Occurrence of a Pyogenic Granuloma in Relation to a Dental Implant

Ivan Dojcinovic, MD, DMD,* Michel Richter, MD, DMD,† and Tommaso Lombardi, MD, DMD‡

Pyogenic granuloma is an inflammatory vascular hyperplasia often occurring in the oral cavity. It appears in response to various stimuli such as low-grade local irritation, trauma, or female steroid hormones. A 32-year-old man sought care for a tender and bleeding lesion of the left posterior maxillary gingiva. The intraoral examination showed an exophytic ulcerated nodule measuring 1.0 cm, related to a dental implant placed in the upper left second premolar position. Radiographic examination showed an oversized healing cap. A provisional diagnosis of reactive inflammatory hyperplasia was made, and the lesion was excised and submitted for histologic examination. On microscopy, the surgical specimen showed an ulcerated nodule consisting of a delicate connective tissue stroma containing numerous blood vessels with plump endothelial cells, intermingled with abundant polymorphonuclear lymphocytes. A diagnosis of pyogenic granuloma associated with a dental implant was made. In this case it was the result of an inappropriate choice of a healing cap, thus allowing an accumulation of dental plaque and sustained chronic inflammation of the peri-implant tissue. A conservative excision and replacement of the healing cap were sufficient for definitive treatment.

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We describe a case of PG related to a dental implant. To our knowledge, similar cases have not been reported.

Patient and Methods

A 32-year-old man in good general health presented with a localized soft tissue mass around the closure screw of a dental implant (Fig 1). The lesion was tender and bled intermittently upon touching. His dental history showed a left maxillary sinus lift along with lateral ridge augmentation with autologous bone harvested from the iliac crest. Six months later, 3 SLA Standard Plus Straumann (Straumann Institute, Basel, Switzerland) implants (4.1 × 10-mm regular neck, 4.8 × 10-mm regular neck, and 4.8 × 10-mm wide neck) were placed in the upper left first and second premolars and upper first molar positions. After 5 months, healing caps were placed at the second stage of surgery. Four weeks later, impressions for permanent prosthetic treatment were taken.

Clinical examination showed an exophytic nodular lesion measuring 1.0 × 0.6 cm on the vestibular gingiva of the second implant placed in the upper left second premolar position.

Radiographic examination showed an oversized healing cap on the middle implant (Fig 2). There was no evidence of horizontal bone loss. In addition, the healing caps of the second and third implants were in tight contact with each other.

A provisional clinical diagnosis of inflammatory hyperplasia was made, and the patient underwent a
debridement flap surgery that included a complete excision of the lesion under local anesthesia, curetage of the surrounding tissues, and replacement of the oversized healing cap. The lesion was then submitted for histologic examination.

Microscopic examination showed a largely ulcerated nodule featuring an edematous granulation tissue rich in small blood vessels with prominent endothelial cells, intermingling with numerous polymorphonuclear leukocytes (Fig 3). The capillaries showed a radiating pattern extending toward the surface. The deeper part of the mass was fibrous and contained large blood vessels often surrounded by a dense chronic lymphoplasmacytic infiltrate. Besides the ulceration, the epithelium was slightly hyperplastic. These features were consistent with the diagnosis of PG.

No recurrence was seen at the follow-up visit after 18 months.

Discussion

The behavior of the soft tissues around implants is an important factor for long-term success. Peri-implant complications include dehiscence, fistula, mucositis, gingival inflammation with hyperplasia, and peri-implantitis. These peri-implant complications are often associated with poor oral hygiene and/or a gap between components caused by loose abutment screws or framework misfits.

Gingivitis and gingival hyperplasia are the most common soft tissue complications. According to the literature, they are found in 1% to 30% of cases. In some cases, they may result from an inappropriate choice of abutments, healing caps that are too short or too close to each other, the presence of dead spaces under the suprastructure, lack of attached mucosa, or faulty alignment of the implant interfering with correct oral hygiene procedures.

Cases of specific gingival hyperplasia around dental implants have been reported. These include hyperplasia induced by phenytoin medication and one case of allergy to titanium abutments. A few articles on peripheral giant cell granuloma associated with dental implants have also been published.

In our case we found a PG in relation to a dental implant. A literature survey did not find any articles on PG associated with dental implants.

Hartzell was the first author to introduce the term “pyogenic granuloma.” He believed that it represented a nonspecific granulation tissue caused by a pyogenic agent.

Such lesions may be found in the oral cavity or extraorally. Intraorally, the gingiva is the most common site of involvement (about 60%-70%), followed by the lips (14%), tongue (9%), buccal mucosa (7%), and palate (2%).
Clinically, PG is a smooth or lobulated exophytic lesion manifesting as a red nodule or mass with a pedunculated or sometimes sessile base, usually hemorrhagic and elastic in consistency. PGs are often ulcerated because of local trauma.

The etiology of PG is unknown, but PG regresses when the potential initiating stimuli are removed. Trauma and infection have been frequently suggested as triggering factors. A hormonal cause has been suggested in some PGs because, clinically and histologically, similar lesions occur in pregnancy. For this kind of clinical variety, the terms “pregnancy tumors” and “granuloma gravidarum” are used.

About 30% to 50% of patients with PG have a history of local trauma. Poor oral local hygiene may be a precipitating factor in most of these cases. Aguilo and Bagán reported the occurrence of PG as a result of an injury to a primary tooth, as well as 2 cases of PG in the maxillary labial mucosa, which were related to an apical fenestration of a primary incisor tooth. Milano et al described a case of PG in relation to aberrant tooth development.

In our case the implants were stable but the inappropriate healing cap of the second implant resulted in dental plaque accumulation and thus sustained chronic inflammation of the gingival peri-implant tissue, triggering the development of a PG.

PGs are treated by a variety of methods, most commonly surgical excision and elimination of etiologic irritants (plaque, calculus, foreign materials, source of trauma). Other treatment protocols have also been proposed such as cryotherapy, chemical and electric cauterization, and the use of Nd:YAG (neodymium:yttrium-aluminum-garnet) laser and carbon dioxide laser. Meffert et al used the flash lamp pulsed-dye laser on PG in relation to aberrant tooth development. It is important to choose a healing cap that corresponds to the size of the implant used. An ill-fitting cap may lead to the development of a reactive gingival lesion such as PG, which in turn may jeopardize the implant survival.

References