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Peri-implantitis Update: Risk Indicators, Diagnosis, and Treatment

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This review, 30 years after accepted scientific literature, focuses on at-risk dentate sites not only amenable, but underpinned, by a statement with the, to-date, 5-year follow-up data. Peri-implantitis (PI) is a bacterial induced, inflammatory disease affecting the supporting tissues around implants, resulting in progressive bone loss, and if untreated, implant failure. Risk factors, patients' psychosocial habits, and improper implant position [1,2,3,4,5] are implicated in the aetiology of PI. Diagnosis of PI is based on clinical and radiographic assessments, with a probable diagnosis of PI being made when bleeding and/or suppuration on probing, with a probable pocket depth of ≥6 mm, and radiographic evidence of bone loss of ≥3 mm. Treatment options include non-surgical, surgical, and regenerative approaches, with the aim of arresting the disease progression and a limit to bone loss of ≤2 mm. Implant maintenance therapy or supportive therapy [6] increases the implant survival rate. This review, a continuation from the previous review, focuses on the risk factors, diagnosis, and treatment options for PI, with a focus on the latest research, including the recent 5-year follow-up data, and a statement with the, to-date, 5-year follow-up data, and a statement with the, to-date, 5-year follow-up data, and a statement with the, to-date,

Surgical treatment may not be a good option in some peri-implantitis cases. When there is a substantial loss of bone in peri-implantitis (half the length of the implant), the success of surgery is unlikely.[32] Implants that are placed in an inappropriate position can limit treatment outcomes. Additionally, implant mobility indicates advanced bone loss (> 60%) or a lack of osseointegration of the implant. In these cases, implant removal is recommended.[33] If implant removal is necessary, then the second implantation should be performed with a larger diameter implant.[32]

Conclusion

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Peri-implantitis is a common problem resulting in tissue destruction and implant loss. Plaque accumulation and biofilm formation play a major role in its initiation and development. Prosthetic factors such as residual cement and overloading may result in peri-implantitis but need clinical evidence. Routine supportive therapy reduces the risk of the onset of peri-implantitis. The peri-implantitis treatment modality should be selected based on the extent of disease. Nonsurgical mechanical therapy is beneficial and should be the initial treatment. The use of antibiotics limited impact on treatment success in peri-implantitis. Lasers remove the early supragingival biofilm and using a low-intensity laser induces soft-tissue regeneration. Implantoplasty is performed to smoothen the implant surface using rotary instruments. Various regenerative measures have shown partial regeneration, but achieving total tissueregeneration is difficult.

Footnotes

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NoteConflict of Interest The manuscript has not been published or submitted elsewhere. The manuscript has been read and approved by all the authors.

None declared.

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