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Peri-implantitis risk factors: A prospective evaluation

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Abstract

Aim: The aim of the present study was to create a tool to evaluate the risk of peri-implantitis according its severity.

Methods: After ethics committee approval, 43 patients provided signed consent and were included prospectively. Forty-five observations were recorded. The following criteria were recorded: number of implant faces showing bleeding and/or suppuration, pocket depth on at least two faces of the implant, bone loss as a function of the length of the implant evaluated on X-rays, number of implant faces with bacterial plaque, the parameters required for determination of excess cement (screwed or sealed prosthesis, burying of sealed prostheses), periodontal status, glycemia, and annual consumption of tobacco. Each of these parameters was plotted on a chart using Microsoft Excel.

Results: Seventeen of 45 (37.8%) cases were identified as having high peri-implantitis risk, two of 45 (4.4%) had low risk, and 11 of 45 (24.4%) had moderate risk; 33.3% patients did not have peri-implantitis and were considered at very low risk.

Conclusion: The observed results applied to the evaluation model are an effective diagnostic tool in assessing the risk of peri-implantitis. The tool takes into account parameters, which have not been taken into account until now. The information is automatically processed and allows early management of peri-implantitis.

KEYWORDS

evaluation model, implant, peri-implantitis, prospective evaluation, risk assessment

1 | INTRODUCTION

When inserting an implant after obtaining osseointegration, inflammatory complications of infectious origin might appear and affect the peri-implant tissues. The first stage of these complications is mucositis or inflammation of the mucosa without bone loss. Mucositis is reversible with appropriate treatment.[1] The next stage of complications is peri-implantitis. This complication is associated with bone loss and is not reversible. The prevalence of peri-implantitis is estimated at 10% of implants and 20% of patients within 5-10 years after implant placement with an increase in prevalence in case of tobacco-consumption or ancient periodontitis.[2,3] In a recent study, Rokn et al[4] reported a prevalence of 8.8% at 10 years in 20% of patients were followed for evaluation of peri-implant tissues complications without a regular maintenance program. Likewise, in his study of partial edentulous patients, Serino and Strom observed that peri-implantitis was mostly related to accessibility to oral cleaning.[5] The prevalence of peri-implantitis correlated with the number of implants inserted in an individual would increase, but has no influence on the prevalence of mucositis.[6] Peri-implantitis does not affect all patients similarly. Literature reviews have observed a significant

increase in the prevalence of peri-implantitis in patients treated for periodontitis.[7]

There is no real biologic evidence of the disruption of integration nor a description of this phenomenon. In their systematic review and meta-analysis, Clementini et al[8] revealed a multifactorial etiology. In general, peri-implantitis has a marginal bone loss in the form of a crater around the implant. It is most often accompanied by various clinical manifestations, such as excessive plaque, bleeding during probing, marginal recession, and difficulty in implementing appropriate oral hygiene.

This pathology represents a public health issue, because implantology has become a technique of choice to replace lost teeth, and industry data (Xerfi France, 2018, unpublished) show an increase in the use of implant devices. One can argue that these elements are connected to the evolution of the number of peri-implantitis. Moreover, if treatment strategies do not show any total efficiency,[9] different points characterize peri-implantitis.[10]

The absence of gradation, taking into account the various parameters involved in the disease, does not make it possible to differentiate the stages of peri-implantitis; it can result in confusion in the therapeutic management.[11] Standardization in the evaluation of the disease is necessary to implement the most appropriate treatment. Other classifications have already been proposed in the literature,[11,12] but do not take into account all the clinical elements that characterize peri-implantitis. In the present study, additional elements have been taken into account to establish this model. Based on an already-published periodontal risk assessment model,[13] we developed a new tool for assessing the level of risk peri-implantitis in the present study.

2 | MATERIALS AND METHODS

2.1 | Model development

A periodontal risk assessment model (bleeding on probing, prevalence of residual periodontal pockets, loss of teeth, estimation of loss of periodontal support in relation to the age of the patient, evaluation of systemic diseases, smoking habits) was described in 2003,[14] and later modified by adding supplementary parameters: glycemic state, plaque accumulation, socioeconomic factors, and stress.[13] We developed a peri-implantitis chart based on that reported for periodontitis.[13]

This chart takes into account only measurable elements, but excludes socioeconomic factors and stress, as in the Chandra study.[13] This chart included the following conditions: bleeding and/or probing, pocket depth, bone loss, excess cement, oral hygiene, history of periodontal disease, smoking habits, and glycemic status.

2.2 | Bleeding and/or probing

The method of Albrektsson et al was used as a reference.[15] The presence or absence of bleeding on probing is determined by the occurrence of bleeding within 15 seconds following a light probing with a colored and graduated periodontal probe or a foam probe with a 0.4-mm diameter. It is realized on the six side of the implant: mesio-vestibular, vestibular, disto-vestibular, mesio-lingual/mesio-palatin, lingual/palatal, distal-lingual/distal-palatal, measured and expressed in the number of affected implant faces (0-6) (Table 1).

2.3 | Pocket depth

The depth of the pocket can vary depending on the mucosa of coverage, the presence or absence of keratinized tissue, the type of restoration limiting the sounding, and the pressure applied.[16] Peri-implant sounding measurements are more sensitive to force variations than the probing of periodontal pockets, and has been recommended to apply a sample pressure equal to 0.25 N.[17] In order to ensure reproducibility, a probing force of 0.25 N is applied using a graduated periodontal probe. This probing is performed on at least two faces of the implant and is expressed in millimeters (Table 1).

2.4 | Bone loss

Implants vary in shape and design. Several studies have arbitrarily established the diagnosis of peri-implantitis on bone loss ≥1.8 mm (corresponding in average to the third turn of an implant).[18–20] Vertical bone resorption <0.2 mm after the first year of charging of an implant is now a generally accepted criterion of success.[15] However, an accurate assessment of this height is difficult to measure clinically. Therefore, peri-implantitis diagnosis based on a crater ≥1.8 mm after 1 year is difficult.[16,21] Standardized X-ray images can help determine the exact level of bone loss relative to a fixed reference point (eg crown-implant or implant-abutment junction). Nevertheless, it is difficult to measure and compare bone loss in

TABLE 1 Coding system for bleeding on probing and/or suppuration, probing depth, and bone loss

Axis score	Faces with bleeding on probing/presence of pus	Probing depth on at least 2 implant faces (mm)	Bone loss related to implant length (%)
0	0	<4	<10
1	1	≥4	10-20
2	2	≥5	20-30
3	3	≥6	30-40
4	4	≥7	40-50
5	5-6	≥8	>50

millimeters. The evaluation proposal is based on the percentage of bone loss relative to the length of the implant. Thus, the classification category is determined by the most severe deterioration of the implant with all faces combined. In order to avoid misclassification, it is important to measure two faces of the evaluated implant.

2.5 | Excess cement

Prosthetic cemented crowns on the implant are common alternatives to screwed implant crowns. Their use frequently results in excessive cement deposits in peri-implant tissue, despite careful clinical control at the time of sealing.[22] The more the cervical margin of a supra-implant crown is subgingival, the greater the risk of residual excess.[17] Dental X-rays cannot be considered as a reliable method of assessing excessive cementation.[17] Therefore, the following parameters were chosen: determination of the means of assembly of the prosthesis on the implant (screwed prosthesis or sealed prosthesis), and in the case of a sealed prosthesis, the measurement of the position of the cervical margin at the marginal gingiva (Table 2).

2.6 | Oral hygiene

When part of a dental implant is exposed in the oral cavity, bacterial colonization of the exposed surface occurs.[18,23] It has been shown that poor hygiene results in greater peri-implant resorption than when oral hygiene is satisfactory.[19] Poor accessibility to oral hygiene in implanted sites has also been correlated with peri-implantitis.[5] In a study that followed up implants over a 3-year period, dental plaque was identified as a significant factor in the development of peri-implantitis.[20] Hygiene was taken into account for our peri-implantitis risk assessment, and was measured by the presence or absence of subgingival bacterial plaque on the four faces of the implant: mesial, distal, vestibular, and palatal or lingual. The ratio is expressed as the number of affected implant surfaces (0-4); for implant-supported prostheses (Brånemark prostheses, implant-supported bridges), an additional evaluation of accessibility to hygiene is proposed (yes/no): yes, if the patient can brush in the implanted area with a toothbrush or use dental floss; and no, in the case of limited access (related to emergence profile in particular) and/or disability of the patient (Table 3).

TABLE 2 Coding system for excessive cement according to assembly means and cervical margin position

Axis score	Prothesis Type
0	Screw-retained prosthesis
1	Cemented prosthesis with supragingival cervical margin
2	Cemented prosthesis with juxta gingival cervical margin
3	Cemented prothesis with intra-sulcus margin <1 mm
4	Cemented prothesis with intra-sulcus margin of 1-2 mm
5	Cemented prosthesis with intra-sulcus margin >2 mm

2.7 | Antecedents of periodontal diseases

Individuals at risk of periodontitis respond differently to microbial aggressions than patients who are not at risk.[24] Therefore, individuals with a history of periodontal disease might also have an increased risk of developing peri-implantitis. Several studies have concluded that patients with a history of periodontitis have a more significant incidence of depth, marginal bone resorption, and peri-implantitis risk than patients with healthy periodontium.[21,25] The majority of patients with periodontitis have greater implant failure. The presence of periodontal pathogens around failed implants could suggest a direct link between periodontitis and peri-implantitis by the translocation of these species from their intraoral niches to implant sites,[26,27] as suggested by several studies.[18,23,28] Therefore, patients' history of periodontitis was taken into account in the evaluation of peri-implantitis risk; the score was calculated based on the periodontal status (healthy/treated/pathological) and the type or stage of periodontal disease (Table 3).

2.8 | Smoking

Smoking changes various aspects of innate and adaptive immune responses of the host.[29,30] Smokers show an increase in the number of granulocytes and the total number of white blood cells,[31] an increase in the life of polymorphonuclear cells,[32] the production of superoxide anion and hydrogen peroxide, the expression of integrins,[30] and the production of protease inhibitors.[33] The humoral immune response is also disrupted by tobacco smoking.[34,35] Smoking inhibits the proliferation and/or function of B and T lymphocytes.[36] Studies comparing the subgingival microbiota of smokers and non-smokers, however, have yielded conflicting results.[37-39] Although the relationship between smoking and peri-implantitis remains controversial, several studies have reported a statistically-significant difference in the incidence of peri-implantitis between smokers and non-smokers,[21,40] with a strong association between peri-implantitis and smoking.[41] Referring to earlier models, the number of packets of cigarettes per year served as a reference for the study.[42] Considering that the number of cigarettes per pack varies from one country to another, this can be a confounding factor.[43] Similar to the Chandra evaluation model,[13] this criterion is now measured in cigarettes per day, in order to take this possibility into account (Table 3).

2.9 | Diabetes

Severe periodontal disease affects the blood sugar of diabetics. In diabetic patients, there is a direct relationship between the severity of periodontitis and complications of diabetes.[44] Based on the available data on the association between periodontal disease and systemic pathologies, it is logically assumed that these pathologies can also influence the development of peri-implantitis. Ferreira et al[45] demonstrated,

TABLE 3 Coding system for periodontal diseases, glycemic status, smoking, and oral hygiene

Axis Score	Periodontal status	Glycosylated hemoglobin A1c level (%)	Smoking (cigarettes/d)	No. implant faces with presence of plaque
0	Healthy periodontium	≤6	Non-smoker	0
1	Treated periodontitis	6, 1-7	Former smoker	1
2	Slight chronic periodontitis	7, 1-8	<10	2
3	Moderate chronic periodontitis	8, 1-9	10-19	3
4	Severe chronic periodontitis	9.1-10	20	4
5	Aggressive periodontitis	>10	>20	No accessibility to oral hygiene

by univariate analysis, that diabetics are more likely to develop peri-implantitis, which was confirmed by Gomez-Moreno et al[46], who reported that in type 2 diabetes patients who had been followed up for a 3-year period, marginal bone loss and peri-implant bleeding correlated with increased glycosylated hemoglobin A1c (HbA1c). In addition, Aguilar-Salvatierra et al[47] found that type 2 diabetes patients can receive implant treatments with immediate safe loading, provided they have moderate HbA1c levels.

In the evaluation model for periodontal disease proposed by Chandra,[13] the selected parameter was fasting glucose, which was expressed in mg/dL. According to the diagnostic reference values established by the American Academy of Periodontology,[48] blood glucose <110 mg/dL implies a low periodontal risk, and a value >126 mg/dL indicates a high risk; a value between these reflects a moderate risk. However, blood glucose only provides a snapshot of glycemic status, whereas HbA1c is useful for assessing glycemic control over a longer period (approximately 2-3 months). It is therefore a more interesting medical constant; it is a marker of the risk of long-term complications of diabetes: This medical constante was selected for this study (Table 3).

2.10 | Model development and calculation method

2.10.1 | Developed model

From a graphical point of view, the peri-implantitis risk analysis model generates a functional diagram, which according to the limits of the polygon obtained, makes it possible to classify a patient into three categories – low, moderate, or high risk – and materialize it on the graphic by different colors (Figure 1).

2.10.2 | Very low risk of peri-implantitis

Individuals with very low risk of peri-implantitis should have all parameters in the low-risk area. We accept for this category a single parameter in the moderate zone.

2.10.3 | Low risk of peri-implantitis

A peri-implantitis risk is considered to be low if all the parameters are in the low-risk area or if a maximum of two parameters are in the moderate-risk zone and the high-risk area (Figure 2). For example, a patient with low peri-implantitis risk presented with three out of six implant faces with bleeding on probing, a pocket depth <4 mm on at least two faces of the implant, bone resorption <30% of implant length (screwed prosthesis), two implant faces with plaque, and severe chronic periodontitis, and was a non-smoker and non-diabetic.

2.10.4 | Moderate risk of peri-implantitis

A risk of peri-implantitis is considered to be moderate if at least three parameters are in the moderate risk zone and no more than one parameter is in the high-risk area (Figure 3). For example, a patient at moderate risk of peri-implantitis had three implant faces out of six with bleeding on probing, a pocket depth ≥6 mm on at least two faces of the implant, bone resorption >30% of implant length, sealed prosthesis with bury limit under the gingiva <1 mm, two implant faces with plaque, and chronic periodontitis treated in 2008, and was a non-smoker and non-diabetic.

2.10.5 | High risk of peri-implantitis risk

A risk of peri-implantitis is considered high if at least two parameters are in the high-risk area (Figure 4). For example, a patient at high peri-implantitis risk presented suppuration on all implant surfaces, a pocket depth ≥8 mm on at least two faces of the implant, bone resorption >50% of implant length, a screwed prosthesis with intra-sulcus margin of 1-2 mm, dental plaque on one implant surface, and severe chronic periodontitis, and was a non-smoker and diabetic with HbA1c >8%.

2.10.6 | Evaluation of the severity analysis model

Forty-three patients with at least one implant were randomly selected from the Odontology Department at La Timone Hospital in Marseille, France. Among these patients, some came for routine check-ups, and others for an implant problem. For each, an informed consent form and a questionnaire were obtained. The present study was approved by the ethics committee of the University Hospital Institute in Marseille (no. 2016-011). A thorough clinical examination was performed and dental X-rays of the implant were also undertaken. In addition, patients with peri-implantitis were subjected to 3-D cone-beam computed tomography in order

to complete the radiological evaluation. To avoid errors due to variability of the observer, two previously-trained dental surgeons (AM and GA) performed all of the clinical and radiographic examinations.

The recorded parameters included the number of implant faces with bleeding and/or suppuration on probing, the pocket depth on at least two faces of the implant, the bone loss relative to the implant length evaluated on dental X-rays, the number of implant surfaces with plaque, the parameters required for the determination of excessive cement (screwed or sealed prosthesis, bury limit of sealed prosthesis), periodontal status, diabetes, and tobacco consumption. Using Microsoft Excel software (Microsoft, Redmond, WA), each of these parameters was plotted on a radar chart. The results obtained made it possible to evaluate the risk specific to each patient.

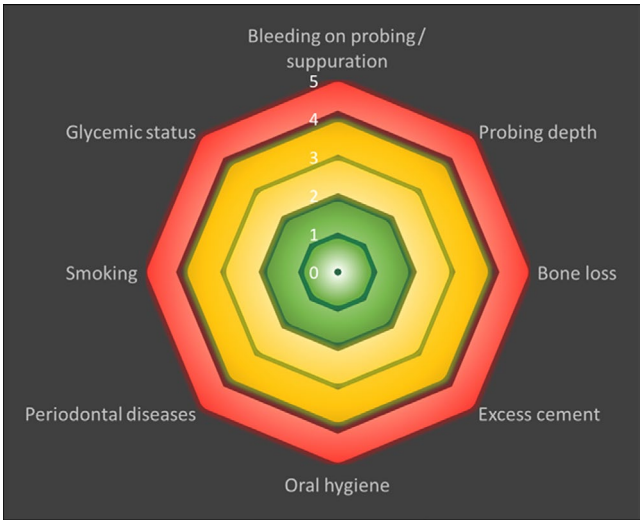

FIGURE 1 Proposed risk diagram with the clinical criteria, radiographic measures, and risk factors divided in eight parameters on three separate risk areas: low, moderate, and high Low-risk area; Moderate-risk area; High-risk

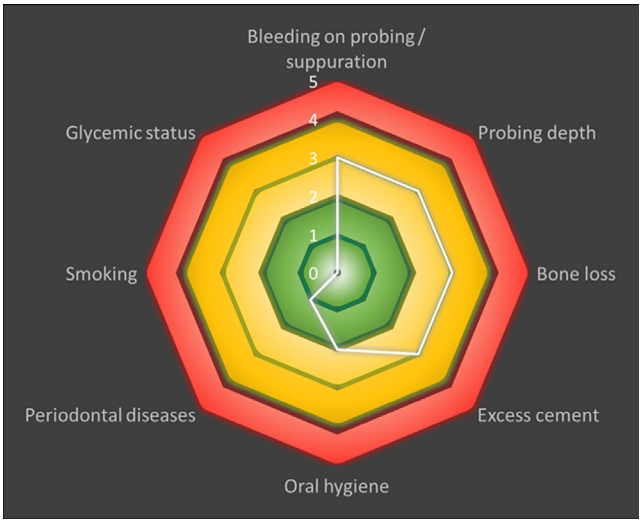

FIGURE 3 Patient with moderate peri-implantitis risk: three of six implant faces with bleeding on probing, a probing depth ≥6 mm on at least two faces of the implant, bone resorption >30% implant length, sealed prosthesis with intra-sulcus margin <1 mm, two implant faces with plaque, and chronic periodontitis treated in 2008; patient was a non-smoker and non-diabetic Low-risk area; Moderate-risk area; High-risk

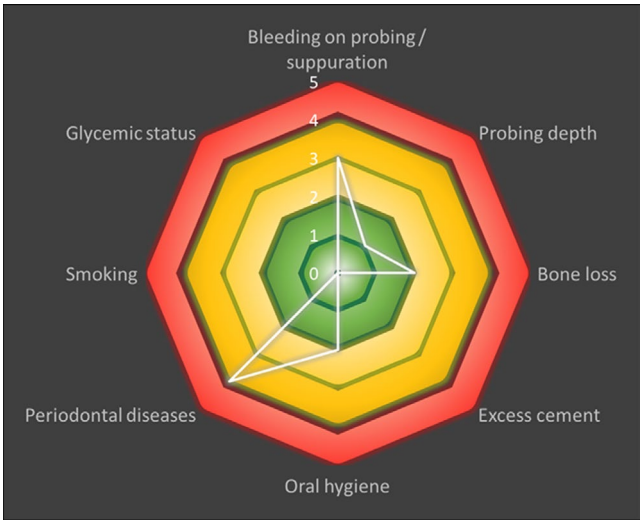

FIGURE 2 Patient with low peri-implantitis risk: three of six implant faces with bleeding on probing, a probing depth <4 mm on at least face faces of the implant, bone resorption <30% of the implant length (screwed prosthesis), two implant faces with plaque, and severe chronic periodontitis; patient was a non-smoker and non-diabetic Low-risk area; Moderate-risk area; High-risk

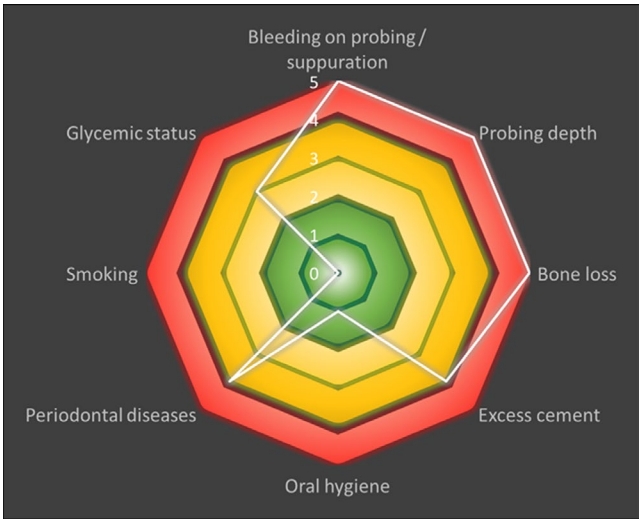

FIGURE 4 Patient with high peri-implantitis risk: suppuration on all implant surfaces, a probing depth ≥8 mm on at least two faces of the implant, bone resorption >50% implant length, a screwed prosthesis with intra-sulcus margin of 1-2 mm, dental plaque on one implant surface, and severe chronic periodontitis; patient was non-smoker and diabetic with glycosylated hemoglobin A1c >8% Low-risk area; Moderate-risk area; High-risk area

TABLE 4 Clinical data collection of pathological implants and controls: sex, bleeding on probing, probing depth, surfaces with presence of plaque (%), presence of pus, number of implants affected by peri-implantitis, mobility, vertical resorption of the supporting bone (% of implant length/largest depth of the peri-implantitis pocket at the end of the implant measured on the radio)

Sample (participant no.)	Sex	Bleeding on probing	Probing depth (mm)	Surfaces with presence of plaque (%)	Presence of pus	Implants affected by peri-implantitis (N)	Presence of mobility	Vertical resorption of the supporting bone (% of implant length)
1	Female	Generalized	8	50	No	4	No	48
2	Female	Generalized	7	100	Yes	2	No	70
3	Female	Generalized	7	25	No	1	No	51
4	Male	Localized	4	100	No	2	No	37
5	Male	Localized	6	100	No	7	No	46
6	Female	Localized	4	50	No	2	Yes	29
7	Female	Generalized	7	50	No	3	Yes	69
8	Female	Localized	8	25	Yes	1	No	51
9	Female	Localized	5	25	No	2	No	64
10	Male	Localized	4	25	No	1	No	46
11	Female	Localized	7	100	Yes	2	No	71
12	Female	Localized	12	25	No	1	Yes	100
13	Female	Localized	10	50	No	5	No	60
14	Female	Generalized	9	100	No	2	Yes	74
15	Female	Localized	6	100	No	1	No	52
16	Male	Generalized	5	100	Yes	6	No	35
17	Female	Localized	7	25	Yes	7	No	67
18	Female	Localized	8	100	Yes	1	No	60
19	Male	Generalized	7	100	Yes	4	No	57
20	Female	Localized	10	100	Yes	2	No	81
21	Female	Localized	6	50	No	1	No	50
22	Male	Localized	6	100	Yes	1	No	35
23	Female	Localized	9	100	No	2	No	40
24, 25, 26	Female	Generalized	10	100	No	4	No	85
27	Male	Localized	6	100	No	2	No	36
28	Male	Generalized	7	100	Yes	2	No	35
29	Female	Generalized	9	50	No	4	No	46
30	Female	Localized	6	50	No	2	No	35
31	Male	Localized	1	25	No	0	No	4
32	Female	—	4	0	No	0	No	15

(Continues)

TABLE 4 (Continued)

Sample (participant no.)	Sex	Bleeding on probing	Probing depth (mm)	Surfaces with presence of plaque (%)	Presence of pus	Implants affected by peri-implantitis (N)	Presence of mobility	Vertical resorption of the supporting bone (% of implant length)
33	Female	Localized	1	25	No	0	No	9
34	Female	—	2	0	No	0	No	11
35	Female	Localized	1	0	No	0	No	17
36	Male	Generalized	1	0	No	0	No	8
37	Female	Localized	3	0	No	0	No	9
38	Male	Localized	1	25	No	0	No	11
39	Female	—	3	0	No	0	No	9
40	Male	—	1	25	No	0	No	2
41	Male	Localized	1	25	No	0	No	5
42	Male	Generalized	1	25	No	0	No	6
43	Male	—	1	0	No	0	No	16
44	Male	—	1	0	No	0	No	2
45	Female	Localized	1	25	No	0	No	0

3 | RESULTS

Sixteen male and 27 female patients aged 35-86 years with an average age of 62.7 years were enrolled in the present study. All patients received implant therapy. Forty-five observations were recorded; three observations of peri-implantitis were found in the same patient. In total, we found 30 individuals with peri-implantitis, 10 had generalized bleeding, and 18 had localized bleeding. Pocket depths ranged from 4 to 12 mm. The number of implants affected by the peri-implantitis varied from one to seven. In four cases of peri-implantitis, the presence of pus was observed. Vertical resorption of the carrier bone (% of implant length) ranged from 29% to 100% (100% in only 1 case; in this case the implant was only adherent to the mucosa and had to be extracted) (Table 4). In addition, four individuals were smokers and two other patients were former smokers. One patient was confirmed to be diabetic. Twenty-two individuals had active chronic periodontitis, including a mild periodontitis form, four moderate, and 17 severe forms. Of the remaining six patients, three had a history of treated and stabilized chronic periodontitis. The latter three patients showed no signs of periodontal disease.Of the 28 patients with peri-implantitis, 25 had one or several peri-implantitis associated with an implant-supported crown or implant-supported bridge. Sealing assembled 13 prosthetic elements and 12 screwed. The remaining three patients had complete bridge Brånemark prostheses.

In the proposed evaluation model, 15 cases were reported to have a very low risk of peri-implantitis, two cases had a low risk of peri-implantitis, 11 were moderate risk, and 17 were high risk. Approximately 37.8% of cases were in the high-risk peri-implantitis category, whereas only 4.4% of cases were in the low-risk category, with 33.3% with a very low risk. The remaining 24.4% of cases were moderate risk (Figure 5).

Among the very low-risk patients, seven presented with localized bleeding and two with generalized hemorrhage; six had no bleeding. Pocket depths ranged from 1 to 3 mm, with the exception of one patient with a pocket that measured 4 mm. The presence of bacterial plaque was observed in seven patients on 25% of the surfaces of the implants. No pus was found in any case.

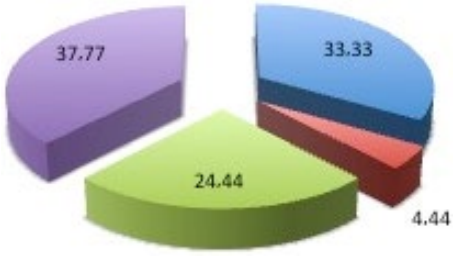

FIGURE 5 Distribution of very low-, low-, moderate-, and high-risk cases according to the proposed assessment model. ■, very low risk; ■, low risk; ■, moderate risk; ■, high risk

4 | DISCUSSION

In the present study, we developed a risk assessment of peri-implantitis. The model developed was tested on 43 patients and 45 observations were made. Of these 45 observations, we observed 30 cases of peri-implantitis: 17 at high risk (37.77%), 11 at moderate risk (24.44%), two at low risk (4.44%), and 15 observations at very low risk (33.33%). This work made it possible to determine four classes of risk levels of peri-implantitis. The last class where the risk was very low could considered as the control group. This proposed chart model is based on the already-published effective model of periodontal risk assessment,[13] which proved to be useful for accurately assessing periodontitis.

In the model, there were eight observed parameters. Few studies have been reported to classify peri-implantitis; however, clinicians emphasize the need for this classification. In the proposed classification by Froum and Rosen,[11] a combination of probing and/or suppuration bleeding, sounding depth, and extent of radiographic bone loss around the implant were used to rank the severity of peri-implantitis. In the present study, we identified three categories of peri-implantitis: early, moderate, and advanced. Although bleeding and/or pus were mentioned in the present study as the best clinical indicators for determining the presence of inflammation, glycemic status and smoking habits were not considered. These two factors are considered important parameters for the evolution of peri-implantitis, as they can constitute a relative contraindication to implant placement (unstable diabetes or a significant daily amount of cigarettes is a strict contraindication to implant treatment, whereas properly-stabilized diabetes or only a few daily cigarettes can be considered for implant treatment). The classes determined by Froum and Rosen,[11] are substantially the same as those in our work; it can nevertheless be noted that the authors did not take into account the history of periodontal disease, which is contrary to our study.

In a study targeting New Zealand oral periodontists and oral maxillofacial surgeons in order to improve the understanding, diagnosis, and management of peri-implantitis, the interviewed clinicians emphasized the importance of assessing peri-implantitis, probing depths, and radiographic evidence of bone loss.[49] This evaluation was decisive for the implementation of the treatment. This present study also confirmed that treatment varies according to the size of the lesions.

In our study, the score obtained provided quantitative information to clinicians, while the polygon from the functional diagram added a qualitative component to the diagnosis. The advantage of the proposed model was therefore twofold: it is a tool to aid diagnosis, while standardizing individual care. After reviewing the literature, eight parameters were selected to be part of the proposed evaluation model; however, several criteria, notably of a clinical nature, were not included. Among them, occlusion and the amount of keratinized mucosa must be mentioned. Concerning the occlusal factor, a biomechanical overload at the bone-implant interface could cause, or at least contribute to, marginal bone loss according to the hypothesis of occlusal surcharge. Some recent studies have suggested that occlusal load/occlusion could be a contributing factor to peri-implantitis.[50,51] However, the question remains, and this is the reason for not including this parameter into this model. Although evidence of the impact of occlusal surcharge on peri-implantitis is lacking, an assessment of patient occlusion during follow-up visits is justify.[52,53]

The presence of keratinized gingiva and its required level of thickness in the peri-implant region has little influence as far as proper oral hygiene is performed. This is in contrast to keratinized mucosa, which when absent can lead to difficulty/sensitivity to brushing and further impair the control of the dental plaque and lead to important tissue lesions. For cases with a keratinized gingiva defect in the proximity of the implant, the study concludes that the absence of tissue does not necessarily negatively affect the health of the peri-implant tissues. In the event that the absence of keratinized gingiva would interfere with the control of the elimination of the dental plaque, this proves to be an indirect impact parameter. According to proposed model, it makes more sense to include a direct assessment of oral hygiene, rather than a clinical criterion influencing it.

Despite the fact that this parameter was not included in the evaluation model, the question of the importance of keratinized mucosa at the peri-implant level persists, and this factor should be discussed in future studies on the incidence of peri-implant diseases. The genotype and its association with tobacco consumption were not included in this model. According to the literature, the polymorphism of the interleukin (IL)-1 gene would have an impact on peri-implantitis, especially in the case of heavy smokers. Feloutzis et al[54] reported significant differences in alveolar bone loss between non-smokers and heavy smokers in the IL-1-positive genotype group and none in the IL-1-negative genotype group. Gruica et al[55] showed that heavy smokers with positive IL-1A and IL-1B genotypes had a greater risk of developing inflammatory complications and increased peri-implant marginal bone resorption compared to non-smokers of the same genotype. In a later study, Laine et al[56] found an association between the polymorphism of the IL-1RN gene and peri-implantitis, particularly in smokers. The proposed evaluation model was based on a simple, rapid, and reproducible measurement of each parameter, while remaining accessible to clinicians in everyday practice. Given the multiple limitations associated with a genotype study, we decided not to include this criterion in the evaluation model.

Tobacco remains a major concern for clinicians: the level of inflammation markers is higher in waterpipe (narghile) smokers with peri-implantitis than in non-smokers with peri-implantitis.[57] In Ata-Ali et al[58]'s study, smokers demonstrated a greater expression of IL-1β, IL-6, IL-10, and tumor necrosis factor-α, but the opposite for IL-8, albeit with no statistical significance. Tobacco alone does not influence the immunological parameters and bacteria of the red complex *Tannerella forsythia*, *Treponema denticola*, and *Porphyromonas gingivalis* of patients with dental implants and healthy peri-implant tissues.

These observations are still debatable. For example, the level of other pathogens identified as biomarkers of periodontitis, such as *Methanobrevibacter oralis*,[59] are not found in peri-implantitis

significantly in smokers,[60] while at the same time, the presence of methanogenic archaea in saliva is influenced by tobacco.[61]

In their cross-sectional study, Pimentel et al[62] noted the shortcoming of standardization to describe peri-implantitis, which was important for the number of peri-implantitis observed, and showed that smoking increased the probability of peri-implantitis by three factors. Tobacco remains to be fully elucidated. Caution is required and clinically-important daily smoking remains a relative or absolute contraindication for certain cases.

It is possible to compare the proposed evaluation model with the work of Froum and Rosen on a new classification of peri-implantitis.[11] They classify the severity of peri-implantitis based on three clinical criteria: bleeding and/or probing suppuration, pocket depth, and peri-implant bone resorption level. The objective of their work was to propose a standardized classification that can be used for communication between researchers and clinicians, allowing better comparison of the results and calibration of the studies. This classification was limited because it defined an implant status by only three criteria, whereas our proposed model allowed us to define a risk of progression of the peri-implantitis based on eight parameters calibrated between them. While Froum and Rosen measured the severity of the pathology at a specific time,[11] our proposed evaluation model assessed its susceptibility to evolution. The comparison of the results of the present study with that of Froum and Rosen makes it possible to demonstrate a link between the severity of a peri-implantitis and its susceptibility to aggravation; the greater the severity of peri-implantitis, the greater the risk of progression.[11] On the contrary, the earlier the diagnosis of peri-implantitis is established, the less likely it is to develop. This correlation confirms the non-linear evolution of peri-implant pathologies, a fortiori if aggravating factors are added (diabetes, tobacco consumption, periodontal disease, residual cement, poor oral hygiene), as shown by the qualitative analysis in Figure 1.

With the exception of very low risk cases (15/45) that can be considered as control group patients without peri-implantitis, the results of the present study highlight the low percentage of patients with low peri-implantitis risk (2/30, 4.44%). This reflects the difficulty in diagnosing early forms, which are often asymptomatic with little or no clinical manifestations and are most often the object of fortuitous discoveries. Moreover, therapy is all the more effective as the management of the peri-implantitis is precocious.[63] As Serino and Turri demonstrated in their study,[64] the proportion of unhealthy implants became healthy again 2 years after surgical treatment; the rate for this was higher for implants with a smaller initial bone crater (2-4 mm) compared to implants with initial bone loss ≥5 mm (74% and 40%, respectively).

From early diagnosis to regular maintenance, to the rapidity of treatments, prevention has an essential role in the management of peri-implantitis and in reducing their occurrence.[9] It is also important to underline the multifactorial aspect of this pathology, thus the importance of an overall understanding of the parameters influencing the level of peri-implantitis risk. The ultimate aim is to categorize each patient and to adapt the therapy individually according to the level of risk. However, the limitations of the present study must be noted because of the small number of cases observed. It is necessary to expand the cohort number to increase the power of the statistical tests. However, the present study had the practical consequence of setting up a specific peri-implantitis consultation in the Odontology Department of the Timone Hospital in Marseille, France. Clinicians have a new assessment tool at their disposal and can plan the treatment according to the level of risk. This tool can also be used for monitoring the disease after treatment (http://diagnostic-tool.pagesperso-orange.fr/).

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