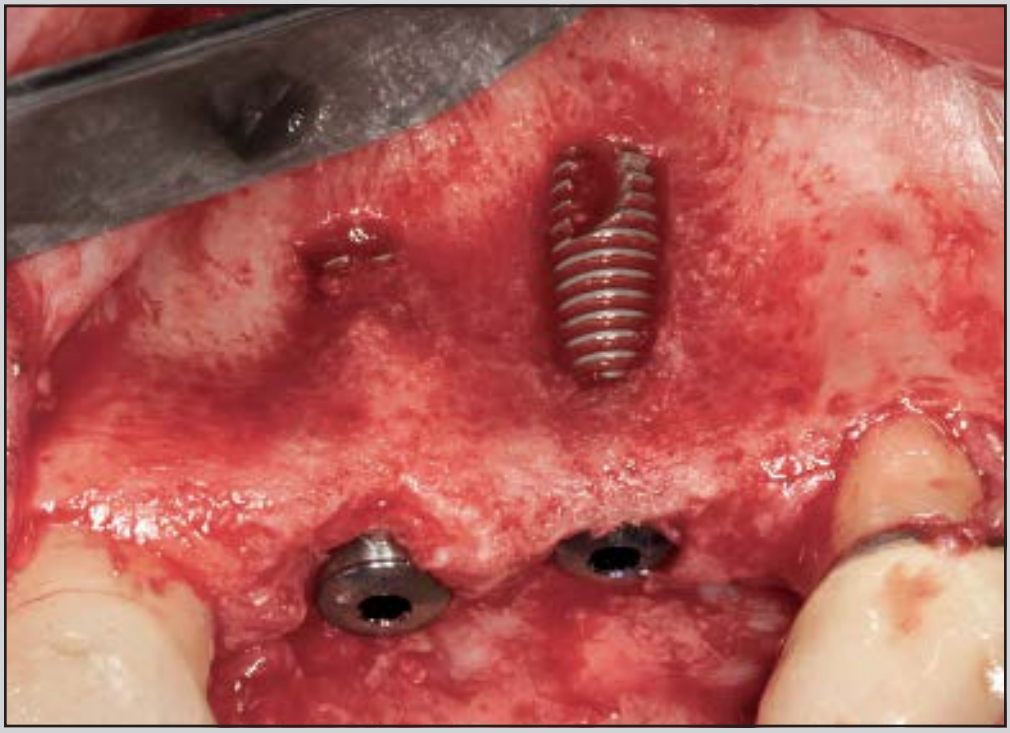
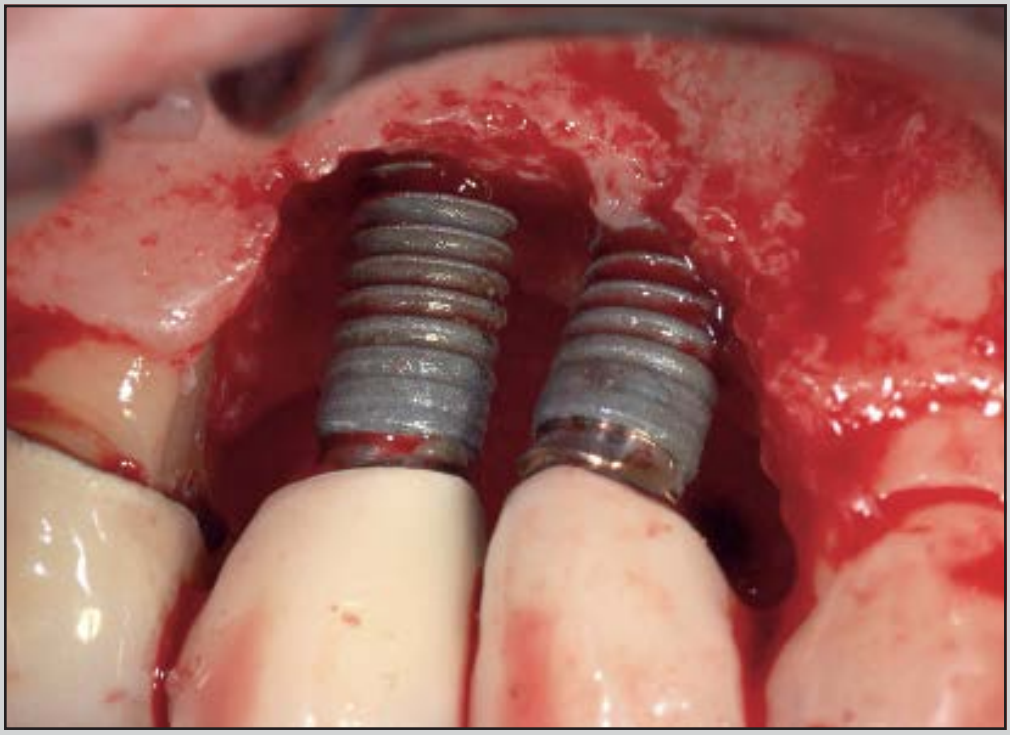
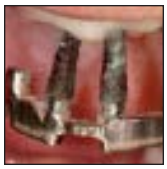




A Classification System for Peri-implant Diseases and Conditions



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Peri-implant bone levels are influenced by pathologic and nonpathologic conditions. The understanding of peri-implant disease has evolved over the past several decades, and the classification of peri-implantitis has been limited to descriptions of disease progression or those involving soft and/or hard tissues (peri-implant mucositis or peri-implantitis). However, no classification system has been established based on etiology. The objective of this study was to identify various etiologies for peri-implantitis and to establish a classification system based on the pathogenesis. The results indicate that the majority of bone loss was related to biofilm, followed by iatrogenic factors, exogenous irritants, absence of keratinized tissue, and extrinsic pathology. The proposed classification system will allow the clinician to properly diagnose peri-implant diseases in relation to etiology. These conditions may respond differently to applied therapies. Int J Periodontics Restorative Dent 2016;36:699–705. doi: 10.11607/prd.2918

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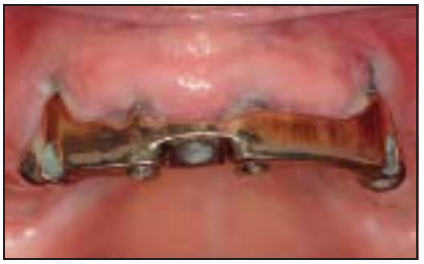
The long-term maintenance of bone around an osseointegrated implant is critical to clinical success.[1,2] Peri-implant bone levels are influenced by pathologic and nonpathologic conditions. Several factors may affect peri-implant bone resorption, including local, surgical, implant, and postrestorative factors.[3,4] Systemic diseases such as diabetes and osteoporosis, genetics, certain medications, and smoking also play a role as cofactors.[5,6] Clinical peri-implantitis has been characterized as an infectious condition of the tissues around osseointegrated implants with signs of inflammation (bleeding and/or suppuration on probing) and progressive radiographic loss of supporting bone.[7] The prevalence of peri-implantitis varies among studies from 10% to 92% of implants, from 12% to 100% of patients, and from 5 to 10 years after implant placement.[8–12] Generally, the prevalence has been based on the patient pool number, prior history of periodontitis, smoking, and type and frequency of maintenance.[8] The occurrence of peri-implantitis will also vary depending on the bone loss and/or probing depth thresholds used and on the cut-off values for clinical parameters reported in the different studies.[13]

The present understanding of peri-implant disease has evolved over the past several decades. Early

Table 1 Peri-implantitis etiology classifications

Origin	Definition	Example
Peri-implantitis induced by pathogenic bacteria/biofilm	Any implant with an etiology due to plaque- or bacteria-induced biofilm	Plaque, biofilm, calculus, host susceptibility to periodontitis (previous/active)
Peri-implantitis induced by exogenous irritants	Implants that show signs of residual cement or other exogenous irritants	Residual cement, smoking, impacted food debris
Peri-implantitis induced by iatrogenic factors	Implants that present with bone loss due to iatrogenic damage, implant malposition, or functional occlusal overload	Buccal implant placement, inadequate interimplant distance, overheating during surgical placement, poorly fitting restorations
Peri-implantitis induced by extrinsic pathology	Implants that present with bone loss caused by an unrelated pathology, systemic disease, and/or papilla migration to an implant	Proximal periapical pathology, proximal carcinoma, latent endodontic lesion postextraction
Peri-implantitis induced by absence of keratinized tissue (AKT)	Implants presenting with an AKT and/or no gingival attachment	Absence of attached gingiva, lack of keratinized tissue with or without muscle attachment



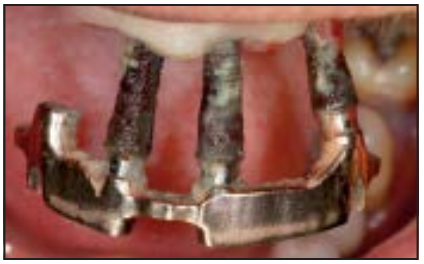


Fig 1 *A gross failure in which three out of four implants were coated with biofilm in a nonattending overdenture patient who had not taken out her teeth for cleaning in the previous 5 years, since she was last seen by her dentist.*

studies described peri-implant disease as early loss of marginal bone as part of the normal healing process,[14] loss of osseointegration due to overload,[15–17] or a bacterial process similar to periodontitis.[7] In periodontitis, the classification system described by Armitage recognizes that several etiologic factors or conditions can result in the loss of periodontal support.[18] Currently, the classification of peri-implant bone loss is generally limited to the inflammatory process around an implant.[19] In the past it has been limited to descriptions of disease progression (early, moderate, or advanced) or involvement of soft and/or hard tissues (peri-implant mucositis or peri-implantitis) as defined by probing depth, bleeding on probing, presence of suppuration, and bone loss.[20] However, no classification system has been established based on different and distinct etiologies or conditions that result in a peri-implant inflammatory response with bone loss. Therefore, the objective of this study was to identify various etiologies for peri-implantitis and establish a classification system based on the pathogenesis.

Materials and methods

The protocol was approved by the Institutional Review Board at the University of Pennsylvania. In this retrospective study, patients were screened for peri-implantitis. Data collection included medical and dental information. Medical data included smoking history and systemic conditions. Dental implant health parameters were assessed by presence or absence of radiographic peri-implant bone loss (crestal and/or noncrestal) and presence or absence of keratinized tissue. A clinical examiner measured the deepest probing depth (UNC 15, Hu-Friedy) for each implant, which were categorized into < 4 mm, 4 to 5 mm, and > 5 mm. Dental implants with peri-implantitis were classified into five potential etiologic categories: biofilm, exogenous irritants, iatrogenic factors, implants presenting with an absence of keratinized tissue (AKT)

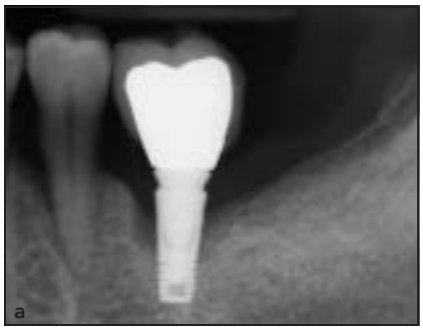
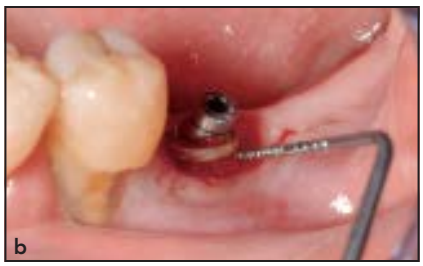


Fig 2 *Radiograph* (a) *and clinical photos* (b, c) *showing excess dental cement associated with signs of peri-implant disease and radiographic bone loss.*



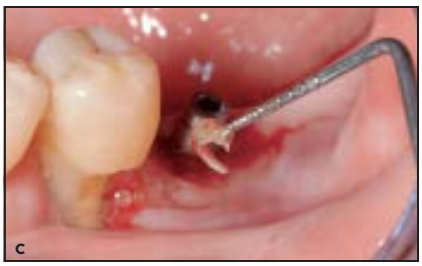
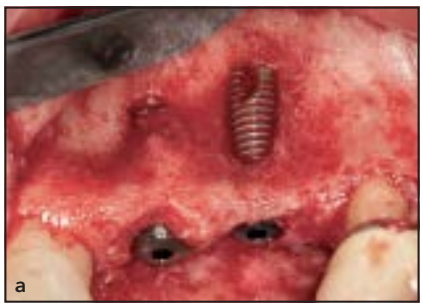


Fig 3 *Implants placed too buccally* (a) *and with inadequate interimplant distance* (b) *are examples of peri-implantitis induced by iatrogenic factors. Operator error may frequently be the cause of bone loss and ultimately peri-implantitis.*



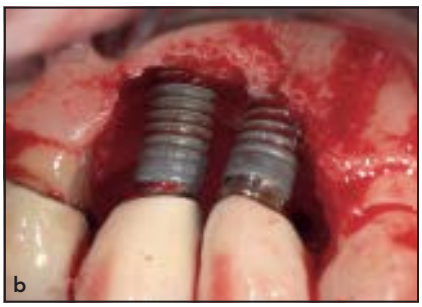
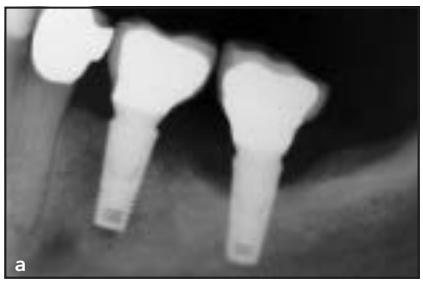
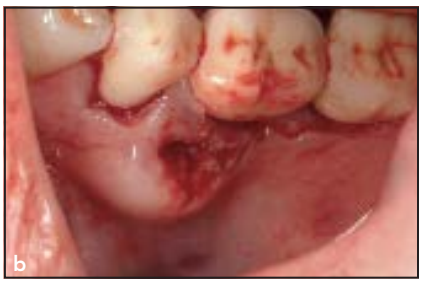
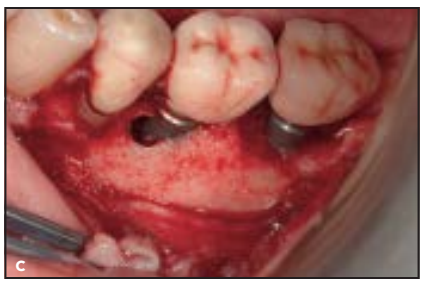
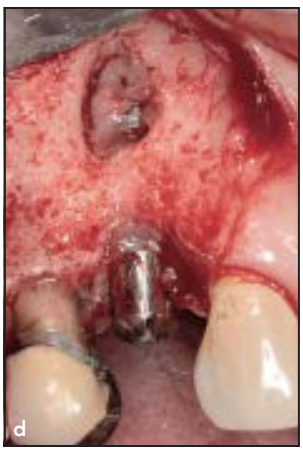


Fig 4 (a) *A radiographic and clinical diagnosis of peri-implantitis with acute abscess formation was made for this case, and it was treated accordingly.* (b) *However, subsequent surgical investigation revealed a solid nonfluctuant mass with associated peri-implant infrabony defects.* (c,d) *Concern was immediately raised and a biopsy taken. The result was squamous cell carcinoma in situ. The patient underwent a partial mandibulectomy without involvement of the tongue. The disease recurred, and even after block neck dissection and radiotherapy the patient eventually presented with multiple metastases and died of her condition 4 years after the first diagnosis.*









with muscle attachments, and extrinsic pathology (Table 1 and Figs 1 to 6). Parameters were analyzed with mean and standard deviation and outcome of categorization expressed as percentage frequency.

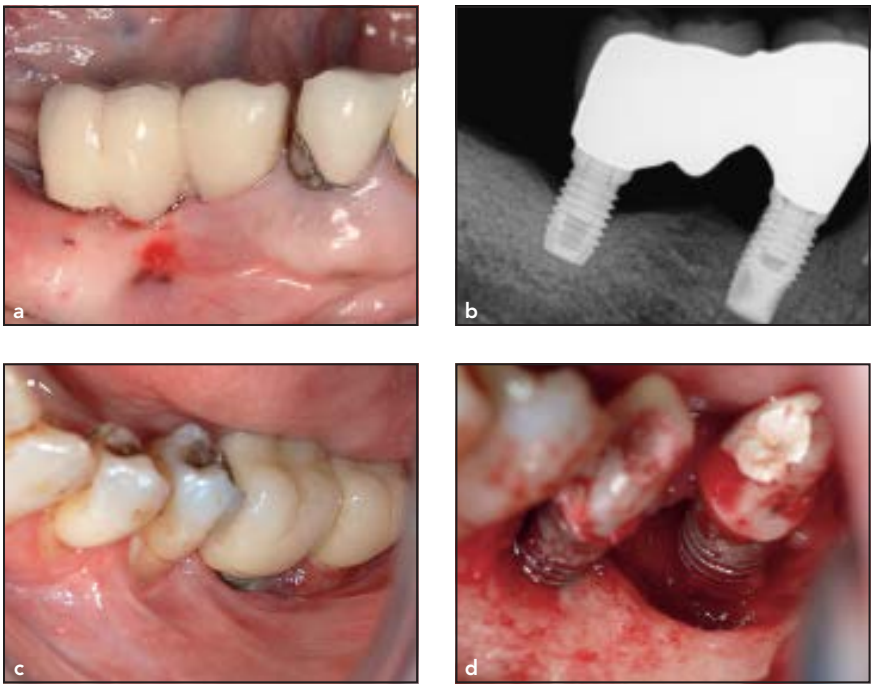


Fig 5 (a) *Clinical image showing lack of keratinized tissue and attached gingiva causing peri-implant pathologic pocket probing depth and* (b) *radiograph showing bone loss.* (c,d) *Clinical images showing absence of keratinized tissue with the presence of muscle pull as the causative factor for peri-implantitis and significant associated bone loss.*

Table 2 **Peri-implant probing depths for the implants included in the study**

Peri-implantitis etiology	< 4 mm	4–5 mm	> 5 mm
Pathogenic bacteria	0	18	195
Exogenous irritants	0	0	15
Iatrogenic factors	0	0	23
Extrinsic pathology	2	1	3
Absence of keratinized tissue (AKT)	0	0	13
Total	2	19	249

Results

A total of 152 patients (58% male and 42% female) with a mean age of 48.7 years were included in the study. The subjects had a total of 270 ailing implants from various manufacturers. All implants were considered to have a rough surface. The implants were restored with a cemented (32%) or screw-retained (68%) prosthesis. There were 32 smokers and 19 diabetic patients in the study. Peri-implant probing depths have been summarized in Table 2. When implants with radiographic bone loss were classified according to etiology, the majority (213 implants [78.8%]) were found to be related to biofilm or bacteria-induced inflammation with bone loss. Bone loss related to exogenous irritants such as dental cement was found for 15 implants (5.5%); bone loss related to iatrogenic factors, such as thinning out the buccal plate or placing an implant too buccally, was found for 23 implants (8.52%); and bone loss related to extrinsic pathology, such as apical periodontal lesions of adjacent teeth, was found for 6 implants (2.2%). Lastly, peri-implantitis related to AKT with mobile mucosa and/or muscle attachments was found with 13 implants (4.8%) (Table 3).

Discussion

Classification of peri-implant diseases and conditions is critical for communication, presentation, formulation of therapy, and prediction of treatment outcomes. In this study, the majority of bone loss was related to biofilm or bacteria. However, both nonpathologic and pathologic factors were important. While the classification or description of peri-implantitis has evolved over the past several decades, the methodology has been based on clinical periodontal parameters. Meffert described the ailing implant as having bone loss with pocketing but static at maintenance visits, whereas the failing implant demonstrated bone loss with pocketing but also presented with bleeding on probing, purulence, and continued bone loss despite therapy.[21]

Table 3 Etiology of peri-implantitis for the implants included in the study

Etiology	Patients (n)	Implants (n [%])	Diabetes (n)	Smoker (n)
Pathogenic bacteria	102	213 (78.8)	17	27
Exogenous irritants	14	15 (5.5)	0	1
Iatrogenic factors	20	23 (8.5)	1	3
Extrinsic pathology	5	6 (2.2)	0	0
Absence of keratinized tissue (AKT)	11	13 (4.8)	1	1
Total	152	270	19	32

Misch also used clinical parameters to assess implant health, describing a continuum from health to disease with disease status related to a progressive worsening of clinical parameters such as probing depth, bone loss, and pain.[22] Nogueira-Filho et al proposed a prognostic classification system based on peri-implant mucosal inflammation. As noted, many implants with signs of soft tissue inflammation had a poorer prognosis. However, the authors concluded that limited evidence exists to support a classification of mucosal inflammation and a specific related prognosis.[23] The absence of a relationship between classification and prognosis may be due to a failure to identify etiologies requiring separation in the classification, as has been set out in this study. Froum and Rosen established thresholds for clinical parameters as a basis for classification of peri-implantitis. Combinations of bleeding on probing and/or suppuration, probing depth, and radiographic bone loss were used to classify peri-implantitis into early, moderate, and advanced categories.[20] Recently, Kadkhodazadeh and Amid proposed a classification system for peri-implant disease in association with natural teeth termed *peri-implant soft tissue (PIST)*. The authors concluded that the system can assist in improving diagnosis, comparison, and subsequent treatment selection. However, the utility of this classification could be limited due to the absence of etiologies such as nonpathologic factors.[24]

In many cases of peri-implant disease, bone loss may be related to noninflammatory factors and may or may not demonstrate crestal radiographic bone loss since bone loss on the lingual or labial side remains invisible on two-dimensional radiographs. Dental implants with periapical infrabony radiolucencies commonly have been documented. Jalbout and Tarnow described four cases of implant failures with periapical lesions.[25] In 2005, Quirynen et al reported on predisposing conditions that led to retrograde peri-implantitis and proposed treatments,[26] while McAllister et al reported on two implant cases in which periapical radiolucencies developed with sinus tracts while implants were still submerged.[27] The failure of dental implants has also been related to practitioner factors, implant position, residual dental cement, and prosthesis design, which have been documented as contributing to increased soft tissue inflammation and peri-implant bone loss.[27] Linkevicius et al evaluated the relationship between patients with periodontitis and the development of cement-related implant disease. A prior history of periodontal disease was found to be a risk factor for implants with residual cement.[28]

Unfortunately, none of the current classifications of peri-implantitis have included etiology. Periodontal classifications, on the other hand, include various etiologies, from plaque induced to foreign body reactions. The etiology of peri-implant bone loss is crucial to development of an appropriate treatment plan and treatment therapy. As indicated in this study, a majority of bone loss is related to an inflammatory process. When the diagnosis is related to a biofilm or bacterial component, the clinician can use nonsurgical or surgical therapies or a combination of both to slow or arrest the disease progress. However, in a recent study by Jemt et al, progressive bone loss, while slow, was ongoing regardless of whether or not the patients received periodontal supportive therapy.[29]

In addition to creating a more targeted therapy for the treatment of peri-implant bone loss, the clinician should have an enhanced sense of the predictability of the intervention and prognosis of the implant. Implants with bone loss related to an inflammatory response to bacteria may have a lower therapeutic efficacy and a diminished long-term prognosis. An implant with bone loss related to residual cement may be easier to treat, resulting in a more predictable response to therapy and an improved prognosis.

Furthermore, systemic and habitual cofactors such as diabetes and smoking may attenuate the tissue response to these etiologies. However, these cofactors alone are not considered a class of etiology since many smokers and diabetics do not succumb to peri-implantitis and the presence of such cofactors can profoundly influence the severity of the disease regardless of etiology.

Clinicians have used dental implants for single or multiple teeth indications for many years with strong outcomes. The current challenge is to evaluate the status of an implant and control disease progression. The results of this study indicate that the proposed classification system will allow the clinician to properly diagnose peri-implant diseases relative to etiology. Indeed, the authors propose that it may no longer be appropriate to include all these entities under the umbrella of peri-implantitis, since their etiologies and potential response to therapy are very different.

Conclusions

Although there is insufficient evidence to understand the precise mechanism of peri-implantitis, in the future peri-implantitis may be defined by the causative etiology based on the proposed classification. This will allow better communication and understanding of the pathogenesis, recommended therapy, and anticipation of prognostic outcome.

Acknowledgments

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