Periodontal inflamed surface area: quantifying inflammatory burden


Abstract

Background: Currently, a large variety of classifications is used for periodontitis as a risk factor for other diseases. None of these classifications quantifies the amount of inflamed periodontal tissue, while this information is needed to assess the inflammatory burden posed by periodontitis.

Aim: To develop a classification of periodontitis that quantifies the amount of inflamed periodontal tissue, which can be easily and broadly applied.

Material and Methods: A literature search was conducted to look for a classification of periodontitis that quantified the amount of inflamed periodontal tissue. A classification that quantified the root surface area affected by attachment loss was found. This classification did not quantify the surface area of inflamed periodontal tissue, however. Therefore, an Excel spreadsheet was developed in which the periodontal inflamed surface area (PISA) is calculated using clinical Attachment Level (CAL), recession and bleeding on probing (BOP).

Results: The PISA reflects the surface area of bleeding pocket epithelium in square millimetres. The surface area of bleeding pocket epithelium quantifies the amount of inflamed periodontal tissue. A freely downloadable spreadsheet is available to calculate the PISA.

Conclusion: PISA quantifies the inflammatory burden posed by periodontitis and can be easily and broadly applied.

Periodontitis is a chronic inflammatory disease of the supporting tissues around the teeth. Severe generalized periodontitis affects 5–15% of any population worldwide and is a major cause of tooth loss (Yoshida et al. 2001, Al-Shammari et al. 2005, Burt 2005). Periodontitis may, however, affect more than teeth and their surrounding structures. Periodontitis is claimed to be a risk factor for a broad range of diseases such as cardiovascular diseases and stroke (Janket et al. 2003, Scannapieco et al. 2003a, Khader et al. 2004, Bahekar et al. 2007) diabetes (Saremi et al. 2005, Khader et al. 2006), pneumonia (Scannapieco et al. 2003b) and preterm low birth weight (Scannapieco et al. 2003c, Khader & Ta’ani 2005).

The biological model for the plausibility of periodontitis as a risk factor for other diseases holds that periodontitis causes an inflammatory burden by eliciting bacteraemia (Chiu 1999, Madianos et al. 2001), systemic inflammatory responses (Kweider et al. 1993, D’Aito et al. 2004, Beck et al. 2005, Montebugno et al. 2005, Southerland et al. 2006), or cross-reactivity leading to auto-immune reactions (Rosenstein et al. 2004, Bartold et al. 2005). This inflammatory burden in turn causes damage to the human body far beyond the oral cavity. Following this biological model, the larger the amount of inflamed periodontal tissue is, the larger the chances are of periodontitis eliciting bacteraemia, systemic inflammatory responses or cross-reactivity. Therefore, any classification of periodontitis as a risk factor for other diseases should quantify the amount of inflamed periodontal tissue in order to quantify the inflammatory burden.

Many studies express periodontitis as a non-continuous variable. These

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studies use cut-off points to classify patients as being either unaffected or affected by mild, moderate or severe periodontitis. By definition, these classifications do not quantify the amount of inflamed periodontal tissue (Janket et al. 2003, Scannapieco et al. 2003a, b, c, Khader et al. 2004, Khader & Ta’ani 2005, Khader et al. 2006, Babekur et al. 2007, Merchant & Pithiphat 2007). Some studies linking periodontitis to other diseases do quantify periodontitis as a continuous variable, i.e. using mean probing pocket depth (PPD) or mean clinical attachment level (Janket et al. 2003, Scannapieco et al. 2003a, b, c, Khader et al. 2004, Khader & Ta’ani 2005, Khader et al. 2006, Babekur et al. 2007, Merchant & Pithiphat 2007).

Although these are continuous variables, this does not necessarily mean that these outcome measures quantify the amount of inflamed periodontal tissue. Thus, none of the classifications currently used expresses periodontitis as a continuous variable that is a measure of the amount of inflamed periodontal tissue. Hence these classifications may not quantify the inflammatory burden.

In addition, a great variety of classifications is used. In research on periodontitis as a risk factor for preterm low birth weight for example, 13 different classifications of periodontitis are being used (Scannapieco et al. 2003c, Khader & Ta’ani 2005, Vetter et al. 2008). Broad application of just one classification of periodontitis would allow for a better comparison of these studies, which may in turn provide decisive conclusions on periodontitis as a risk factor for other diseases.

The goal of this paper was to develop a new classification of periodontitis that quantifies the amount of inflamed periodontal tissue and which can be easily and broadly applied.

Material and Methods

Periodontal inflamed surface area (PISA)

Because no gold standard for periodontitis as a risk factor for other diseases exists, a list of demands was assembled for the construction of a new classification of periodontitis. The first and foremost demand was that the new classification should adequately quantify the amount of inflamed periodontal tissue. Second, the classification should be easy to use and broadly applicable. This means that the classification should make use of clinical measurements commonly used to establish periodontitis, i.e. Clinical attachment level (CAL), recession and bleeding on probing (BOP) measurements. A literature search was performed to look for a classification of periodontitis that met these demands. These in turn trying to enter the circulation (Amato et al. 1986, Thilo et al. 1986, Caton et al. 1988). Therefore, part of the PESA that consists of healthy epithelium may not contribute to the inflammatory burden.

The inflamed part of the PESA on the other hand, does theoretically pose an inflammatory burden. To establish a new classification of the PESA we propose calculating part of the PESA that is affected by BOP. After all, BOP reflects decreased collagen density, increased blood vessel density and fragility and a reduction of epithelial thickness and epithelial integrity (Greenstein et al. 1981, Polson et al. 1981, Davenport et al. 1982, Muller-Glauer & Schroeder 1982, Amato et al. 1986). Thin, fragile or even discontinuous pocket epithelium, may serve as an entrance for oral bacteria into the systemic circulation. Furthermore, BOP is characterized by a dense infiltration of inflammatory cells (Thilo et al. 1986, Caton et al. 1988). These inflammatory cells may play a key role in eliciting a systemic inflammatory response or cross-reactivity. Thus, the bleeding surface area, the periodontal inflamed surface area (PISA), may be thought of as the main contributor to any systemic inflammatory burden posed by periodontitis. Therefore, the PISA is defined as a classification of periodontitis that quantifies the amount of inflamed periodontal tissue and as such, quantifies the systemic inflammatory burden.

Results

Calculating PISA

Using the formulas described by Hjøiøl et al. (2001), a Microsoft Excel spreadsheet was constructed to facilitate PISA calculation. PISA is calculated in seven steps:

1. After filling in CAL measurements at six sites per tooth in the Excel spreadsheet, the computer calculates the mean CAL for each particular tooth.
2. The mean CAL around a particular tooth is entered into the appropriate formula for the translation of linear CAL measurements to the ALSA for that specific tooth (Hjøiøl et al. 2001).
3. After filling in recession measurements at six sites per tooth, the computer calculates the mean recession for each particular tooth.

4. The mean recession around a particular tooth is entered into the appropriate formula for the translation of linear recession measurements to the RSA for that specific tooth (Hajjøel et al. 2001).

5. The RSA for a particular tooth is subtracted from the ALSA of that particular tooth, rendering the PESA for that specific tooth, i.e. PESA = ALSA – RSA (Fig. 1a and b).

6. The PESA for a particular tooth is then multiplied by the proportion of sites around the tooth that was affected by BOP. For example, if three out of the maximum of six sites were affected by BOP, the PESA of that particular tooth was multiplied by 3/6, thereby rendering the PISA for that specific tooth.

7. The sum of all individual PISAs around individual teeth is calculated, amounting to the total PISA within a patient’s mouth.

Next, PISA was calculated for three different patients. Patient 1 has a healthy periodontium; exhibiting limited BOP with CAL not exceeding 3 mm (Fig. 2a). Patient 2 has severe local periodontitis; exhibiting BOP with CAL ranging from 6 to 10 mm locally (Fig. 2b). Patient 3 has severe generalized periodontitis, exhibiting widespread BOP with CAL ranging from 3 to 10 mm (Fig. 2c). PISA may thus range from 20.6 mm² (±0.3 cm²) in healthy individuals to 3,899 mm² (±39 cm²) in patients with severe generalized periodontitis (Fig. 2).

Spreadsheets are freely available from our website: www.parsproto.to.info. All it takes to calculate PISA is filling in CAL, recessions and BOP on six sites per tooth in this freely downloadable spreadsheet. In addition to research purposes, the spreadsheet might also be used to show patients their surface area of bleeding pocket epithelium in millimetres square, illustrating the inflammatory burden periodontitis potentially poses to their body.

Discussion

The great variation in periodontal classifications used in the various studies and the lack of a tool that adequately assesses the inflammatory burden of periodontitis is a major drawback of the studies published on the periodontal inflammation–systemic disease interaction. Therefore, a new measure of periodontitis as a risk factor for other diseases was developed, the PISA. PISA reflects the surface area of bleeding pocket epithelium in square millimetres. PISA is calculated using conventional CAL, recession and BOP measurements. PISA quantifies the amount of inflamed periodontal tissue, thereby quantifying the inflammatory burden posed by periodontitis. A freely downloadable spreadsheet is available to calculate PISA, therefore PISA can be easily and broadly applied. Broad application of PISA may provide decisive conclusions on periodontitis as a risk factor for other diseases. An additional advantage of the PISA is that it can be retrospectively calculated using existing research data containing CAL, recession and BOP measurements.

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Fig. 2. (a) Full mouth clinical attachment level (CAL), recession and bleeding on probing (BOP) measurements on six sites per tooth from a healthy patient. The periodontal epithelial surface area (PESA) is 1,125.9 mm² (≈11 cm²), the PISA is 28.6 mm² (≈0.3 cm²). (b) Full mouth CAL, recession and BOP measurements on six sites per tooth from a patient with severe localized periodontitis. The PESA is 1,878.6 mm² (≈19 cm²), the PISA is 1,048.6 mm² (≈10 cm²). (c) Full mouth CAL, recession and BOP measurements on six sites per tooth from a patient with severe generalized periodontitis. The PESA is 3,899.1 mm² (≈39 cm²), the PISA is 3,704.2 mm² (≈37 cm²).
Although PISA is thought to be the best tool for assessment of the amount of periodontal inflamed tissue that is currently available, for several reasons PISA might not precisely quantify the amount of inflamed tissue. First, CAL, recession and BOP measurements used for the calculation of PISA always include measurement errors related to observer, instrument, teeth patients and their interactions. Second, the formulas transforming CAL and recession to surface area, use population-based mean values of both root surface areas and root lengths. Thus, individual variations in root surface area and root length are not taken into account when calculating PISA. Third, PISA quantifies the amount of inflamed periodontal tissue in two dimensions, whereas in fact periodontitis is a three dimensional inflammatory process, i.e. extending into the connective tissue around the root. For these reasons, PISA may not precisely quantify the amount of inflammatory tissue. However, PISA likely quantifies the amount of inflamed periodontal tissue for each individual patient more accurately than any classification currently used.

PISA is, however, unable to accurately quantify the amount of inflamed periodontal tissue in case of pseudopockets or gingival overgrowth, i.e. when the gingival margin is located above the CEJ (Fig. 1c). In these cases, CAL will be smaller than PPD and ALSA will consequently be smaller than PESA. Therefore, using only CAL will underestimate the true PESA and thereby underestimate true PISA. Using PPD instead of CAL, i.e. entering PPD into the formula for CAL, will diminish this underestimation. However, given the conical shape of a root, it will still lead to a slight underestimation of true PISA. Fortunately, cases of gingival overgrowth are relatively rare, and often due to usage of medication (e.g. antiepileptic drugs, immune suppressive drugs, calcium blockers) or presence of certain diseases (e.g. leukaemia). These cases are not representative for the general population. Therefore, PISA can be used in the majority of cases, i.e. when the gingival margin is located at or below the CEJ. In case the gingival margin is located above the CEJ, PISA might still be used by replacing CAL with PPD, taking into account that it will result in an underestimation of true PISA. Finally, PISA might not adequately predict the probability of periodontitis to cause other diseases, even if it would be possible to measure precisely the amount of inflamed periodontal tissue. For example, the type of inflammation might be more important in causing other diseases than the amount of inflammation. Certain cells, proteins or inflammatory mediators might play a key role in causing other diseases. Furthermore, there might be a critical level of inflammation that functions as a threshold. When the threshold is passed, a response is initiated that causes damage to the human body beyond the oral cavity. Finally, PISA does not take into account the type of microbiological flora. Certain oral microorganisms might play a key role in causing other diseases, e.g. Campylobacter rectus, Prevotella intermedia, Porphyromonas gingivalis and Peptostreptococcus micros (Chiu 1999, Mädianos et al. 2001, Beck et al. 2005). For these reasons, the PISA might not be the only determinant in predicting the probability of periodontitis to cause other diseases. When additional information on the role of certain inflammatory mediators or microorganisms in causing other diseases becomes available, this information and the PISA can be incorporated in a new model that predicts the probability of periodontitis to cause other diseases even more accurately.

Although the PISA still has shortcomings, theoretically it appears to be a far better classification of periodontitis as a risk factor for other diseases than any classification currently used (face validity). The next step is to perform studies to analyse construct validity by means of correlating PISA with
measures of the activity, severity or presence of other diseases.

References


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Clinical Relevance

Scientific rationale for the study: Currently, all classifications of periodontitis fail to quantify the amount of inflamed periodontal tissue, while this may be the link between periodontitis and other diseases.

Principal findings: The PISA reflects the surface area of bleeding pocket epithelium in millimetres square, thereby quantifying the probability of periodontitis to cause other diseases. Practical implications: A freely downloadable spreadsheet is available to calculate PISA using CAL, recession and BOP measurements. This spreadsheet can be used to show patients their surface area of bleeding pocket epithelium, illustrating the inflammatory burden periodontitis potentially poses to their body.

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