Short Communication

Managing peri implant diseases

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1. Introduction

Teeth replacement with dental implant supported prosthesis is now a widely established modality with good long term survival and success. The biological complications related to dental implants are mainly inflammatory, associated with dental biofilm accumulation, but rarely other soft tissue complications like epulis or squamous cell carcinomas are also reported.1 Peri implant mucositis and peri implantitis, the peri implant diseases, have to be clearly distinguished from peri implant health.

1.1. Diagnostic criteria

1.1.1. Peri implant health

1. Absence of visual signs of inflammation in the peri implant mucosa including redness, swelling, suppuration.
2. Absence of profuse bleeding (line or drop bleeding) on probing with a light probing force of 0.25 N.
3. Probing pocket depth unchanged compared to baseline (soon after placement of the prosthetic crown).
4. Absence of radiographic bone loss more than 2mm compared to baseline.

1.1.2. Peri implant mucositis

1. Visual signs of inflammation in the peri implant mucosa including redness, shininess and swelling.
2. Definite evidence of bleeding (line or drop) on probing with a light force of 0.25N.
3. Suppuration after probing or on application of digital pressure
4. An increase in probing depth compared to baseline.
5. Absence of radiographic bone loss more than 2mm compared to baseline.

Soreness is a common presenting complaint. Peri implant tissues are less resistant to probing and can be easily traumatized with excessive probing force. A resulting local bleeding dots should be differentiated from profuse line or drop bleeding associated with peri implant mucositis.
1.1.3. Peri implantitis

1. Visual signs of inflammation in the peri implant mucosa combined with profuse bleeding on probing and/or suppuration.
2. Increase in probing pocket depth as compared to baseline.
3. Radiographic evidence of progressive bone loss (more than 2mm) as compared to baseline.

If baseline records are not available bone loss > 3mm and probing depth >6mm with profuse bleeding is diagnostic of peri implantitis.

1.2. Management

1.2.1. Peri implant mucositis

There is strong evidence to support dental biofilm as the etiologic agent for peri implant mucositis and therefore demonstration of plaque and clinical signs of inflammation are necessary for the diagnosis. Other major local predisposing factors include poor compliance/ non compliance with supportive periodontal therapy (SPT), implant surface characteristics like roughness, prosthetic design features complicating plaque control, keratinized gingiva (KT) width and excess cement in the peri implant sulcus.

Once the prosthetic supra structure is inserted, baseline records should be taken including a four point pocket chart and a standardized periapical radiograph. Patient education and plaque control instructions are given and the patient should be enrolled into a tailor made SPT schedule of three, six or 12 months based on their risk profile.

Management of peri implant mucositis begins with focused oral hygiene instructions including the use of manual tooth brush with small head and medium bristles or an electric tooth brush with a rotating and oscillating head. Adjunctive use of single tufted brushes and chlorhexidine mouth rinse or gel is advised. Local risk factors including excess cement or poorly designed prosthesis impairing oral hygiene should be corrected. Systemic risk factors like smoking and diabetes should be controlled. Re institution of plaque control can lead to resolution of mucositis in not less than 3 weeks. For professional supra gingival plaque control, pumice slurry and polishing cups can be used. Sub mucosal debridement may be carried out with titanium scalers, plastic curettes, teflon coated curettes, carbon fibre curettes, gold plated curettes or plastic tipped ultrasonic inserts.

There is not enough evidence for the benefit of adjunctive use of local/systemic antimicrobials.

1.2.2. Peri implantitis

Peri implantitis usually develops within the first few years of implant insertion. Unlike periodontitis the inflammatory lesion is not “walled off” and extends circumferentially resulting in saucer shaped/funnel shaped bone loss in bucco lingually wider ridges. In thin ridges the bone loss is horizontal. The evidence is strong to consider poor oral hygiene (Odds ratio 14) and previous history of periodontitis (Odds ratio 4) as important risk factors. It is important to properly seal the access hole in screw retained restorations to prevent micro leakage.

The first line of management for peri implantitis is non surgical as described for peri implant mucositis. But the deepened pockets, the implant thread design and surface roughness compromise accessibility and make surface decontamination difficult. Therefore meticulous oral hygiene, systemic risk factor control and non surgical therapy are only modestly effective for peri implantitis management which often warrants surgical debridement. The objectives of surgical approach are not only to improve access and reduce pocket depths but also to attempt re osseointegration. Surgical approaches for various clinical situations are as follows

1.2.3. Minimal bone loss in the esthetic zone

Access flaps using inverse bevel incision are employed for shallow defects in the upper anterior teeth.

1.2.4. Supra bony defects when esthetics is not a priority

Large flaps are reflected buccally and lingually and defects debrided thoroughly. Ostectomy or osteoplasty is done if indicated. After implant surface decontamination flaps are sutured apically. Many agents have been used to decontaminate the implant surface including saline, hydrogen peroxide, citric acid, abrasive pumice, chlorhexidine, air powder polishing, lasers and antimicrobials. There is insufficient evidence to suggest one decontamination agent/technique to be better over the other. Implantoplasty (implant thread removal and polishing) may be done using carbide/diamond burs.

1.2.5. Re osseointegration for intra bony defects

For walled bone defects associated with circumferential bone loss, guided bone regeneration (GBR) is employed.

<table>
<thead>
<tr>
<th>Table 1: Ten point implantcheck up protocol for SPT visits.</th>
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<tr>
<td>1. Plaque and calculus assessment</td>
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<td>2. 4 point probing</td>
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<td>3. Bleeding/suppuration</td>
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<td>4. Mucosal recession</td>
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<td>5. Mobility</td>
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<td>6. Occlusion- passive, light contact</td>
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<td>7. Contacts- definite interproximal contacts needed</td>
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<td>8. Percussion sensitivity</td>
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<td>9. Radiographs – only if points 1-8 suggests clinical change</td>
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<td>10. Instrumentation</td>
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Autogenous or xenogenic grafts can be used alone or in combination with resorbable/non resorbable membranes. Alternatively porous titanium granules may be used for defect fill with some success. Primary closure and submerged healing are advised for better outcomes.

There is little additional benefit for the routine use of adjunctive antibiotics and the risk of antimicrobial resistance should be considered.

In worse cases explantation of severely compromised implants is beneficial when implant retention is considered more riskier.

Strict adherence to patient tailored regular follow up interventions like Cumulative Interceptive Supportive Therapy (CIST) would lead to long term survival of dental implants.6

2. Source of Funding

None.

3. Conflict of Interest

None.

References

Author biography
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