The participants were grouped as periodontitis (case), non-surgically treated periodontitis and healthy (control). Volatile sulfur compounds were measured in parts per billion (ppb) as a caliber for halitosis for each group. He concluded that halitosis is directly related to periodontitis and periodontal pocket depth among the adults, which can be successfully diagnosed by the gold standard method, halimeter. (Hossam, 2014) Organoleptical method and halimeter were used by Evirgena et al for the assessment of halitosis. Out of the 38 patients, 14 were diagnosed with halitosis by 6 clinicians using a halimeter. The highest sensitivity (89%) was found for clinician No. 6, followed by clinician No. 5 (78%). Specificities were 57% for clinician No. 4 and 36% for clinician No. 1 and No. 5. The
most correct positive predictive value (halitosis according to halimeter readings) was made by clinician No. 6 (65%), who also had the highest rates (83%) of negative predictive value (no halitosis according to halimeter readings). There were no statistically significant differences (P > 0.05) between the diagnoses of clinicians No. 1, 2, 3, 5, or 6. This study indicates that calibration of clinicians is a significant factor in the organoleptic evaluation of halitosis and he considered halimeter as the gold standard for char side diagnosis of halitosis. (Sehrazat Evirgena et al., 2013) OralChromat™ and Halimeter were compared by Salaki et al for the assessment of the ability of common cultivable oral anaerobic bacteria to produce malodorar volatile sulfur compounds from cysteine and methionine. The major VSC producers identified by both Halimeter and Oral Chroma with LT cysteine as substrate were Campylobacter ureolyticus, Porphyromonas gingivalis, Tannerella forsythia, Prevotella intermedia, Aggregatibacter actinomyctetomomas and Gemellamobillorum. The concentrations of hydrogen sulfide recorded by both Halimeter and Oral Chroma were essentially identical. With LTmethionine as substrate, both Halimeter and Oral Chroma identified different complements of anaerobes with C. ureolyticus, P. gingivalis, Fusobacteriumnucleatum and P. intermedia as major VSC producers. The concentrations of methyl mercaptan recorded by the Halimeter were lower compared to those assessed by the Oral Chroma. (Salaki, 2011)

Dentocult® SM test and PerioCheck® test were used for measuring caries-related organisms and periodontal disease-related organisms respectively. Huang et al studied Chair-Side Quantitative Oral-Micro flora Screeing for Assessing Familial Correlation of Periodontal Status and Caries Prevalence using the above tests. He enrolled 30 parent-child pairs, with the children exhibiting complete deciduous dentition or mixed dentition with only permanent first molars. Clinical statuses were evaluated using caries and periodontal disease indicators, including the sum of decay and the number of missing or filled teeth (DMFT) for adults, decay, extraction caused by dental disease, and filled teeth (deft), for children, probing depth, and plaque control record (PCR). Supra- and sub-gingival bacteria were determined based on semi-quantitative measurements of microbial infection by using data from the Dentocult® SM test and the PerioCheck® test. No statistically significant relationship was detected between the prevalence of periodontal pathogens and that of cariogenic pathogens in the oral cavity. The study showed that the quantity of caries pathogens were not significant related to periodontal pathogens, but the caries clinical outcome is negative related with periodontal clinical outcome between familial pairs. (Yung-Kai Huang et al., 2014) The PerioWatch was developed as a simple method of analyzing Aspartate aminotransferase (AST) at the chairside. Rez et al assessed the presence of aspartate aminotransferase (AST) in peri-implant crevicular fluid, with or without clinical signs of mucositis, to determine its predictive diagnostic value, sensitivity, and specificity. The AST levels were determined (at a threshold of 1200 mU/mL) using commercial AST test (Pocket-Watch, Sterio-Oss, Loma Linda, Calif) for 60 clinically successful implants in 25 patients with or without peri-implant mucositis. Samples were taken prior (AST1) to peri-implant probing with a manual constant-pressure probe (0.2 N) and 15 minutes after probing (AST2). Clinical assessments included radiographic determination of preexisting bone loss, probing, and the evaluation of mucositis, plaque, and bleeding upon probing. There were a significant difference between AST1 and AST2 at both levels. Aspartate aminotransferase diagnosed with Pocket watch was a reliable predictor of patients with mucositis. (Arturo Sa’nchez-Pe rez et al., 2012) The Time resolved immunofluorometric assay (IFMA), MMP- 8 specific chair-side dip-stick test, dentoAnalyzer device and the Amersham ELISA kit were compared for gingival crevicular fluid (GCF) matrix metalloproteinase (MMP)-8 detection by Sora et al. Matrix metalloproteinase-8 levels from 20 GCF samples from two periodontally healthy subjects, 18 samples from two patients with gingivitis and 45 samples from six patients with moderate to severe periodontitis, altogether 83 samples, were analysed. Western immunoblot using same monoclonal anti-MMP-8 as in IFMA and dentoAnalyzer was used to identify molecular forms of MMP-8 in GCFs. Immunofluorometric assay and dento- Analyzer can detect MMP-8 from GCF samples and these methods are comparable. Using Western immunoblot, it was confirmed that IFMA and dentoAnalyzer can detect activated 55 kDa MMP-8 species especially in periodontitis-affected GCF. Dento Analyzer is among the first quantitative MMP-8 chair-side testing devices in periodontal and peri-implant diagnostics and research. (Sorsa et al., 2010)

Mobilometer and Florida probe were used by Doshi et al to evaluate the association between bone loss and Periotest values. A total number of 60 patients between the age group of 18 and 55 years were divided into subjects with healthy periodontium (Group A), and those with Chronic Generalized Periodontitis (Group B), having a generalized pocket probing depth of 3 – 8 mm, respectively. With the help of the Periotest, Florida probe, and radiographs the assessment was carried out. He found that the values were highly significant in the incisor, premolar, and molar areas. The anterior teeth showed higher PTV compared to the posterior teeth; and the mandibular teeth showed higher PTV compared to the maxillary teeth. There was no association between the clinical indices and Periotest values. (YogeshDoshi et al., 2010) Specificity of Meridol Perio Diagnostics was verified with purified genomic DNA from several bacterial and fungal species as well as with human DNA by Recani et al. In fifty one dentate patient, 136 implants were inserted either in the upper or lower jaw in the place where molars were missing. Cemented suprastructure was put in 32 patients and screw retained suprastructure in 19 patients. Samples were taken with sterile paper points before abutment fixation and six months later (three times during ten seconds from the gingival sulcus) and analyzed with real-time polymerase chain reaction. The frozen culture (1.5 ml) was sent to Carpegen GmbH, and 0.5 ml of the defrosted dilution was used for real-time PCR analysis. The cells were harvested by centrifugation (15,000 g at 41C) for 10 min and immediately subjected to the automated process of the Meridol Perio Diagnostics (GABA International, Munchenstein, Switzerland) analysis. This real-time PCR based analysis was developed and validated by Carpegen GmbH. There was a significant increase in bacterial count in persons with cardiovascular, rheumatic diseases and in those who took medications and were older. (Recani et al., 2014) Dip-stick test was used to estimate the concentration of MMP-8 in gingival crevicular fluid in smoking (S) and nonsmoking (NS) patients with chronic periodontitis by Mantalya et al. Clinical parameters, MMP-8 test results and concentrations were monitored in 16 patients after initial treatment and in 15 patients after scaling and root planing (SRP), every other month, over a 12-month time period. SRP reduced the mean GCF MMP-8 levels, test scores, probing depth (PD), attachment loss (AL) and bleeding on probing (BOP). In sites of periodontal disease progression, the
distribution of MMP-8 concentrations was broader than in stable sites, indicating a tendency for elevated concentrations in patients with periodontal disease. The mean MMP-8 concentrations in smokers were lower than in nonsmokers, but in smokers and nonsmokers sites with progressive disease, MMP-8 concentrations were similar. (Mäntylä et al., 2006)

The Diamond Probe/Perio 2000 System was used to evaluate the relationship between volatile sulfur compounds (VSC) and gingival health status by Pavolotskaya et al. A split-mouth design with randomly selected quadrants of the mandibular arch enabled 39 participants to serve as their own controls. At baseline and at three subsequent appointments (days 7, 14, and 21) gingival inflammation (GI), bleeding on probing (BOP), and sulfide levels (SUL) were measured using the Gingival Index and the Diamond Probe/Perio 2000 System. For three weeks, participants refrained from brushing and flossing one randomly selected quadrant of the mandibular arch. Data suggest that SUL correlate positively to GI and BOP on both sides; however, the strength of the correlation was stronger for the NH side. Based on study outcomes, the Diamond Probe/Perio 2000 System demonstrated the ability to detect sites with elevated SUL; therefore, SUL may be a useful adjunctive indicator of early plaque-induced gingivitis. (Aleksandra Pavolotskaya et al., 2006) Comparison of Periocheck (neutral protease) and Perioscan (BANA hydrolase) kits with traditional clinical methods of detecting periodontal disease and to monitor the ability of the kits to reflect the response to initial therapy were done by Hemmings et al. 19 patients with moderately severe chronic periodontitis were seen before and after a course of oral hygiene and root instrumentation consisting of 4 appointments. Clinical measurements and test assays were collected at 5 diseased sites and 2 healthy sites in each subject. At baseline Periocheck had a sensitivity of 88% and a specificity of 61% whereas Perioscan had a sensitivity of 99% and a specificity of 55%. When related to the clinical diagnosis. The probability that the tests agreed with the clinical outcome after treatment, was calculated as 50.4% for Periocheck and 52% for Perioscan. (Hemmings et al., 1997)

Future perspectives

It can be interpreted that new technologies that have been developed or are in development can be used to enhance the ability to predict, diagnose and treat periodontitis. Moreover, new diagnostic technologies like nucleic acid and protein microarrays and microfluidics are under development for risk assessment and comprehensive screening of biomarkers. These will provide practitioners with more effective means of prevention, detection and treatment of periodontitis that are currently available. These recent advances are leading to the development of more powerful diagnostic tools for practitioners to optimize their treatment predictability. Future developing chair side diagnostic kits should focus on more reliable predictive tests so that it can predict the future periodontal activity and thus enable administration of the treatments tailored to specific sites before irreversible damage has occurred.

Conclusion

In Periodontology, the success of any treatment is dependents upon the accuracy of the initial diagnosis. At present, the majority of chronic periodontitis cases can be adequately managed using existing diagnostic methodology, although it is clearly more desirable to be able to diagnose “active disease” as it occurs, rather than months later. Gingival crevicular fluid (GCF) became an early medium to examine for biomarkers due to its location within the sulks and easy accessibility. Chapple (2009) states the advantages of using GCF “The biomarkers found in GCF indicate the presence or absence of periodontal pathogens, gingival and periodontal inflammation, the host inflammatory-immune response to certain pathogenic species and host tissue destruction.” The disadvantages of using GCF is that it is expensive, time consuming, requires multiple samples of individual tooth sites and requires laboratory processing. Saliva contains a plethora of biomarkers for periodontal disease and has emerged as a medium of choice for periodontal disease. Oral DNA labs (Brentwood, Tenn) offer two salivary tests that evaluate for periodontal disease. MyPerioPath is a DNA test that uses saliva to determine an individual risk for periodontal disease by identifying the specific bacterial pathogens (microbial biomarkers) associated with the disease. MyPerioID uses salvia to determine the patient’s genetic susceptibility for periodontal disease by testing for a genetic biomarker. It is believed that 30% of the population carries this genetic variation. While both tests provide useful information regarding an individual risk for periodontal disease, they require the use of laboratory. Significant advances are in development for screening of periodontal disease.

Researches have also reported high levels of inflammatory markers like C reactive protein (CRP) in association with chronic and aggressive periodontal disease 10(S). Researchers have developed a lab-on—a chip system to determine the difference of CRP levels between healthy individuals and patients with periodontal disease. The University of Michigan in collaboration with NIDCR has developed a rapid device, known as integrated microfluidic platform for oral diagnostics. This handheld, pocket sized test determines the amount of enzymes like MMP 8 in saliva in less than 10 minutes. Herr (2007) states that “MMP 8 has been identified as major tissue destructive enzyme in periodontal disease. Consequently, MMP 8 is a promising candidate for diagnosing and possibly more importantly, assessing the progression of periodontal disease”. Many of the biochemical chairside diagnostic test kits have been marketed. The newly commercially available chairside tests for host and bacterial markers of periodontal disease offer prospects which would make the monitoring of specific sites possible.

REFERENCES


