



RESEARCH ARTICLE

DENTAL IMPLANTS: MAXILLOFACIAL PATHOLOGIES: A REVIEW OF COMPLICATIONS AND ADVERSE EFFECTS

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ABSTRACT

Dental implants have been a successful treatment alternative for restoring missing teeth. However, treatment is not always successful, as evidenced by reports reviewing the reasons for implant failures/complications. The aim of the present article is to discuss the less often seen, grey aspects of dental implants, like some of their local and systemic complications.

Key words:

Dental implants, Maxillofacial pathology, Implant failures, Biological complications, Mechanical complications, Oral complications.

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INTRODUCTION

Dental implants have been a successful treatment alternative for restoring missing teeth. Since the introduction of the concept of osseointegration, the success of dental implants has increased dramatically because of better understanding of bone response and the improvement in bone loading concepts. Their benefits have been spoken of so often that they seem to have no complications at all. Like every coin has two sides, so do implants. Treatment is not always successful, as evidenced by reports reviewing the reasons for implant failures/complications. In recent years, a number of authors have specifically looked at implant related complications and maintenance requirements. The literature has reported biological complications which may include adverse soft tissue reactions, sensory disturbances, progressive marginal bone loss and loss of integration. Mechanical complications may include fractures or loosening of components in the system. (Berglundh *et al.*, 2002)

Complications associated with dental implants

The classical subdivision of complications into iatrogenic factors, biological and mechanical, does not fully reflect their nature. Since complications considered as biologically caused

during the stage of healing with formation of connective tissue, may be caused due to overheating during the preparation of the implant bed with tissue necrosis, which is included in the iatrogenic factors. And hence the classification of complications may be only arbitrary.

Iatrogenic complications

Bleeding

Bleeding and the formation of massive hematomas in the floor of the mouth are the result of an arterial trauma. A vascular wound may occur after detrimental surgical manipulations or tearing of the lingual periosteum, but in most cases, it is attributed to perforations of the lingual cortical plate. Mechanical pressure exerted by the expanding hematomas displaces the tongue and floor of the mouth both superiorly and posteriorly. (Kalpidis and Setayesh, 2004) The surgeons also should consider other sources of potential hemorrhage and subsequent hematoma formation, including injuries to muscles or other soft tissues. (Isaacson, 2004) This occurrence may lead to extensive bleeding into the submandibular space, resulting in a life-threatening acute airway obstruction within the first few hours after surgery. (Goodacre *et al.*, 1999) In the literature there are described 5 cases of dangerous bleedings, occurring at the stage of surgical insertion of the implant.

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Table 1. Complications associated with dental implants

Type of complication	Examples
Iatrogenic	1. Bleeding 2. Nerve injury 3. Injury to adjacent teeth 4. Schneiderian membrane perforation
Mechanical	1. Overloading 2. Screw loosening, screw fracture, 3. Corrosion 4. Fracturing of veneering porcelain and framework fracture 5. Galvanism
Biologic	1. Flap dehiscence and exposure of graft material 2. Peri-mucositis 3. Peri-implantitis 4. Post operative maxillary sinusitis
Maxillofacial pathology	1. Osteomyelitis 2. Peripheral giant cell granulomas 3. Squamous cell carcinomas 4. Osteosarcoma

Similar complications occur when the compact layer of the bone is perforated. (Georgiev and Nogalchev, 2010) The hemorrhage can easily spread in the loose tissues of the floor of the mouth, the sublingual area, and the space between the lingual muscles, which may require intubation or an emergency tracheostomy. (Dubois *et al.*, 2010)

Table reference (Park and Wang, 2005)

Neurosensory disturbances

A mean incidence of neurosensory disturbance incidence after implant surgery was 6.1% to 7% with a range between 0.6% and 39%. Nerve damage can have results ranging from mild paresthesia to complete anesthesia or even disabling dysesthesia. (Goodacre *et al.*, 1999; Goodacre *et al.*, 2003) Possible causes of nerve injury include poor flap design, traumatic flap reflection, accidental intraneural injection, traction on the mental nerve in an elevated flap, penetration of the osteotomy preparation and compression of the implant body into the canal. Nerve injuries may be caused indirectly by postsurgical intra-alveolar edema or hematomas that produce a temporary pressure increase, especially inside the mandibular canal. Direct traumas are the most frequent causes of nerve injury, and they may occur through five mechanisms: compression, stretch, cut, overheating, and accidental puncture. (Misch and Wang, 2008) The mental nerve is at particular risk of iatrogenic injury because it arises from asymmetric foramina and forms a concave loop anteriorly. In edentulous patients, it may be very close to the bone surface or the top of the crest. The nerve injury may cause one of the following conditions: parasthesia (numb feeling), hypoesthesia (reduced feeling), hyperesthesia (increased sensitivity), dysthesia (painful sensation), or anesthesia (complete loss of feeling) of the teeth, the lower lip, or the surrounding skin and mucosa. (Greenstein and Tarnow, 2006) For implants placed in the atrophic posterior mandible, the routine use of intraoperative periapical radiographs during the drilling sequence can help avoid the risk of injury to the inferior alveolar nerve. Periapical radiographs used intraoperatively to obtain working length measurements are similar in concept to techniques used in root canal therapy. This method can reliably determine safe distances between the

implant and the inferior alveolar canal, thus avoiding the risk of injury to the nerve altogether. (Burstein *et al.*, 2008)

Injury to adjacent teeth

Damage to teeth adjacent to the implant site may occur subsequent to the insertion of implants along an improper axis or after placement of excessively large implants. This problem arises more frequently with single implants. (Annibaldi *et al.*, 2009) Adjacent teeth should be evaluated before implant placement. Dilacerated roots and excessive tilting in the mesiodistal direction that invades the implant space often prevent ideal placement. (Misch and Wang, 2008) The tilt of adjacent teeth should be assessed before drilling. The damage of an adjacent tooth by implant placement may cause the tooth to become non-vital, and the tooth may require subsequent endodontic treatment. (Sussman, 1998) Use of a surgical guide, radiographic analysis and CT scan can help locate the implant placement, thereby avoiding damage to adjacent teeth. The angulation of adjacent teeth and dilacerations of roots must be radiographically assessed prior to implant placement. Ideally, 1.5 to 2 mm of bone should be present between an implant and the adjacent tooth. Furthermore, inspection of a radiograph with a guide pin at a depth of 5 mm will facilitate osteotomy angulation corrections. To prevent a latent infection of the implant from the potential endodontic lesion, endodontic treatment should be performed. (Sussman, 1998; Greenstein *et al.*, 2008) Discrepancies between the apical and crestal interdental spaces as a result of mesial or distal tipping of the roots can be corrected orthodontically. (Annibaldi *et al.*, 2009)

Schneiderian membrane perforation

The Schneiderian membrane, which is characterized by periosteum overlaid with a thin layer of pseudociliated stratified respiratory epithelium, constitutes an important barrier for the protection and defense of the sinus cavity. The integrity of the sinus membrane is essential in maintaining the healthy and normal function of the maxillary sinus. (Ardekian *et al.*, 2006) The most common intraoperative complication seems to be Schneiderian membrane perforation, which occurs in 10% to 60% of all procedures (Ardekian *et al.*, 2006; Pikos, 1999; Proussaefs *et al.*, 2004). The mucociliary apparatus protects the sinus against infection while the membrane also acts as a biologic barrier. If a perforation occurs, the membrane perforation could represent a window for bacterial penetration and invasion into the grafted area. (Zijderveld *et al.*, 2008) Failure to atraumatically elevate the Schneiderian membrane may result in graft migration or loss, exposure of the graft or the implant to the sinus, and postoperative site infection. In addition to contaminating the recipient site, disruption of the mucosa may alter the normal mucociliary flow patterns, causing retention of secretions and infections around the foreign body. (Ward *et al.*, 2008) To minimize Schneiderian membrane perforations, surgeons must evaluate the maxillary sinus anatomy while considering the lateral thickness of the lateral wall, slope of the sinus wall, location of septa, membrane thickness through the radiography and CT analysis before maxillary sinus augmentation. (Zijderveld *et al.*, 2008; Ward *et al.*, 2008)

Mechanical complications

Biomechanical failures range from loosening of screws to breakage of implant components and implants. These types of

failures can be avoided with proper treatment planning, a good understanding of screw joint mechanics and knowledge of the implant system used.

Overloading

Implant success is reported to depend on both biologic tissue (soft tissues and bone) response and mechanical components strength (implant components and superstructure). The soft tissue is more susceptible to invasion by bacteria, whereas bone may be more susceptible to loading. Occlusal overload is often regarded as one of the main causes for peri-implant bone loss and implant/implant prosthesis failure. Studies have suggested that Occlusal overload may contribute to implant bone loss and/or loss of osseointegration of successfully integrated implants. (Adell *et al.*, 1990; Adell *et al.*, 1981)

Screw loosening & screw fracture

Screw loosening is an often reported problem with implant supported restorations, especially with single tooth restorations. (Henry *et al.*, 1996; Jivraj and Chee, 2005) This is largely due to clinicians not having a good understanding of the mechanics of a screw joint and the implant manufacturers not providing components and instrumentation that would allow clinicians to maximise the retentive properties of the screw. (Winkler *et al.*, 2003) Care must be taken when threading screws into implants; when screws are cross threaded, damage to the implant can occur and when too much force is applied, breakage of the screw can occur. When screws are cross threaded and broken they are difficult to remove and can render the implant unusable. (Chee and Jivraj, 2007)

Implant breakage

Breakage of implants and implant components can also occur; Often this is due to poor treatment planning and exposing implants to excessive forces. (Eckert *et al.*, 2000)

Corrosion

Corrosion can severely limit the fatigue and ultimate strength of the material leading mechanical failure of the implant. It has been found that the metal fatigue can lead to implant fracture. Yokoyama *et al.* studied the delayed fracture of titanium in a biological environment absorbs hydrogen and this may be the reason for delayed fracture of a titanium implant. (Yokoyama *et al.*, 2002) Although titanium alloys have better corrosion properties compared to Co-Cr and stainless steel (other implant materials) their corrosion leads to dissolution of titanium and other alloying elements like aluminum, vanadium, niobium, molybdenum] causing localized to generalized host response. The leached ions may induce potentially osteolytic cytokines into tissues leading to implant loosening and may even cause severe allergic reactions or hypersensitivity. (Rogers *et al.*, 1997)

Galvanism

Galvanic corrosion occurs when dissimilar alloys are placed in direct contact within the tissues. The complexity of the electrochemical process involved in the implant-superstructure joint is linked to the phenomenon of galvanic coupling and pitted corrosion. (Reclaru and Meyer, 1994)

Biological complications

Flap dehiscence and exposure of graft material

The most common postoperative complication is wound dehiscence, which sometimes occurs during the first 10 days. (Greenstein *et al.*, 2008) Contributing factors of dehiscence and exposure of the graft material or barrier membrane include flap tension, continuous mechanical trauma or irritation associated with the loosening of the cover screw, incorrect incisions and formation of sequestration of bone debris. Premature exposure of barrier membranes may also cause contamination of the graft and its eventual loss. (Park and Wang, 2005) To avoid wound dehiscence, tension-free closure using a buccal releasing incision is most important. Dentures should be relieved with a tissue conditioner. Mattress sutures combined with interrupted sutures are also useful. When the dehiscence is small and occurs within 24 to 48 hours, the clinician can immediately resuture the dehiscence. Once the diameter of the wound is large (2 to 3 cm) or the time elapsed is > 2 days, it is suggested that the margins of the wound be excised and resutured. If the suture is not possible, chlorhexidine rinses twice a day and/or systemic antibiotics should be considered. (Greenstein *et al.*, 2008)

Peri-mucositis

Biofilms form on all hard non-shedding surfaces in a fluid system, i.e. both on teeth and oral implants. As a result of the bacterial challenge, the host responds by mounting a defence mechanism leading to inflammation of the soft tissues. In the dento-gingival unit, this results in the well-described lesion of gingivitis. In the implanto-mucosal unit, this inflammation is termed "mucositis". If plaque is allowed to accumulate for prolonged periods of time, experimental research has demonstrated that "mucositis" may develop into "periimplantitis" affecting the periimplant supporting bone circumferentially. (Lang *et al.*, 2000)

Peri-implantitis

Pathologic alterations in the tissues that contact a dental implant fall under the definition of peri implant pathology. The development of inflammatory process that is limited to the peri-implant soft tissue can be defined as peri-implant mucosites. The progressive peri-implant bone loss is referred to as peri-implantitis. (Lang *et al.*, 2000)

Post operative maxillary sinusitis

Timmenga *et al.* reported that the occurrence of postoperative sinusitis after bone grafting of the sinus floor is limited to patients with a predisposition for sinusitis. To minimize the occurrence of a postoperative infection, possible causes should be removed prior to sinus augmentation. (Timmenga *et al.*, 1997)

Fracture

Although mandibular fractures, secondary to the placing of implants are an infrequent complication, they have been widely described in the literature in the past. This complication occurs when implants are placed in atrophic mandible, and usually occur in elderly patients seeking implant treatment in the anterior area in order to improve adaptation of complete lower

prostheses. Raghoebar *et al.* describe four cases of mandibular fractures related to the placing of implants in atrophic alveolar processes. (Raghoebar *et al.*, 2000) Presentation of this complication may occur sometime after surgery, and to minimize these complications, implant placement in atrophic mandible requires a minimum bone height and width of 7 and 6 mm respectively. (Raghoebar *et al.*, 2000) When placing implants in atrophic mandible, periodic follow-ups including clinical and radiographic examination become even more necessary, as well as instructing the patient to avoid occlusal overloading during the osseointegration period. On other occasions, the mandibular fractures are related to complex surgical techniques, such as the transposition or lateralization of the inferior alveolar nerve. (Karlis *et al.*, 2003; Morrison *et al.*, 2002)

Maxillofacial pathology

Osteomyelitis

Periodontal disease, including the presence of plaque in the gingival sulci of the extracted teeth, is considered a risk factor for infection and failure of immediate implants. Especially in patients with previous periodontal disease, peri-implant microbiota have been found to be similar to that found in periodontally diseased sites. (Sussman and Moss, 1993; Mombelli *et al.*, 1995) Immediate placement of implants into extraction sockets is described as a safe procedure. Nevertheless, infection and implant loss are well documented sequelae in several case reports. Sussman and Moss reported failure of an immediate implant due to spread of infection from an adjacent tooth to the site of an implant and causing an extensive localized osteomyelitis. (Sussman and Moss, 1993) In histomorphologic examinations of cases of implant failure, osteomyelitis, not periodontitis, has been observed in the peri-implant tissue. In contrast to periapical lesions around natural teeth, Lindhe *et al.* characterized peri-implant infections extending to the bone marrow and described those lesions as osteomyelitis instead of periodontitis. Despite this histomorphologic distinction, a case of postimplantation osteomyelitis of the severity of the present case, with eventual loss of the mandible as observed in this case, has yet to be reported in the literature. (Esposito *et al.*, 1999; Lindhe *et al.*, 1992)

Peripheral giant cell granulomas

Titanium is the osteointegratable metallic biomaterial most used in dental implants as a therapeutic alternative. Although osseointegration can be successful initially, factors inherent to the implant material or the peri-implant milieu can lead to partial or complete loss of osseointegration and subsequent implant failure. Gingival hyperplasia, mucositis and peri-implantitis have been described among the soft tissue complications associated with dental implants. (Adell *et al.*, 1981; Lang *et al.*, 2000) Pure titanium is the metal most used in dental practice in direct contact with host tissues. In vivo, no metal or alloy are completely inert. Corrosion constitutes one of the possible causes of implant failure after initial success, with the release of ions/ particles into the biologic milieu. These metal ions/ particles that are released can be locally confined and/ or migrate systemically. (Olmedo *et al.*, 2003; Olmedo *et al.*, 2008) Macrophages loaded with titanium particles have been detected in the peri-implant tissue of failed

dental implants. (Olmedo *et al.*, 2003) Numerous case reports in the literature describe histological evidence of inflammatory response and the presence of metallic ions or particles in tissues adjacent to orthopedic prosthesis of titanium or titanium based alloys. (Jacobs *et al.*, 1998) Different researchers have found metal ions in organs and body fluids. In autopsies Urban *et al.* found metal and plastic particles from coxofemoral prostheses and knee replacements in organs such as liver, spleen and lymph nodes. (Urban *et al.*, 2000) The presence of metal particles in tissues in the vicinity of an implant can be the result of electrochemical corrosion, frictional wear or a synergistic combination of the two. Mechanical disruption during insertion, abutment connection or removal of failing implants has been described as a possible cause for the production of metal particles. (Flatebo *et al.*, 2006; Jacobs *et al.*, 1998) The products of corrosion of the metal implant may behave as haptens generating a hypersensitivity reaction with the release of inflammatory mediators: cytokines and macrophage recruitment. Flatebo *et al.* evaluated histologically non perforated oral mucosa covering submerged titanium implants and found macrophages loaded with titanium particles as indicators of the corrosion process in the soft peri-implant tissue of failed human dental implants. The number of macrophages was greater in the proximity of the metal surface of the implant than at a distance. (Flatebo *et al.*, 2006) In addition to reactive lesions different pathologies have been reported in association with titanium implants such as periapical lesions, cystic lesions, neoplastic lesions such as squamous cell carcinoma, Osteosarcoma and plasmacytoma of the mandible. As the use of dental implants continues to expand dentists need to be aware of these complications. (Tozum *et al.*, 2006; Casado *et al.*, 2008; Gallego *et al.*, 2008; McGuff *et al.*, 2008; Poggio, 2007)

Squamous cell carcinomas

Local irritation, tobacco and alcohol exposure and local infections have been thought to be linked to the development of SCC. Titanium osseointegrated implants have been used widely since 1965, and their usefulness and practicality are unquestionable. OLP and history of smoking also could be associated with higher risk of developing a malignant lesion in the vicinity of a dental implant. (Lorena Gallego *et al.*, 2008) Investigators have shown in animal and human studies that many implant materials—such as stainless steel, chromium, cobalt, iron, lead, nickel, manganese, selenium, zinc, beryllium, cadmium, silicon and titanium—have potential oncogenic properties. (Lorena Gallego *et al.*, 2008) The development of neoplasia associated with alloplastic implant materials is a rare complication that a number of clinicians and researchers have reported, primarily in the orthopedic literature. (Abu El-Naaj *et al.*, 2007; Czerninski *et al.*, 2006; Shaw *et al.*, 2004; Block and Scheufler, 2001; Moxley *et al.*, 1997) The development of squamous cell carcinoma (SCC) around dental implants, either endosseous or transosseous, is an uncommon pathological manifestation with only a few cases described in the literature. Squamous cell carcinoma is the malignant neoplasm most commonly reported to involve dental implants. In many of these cases, the clinical presentation of the carcinoma was similar to that of peri-implantitis, leading to a potential delay in the clinician's recognition and diagnosis of the malignancy. (Moxley *et al.*, 1997; Clapp *et al.*, 1996) A possible role for the implant biomaterials in carcinogenesis has not been definitively demonstrated by researchers, as the majority of these patients have one or more known risk factors for oral squamous cell

carcinoma. However, it is well-known that squamous cell carcinomas may arise in sites with persistent inflammation and epithelial turnover, as has been seen in fistula tracts draining chronic osteomyelitis. (Abu El-Naaj *et al.*, 2007; Czerninski *et al.*, 2006; Moxley *et al.*, 1997; Clapp *et al.*, 1996)

Osteosarcomas

The development of malignancy in association with implanted orthopedic hardware is a rare but well-known and devastating complication. (Clapp *et al.*, 1996; Keel *et al.*, 2001; Visuri *et al.*, 2006; Scheon *et al.*, 2004; Kirkpatrick *et al.*, 2000; Paavolainen *et al.*, 1999; Case *et al.*, 1996; Jaffe *et al.*, 1994; Jacobs *et al.*, 1992; Jacobs *et al.*, 1991; Khurana *et al.*, 1991; Ward *et al.*, 1990; Hughes *et al.*, 1987; Memoli *et al.*, 1986; Pedley *et al.*, 1981; Sinibaldi *et al.*, 1976; Sunderman, 1971; Cole *et al.*, 1997; Brien *et al.*, 1990; Harris, 1990) Implant-related sarcomas arise in bone or soft tissue contiguous with the implant hardware. Most tumors are high-grade malignancies and have included pleomorphic sarcoma (malignant fibrous histiocytoma), osteosarcoma, Ewing sarcoma, angiosarcoma, fibrosarcoma, malignant peripheral nerve-sheath tumor, synovial sarcoma, epithelioid sarcoma, epithelioid hemangioendothelioma, chondrosarcoma and lymphoma. (Moxley *et al.*, 1997; Lamovec *et al.*, 1988; van der List *et al.*, 1988; Weber, 1986; Dodion *et al.*, 1982; McDonald, 1981; Tayton, 1980) The majority of endosseous dental implants consist of titanium, which is considered a highly biocompatible material that will promote bone growth and osseointegration. Most dental implant fixtures are manufactured from commercially pure titanium and various titanium alloys, which may contain variable amounts of iron, oxides, aluminum, vanadium, copper, palladium, zirconium and molybdenum. Implant superstructure, attachment and restorative elements may consist of various metallic, polymeric and ceramic materials. Implant fixtures also may undergo a variety of surface treatments—such as passivation, anodization and ion implantation—to enhance the surface oxide layer and prevent the release of metallic ions through corrosion. Texturing to increase surface area may be accomplished by plasma spraying, acid etching and blasting with ceramic material. Manufacturers also may coat implants with bioactive materials to enhance osseointegration (Esquivel-Upshaw, 2003; Ratner *et al.*, 2004). The International Agency for Research on Cancer has classified titanium in Group 3 (meaning that the agent is not classifiable regarding its carcinogenicity to humans), and titanium generally is considered to be a safe biomaterial. However, in isolated reports, investigators have suggested some potential for titanium to induce neoplasia. (McGregor *et al.*, 2000; Williams, 1981; Williams, 1981; Noguera *et al.*, 2000; Yamadori *et al.*, 1986) Researchers have demonstrated the dissolution of metallic corrosion products into adjacent peri-implant tissues. Titanium levels can reach up to 300 parts per million in tissues around implants and can produce a clinically visible discoloration. Researchers also have found high levels of titanium in the spleen and lungs of laboratory animals after implant placement. Others demonstrated precursor B-cell proliferation associated with titanium implants in a mouse model. Those investigators believed this phenomenon to be mediated by cytokines released from macrophages or multinucleated giant cells that were present at the implant/bone marrow interface. (Rahal *et al.*, 2000) Additional factors may be involved in implant related tumor induction. Implanted biomaterials may release trace amounts of residual compounds such as monomers, catalysts, plasticizers and antioxidants that

were used during the implant manufacturing process. It is possible that such contaminants could be associated with sarcoma development.⁸ Research also has shown that, through a phenomenon known as the Oppenheimer effect, implants of solid materials with a large surface area in soft tissue have induced sarcomas in rodent animal models, even though the material has no inherent toxic or tumorigenic properties (Kirkpatrick *et al.*, 2000; Paavolainen *et al.*, 1999). Other contributing or etiologic factors that could have played a role in the development of osteosarcoma in the case reported here include localized osteonecrosis related to implant placement, persistent chronic osteitis and the cumulative low-dose radiation exposure related to multiple radiographic imaging studies of the involved area. Any role played by the osteoconductive graft material placed in the extraction site is undetermined, as we did not find any reports of neoplasia associated with the use of deproteinized bovine bone mineral graft material. (Kirkpatrick *et al.*, 2000; Paavolainen *et al.*, 1999) The accumulated clinical and scientific evidence supports the concept that the development of malignancy in relation to the use of implanted biomaterials, while a rare occurrence, is a potential complication of the use of these materials. (Paavolainen *et al.*, 1999)

Conclusion

The use of endosseous dental implants has evolved rapidly in the past several decades and has revolutionized and greatly enhanced the prosthetic rehabilitation of the dentition. Dental implant systems have undergone rigorous in vitro and in vivo testing and have proven to be safe and biocompatible. The overall success rate is good. However, Dental implant placement is, not without its complications. Complications, include postoperative infection, implant fracture, peri-implantitis, bone loss and failure of osseointegration with loosening of the implant, thus requiring its removal. The development of neoplasia associated with alloplastic implant materials is a rare complication that a number of clinicians and researchers have reported, primarily in the orthopedic literature.

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