Apical periimplantitis—also known as periapical implant lesions—develops in the tissues around the apex of an implant after placement, while the bone architecture in the coronal portion is maintained. If left untreated, this pathology eventually causes osseointegration failure. The diagnosis of apical periimplantitis is based on the clinical and radiographic findings. Clinically, early apical periimplantitis is characterized by symptoms (pain and tightness) and signs (swelling, fistula and drainage) of variable intensity, depending on the stage of the lesion. Clinically, the patient complains of pain and inflammation appearing, although in the early phase, there may be pain but not inflammation. Radiographically, a radiolucency around the implant apex may be observed (although it is not necessary—the same happens in acute periapical periodontitis, which may have symptoms without radiographic alterations). The use of new imaging technologies such as small-volume cone beam computed tomography is helpful in establishing an early diagnosis, showing a clear clinical image of periapical implant bone loss.

In the literature, there are few papers on diagnosing this disease and these lack homogeneity of diagnosis criteria. Diagnosis of apical periimplantitis involves clinical and radiographic evaluation, and the treatment will vary according to the findings:

a) If the implant has a radiolucent area (not present after surgery owing to overdrelling and manifesting over time) without pain, monitoring of the lesion is recommended, without medical treatment.

b) If the radiolucency has increased in size or if the patient develops pain, medical and surgical treatment are indicated.

Early diagnosis and management of active apical periimplantitis lesions (nonsuppurative phase with symptoms, acute suppurative and subacute phases) includes the surgical approach and its follow-up to evaluate the success of the treatment and avoid implant failure.

The literature describes medical and surgical approaches to treating periapical implant lesions. Medical treatment using antibiotics (amoxicillin, amoxicillin/clavulanate, metronidazole and clindamycin) alone has proved ineffective in controlling symptomatic or active lesions, and surgical access must be performed. There is no established gold standard treatment, so the goal is to eliminate the area of infection. Surgical treatment entails anesthesia, incision, full-thickness flap elevation, ostectomy, apical curettage and abundant irrigation. After debridement, some authors have described irrigation of the bone defect with saline solution or with chlorhexidine. Other agents have been suggested for local decontamination of the implant surface, such as chlorhexidine, calcium hydroxide paste and tetracycline pastes. There is no clinical evidence on the efficacy of any of these agents. Some studies describe the use of biomaterials, with or without membranes, in order to achieve complete bone regeneration of the defect. Resection of the apex of the implant is recommended in those cases where access for removal of the granular tissue is not otherwise ensured, likewise when there is an anatomical relationship with the maxillary sinus or nasal cavity.

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