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Case report

Plasmacytoma of the mandible associated with a dental implant failure: a clinical report

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Abstract: The case report of a patient is presented who had been suffering from a plasmacytoma of the spine several years back, and who had developed a new plasmacytoma of the mandible, 3 years subsequent to the insertion of a dental implant. This second solitary lesion occurred 15 years after the first one, and without signs of conversion to multiple myeloma. Research in animal models has shown multinucleated giant cells, belonging to the monocyte–macrophage lineage, persisting between the titanium surface and the lymphohemopoietic compartment, at least 1.5 years after implant insertion. Factors that increase the proliferative activity of precursor B cells, for example a protracted macrophage activation, are likely to increase the risk of B cell oncogenesis. A possible role of the titanium surface in an increase of precursor B cell proliferative activity, thus facilitating a new localization, was evaluated.

The interaction between an implant surface and bone marrow cells has been considered in very few studies. Some concern exists that dental implants might perturb local lymphohemopoiesis, possibly even inducing neoplasia in the B lymphocyte lineage (Rahal et al. 2000). The relationship between dental implants and tumors has been studied mainly from the standpoint of osseointegration in irradiated bone (Watzinger et al. 1996), or apropos implant survival rates in head and neck tumor patients (Kovács 2000). Plasmacytomas are B lymphocyte lineage tumors composed of plasma cells. They may occur as one of three different entities: multiple myeloma (MM), solitary plasmacytoma of the bone (SPB) or extramedullary plasmacytoma (EP) (Pisano et al. 1997). Both SPB and EP can convert to MM, although with different natural behaviors and frequencies (Holland et al. 1992). Notwithstanding that the disease is chronic, thanks to treat-

ment, patients may sustain a long-term prognosis. Considering the average age of onset (58–60 years according to Holland et al. 1992), those patients are likely to seek prosthodontic therapy. A clinical report exists with regard to implant therapy in respect of an MM patient (Sager & Theis 1990). The case report of a patient who had been suffering from an SPB several years back, and who had developed a new plasmacytoma, subsequent to the insertion of a dental implant, is presented.

Case report

A 75-year-old patient was referred for prosthetic problems. She had been suffering from SPB 12 years back. The plasmacytoma was located in the spine. The patient had a lower overdenture retained by teeth 4.2 and 4.3. Both teeth had to be extracted, due to severe periodontal breakdown, and

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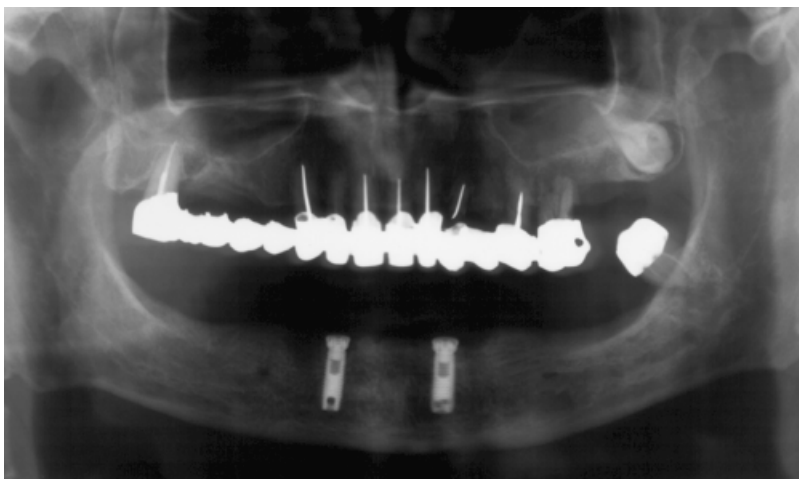


Fig. 1. Orthopantomography after stage I surgery.

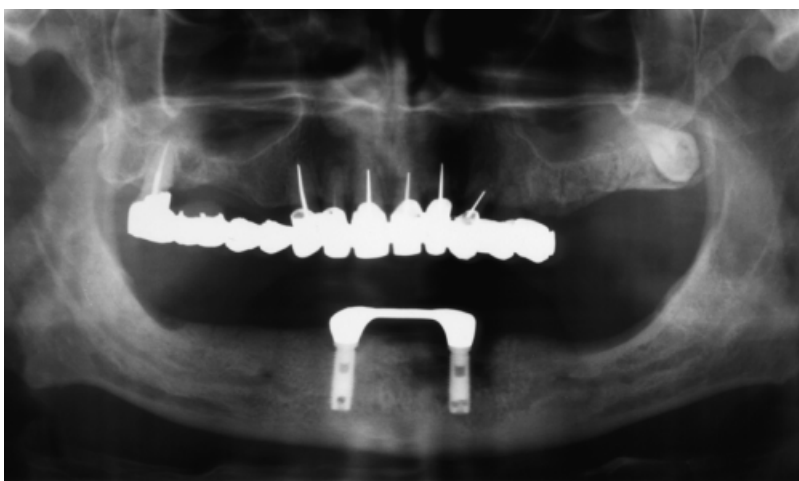


Fig. 2. Orthopantomography before implant removal.

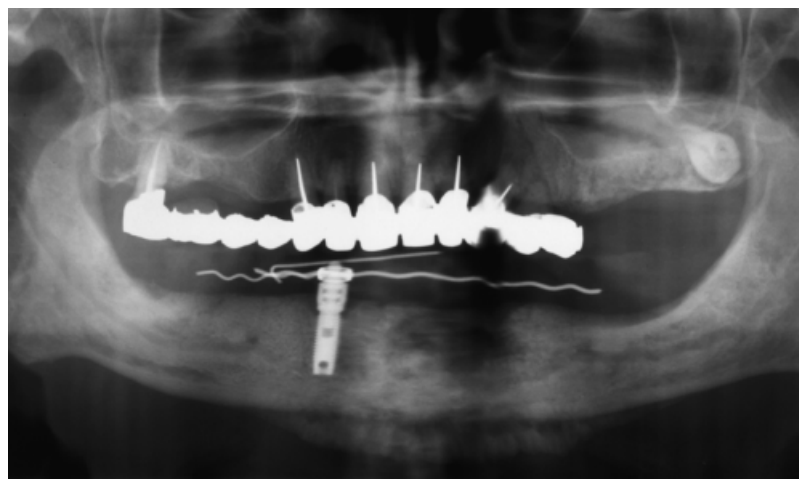


Fig. 3. Orthopantomography 6 months after implant removal. Radiopaque formations at the lower border of the mandible, where the mass was clinically evident.

the prosthesis was converted to a complete denture. Owing to the lack of stability of the denture and high patient discomfort, two implants with titanium-machined sur-

faces (Implant Innovations Inc., Palm Beach, FL, USA) had to be inserted into the mandible (Fig. 1). After 4 months of integration, the implants were connected

by a gold cast bar and a new implant-retained overdenture was inserted. No treatment was performed on the upper arch, despite the presence of some dental problems. The patient was enrolled in a standard recall program for implant patients, with oral hygiene every 4 months. Patient compliance with oral hygiene, however, was limited.

After 3 years of utilization, the patient reported a swelling of the chin, and complained of pain in the same area. Extraoral examination revealed a solid mass below the left anterior border of the mandible. The clinical oral examination revealed swelling around the left implant, with considerable bleeding on probing, and with a severe loss of bone. A panoramic radiograph showed a large transparency in relation to the left fixture (Fig. 2). The implant was easily removed and a biopsy was simultaneously taken. The overdenture was modified with a ball-and-ring attachment to the remaining implant. The biopsy revealed a new plasmacytoma. The patient underwent radiotherapy. The remaining implant was stable, and there were no signs of failure 6 months after the radiotherapy. A panoramic radiograph showed the presence of the radio transparency, while the bone formation below the border of the mandible had become apparent (Fig. 3).

Discussion

In this patient, a plasmacytoma, a marrow-derived B lineage neoplasm, had developed in connection with an implant in the anterior mandible. The pattern of bone resorption, the existence of osteoblastic activity below the border of the mandible and of course the biopsy, exclude peri-implantitis as the cause of failure, in an area that has the highest implant survival rate. The remaining implant was not affected by radiotherapy.

Plasma cells produce osteoclast-activating factors, which stimulate the growth of osteoclasts and therefore bone resorption. Plasmacytomas usually appear on radiographic images as radiolucent areas (Matsumura et al. 2000). Plasmacytoma cells have also been reported with a capability of stimulating osteoblastic activity, with new bone formation (Ramon et al. 1978). A

clinical report exists in respect of implant therapy in an MM patient (Sager & Theis 1990), but the focus is only on the possibility of treating terminally ill patients with implants.

The patient had been suffering from an SPB, 12 years before the implant was placed. It is known that conversion to MM depends on the type of plasmacytoma, 53% of patients with SPB converting to MM, vs. 36% of patients with EP. For patients suffering from SPB, there is no evidence of plateau in the diagnosis to conversion time, and conversion can continue to occur even after 17 years (Holland et al. 1992). The study supported the concept of EP having a lower incidence of conversion to MM, and a different natural history from that of SPB, with SPB potentially being MM in evolution. Holland et al., however, also reported a specific subset of patients in whom multiple sequential SPB had developed. In these patients, the time-lapse to the appearance of a new solitary lesion was longer than the usual conversion time to MM. In some of these patients, conversion to myeloma had subsequently occurred, while others had no evidence of conversion with up to three solitary lesions.

In the subject case, a second solitary lesion occurred 15 years after the first lesion, without any signs of conversion to myeloma.

Mandibular lesions are a common finding in MM (Witt et al. 1997). In the jaws, the most common radiographic finding is a radiolucency that is often superimposed on the roots or apices of nearby teeth. The most common symptom is localized pain, and the most frequent clinical sign is a raised red lesion on the alveolar ridge. The disease affects elderly persons, and the posterior mandible is the most frequently reported location. There is a re-

ported correlation between the degree of dysplasia of tumor cells and survival rates (Pisano et al. 1997).

Indeed, it is impossible to tell whether the plasmacytoma in the mandible was somehow promoted by an interaction between the titanium surface and the plasma cells. Actually, factors that increase the number and proliferative activity of precursor B cells, for example a protracted macrophage activation, are likely to increase the risk of B cell oncogenesis.

Mandible localization may be unrelated to the presence of the implant, but we cannot exclude the fact that the titanium surface could have acted as a 'catalyst', increasing the precursor B cell number and proliferative activity, and thus facilitating the localization of a new lesion.

Possible interactions between implant surfaces and bone marrow cells have been described in an animal model (Rahal et al. 2000). Precursor B cells show an early increase in number and proliferative activity. Multinucleated giant cells are observed at the interface between the bone marrow and the titanium implants. After subsequent intervals, however, no significant differences from controls can be observed, and there are no perturbations in the spatial localization of either B lineage cells or DNA-synthesizing hemopoietic cells. Following initial marrow regeneration and fluctuating precursor B cell activity, despite the presence of giant cells, titanium implants are apparently well tolerated by the directly apposed bone marrow cells. Normal hemopoietic cells lie in contact with the implant surfaces; multinucleated giant cells, however, establish themselves between the titanium surfaces and the lymphohemopoietic compartment. These cells belong to the monocyte-macrophage lineage. The significance of their persistence is undetermined. The presence of

activated macrophages within the bone marrow itself, even when restricted to a small region of the bone marrow, raises concerns that the incidence of B cell neoplasms might be enhanced after a latent period. Rahal et al. provide no evidence to suggest that titanium implants, in contact with mouse bone marrow, carry a long-term risk of marrow-derived B lineage neoplasms. They have, however, advocated further studies before complete confidence with regard to myelointegration in humans can be assumed.

In the light of possible interactions between dental implants and local lymphohemopoiesis, care should be exercised when considering dental implants as a treatment option for those patients who have suffered from neoplasia in the B lymphocyte lineage, even though there has been a partial or complete recovery. Moreover, existent dental implants should be carefully monitored as potential sites of localization in patients, where a B lymphocyte lineage tumor has been diagnosed.

要旨

本稿は、数年前に脊椎の形質細胞腫に罹患し、歯科インプラントを埋入した後3年後に下顎にあらたな形質細胞腫を発症した患者の症例報告である。この2つ目の病変は最初の細胞腫の15年後に発症したが、多発性骨髄腫への転換を示す徴候はなかった。動物モデルの研究では、単球-マクロファージ系統の多核巨細胞が、インプラント埋入後少なくとも1.5年後にチタン表面とリンパ球造血性コンパートメントの間に持続していた。前駆B細胞の増殖活性を増加する因子、例えばマクロファージの長期の活性化は、B細胞の腫瘍形成のリスクを増加する可能性がある。チタン表面が前駆B細胞の増殖活性の増加に関与し、新しい病変の定位を促進する可能性について評価した。

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