International Journal of Current Advanced Research
ISSN: O: 2319-6475, ISSN: P: 2319-6505, Impact Factor: 6.614
Available Online at www.journalijcar.org
Volume 10; Issue 02 (A); February 2021; Page No.23778-23799
DOI: http://dx.doi.org/10.24327/ijcar.2021.23799.4713

Review Article

ORAL IMPLANT COMPLICATIONS AND ITS MANAGEMENT

Kavyashree PM and Nagarathna DV

Dept of Periodontics, A.J Institute of Dental Sciences, Mangaluru-575004

ARTICLE INFO

Article History:
Received 6th November, 2020
Received in revised form 15th December, 2020
Accepted 12th January, 2021
Published online 28th February, 2021

Key words:
Dentistry

ABSTRACT

Dentistry has undergone many changes during the past in the field of implant dentistry. Patients are able to benefit from the implant therapy while Clinician is faced with complex options. Though the success rates are reported with the form of therapy, failures do occur. Hence thorough knowledge regarding the various aspects of failure is necessary.
Dental implants have been a successful treatment alternative for restoring missing teeth. Osseo integrated dental implants represent a widely accepted and documented treatment modality for the rehabilitation of the partially or totally edentulous ridge.
However, treatment is not always successful, because implant is a foreign body. The focus of implant research is shifting from descriptions of clinical success to the identification of factors associated with failure and its management.

INTRODUCTION

Dental implants have been a successful treatment alternative for restoring missing teeth. Osseo integrated dental implants represent a widely accepted and documented treatment modality for the rehabilitation of the partially or totally edentulous ridge.

However, treatment is not always successful, because implant is a foreign body. The focus of implant research is shifting from descriptions of clinical success to the identification of factors associated with failure.[1]

Primary predictors of implant failures are poor bone quality, chronic periodontitis, systemic diseases,smoking, unresolved caries or infections. Improper patient selection, poor oral hygiene, traumatic occlusion, iatrogenic causes of Plaque retention and bone preparation without the use of internally cooled, high torque, slow speed hand pieces, have been the factors contributing to the breakdown of otherwise successful implants

Acentric loading an inadequate number of implants, parafunctional habits and absence /loss of implant integration with hard and soft tissue, inappropriate prosthetic design may contribute for implant failures. When these complications arise, many dentists placing or restoring implants have little or no experience on how to handle the problem.[2]

Corresponding author:* **Kavyashree PM
Dept of Periodontics, A.J Institute of Dental Sciences, Mangaluru-575004

While placing or restoring implants clinician must be prepared for the possibility of potential complications. These may be minor or major, reversible or irreversible in nature.

The 'treatment' of the problem of an increasing incidence of complication occurrence is ironically in the 'prevention' of these problems from occurring. Better case selection, knowledge of systemic problems that can result in complications and better treatment planning are all essential to reduce the risk of complications[3].

Use of available technology and diagnostic tools, that is computer axial tomographic scans, cone beam scans, surgical guides, computer treatment planning, and aids to assess primary implant stability (Periotest, Ostell) along with piezoelectric surgical machines, can aid the clinician in obtaining more predictable planning, placement, and restoration of implant-supported restoration.

Complication can arise in any area of biological function. Implant dentistry has been fraught with compromise and complication. Avoiding those condition that contribute to poor result, choosing patients that offer ideal surgical and prosthetic circumstances and Complex clinical challenges can improve favorable outcome. It is mandatory for every clinician to know how and why the complications occur and how best one can prevent progress of the implant failures.

CLASSIFICATION OF IMPLANT COMPLICATIONS

1. In 1981, Adell classified complications into 3 categories:

- Loss of osseointegration
- Gingival complications
- Mechanical complications
- This classification was expanded by Balshi, who included esthetic, phonetic, functional, and ergonomic complications.

2. In 2008, Kelly Misch *et al*[27], had classified implant complications as:
   - Treatment plan related (wrong angulation, improper implant location, lack of communication),
   - Procedure related (lack of primary stability, mechanical complications, mandibular fracture, ingestion/aspiration)
   - Anatomy related (nerve injury, bleeding, cortical plate perforation, sinus perforation, devitalization of adjacent teeth) and others (iatrogenic, human error)
3. According to tissue affected (Greenstein, 2008), it can be classified as[28]:
   - Soft tissue complications
     a. Haemorrhage
     b. Neurosensory disturbances
     c. Tissue emphysema
     d. Infection
     e. Wound dehiscence
     f. Aspiration or ingestion of surgical instruments
     g. Post-operative pain
   - Hard tissue complications
     a. Peri-apical implant pathosis and endodontic considerations
     b. Lack of primary implant stability
     c. Inadvertent penetration into the maxillary sinus or nasal fossa
     d. Sinus lift predicaments
     e. Mandibular fracture
4. In 2010, Stuart J Froum[29], stated implant complications as:
   - Associated with systemic disorders and medications
   - Associated with implant planning
   - Implant fractures
   - Implant failures
   - Peri-implantitis
   - Esthetic complications due to implant malposition
   - Related to immediate implant placement into extraction sites
   - Related to immediately loaded dental implants
   - Complications can be described as those occurring during first stage surgery, second stage surgery, abutment connection, prosthetic procedure, control after prosthesis placement
5. In 2014, Joan Anfruns[30], proposed classification of implant complications as:
   - Pre-operative complications
     a. Associated with systemic disorders and medications
     b. Associated with implant planning
     c. Diagnostic errors
   - Intra-operative complications
     a. Bleeding
     b. Adjacent soft tissue damage
     c. Damage to adjacent teeth
     d. Broken instruments and aspiration of components
     e. Nerve damage
     f. Perforation of sinus membrane
     g. Associated with bone augmentation
   - Post-operative complications:
     a. Infection
     b. Wound dehiscence
     c. Implant fracture
     d. Implant failure
     e. Esthetic complication due to malposition
     f. Prosthetic complications
     g. Peri-implantitis
     h. Associated with maintenance therapy

Pre-operative complications

With the patient selection being the critical factor for implant success or survival, the medical condition, pharmacologic implications, and overall health of the patient cannot be overemphasized.

Complications associated with systemic disorders & medications

The systemic conditions leading to complications are

- Myocardial infarctions
- Cerebrovascular accidents
- Valvular prosthesis placement
- Osteoporosis
- Paget's disease
- Psychiatric disorders
- Parkinson's disease
- Diabetes
- Smoking
- Immunodeficiency

Pharmacologic considerations

Corticosteroids

Corticosteroids are a common treatment for various systemic diseases. Their use often leads to suppression of a patient's immune response and makes them prone to developing bacterial, viral, and fungal infections. These infections can be difficult to treat with conventional therapy and patients taking exogenous steroids are at risk for osteopenia and osteoporosis. Hence implant placement in such patients can increase risk of infection and implant failure.[46,47]

Bisphosphonates

Oral bisphosphonates are used frequently to treat osteoporosis and osteopenia, and include alendronate (Fosimax), etidronate (Didronel), residronate (Actonel), tiludronate (Skelid).

Patients under treatment with oral bisphosphonates are at a considerably lower risk for osteonecrosis of the jaw than patients treated intravenously.[49]

A patient is considered to have bisphosphonate related osteonecrosis of the jaw if they have the following three characteristics:

- current or previous treatment with a bisphosphonate
- exposed, necrotic bone in the maxillofacial region that has persisted for more than 8 weeks

- no history of radiation therapy to the jaws.
  Hence, implant placement is contraindicated if the patient shows the above characteristics of osteonecrosis.

Anticoagulants

The three main anticoagulants are coumarin, heparin, and aspirin. They are usually prescribed to treat a number of cardiac or vascular disorders, including atrial fibrillation, ischemic cardiac disease, cardiac valvular disease, prosthetic cardiac valves, post Myocardial infarction, deep venous thrombosis, and many others.[50]

The patients taking aspirin for its analgesic and/or anti-inflammatory properties, and do not have thrombotic concerns could discontinue aspirin before dental implant surgery, as these patients are not at known risk for thrombosis.

Antibiotics:[52]

The principle of antibiotic prophylaxis before oral surgical procedures, including dental implants, in patients at risk for endocarditis or in those who are severely immunocompromised is well established. However, it is widely agreed that total use of antibiotics should be reduced to minimize the emergence of resistant bacterial strains.

American Heart Association Recommended Antibiotic Prophylaxis Guideline

Prophylactic antibiotics no longer recommended for patients with these conditions:

- Mitral valve prolapsed
- Rheumatic heart disease
- Bicuspid valve disease
- Calcified aortic stenosis
- Congenital heart conditions such as ventricular septal defect, atrial septal defect, and hypertrophic cardiomyopathy

Prophylactic antibiotics no indicated for patients with these conditions (high risk)

- Artificial (prosthetic) heart valves
- History of infectious endocarditis
- Unrepaired or incompletely repaired cyanotic congenital heart disease including shunts and conduits
- Congenital heart defects repaired with prosthetic material or device
- Cardiac transplantation recipients who develop cardiac valvulopathy

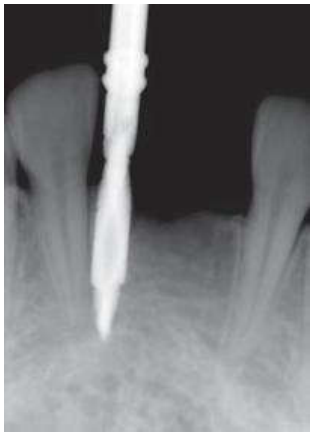
Antibiotic Regimens for Heart Conditions Requiring Prophylaxis

Situation of patient	Agent	Regimen
Standard general prophylaxis	Amoxicillin	Adults: 2.0 g, 1 hr before procedure
Unable to take oral medications	Ampicillin	Adults: 2.0 g IM or IV
Allergic to penicillin	Clindamycin	Adults: 600 mg, 1 hr before procedure
	Cephalexin/ Cefadroxil	Adults: 2.0 g, 1 hr before procedure
	Azithromycin/ Clarithromycin	Adults: 500 mg, 1 hr before procedure
Allergic to penicillin and unable to take oral medication	Clindamycin	Adults: 600 mg IV within 30 min before procedure
	Cefazolin	Adults: 1.0 g IM or IV within 30 min before procedure

Complications associated with implant planning

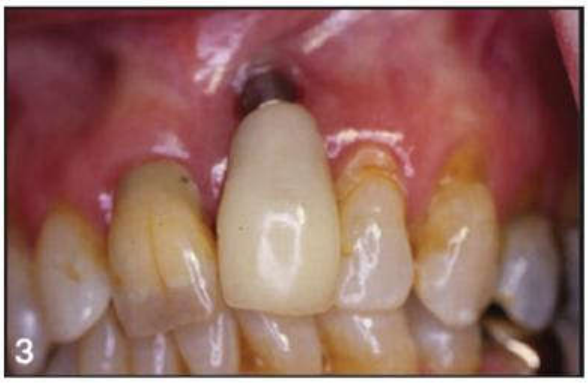
Improper angulation:[53]

Tooth or root proximity to a planned implant site can cause adjacent tooth devitalization.



Improper angulation

Improper implant location



Implant positioned too buccally

- An adjacent tooth with an undiagnosed periapical lesion could lead to implant failure, when theinfection spreads and reaches the implant surface.

Adjacent teeth should be at least 1.5 mm from the implant body and more than 3 to 4 mm between adjacent implants to prevent horizontal bone loss as well as to preserve aesthetics.

Perforations of the inferior alveolar canal
Prevention:[54]

Avoiding implant complications caused by planning during surgical phase requires meticulous preparation.

1. First, use surgical planning tools that can help plan the surgery and reveal potential complications. These tools consist of, periapical and panoramic radiographs, radiographic surgical templates to identify ideal implant locations, and 3D computer software for positioning implants and avoiding vital anatomic structures.
2. Second, consider all manufacturers' recommendations that may be specific to the implant system being used and determine whether it is a one- or two- stage approach. Ideally, the implant platform should be positioned 2-3 mm apically to the buccal cementoenamel junction level of the adjacent teeth. If the position is more coronal it could allow the metal platform to become supragingival, affecting esthetics. If the position is more apical it makes it more difficult to fit prosthetic parts onto the implant, and may create a more difficult maintenance issue.
3. Third, use correct surgical techniques for implant placement. This encompasses all phases of the surgery from the proper incision line to elevating the flap,

osteotomy preparation, implant placement, and final closure.

4. The next important point is to match surgical skills to the level of care required for different anatomic groups.

- The anterior maxilla is considered to be the most challenging area for implant treatment. This is due to issues pertaining to the volume of bone, angulation of the ridge, and the esthetic ramifications.
- The anterior mandible would be least challenging. Here the quality of bone, volume of bone, and lack of anatomically vital structures reduce the implant placement challenges.
- The posterior mandible is considered the highest risk area for implant treatment. This is because of the proximity to the mandibular alveolar canal and the mental foramen. In addition, the mandible can have lingual undercuts, hence increasing the potential risk of perforation of the lingual cortex.
- The second most dangerous area to treat is posterior maxilla. The maxillary sinus is at risk of perforation and having implants unintentionally displaced into the sinus cavity

Complications associated with diagnostic imaging

Due to scanographic templates:[54]

Complications can occur if the template is not properly fabricated or does not fit precisely, leading to movement during the scanning process. In addition, if the patient's existing denture does not represent the proper tooth position or the wrong plane of occlusion, the location of the subsequently placed implants will be incorrect.

Due to 2-D imaging

Problems associated with 2D imaging modalities include various distortion factors which can differ with anatomic location, foreshortening, elongation, overlapping of adjacent structures, lack of density determination, no determination of bone width or quality, and poor spatial relationship of vital structures.

For prevention of such complications, 3D imaging is considered as the most valuable tool. In addition, postoperative CT/CBCT images are very important confirming that implants have been properly positioned in relationship in the host bone and the desired prosthetic restoration.

Limitations of 3-D imaging:[55]

Cone Beam CT scan has some of the same limitations inherent to all imaging modalities. The most important limitations for implant planning are the lack of accurate representation of soft tissue structures, such as gingival, and the various artefacts produced primarily by metal restorations; such artefacts may interfere with the diagnostic process by masking underlying structures. The post-surgical radiolucency around implant may be misdiagnosed for periapical lesion. Furthermore, the cost of CBCT is higher than that of other traditional modalities. The advent of CBCT has also introduced the issue of liability in interpreting the image.

Intra Operative Complications

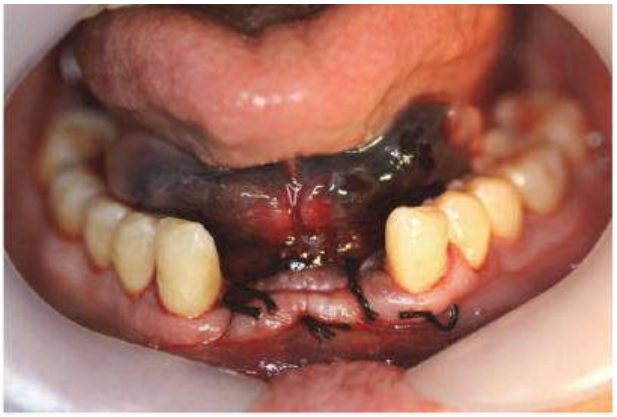
Complications that occur during surgery have the potential to become the most serious. These events may be a result of inadequate planning, mishandling of surgical instruments, anatomical variations, inexperience of the operator, or simply the risks of the procedure itself.

Haemorrhage(bleeding)

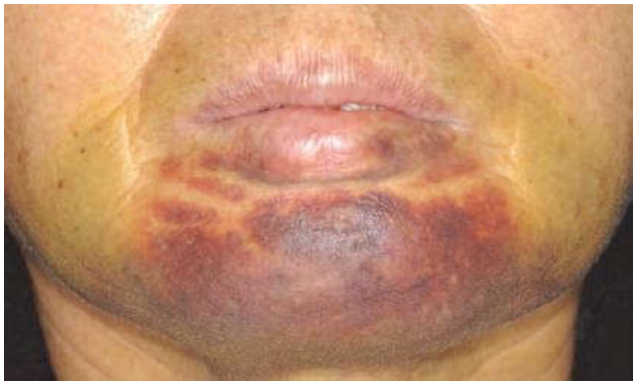
Bleeding during surgery is expected and usually easily controlled. The amount of bleeding associated with a surgical procedure is dependent on numerous factors as extent of flap reflection, soft tissue management, the patient's anatomy, and systemic health.[56]

Severe bleeding and the formation of massive hematomas in the floor of the mouth are the result of an arterial trauma.

The haemorrhage 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.[19]Surgeons also should consider other sources of potential haemorrhage and subsequent hematoma formation, including injuries to muscles or other soft tissues.[58]



A severe hematoma on the anterior floor of the mouth after implant placement



Ecchymosis on the chin after implant placement in the anterior mandible.

When there is massive bleeding and progressive respiratory distress then it resembles the clinical development of Ludwig's angina.

Management

Most important is the immediate bimanual compression at the suspected site of perforation and transport of the patient to the nearest hospital to secure the airway without delay.[19] Once the airway is controlled, efforts are taken for definitive resolution of the haemorrhage. Haemorrhages can be controlled by gauze tamponage, application of haemostatic agents, cauterization, or digital compression.

If haemorrhage cannot be controlled by these methods, ligation of the bleeding vessel should be performed.

To prevent unintentional haemorrhages in cases involving the immediate placement of implants or recent tooth extractions, the practitioner should be careful not to use the extraction socket as a guide for angulation because this may lead to the perforation of the lingual cortex.[58]

Bleeding site during implant osteotomy	Arteries	Treatments
Posterior mandible	Mylohyoid	Finger pressure at the site
Middle lingual of mandible	Submental	Surgical ligation of facial and lingual arteries
Anterior lingual of mandible	Terminal branch of sublingual or submental	Compression, vasoconstriction, cauterization, or ligation
Invading the mandibular canal	Inferior alveolar artery	Bone graft

Table 1. Treatment of a hemorrhage at an implant osteotomy site (Park & Wang, 2005)

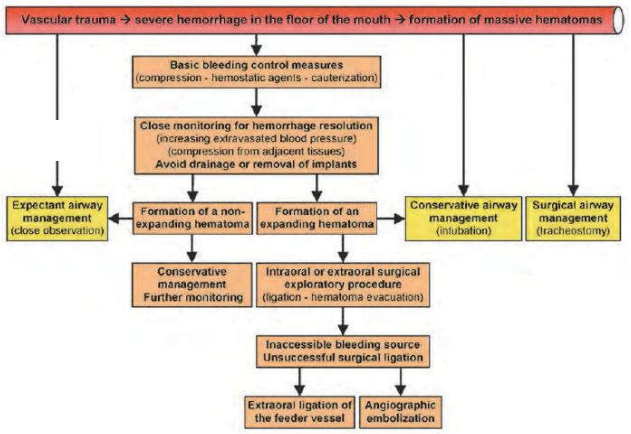


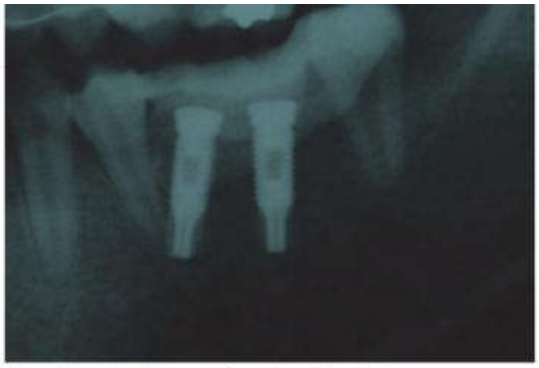
Fig. 4. A flow diagram of airway management and control of massive hemorrhage in the floor of mouth associated with implant placement in the anterior mandibular region (Kalpidis & Setayesh, 2004).

Adjacent soft tissue damage

Damage to adjacent soft tissues is usually related to mishandling of surgical instruments.

Damage to adjacent teeth or structures

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 (**Annibali** *et al*., 2009)[56]. Adjacent teeth should be evaluated before implant placement.



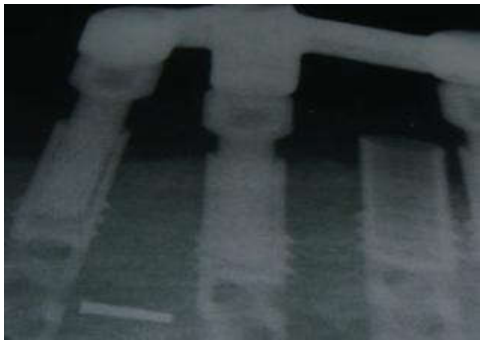
Injury of an adjacent tooth by a malpositioned implant.

Management

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 (Greenstein *et al*., 2008)[28]. To prevent a latent infection of the implant from the potential endodontic lesion, endodontic treatment should be performed (**Sussman,** 1998).[61]

Broken instruments and aspiration of foreign components:[60]

Intraoperative ingestion or aspiration of a dental screwdriver or an implant can present a life-threatening complication. Usually, aspiration of a foreign body will be accompanied by coughing. However, it is possible for a patient to aspirate an object without coughing.



Prevention

Aspiration can be avoided if a piece of silk suture or floss is tied to the screwdriver or another device before it is inserted into the mouth. This provides the clinician a fast way to identify and retrieve a dropped instrument. In addition, it is sensible to place a large piece of gauze into the patient's mouth so that when an object is dropped, it is easily retrieved.

Management

If a device is aspirated, it is necessary to refer the patient to an otolaryngologist for evaluation and treatment. Aspiration usually requires bronchoscopic retrieval. We can place a large piece of gauze into the patient's mouth so that when an object is dropped, it is easily retrieved. Whenever a radiopaque object is suspected medical radiographs are an absolute necessity.

Nerve damage

Neurosensory alterations may occur subsequent to implant therapy. It can occur intra operatively or post operatively. Intra-operative damage can occur during soft tissue manipulation or implant osteotomy preparation. Post-operative sensory disturbances are usually caused by edema and compression of the nerve in the days following surgery and do not require any intervention. These disturbances can lead to paraesthesia, dysesthesia, hypoesthesia, and anaesthesia. Intrusion into the inferior alveolar or mental canal during osteotomy development can cause transection, tearing, or laceration of nerves.

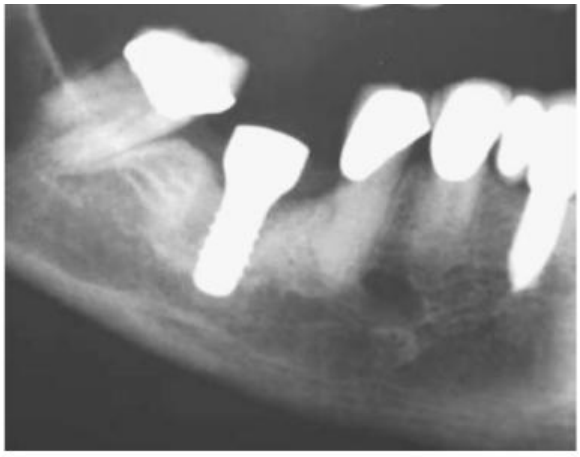


Figure 3 Dental implant penetrating into the inferior alveolar canal

Implant insertion can also result in bone compression on the nerve. In addition, within the soft tissue, the lingual or mental nerve may be injured by compression, stretching, the scalpel, or needle penetration.

Neuropraxia	There is no loss of continuity of the nerve, it has been stretched or has undergone blunt trauma. The paraesthesia will subside, and feeling will return in days to weeks.
Axonotmesis	Nerve is damaged but is not severed; feeling returns in days to weeks.
Neurotmesis	Severed nerve; poor prognosis for resolution of paraesthesia

Classification of nerve injuries (Greenstein & Tarnow, 2006)[15]

Neurological sequelae of nerve injury

After nerve injury, the patient will manifest one or more of the following symptoms: paresthesia (numb feeling, burning, and prickling), hypoesthesia (reduced feeling), hyperesthesia (increased sensitivity), dysesthesia (painful sensation), or anesthesia (complete

Postoperative management after neurosensory alteration

Whenever there is nerve damage occurred during osteotomy development and the implant was inserted, radiographs should be taken to ascertain the implant's position. If it is intruded into a nerve canal, the implant should be slightly withdrawn a couple of turns or removed altogether. The next day, if a patient relates symptoms of altered perception, it needs to be determined whether they are due to the presence of the implant or sequelae of soft tissue manipulation or edema. If it is due to implant,it should be removed. If the twist drill or the implant did not encroach upon the canal, it is possible that bone was compressed, there by placing pressure on the nerve. The implant should be slightly withdrawn several turns. In the event of uncertainty with regard to implant penetration into a nerve canal, a CT scan may be needed to provide additional information.

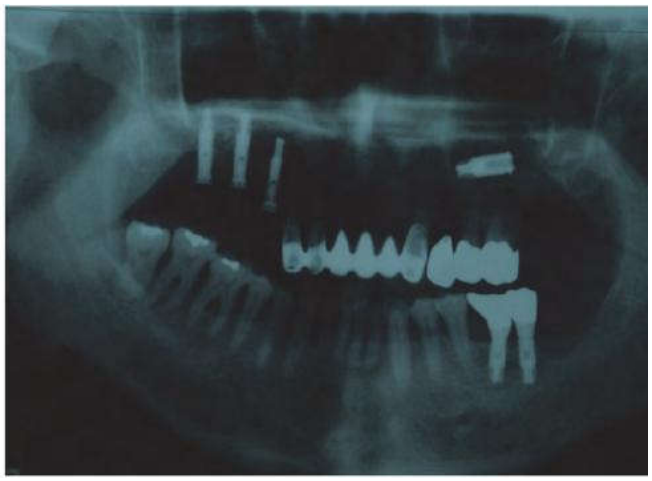
We should document the level of neurosensory dysfunction. Several tests can be used to evaluate neural impairment. The clinician should know the depth and extent of the sensory dysfunction. Altered sensation regarding the lip and tongue and drooling should be documented. Numbness for 16 weeks suggests that the nerve sheath was disrupted, and the patient should be referred for possible microsurgery.

Tests to Discriminate if Neurosensory Damage Has Occurred

1. Light touch test: a soft brush is applied to the lip, and the patient is asked in which direction the stimulus was applied.[18]
2. Pain test: a 27-gauge needle can be used to determine whether the patient perceives pain.[18]
3. Two-point discrimination test: calipers are opened progressively at 2-mm increments until the patient is able to discriminate the caliper ends as two separate points of contact.
4. Ice or a heated mirror handle (43°) can be used to determine whether the patient is able to discriminate between hot and cold.[18]

Perforation of sinus membrane

In the maxillary posterior, the proximity of the sinuses can create a problem for dental implants if there is minimal residual crestal bone (less than 5 mm) for stability. Perforation of the Schneiderian membrane during sinus augmentation procedures is a common finding.



Displacement of implant into the maxillary sinus

Various mechanisms proposed to explain the migration of an implant into the maxillary sinus are:

- changes in intrasinus air pressure
- nasal pressures
- autoimmune reaction to the implant, causing peri-implant bone destruction and compromising osseointegration
- resorption produced by an incorrect distribution of occlusal forces (Galindo *et al*., 2005). [69]

The changes in intrasinus and nasal air pressures produce a suction effect because of the negative pressure exerted by these cavities. A portion of the bone grafting material can become dislodged in the maxillary sinus at either the initial ridge augmentation or during the implant placement surgery. The natural ciliary movement in the maxillary sinus will transport foreign material toward the ostium (Hunter *et al*., 2009).[70] In cases with less than 5 mm of bone, mastication can cause the implants to move during the graft maturation timeframe (Peleg *et al*., 2006).[71]

Prevention

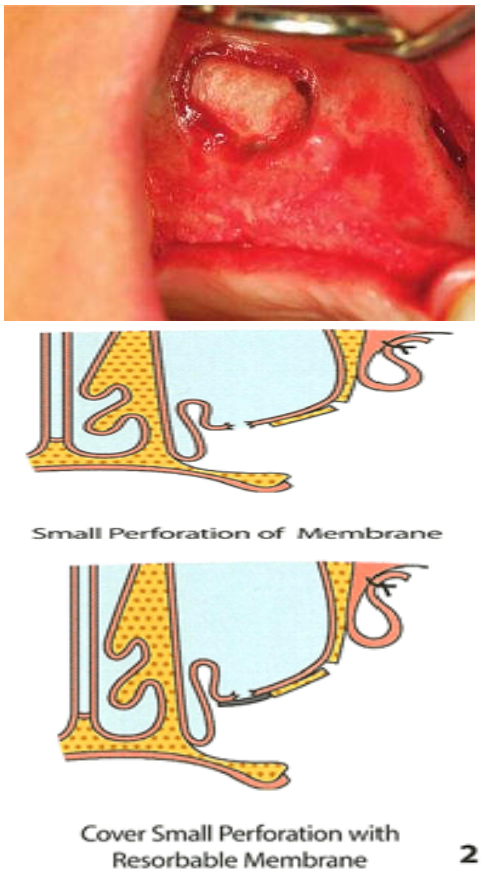
A thorough knowledge of the 3D anatomy of the sinus is essential if the perforation is essential if the perforation rate is

to be kept minimum. A CT analysis will give information relating to the thickness of the crest and lateral walls, presence of discontinuities in the bony walls, width of sinus, slope of the anterior sinus wall, membrane thickness, and the presence, size and location of septa.

Management

If a perforation is small (<5mm in diameter), the problem may be closed by using tissue fibrin glue, suturing or covering with a resorbable barrier membrane.

If the perforation is large (>5mm in diameter) larger barrier membranes, lamellar bone plates or suturing may be used either alone or in combination with tissue fibrin glue to provide a superior border for the grafting material.



If a tear in the membrane occurs along the periphery of the osteotomy and it is difficult to reengage the membrane, this situation can be managed by extending the osteotomy outline several millimetres past the original window and re-establishing contact with the membrane.

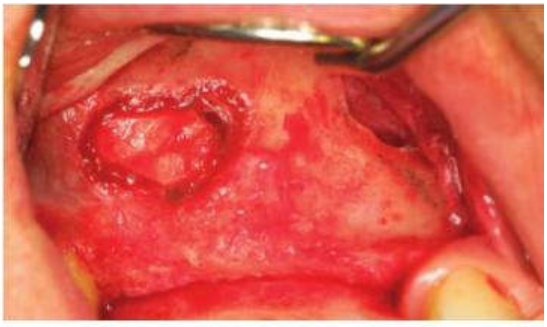


Figure 12 Extension of osteotomy to recapture perforated membrane

Bleeding from the membrane can be managed by placing gauze that is saturated with aesthetic solution (contains 1/50,000 epinephrine) directly onto the membrane. Bleeding from the bone requires direct pressure with an instrument (e.g., a haemostat), and it can be touched with a cautery unit. If the osteotomy is developed, another way to manage an intraosseous arterial bleeder is to displace the membrane and clamp the bone with a mosquito haemostat, therebycrushing the bone and occluding the bleeding blood vessel.

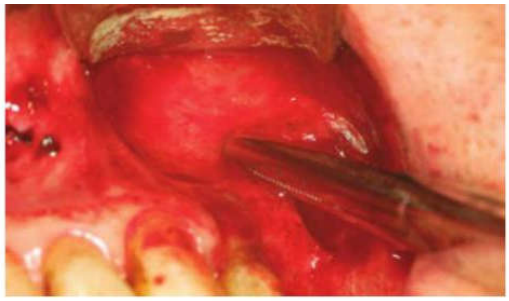


Figure 13 Haemostat used to clamp the bone after the Schneiderian membrane was displaced

Complications associated with bone augmentation procedures

Recipient site complications: Early healing complications

Graft contamination

To maintain cellular viability bone graft should be stored in sterile normal saline after harvest, rather than sponge or towel.[74] Minimal time should elapse between graft harvest and placement. Soaking the graft in 10% povidone-iodine solution for 10 minutes has been found to eliminate surface bacteria without altering the histologic integrity of the graft.[76]

Complication: graft contamination

Etiology: mishandling graft

Prevention: sterile drapes, use of bone clamp or Allis forceps, separate protected graft container, remove powder on surgical gloves

Treatment: povidone-iodine, reharvest additional graft.

Wound Dehiscence

Complete flap coverage and tension free closure are essential to the successful incorporation of the bone graft. Incision line opening with graft exposure is the most common recipient site complication with onlay bone augmentation. Revascularization of the bone graft is essential for incorporation into the recipient site. Therefore, exposure of the bone graft is detrimental to the prognosis of the graft and often leads to graft failure.

The postoperative management of wound dehiscence after that onlay bone grafting is based on the biologic principle that the graft is non-viable until revascularized. No attempt should be made to resuture or manipulate the surrounding flap as the edematous soft tissue is inflamed and friable. Once exposed to the oral cavity the microporous surface of the bone graft is contaminated with a biofilm of bacteria. As such, the exposed bone is no longer biocompatible and the surrounding soft tissue will not accept attempts of recovery. In addition, epithelium will not grow over the exposed bone. The clinician should let the wound declare itself and closely monitor the healing. Sharp protruding edges of the bone graft may be smoothened and reduced with a coarse diamond bur. If more than half of the bone graft becomes exposed the prognosis is poor and graft removal should be considered. Cancellous bone

grafts tolerate exposure better than cortical bone grafts as they revascularize more quickly.[78]

Infection

The incidence of postoperative infection after onlay bone graft surgery is low.[79] Infections may occur within the graft donor or recipient site.

As the consequences of postoperative infection are detrimental to graft success, the patient should be placed on prophylactic antibiotics starting with a loading dose 1 hour before surgery and continuing for 1 week.[79] Amoxicillin is commonly used as it is well absorbed and only requires administration 3 times a day. Preoperative chlorhexidine rinsing can reduce the bacterial contamination of intraorally harvested bone grafts.[81] Chlorhexidine rinse is used thereafter twice daily following surgery as oral hygiene procedures, such as brushing and flossing, are avoided around the surgical site.

Recipient site complications: late healing complications

Bone graft incorporation and resorption

Free autogenous bone grafts must be revascularized in order to incorporate. The cancellous portion of the graft revascularizes more rapidly than the cortical bone.[83]

Membranous bone grafts, from the mandible or calvarium, have been found to reveal less resorption than grafts from endochondral sites, such as the iliac crest.[84] Denser cortical bone grafts when used for onlay bone augmentation.[85] Cortical bone grafts from the mandible exhibit minimal resorption and maintain their dense quality, making them ideal for onlay augmentation before implant placement.[86] Whereas the greatest change in the width of a corticocancellous graft occurs in the first 3 months the volume loss in height stabilizes after 1 year.[87] It is prudent to overbuild the reconstructed ridge slightly, in anticipation of some volume loss upon healing.

The osseous recipient bed is prepared to improve the fit and contact of the bone block graft. Perforation of the cortex in the recipient site with a small round bur releases growth factors, expedites revascularization of the graft, and improves the graft incorporation.[88]

Block bone grafts do not tolerate micromovement and will resorb unless rigidly fixated. The graft is mortised into the recipient bed and fixated to the ridge with screws rigidly. Fixatin screws typically range from 1 to 2 mm in diameter. A screw length that maximizes retention within the native bone should be selected. The use of barrier membrane has been suggested as a strategy to reduce resorption of block bone grafts.

It is imperative that the onlay bone graft remains immobilized during healing. A fixed provisional prosthesis, such as a temporary bridge or bonded prosthesis, is preferred for tooth replacement over the grafted sites, with minimal contact with the grafted site.

Unfavourable concentration of forces from the opposing dentition should be avoided and a broad distribution of occlusal contacts is preferred. Bruxism has been found to impact outcomes negatively in grafted patients.[82] It requires 2 months for the onlay graft to form union to the host bone and after this it rely less on the fixation screws for immobility.

Etiology: graft remodelling, graft character (cortical, cancellous), poor fixation, graft loading.

Prevention: recipient site preparation, fixation screws, barrier membrane, overbuild graft site, fixed provisional prosthesis or disuse and modification of removable prosthesis, minimize re-entry flap reflection.

Treatment: shorter implants, narrower implants, regraft at implant insertion.

Complications associated with Guided bone regeneration:
The most common complication is the premature exposure of the membrane to the oral environment and its sequelae. Once exposed to the oral environment, micro-organisms can invade the surface and pass through the membrane. The colonization the regenerating tissue starts 3-4 weeks after exposure. This period can be assumed as the critical time for membrane to avoid infection to the deeper tissues and further leading to implant failure.

On the basis of the evidence emerging from clinical practice, a possible classification of complication in GBR with non-absorbable membranes can be suggested:[89]

- Exposure and infection of the membrane:
  1. Class I: small membrane exposure (_< 3mm) without purulent exudation
  2. Class II: large membrane exposure (> 3 mm) without purulent exudation
  3. Class III: membrane exposure with purulent exudation
  4. Class IV: abscess formation without membrane exposure

Better results with non-absorbable membranes than with absorbable ones are due to better space-maintaining abilities, controlled time of barrier function and lack of resorption process.

Class I

It can be maintained with a focussed hygiene regiment consisting of topical application of 0.2% chlorhexidine gel twice daily to reduce plaque formation and avoid inflammation of the surrounding tissues. The membrane can be left in place for maximum of 3-4 weeks. After this period, membrane removal must be performed. Because of its osteogenic potential, the soft tissue under the membrane must not be removed, to avoid damage to the regenerating tissue.

Class II

In cases exhibiting an exposure larger than 3 mm, the membrane must immediately be removed to avoid infection of the regenerating tissue. If the underlying bone graft is not compromised, the flaps should be closed to allow the grafted area to heal for at least 4-5 months. Antibiotics coverage with amoxicillin and clavulanic acid is also suggested.

Class III

If the membrane exposure is associated with a purulent exudate, the membrane must be removed immediately to limit the damage caused by the infection spreading to the underlying regenerating tissue. After membrane removal, a gentle curettage of the graft is essential to remove the infected particles and inflammatory tissue that could jeopardize the regenerative process. Amoxicillin (875 mg) and clavulanic

acid (125 mg) should be prescribed twice a day for at least 5 days.

Class IV

This is rare, albeit severe clinical complication, characterized by the formation of an abscess in the surgical area without the exposure of the membrane. The etiopathogenesis of this phenomenon may include any or more of the following:

- Bacterial contamination of the e-PTFE during membrane handling
- Bacterial contamination of the bone graft
- Improper suture removal
- Endodontic/periodontic infections from adjacent teeth
- Inadequate prosthetic margins
- Patient inoculation of the area with exogenous bacteria

The membrane must be immediately removed and all the infected tissue curetted. The use of a rifamycin or tetracycline antibiotic wash is also suggested to reduce bacterial contamination of the treated area. The patient should be placed on a regimen of antibiotics for 5 days.

Post-surgical complications

Infection and wound dehiscence

These complications are earlier dealt under intra-surgical complications.

Implant Fracture

Introduction

One of the most severe complications is the fracture of a dental implant that has undergone osseointegration,Since fracture is often associated with sustained or intermittent force application, the loss of one implant may condemn the prosthesis to imminent failure. The remnants of the implant, remaining integrated with bone, must be surgically resected and are then subject to postsurgical morbidity that could include pan, infection and possible jaw fracture.

Etiology

Bone loss versus abutment screw configuration

Bone loss may be a factor that is associated with implant fracture. There is a specific pattern of bone loss often seen in cases where implants have undergone fracture.[93]In cases where this bone loss advances to the level past engagement of the abutment screw, this area seems to be most vulnerable to cyclic fatigue as itis the thinnest portion of the implant. In addition, the modulus of elasticity of titanium is in the order of ten times higher than that of bone, thereby predisposing both to shear forces.

Bone, being a dynamic tissue, is capable of adaptation to the forces placed upon it within the normal confines of the prosthesis-implant complex. However, outside of what would be considered normal, the metal may undergo cyclic fatigue and ultimately failure under the bending forces placed upon it. Frequently, this seems to occur at the same level as the base of the osseous defect, making this a fulcrum point for bending forces. Indeed, microstructural analysis has demonstrated that fragments from fractured implants showed patterns compatible with fatigue failure.

Although early theories suggest that bone loss makes the implant more susceptible to fracture[93], it is possible that bone loss may be secondary to microfracture of alloy microstructure and be a sequel of the fracture itself.[94] Retrograde infection of the site from the intaglio surface of the implant through the fracture may induce inflammation and be responsible for some of the bone loss. Often, commercially pure titanium will tear under chronic cyclic overload and create a notch. If a defect notch starts at an interface and has a degree of micromotion or acts as a conduit for inflammatory mediators, it would be evident that bone loss would ensue. As a result of further propagation, the bone loss becomes a secondary factor for initiation by the microfracture.

Iatrogenic implant placement or manipulation

Cross-threading the internal threads of the implant may also lead to component complication, increasing the potential for fracture. This may occur either because cross-threading leads to more frequent screw loosening or because the cross-threading is rectified through the use of a tap to recreate the appropriate threads, thereby removing some material from the internal surface of the implant. Gentle, deliberate placement of healing abutments, impression components, and restorative abutments will preserve the pristine nature of these mechanical connections, avoiding the potential for an unstable connection.

Manufacturing defects

Defects in the raw materials and in the manufacturing, process are certainly possible when implants are made. Clinicians are cautioned to purchase implants from manufacturers who demonstrate good manufacturing practices in keeping with International Organization for Standardization or Food and Drug Administration Standards.

Biomechanics

It has been documented that the human bite force potential can exceed several hundred Newtons of force in the molar region, thereby transferring a high magnitude of force to the implant. Typically, fractured implants are found in the molar areas where this force potential is quite high. Other sources of fractures may be adjacent to cantilever extensions where force application has the potential to cause bending moments, precipitating fracture. Limitations in the length and use of cantilever extensions may be judicious where concern exists over bite force potential.

Patient-related factors

Parafunctional activities can lead to excessive forces on the implants. In addition to bruxism and clenching, some patients simply chew with excess force.

Prevention

If parafunctional activities are noted it is appropriate to provide the patient with a protective occlusal guard that can be used during sleeping hours. Likewise, protective guards could be used during waking hours for patients who repeatedly clench or grind during these times. Counselling may be beneficial for patients who chew with excessive force. There is, however, no panacea for this patient group. It may be more appropriate for patients in this category to consider a greater number of implants to share in the functional load and to consider implant of larger diameter, thereby making the implant more resistant to the forces that could cause fracture.

Implant Failure

Definition

Implant failure refers to the state where the implant has lost integration at a time-point following implant placement.

Classification

There are two commonly used periods to assess an implant failure that relate to the time of occurrence:

- Early failures: failures before osseointegration, primarily the result of surgical and/or post-operative complications.
- Late failures: failures after the osseointegration period, usually arising during and after the restorative phase.

Etiology

Implant failure can be caused by several factors, including,

- Infection
- Tissue trauma
- Overload
- Iatrogenic
- Guided bone regeneration

Infection

Periimplant bone loss due to infection leading to implant failure is known as peri-implantitis and is dealt later under peri-implantitis.

Tissue trauma

An important factor in the etiopathogenesis of early implant failure is the overheating of the bone at surgical site. The critical temperature above which bone necrosis occurs is 47^{o}C for 1 minute.[102]

Pathologic features of bone loss caused by tissue trauma includes:[103]

1. Presence of bone sequestra
2. No regeneration of the peri-implant bone
3. Presence of an inflammatory infiltrate in the gap between bone and implant
4. No organization of the peri-implant bone clot
5. Presence of a compact and mature bone around the implant, and
6. Presence of bacteria and necrotic bone around the implant

Overload

Transmucosal loading and occlusal trauma can lead to implant failure by causing implant bone loss.

Iatrogenic factors

Before the surgical stage there are appropriate and accurate imaging tools for accurate diagnosis and planning. If those are lacking, there is limited ability to diagnose bone morphology, existing pathology, and anatomic aberrations. This becomes especially important when dealing with a site in close proximity to significant vital anatomic landmarks. The resultant incorrectly placed implant would be considered a failure at the time of placement, i.e. an iatrogenic failure.

Prevention

Many of situations that lead to implant failure can be avoided by meticulous planning and execution. Planning of the case using diagnostic radiographs, particularly CT scan imaging, wax-ups and attention to detail before and during implant procedures can minimize problems. It is of utmost importance for the clinician to assess the risk-to-benefit ratio in each case, and plan the case such that the relevant risk factors are modulated before treatment.

Treatment

The first step in treatment is to diagnose and identify the failed implant. In any case of implant failure where mobility is apparent, the implant should be removed immediately. Then the treatment sequence following depends on the site as well as the amount of the tissue loss and the ability to provide primary stability for the replacement implant.

The treatment options for managing implant failure include the following:

- Immediate replacement of failed implant with a wider diameter implant
- Simultaneous replacement of a failed implant with a guided bone regeneration procedure
- A staged approach where the lost tissue is first rebuilt, and the implant is then placed following site healing.

Esthetic Complications Due To Malposition

Introduction

Esthetic complications can be caused either malpositioned implants, inappropriate number and/or site of utilized implants, or peri-implant infection progressively leading to the destruction of peri-implant bone, or by existing bone or soft-tissue deficiencies in the alveolar process.

Preoperative planning: clinical assessment

Conditions comprising systemic, extraoral and intraoral factors, and patient expectations, form the basis of the aesthetic risk assessment (ERA) for implant therapy proposed by Martin *et al*.[117] The key intraoral or local site factors are as follows:

- *Gingival biotype*: Thin gingival biotype situations present with a much higher risk of mucosa recession than thicker gingival biotypes.
- *Shape of tooth or crowns:* Replacement of teeth that are more triangular in shape present with higher esthetic risk than teeth with a rectangular outline. Greater challenges are presented to the clinician in closing embrasure spaces and creating narrower cervical contours when replacing triangular teeth.
- *Infection at the implant site:* In dentate sites, the presence of acute infection increases the difficulty of managing the peri-implant soft tissues during surgical procedure and the risk of complications postoperatively.
- *Bone level at adjacent teeth:* The bone level at proximal surfaces of adjacent teeth dictates the height and form of the implant-tooth papilla after restoration of the implant. Natural teeth that have compromised proximal bone increase the risk of a reduced or absent papilla.

- *Restorative status of neighboring teeth:*When teeth adjacent to the proposed implant site have been crowned, there is an elevated risk of recession occurring post-operatively and exposure of the crown margins after healing.
- *Width of edentulous span:* In general, multiple adjacent missing teeth are a much greater challenge esthetically than single-tooth replacements. The principal challenge is in creating a papilla between two adjacent implants, or between an implant and a pontic.
- *Soft-tissue anatomy:* If the pre-existing site presents with soft-tissue deficiencies in a horizontal and/or vertical plane, this increases the difficulty in achieving ideal esthetic outcomes. Adjunctive hard- and soft-tissue graft procedures are often required.
- *Bone anatomy of the alveolar crest:* This factor is closely related to the soft-tissue anatomy. Where there is a significant effect in the bone, adjunctive hard-tissue grafting procedures are usually required.

Prosthetic-Related Dental Implant Complications

Complications have been defined as secondary conditions that develop during or afterimplant surgery or prosthesis placement.

Mechanical complications

a. Complications caused by unfavorable implant placement:
b. Complication attributable to the prosthesis: overdenture attachment complications
c. Prosthesis fractures
d. Screw loosening and fractures:

Biologic complications attributable to the prosthesis

Gingival inflammation and proliferation:

Etiology

Gingival inflammation and proliferation around the implants have been noted when implant overdenture bars or frameworks associated with implant fixed complete dentures are placed too close to the tissue.

Gingival inflammation and proliferation have been more commonly observed with implant overdentures and implant fixed complete dentures than with other implant prostheses.

Prevention and treatment

Bars for implant overdentures and the cantilever extensions of fixed complete dentures should be located 1-2 mm above the soft tissue. The patient must be shown how to clean adequately around their prostheses and they must be encouraged to maintain a high level of homecare. It has been stated good oral hygiene is the main factor in preventing adverse soft tissue responses.[127]

A loose or fractured abutment screw can produce localized gingival inflammation and proliferation. If a screw has come loose or has fractured, it should be tightened or replaced and that usually eliminates the soft tissue complication. Extensive soft tissue proliferation as a result of long-standing poor hygiene may require soft tissue surgery to remove the proliferative, unhealthy tissue.

Fistulae

Etiology

Fistulae have been noted when there are loose and/or fractured screws that attach a crown, prosthesis or prosthetic component to an implant. The fistula is commonly located at the level were the mechanical deficiency is located. It has also been noted around cemented crowns and prostheses when there is excess cement retained subgingivally.[128] The problem is exacerbated by deep subgingival margins that make it difficult to remove excess cement.[128]

In situations where two-stage surgery is used, insufficient tightening of the implant cover screw may result in its loosening and the development of a fistula within few days of surgery.

Prevention and treatment

Loose and/or fractured screws should be tightened or replaced and this will resolve the fistula. When implant crowns or prosthesis are cemented, the excess cement should be carefully removed and the sulcus inspected for any remnants of cement left behind. It is wise not to place crown or prosthesis margins too deep into the sulcus as it becomes difficult to determine whether excess cement is present. Some fistulae related to excess cement require surgical flap reflection to remove the excess cement.

A provisional cement is recommended where there is sufficient retention provided by the abutment. When a definitive cement is needed because of a short abutment, zinc phosphate is recommended as opposed to glass-ionomer or resin cements. It has been shown that zinc phosphate cement is easier to remove and resin cements are the hardest to remove.[129]

Swallowing an instrument or implant component

Etiology

An instrument such as Hex driver or any of the implant components can be dropped while placing into the mouth and removing them from the mouth. They can be inadvertently swallowed when they land in the back of the mouth.

Prevention and treatment

Good surgical technique, including the use of a throat pack when small instruments, screws, abutments, or implants are inserted in the oral cavity, is the best way to avoid inadvertent swallowing or aspiration. Using floss in directional or indicator instruments can make retrieval of these easy if they shift position, or go under or over the tongue.

Floss should always be tied to screwdrivers to permit retrieval should they inadvertently slip out of the fingers. Throat packs or pharyngeal screens are also effective for intubated patients.
If an instrument or other foreign body such as implant component is inadvertently swallowed, a chest radiograph should immediately be made and evaluated. A specialist should be consulted to determine whether the foreign body should be removed or whether it is likely to pass through the gastrointestinal tract.

Peri-Implantitis

Etiology

Microbiological aspects

Biofilm formation

A layer of glycoproteins will coat the implant surfaces that are exposed to the oral environment. Single bacterial colonies will adhere to the pellicle coat following which larger and more expansive aggregates of oral bacteria is formed. Such early colonization is usually predominated by a Gram positive coccoidal and rod microbiota. As time passes, the biofilm development will result in a more complex microbiota, the composition of which is dependent on the microbiota of the entire oral ecosystem.

Microbiota associated with peri-implant infections:[130]

Bacterial infections play the most important role in the failure of dental implants. Bacterial flora which are associated with periodontit is and peri-implantitis, are found to be similar. Studies have shown that the bacterial flora at the failing implantsites consist of gram-negative anaerobic bacteria including porphyromonasgingivalis, prevotella intermedia and actinobacillusactinomycetemcomitans, which resemble the pathogens in periodontal disease.

It has been demonstrated that the bacteria which are found in the implant sulcus in the successful implant cases, are basically the same flora as are found in the natural tooth sulcus in a state ofhealth. The implants in partially edentulous patients appear to beat a greater risk of peri-implantitis than the implants in completelyor fully edentulous patients.

Differences may exist in how microorganisms colonize on a tooth compared with a titanium implant surface. Thus, staphylococcus aureus, a pathogen commonly not considered in periodontal microbiologic research, is known to have an important ability toattach to almost any biofilm on titanium.

Such infections are difficult to eradicate because bacteria that cause these infectins live in well-developed and protective biofilms. Staphylococcus aureus autolysin may be an important factor in the early colonization of such implant devices, including oral titanium implants.

Biomechanical factors such as an occlusal overload may play a significant role in the failure of the implant. The occlusal overload may result in progressive bone loss around the implant, thus leading to the failure of the implant. The implants which suffer from traumatic failure have subgingival microflora resembling that which is present in a state of periodontal health, with cocci and nonmotile rods as the predominant morphotypes i.e. streptoccous and actimomycesspecies as the predominant microflora.

The other aetiological factors are patient related factors that include

- systemic diseases e.g. diabetes mellitus, osteoporosis, etc;
- social factors- such as inadequate oral hygiene, smoking anddrug abuse;
- para functional habits e.g. bruxism and iatrogenicfactors such as lack of primary stability and premature loading during the healing period.

Risk factors:[131]

Smoking

Smoking is a significant risk factor for dental implant therapy and augmentation procedures accompanying implantations. Smoking has an adverse affect on implant survival and success. The effect of smoking on implant survival appeared to be more pronounced in areas of loose trabecular bone.

Genetic factors

Results provide evidence that IL-1RN gene polymorphism is associated with peri-implantitis and may represent a risk factor for this disease. (*LaineMLet al.)*

Diabetes

Poorly controlled diabetes may be considered a risk factor for increased severity of periodontitis. Large scale randomized-controlled clinical trials is needed.

Occlusal overload

Occlusal overload less than 100µm – doesn't cause bone loss
Occlusal load 180 µm or more – may cause vertical bone loss
Implants are less tolerable to non-axial occlusal load because of lack of PDL. Occlusal load is more concentrated on the marginal bone

Poor plaque control

- Patient's ability to mechanically clean the site with brushes, interdental brush, and floss is related to implant positioning and meeting patient expectation for esthetics, phonetics, and function.
- Moreover, prosthesis design can also preclude clinical evaluation with probing and adequate home-care procedures. These concerns must be factored in the prosthetic decisions to facilitate daily oral hygiene. (*Serino G 2009)*
- Dentist should educate the patient in proper plaque control and to ensure the establishment of regular periodontal maintenance. This will help to assess the adequacy of plaque removal efforts and to intervene as early as possible if problems are detected.

Residual cement

- Incomplete removal of cement left in the subgingival space around dental implants. The cementation of crowns on implants is a common practice. It is quite plausible for cement to be left behind because of implant positioning and the subsequent suprastructure design, which may hamper mechanical non-surgical therapy efforts to access the subgingival space. *(Linkevicius T 2012)*
- Dental cement causes of inflammation and disease may be related to its roughness; however, its surface topography may provide a positive environment for bacterial attachment.

Pathogenesis

At the First European Workshop on Periodontology in 1993, two disease patterns associated with oral implants were identified and defined.

Peri-imlant mucositis is a term used to describe reversible inflammatory reactions in the mucosa adjacent to an implant.

*Per-implantitis*is defined as an inflammatory process that (i) affects the tissues around an osseointegrated implant in function and (ii) results in loss of supporting bone.

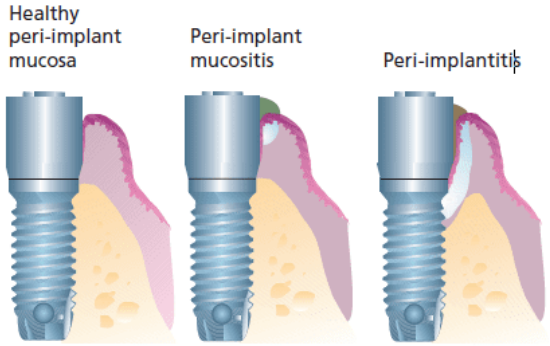


Fig. 24-1 Schematic drawing illustrating healthy peri-implant mucosa, peri-implant mucositis, and peri-implantitis.

Features	Periimplant mucositis	Periimplantitis
Periimplant disease	Disease related to soft tissues around implant	Disease related to soft tissues & its underlying bone
Similarity irt tooth	Similar to gingivitis	Similar to periodontitis
Clinical features	BOP, Inflammation	BOP. Inflammation , Pocket depth, and bone loss.

For ethical reasons, experimental studies of peri-implant infections cannot be conducted in humans. Hence, the information gathered in this field must rely on animal studies.

The biopsy samples from a dog study revealed the lesions in the periodontal sites was consistently separated from the alveolar bone by a zone of non-inflamed connective tissue, while the lesions in the peri-implant tissue in most situations extended into and involved the marrow spaces of the alveolar bone.

It was concluded that the pattern of spread of inflammation was different in periodontal and peri-implant tissues. The lesions in plaque-associated periodontitis were limited to connective tissues, while in the peri-implant tissues the lesions also involved the alveolar bone. In contrast to the periodontal tissues, the peri-implant tissues appeared to be poorly encapsulated to resolve progressive, plaque-associated lesions and extend into the marginal bone tissue and may, if they are allowed to progress, lead to loss of implant.

Diagnostic aspects

Mobility

Loss of clinical stability as a result of complete loss of osseointegration would be reflected in a sudden increase in implant mobility. Therefore, an increase in clinical mobility represents a highly specific, but not at all sensitive, parameter for monitoring clinical stability.

Bleeding on probing

The diagnostic accuracy of BOP was significantly higher than that of teeth. Hence, from a clinical point of view, absence of BOP around implants would indicate healthy peri-implant tissues.[132]

Probing depth and loss of attachment

Probing around oral implants must be considered a sensitive and reliable clinical parameter for long term clinical monitoring of peri-implant mucosal tissues. Presence of soft tissue seal inhibits probe tip penetration in healthy or slightly inflamed peri-implant soft tissues, but did not do so in peri-implantitis. Repeated subsequent comparisons of probing depth and loss of implant support in comparison with baseline measurements are highly recommended.

- ***Pus formation***

Pus formation is always a sign of infection with active tissue destructive process taking place. Periimplantitis lesions usually yield some pus formation upon provocation by pressing on mucosal tissues, while mucositis lesions may not.

- ***Radiographic interpretation***

For two staged implant surgery, the distance from the implant shoulder to the alveolar bone crest represents a reliable radiographic parameter for long term monitoring in clinical practice,[133] provided that optimal exposure geometry has been achieved. For immediately loaded implants radiograph is taken on the day of implant placement and during recall visit. To detect bone loss, the alveolar bone level is compared in both the radiograph. Conventional radiographs have a low proportion of false-positive findings and, hence yield high specificity for the detection of peri-implant bone loss. However, this characteristic limits radiographs to being confirmatory rather than exploratory.

- ***Prophylactic procedures***

Instruction in oral hygiene and patient motivation

To provide a good long-term prognosis, the dentition has to be free of oral diseases before the actual implant installation. Plaque control is recognized as an integral part of periodontal treatment and forms the basis for the prevention of future disease.[134]

Cleanable reconstructions

It is well established that over contoured reconstructions, particularly in the proximal region, and also, sub-gingivally placed reconstructions with imprecise margins will prevent the patient from attaining optimal oral hygiene, thereby jeopardizing the health of abutment teeth and their surrounding tissues. Hence, reconstructions must meet high standards of marginal precision, especially in situations where esthetic aspects demand slightly sub gingivally placed margins as this would influence the subgingival microbiota.[135]

Furthermore, interproximal contours adjacent to abutment teeth or implants have to be shaped to accommodate appropriate cleaning devices.

Maintenance care

After successful periodontal and implant therapy the patient should be offered a maintenance care program adequately designed to fit their individual needs. This will provide optimal preventive services and facilitate the treatment of ongoing or emerging disease process by providing appropriate supportive therapy.

A recall visit may be divided into four different phases:

- Examination, re-evaluation, diagnosis
- Motivation, reinstruction, instrumentation
- Treatment of infected sites
- Polishing, fluoridation, determining recall visits.

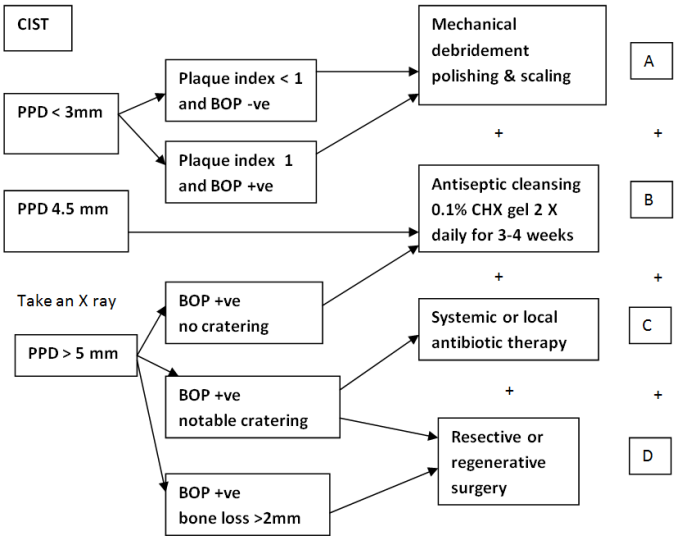
Therapeutic Strategies

Stages of treating periimplantitis

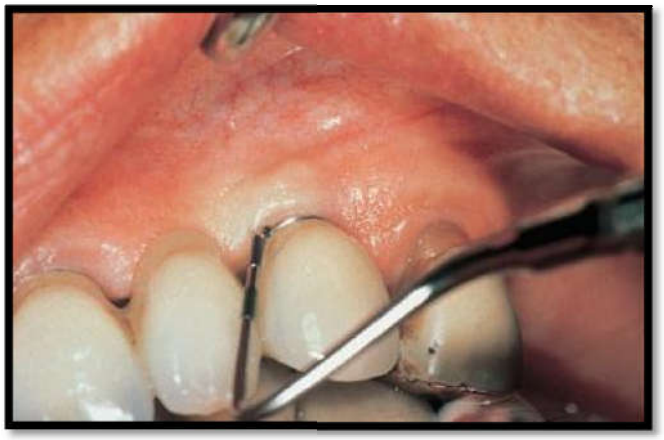
1. Treatment of acute complications of chronic marginal periodontitis.
2. Cleaning performed by a doctor to remove microbial natural factors:
   - gingival debridement by removing plaque
   - biofilm;
   - supragingival scaling;
   - professional subgingival scaling;
   - suppression of inflammatory processes caused by root debris.
1. Patient education for learning a sanitation system mainly by brushing and by using secondary hygiene aids.
2. Detection and removal of iatrogenic factors.
3. Antimicrobial medication therapy of chronic gingivitis and periodontitis marginal superficial.
4. Reduction of inflammatory exudate from periodontal pockets by antibiotic treatment.
5. Surgical suppression of inflammatory sites other than periodontal pockets and gingival hyperplasia.
6. Suppression of the actual surgical periodontal pockets and gingival hyperplasia.
7. Occlusal balancing.
8. Prosthetic restoration.
9. Biostimulative treatment.
10. Maintenance of the results through preventive measures and further curative procedures.

Cumulative Interceptive Supportive Therapy: (CIST)

Depending on the clinical and radiographic diagnosis, a protocol of therapeutic measures, called cumulative interceptive supportive therapy, has been designed to head off the development of peri-implant lesions.[136]

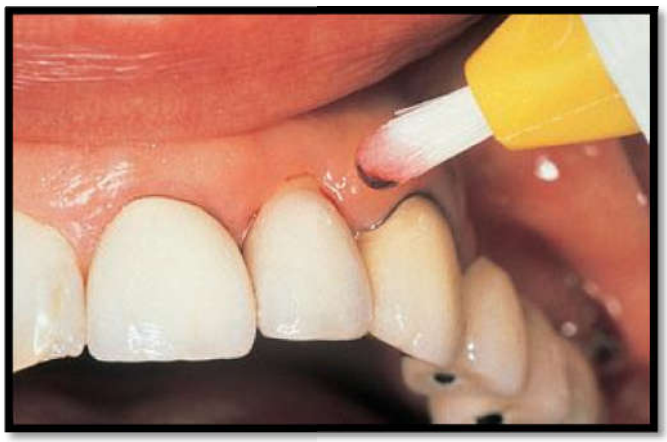


Implants with plaque and calculus deposits and surrounded by a mucosa that is BOP positive but suppuration negative and with a PPD ≤4 mm are to be subjected to mechanical debridement as described above



Antiseptic therapy; CIST protocol A+B

At implant sites which are BOP positive, exhibit an increased probing depth (4–5 mm) and may or may not demonstrate suppuration, antiseptic therapy is delivered in addition to mechanical debridement. A 0.2% solution of chlorhexidine digluconate is prescribed for daily rinsing, or a 0.2% gel of the same antiseptic is recommended for application to the affected site. Generally, 3–4 weeks of antiseptic therapy are necessary to achieve positive treatment results.[137]



Antibiotic therapy; CIST protocol A+B+C

At BOP-positive implant sites with deep pockets (PPD ≥6 mm) (suppuration may or may not be present), there are frequently also radiographic signs of bone loss. Such pockets represent an ecologic habitat which is conducive for the colonization of Gram-negative and anaerobic putative periodontal pathogens. Anti-infective treatment must include the use of antibiotics to eliminate or reduce the pathogens in this habitat. This, in turn, will allow soft tissue healing as demonstrated in a clinical study by Mombelli and Lang (1992)[23].

Prior to administering antibiotics, the mechanical (CIST A) and the antiseptic (CIST B) protocols have to be applied.

Regenerative or resective therapy; CISTprotocol A+B+C+D

It is imperative to understand that regenerative or resective therapy is not instituted until the periimplant infection is under control. Thus, before surgical intervention is planned, the previously diseased site should have become BOP negative, exhibit no suppuration, and have a reduced probing depth. Depending on the extent and severity of the local bone loss, a decision is made whether regenerative or resective measures are to be applied.

In this context it must be realized that the goal of regenerative therapy, including the use of barrier membranes, is new bone formation in the crater-like defect around the implant.

Non-Surgical:[24]

Mechanical debridement – using plastic, wooden curettes

Peri-implantitis is a frequent finding in patients with dental implants. The present study compared two non-surgical mechanical debridement methods of peri-implantitis. No group differences were found in the treatment outcomes. While plaque and bleeding scores improved, no effects on PPD were identified.

Local drug delivery/ systemic antibiotics

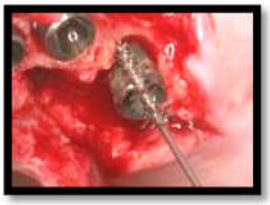
Local drug delivery- Actisite (fiber containing polymeric tetracycline HCL), Arestin, Doxycycline, Amoxycillin, Metronidazole, Cefazolin.

The systemic administration of antibiotics that specifically target gram-negative anaerobic organisms has shown an alteration in the microbial composition and a sustained clinical improvement over a 1-year period

Surgical

Open debridement

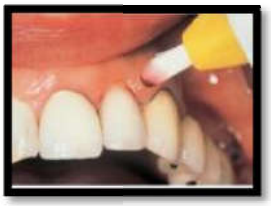
Mucoperiosteal flaps were elevated, the granulation tissue within the bone craters was curetted and the abutments were removed. The fixtures were cleaned with a detergent (delmopinol-HCl). The abutments were cleaned, autoclaved and reconnected to the fixtures. Treated sides were then subject to a careful plaque control program



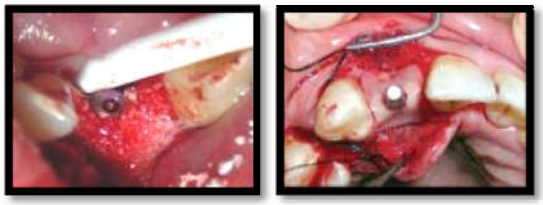
Implant Detoxification:[1]

CHX, Saline, Citric Acid, stannous fluoride, tetracycline HCL, hydrogen peroxide.

Various chemotherapeutic agents like contact with a supersaturated solution of critic acid (40% concentration; pH 1) for 30-60 seconds have been used for the preparation of the implant surfaces, as they have the highest potential for the removal of endotoxins from both the hydroxyl apatite and the titanium implant surfaces. Soft laser irradiation has also been used for the elimination of the bacteria which are associated with peri-implantitis.



Bone grafts and Barrier membranes

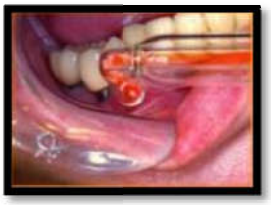


Various bone graft techniques and guided bone regeneration (GBR); even in conjunction with platelet rich plasma (PRP), have been successfully used for the regeneration of lost bones in 3 wall or circumferential defects. It is advisable to remove the prosthesis at the time of regenerative surgery; nevertheless, peri gingival regenerative therapy for one stage implants or for implants with non-retrievable prosthesis can also be done. A thorough preparation of the implant surface should be followed by an elaborate rinsing with saline solution.

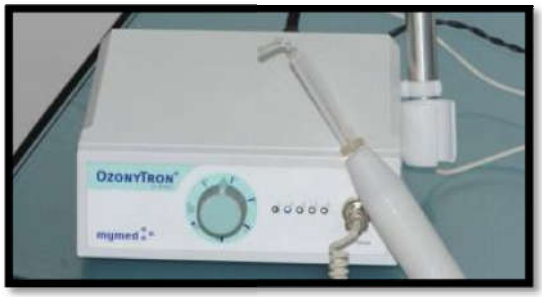
Roughening of the bone surface can be done by penetration with round burs to increase the accessibility to the osteogenic cells. The membranes which are placed should ensure the complete coverage and the isolation of the bony defect. The reflected flap should be closed primarily over the site with a mattress and interrupted sutures. The membrane shouldbe left undisturbed for 4-6 weeks.

Recent Advances

Ozone therapy (OZOTOP)



A sterile specially formed perio-tip, attached to the hand piece, was hand-guided over the whole specimen area analogous to clinical procedure. Applied with the two minimal and maximal treatment times of 6 and 24 s preselected by the manufacturer.



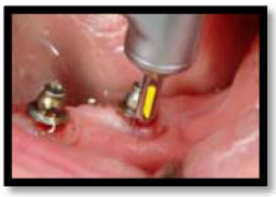
For the prevention of periimplantitis an adequate and steady plaque control regimen must be ensured.

Ozone, a powerful antimicrobial kills the microorganisms causing periimplantitis. In addition, ozone shows a positive wound healing effect due to the increase of tissue circulation. Gasiform ozone or ozonized water shows an increased healing compared to wound healing without ozone therapy

Photodynamic therapy

PDT is used to prevent periimplantitis. Laser photo biomodulation can be successfully used to improve bone quality around dental implants, allowing early wearing of prosthesis.

Laser

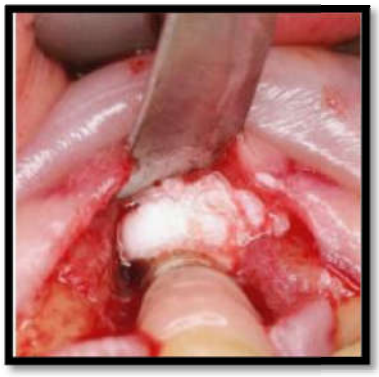


Er: YAG laser (Key Laser 3 Perio, KaVo) at an energy level of 100 mJ/ pulse and 10 Hz (12.7 J/cm2) using a cone-shaped sapphire tip. The instrument tip was used in a parallel mode using a semi-circular motion around the circumferential pocket area of the implant. Routine local anaesthesia is needed.

Nano crystal granules

Full -thickness mucoperiosteal flap was raised. The implant was curetted with area- specific titanium curettes (Langer and Langer, Rønvig). The depth and width of the osseous defects has to be measured.

The implant surfaces should be conditioned using 24% ethylenediaminetetraacetic acid gel (PrefGel, Institut Straumann) for 2 minutes and then rinsed with sterile saline. If necessary, to achieve satisfactory blood supply to the defect, the cortical bony wall was perforated with a sharp instrument. Porous titanium granules are applied to the osseous defects using a surgical stainless-steel instrument (#1 Woodson, Hu-Friedy). Any excess material was carefully removed, i.e., the defects were not overfilled. Repositioning of flap with resorbable suture



Prosthetic Treatment

The first phase involves an analysis of the fit of the prosthesis, the number and position of the implants, and an occlusal evaluation. Occlusal equilibration; improvement of the implant number and position, and changes in the prosthetic design can contribute to arrest the progression of the peri-implant tissue breakdown.

The second phase includes a surgical technique to eliminate the deep peri-implant soft tissue pockets or to regenerate the bone around the implant.

Treatment steps include (fistula Tract)-

1. cleaning and sterilization of the abutment
2. application of sealing agents between the abutments and prosthesis,
3. improvement of oral hygiene,
4. if necessary, surgical soft tissue corrections.

Explantation

If a previously Osseo integrated oral implant is clinically mobile, explantation is mandatory. The peri-implant lesion involves the entire length and circumference of the implant. Radiographically, this may be visible in a radiolucency surrounding the entire outline. Explantation may also be necessary if the peri-implant infection has advanced to a degree where it cannot be controlled by the therapeutic protocols proposed. Such a situation is clinically characterized by the presence of suppurative exudates, overt BOP, and severely increased peri-implant probing depth (usually >/= 8mm), eventually reaching perforations or vents of hollow body implants, and may be associated with pain.

CONCLUSION

Owing to the infectious nature of peri-implant mucositis and peri-implantitis, preventive procedures have to be rendered in a well-organized recall program to assure adequate supportive therapy for a lifetime. Depending on continuing diagnosis during maintenance, developing peri-implant lesions should be treated according to the CIST protocols. It is evident that preventive measures have to be reinstituted after such therapy.

Complications Related To Maintenance Therapy

The maintenance phase of implant dentistry encompasses the preventive care necessary to preserve the health and integrity of the soft and hard tissues surrounding the implant and the procedures required to sustain the function and esthetics of the restoration. This is the longest phase of involvement for the patient treated with implant dentistry, and has the greatest impact on achieving the long-term prognosis of an implant-supported restoration.

The preventive protocol used to preserve the health of peri-implant tissues consists of two phases. The first, the assessment phase, is to differentiate between presence of health and disease. In addition, etiologic factors or risk factors that can be responsible for deviations from health should be identified. The second phase is the hygiene phase, consists of training and directing the patient to control the potential etiologic factors that can result in peri-implant disease or damage to the restoration. Included in this phase is debridement, at appropriate intervals.

Etiology

Complication	Clinical presentation	Etiology
Inflammatory peri-implant disease:	Bleeding/suppuration on probing, changes in color, form, texture of peri-implant mucosa	Plaque, cement, loose restorative components
Mucositis	In addition, loss of bone as detected on X-rays compared to previous X-rays.	In addition, smoking, alcohol, systemic disease
Peri-implantitis		
Peri-implant mucosal hyperplasia	Tissue overgrowth	Overdenture, Ill-fitting prosthesis, medications
Loss of stability of the restorative components:	Peri inflammation, loose prosthesis, bleeding/suppuration from crevice, bone loss	Loose/fractured screw, fractured implant fixture, loss of cement adhesion, non-integrated implant
Non-maintainable environment	Bleeding/suppuration from crevice, change in color, form, texture of peri-implant mucosa, non-adherent tissue around implant, deep peri-implant probing depths, bone loss	Implant too close to adjacent implant/tooth, prosthetic design ridge lap, interproximal spaces closed by ceramic restoration or pink porcelain

Prevention

Managing the soft tissue relative to crevicular depth and ensuring a circumferential zone of attached keratinized tissue are important considerations for long-term implant maintenance. These goals can be accomplished with repositioned flaps (apically positioned), pedicle grafts, free soft tissue grafts or acellular dermal matrix grafts.

Prevention of the recurrence of inflammatory disease involves plaque control and where necessary, treatment to alter the environment to facilitate this goal. There are many plaque controlsimplements to aid in this process.

Type of implement	Examples
Brush (manual)	Imtec Access Brush, End Tuft brush
Powerbrush	Rota-dent, Philips Sonicare, Oral-B Braun Triumph
Floss	Teflon Tapes, Thornton Floss, Periodontal floss, Bridge & Implant cleaner, Oral-B Superfloss
Interdental brush	Tapered, Thinline, Proxytip, GUM Soft picks, Perio-aide

Management

Initially, all plaque and calculus above and below the peri-implant mucosal margin should be removed. This may be accomplished with plaque control implements, hand instruments and power instruments. Antibiotics and antimicrobials can be prescribed. Surgical procedures to treat peri-implant osseous defects include:

- Open flap debridement
- Bone grafting
- Guided tissue regeneration
- Resective osseous surgery
- Combination regenerative therapy

In sites that lack keratinized tissue soft-tissue augmentation procedures may be performed. A variety of techniques, employed for periodontal mucogingival surgery, has been successfully used for this purpose.An exposed implant surface presenting a roughened macroarchitecture and microarchitecture retains plaque and calculus and limits plaque control and debridement. Rendering the surface smooth by grinding and polishing has been suggested to reduce debris retention.

If the inflammation is related to restorative component or design problems appropriate corrections must be made.

Implant complications related to immediate implant placement

Introduction

The advantages of immediate implant placement (IIP)include improved healing without flap advancement, decreased treatment time, fewer surgical procedures, decreased cost, and decreased discomfort.[143]

The most common complications that occur with IIP include:

- Poor implant positioning
- Membrane exposure during healing
- Inadequate bands of keratinized tissue after healing
- Gingival recession
- Implant failure
- Unacceptable esthetic outcomes

Complications related to immediately loaded dental implants

Complications that are associated with the immediate implant loading protocol include:

- failure of the implant to Osseointegrate
- surgical complications
- esthetic complications
- implant malposition
- restorative complications

Failure to achieve osseointegration:

Etiology

Early healing begins with osteoclastic activity causing a reduction in implant stability. This gradual loosening of the implant continues for several weeks until osteoblastic activity deposits new bone onto the implant surface, leading to osseointegration. The length of these periods varies with many factors including implant size, shape and surface morphology, bone density, patient health, osteotomy technique and bone physiology.[145]

Despite the reduction in stability that occurs during normal healing, mobility does not usually increase to a point that would cause implant failure. There is growing evidence that carefully applied forces may accelerate osseointegration and increase bone to implant contact.[146,147]

Prevention

Wound healing studies and clinical recommendations stress the need to maintain implant stability during healing. Therefore, if initial stability is not high, implants should not be restored with an immediately loaded protocol. There are numerous methods to assess implant stability after placement, including insertion torque value, Periotest,[158] and Osstell[149]. Insertion torque is estimated from the surgical drilling unit, which cannot be assumed to be precise or accurate.

Surgical complications related to immediate loading

Patient injury and material failure

Etiology

Injury can result if a surgeon attempts to increase implant stability by placing longer or wider implants than would normally be warranted into the available bone, thus fracturing

the alveolar ridge, perforating cortical plates of bone, or damaging vital structures.

Prevention

To prevent surgical injury to vital structures or trauma to surrounding structures during placement, surgeons must adhere to conventional practices and the manufacturer'guidelines for implant placement. Preoperative diagnosis and planning should create an awareness of anatomic limitations of a particular site, regardless of the loading protocol.

Excessive implant depth (deep placement)

Etiology

Since immediate loading depends on primary stability, there is often an effort to seat implants to the point where they have sufficient torque resistance. This often results in implants that are overtightened or threaded too deeply in an effort to increase initial stability.

Prevention

Implant size and depth should not be altered from ideal in order to increase initial stability. A surgical guide should be used to determine ideal apico-coronal placement. If adequate stability cannot be achieved, then implant should be allowed to heal unloaded or unrestored.

Esthetic complications

Etiology

Few authors indicated that limited recession often occurs with immediate loading which could measure as much as 1.5 mm. Kan *et al*. suggested a correlation between the size and shape of the socket defects and final esthetic outcomes. They noted that larger initial defects produced final results with greater soft-tissue changes and poorer esthetic results.

Prevention

If significant augmentation is required and there is pre-existing bone loss, a stages or sub-merged healing protocol offers the best opportunity to maximize regenerative and esthetic outcomes before implant placement. If the risk of compromised esthetic results is acceptable, then simultaneous hard- and soft-tissue augmentation may be performed at the same time as implant placement and restoration. In cases with greater esthetic demands, a delayed implant placement protocol following augmentations should be considered.

Restorative complications

Etiology

Complications with the provisional restoration can lead to implant failure. When single teeth or small segments are immediately restored, the provisional restorations are usually contoured and adjusted to avoid direct occlusal contact. This does not mean that they are shielded from all contact, since forces can still be applied from the tongue, food, cheeks, or foreign objects. If these secondary forces exceed the theoretical sum of the primary and secondary stability of the implant.

Prevention

Bracing contacts from implant Provisionals to stable adjacent teeth can be added to limit mobility from non-occlusal forces. These wings should be constructed to reduce the ability of the tongue to produce horizontal forces on provisional restorations, but do not need to be visible on the facial surfaces. The extensions should be smooth and unobtrusive to avoid creating an irritant which could paradoxically trigger increased tongue activity.

Treatment

If immediately restored implants are found to be mobile within a short time after placement, it may be possible to save them by eliminating or minimizing forces on them. This can be done by carefully removing the provisional restoration and abutment. If the provisional cannot be removed, then bonding the mobile implant to a stable adjacent tooth may provide sufficient stability to allow the implant to Osseointegrate. Splinting is to be performed within 2-4 weeks of implant placement and loading.

CONCLUSION

Eventhough reported success rates in implant dentistry are higher than ever, so are reported complications. Implant dentistry has gained popularityin the past decade. Likewise, the number of dental implant companies has increased exponentially; they may not submit their products to meticulous research. These two factors combined may relate to the increase in complication rates.

Surgical implant complications are not uncommon and should be addressed immediately. Etiology of these complications can be iatrogenic, due to poor treatment techniques, or lack of communication between dental disciplines. Time should be spent in the implant planning stages, such as tracing preoperative radiographs, measuring models, taking CT scans and making proper surgical guides. Basic anatomy must be considered and should be reviewed by the surgeon in every case.

In addition to emphasizing the importance of self-care, it is incumbent on therapists to continuously monitor and maintain restored dental implant fixtures. Reducing biofilm loads associated with the restored implant - either through self-care, or nonsurgical or surgical intervention - is likely to reduce the risk for progressive bone loss. The first years in function are critical for implant survival, and clinicians should aim for a well-structured maintenance program in order to increase the probability of long-term implant success.

Most of the complications are preventable with adequate patient selection, treatment planning, careful execution of the case and well-structured maintenance program.

References

1. Esposito M, Hirsch J, Lekholm U, Thomsen P.Differential diagnosis and treatment strategies for biologic complications and failing oral implants: a review of the literature.Int J Oral Maxillofac Implants 1999;14(4):473-90.
2. van Steenberghe D.The use of oral implants in compromised patients.Periodontol 2000. 2003;33:9-11.
3. Froum S J. Implant complications: scope of the problem. In: Froum S J. Dental Implant Complications: Etiology, Prevention and Treatment. 2nd ed. United Kingdom: Wiley Blackwell; 2015. p. 6.
4. Khadivi V, Anderson J, Zarb GA. Cardiovascular disease and treatment outcomes with osseointegration surgery. J Prosthet Dent 1999;81:533–536.

5. Morris HF, Ochi S, Winkler S. Implant survival in patients with type 2 diabetes: Placement to 36 months. Ann Periodontol2000;5:157–165.
6. Fiorellini JP, Chen PK, Nevins M, Nevins ML. A retrospective study of dental implants in diabetic patients. Int J Periodontics Restorative Dent 2000;20:366–373.
7. Lambert, P.M., Morris, H.F. and Ochi, S. The Influence of Smoking on 3-Year Clinical Success of Osseointegrated Dental Implants. *Ann Periodontol*2000;5:79-89.
8. Scully C, Diz Dios P, Jumar N. Special care in dentistry: handbook of oral healthcare. Edinburgh: Churchill Livingstone/Elsevier, 2007.
9. Buser D, Dula K, Hirt HP, Schenk RK.Lateral ridge augmentation using autografts and barrier membranes: a clinical study with 40 partially edentulous patients.J Oral Maxillofac Surg. 1996 Apr;54(4):420-32.
10. Esposito M, Hirsch JM, Lekholm U, Thomsen P.Biological factors contributing to failures of osseointegrated oral implants. (I). Success criteria and epidemiology. Eur J Oral Sci. 1998 Feb;106(1):527-51.
11. Goodacre CJ, Kan JY, Rungcharassaeng K.Clinical complications of osseointegrated implants. J Prosthet Dent. 1999 May;81(5):537-52.
12. Goodacre CJ, Bernal G, Rungcharassaeng K, Kan JY. Clinical complications with implants and implant prostheses. J Prosthet Dent. 2003 Aug;90(2):121-32.
13. Proussaefs P, Lozada J, Kim J, Rohrer MD.Repair of the perforated sinus membrane with a resorbable collagen membrane: a human study. Int J Oral Maxillofac Implants. 2004 May-Jun;19(3):413-20.
14. Tiwana KK, Morton T, Tiwana PS. Aspiration and ingestion in dental practice: a 10-year institutional review.J Am Dent Assoc. 2004 Sep;135(9):1287-91.
15. Greenstein G, Tarnow D. The mental foramen and nerve: clinical and anatomical factors related to dental implant placement: a literature review. J Periodontol. 2006 Dec;77(12):1933-43.
16. Wallace SS, Mazor Z, Froum SJ, Cho SC, Tarnow DP. Schneiderian membrane perforation rate during sinus elevation using piezosurgery: clinical results of 100 consecutive cases. Int J Periodontics Restorative Dent. 2007 Oct;27(5):413-9.
17. Rocchietta I, Fontana F, Simion M. Clinical outcomes of vertical bone augmentation to enable dental implant placement: a systematic review. J Clin Periodontol. 2008 Sep;35(8 Suppl):203-15.
18. Testori T, Wallace SS, Del Fabbro M, Taschieri S, Trisi P, Capelli M, Weinstein RL. Repair of large sinus membrane perforations using stabilized collagen barrier membranes: surgical techniques with histologic and radiographic evidence of success. Int J Periodontics Restorative Dent. 2008 Feb;28(1):9-17.
19. Dubois L, de Lange J, Baas E, *et al*.Excessive Bleeding in the Floor of the Mouth After Endosseus Implant Placement: A Report of Two Cases.Int J Oral MaxillofacSurg2010;39 (4),412-5.
20. Rosenberg ES, Torosian JP, Slots J. Microbial differences in 2 clinically distinct types of failures of osseointegrated implants. Clin Oral Implants Res. 1991 Jul-Sep;2(3):135-44.
21. Tarnow DP, Magner AW, Fletcher P. The effect of the distance from the contact point to the crest of bone on the presence or absence of the interproximal dental papilla. J Periodontol. 1992 Dec;63(12):995-6.
22. Esposito M, Hirsch J-M, Lekholm U, Thomsen P. Failure patterns of four osseointegrated oral implant systems. J Mater Sci Mater Med 1997: 8: 843–847.
23. Mombelli A, Lang NP. The diagnosis and treatment of peri-implantitis. Periodontol 2000 1998;17:63-76.
24. Renvert S, Roos-Jansåker AM, Claffey N. Non-surgical treatment of peri-implant mucositis and peri-implantitis: a literature review. J Clin Periodontol. 2008 Sep;35(8 Suppl):305-15.
25. Kan JY, Rungcharassaeng K, Sclar A, Lozada JL. Effects of the facial osseous defect morphology on gingival dynamics after immediate tooth replacement and guided bone regeneration: 1-year results. J Oral Maxillofac Surg. 2007 Jul;65(7 Suppl 1):13-9.
26. Patodiya A. Trouble shooting in implants. Indian J Dent Sciences. 2014 Oct;6(4):92-96.
27. Misch K, Wang HL. Implant surgery complications: Etiology and treatment. Implant Dent 2008;17:159-66.
28. Greenstein G, Cavallaro J, Romanos G, Tarnow D. Clinical recommendations for avoiding and managing surgical complications associated with implant dentistry: a review. J Periodontol. 2008 Aug;79(8):1317-29.
29. Froum SJ. Dental Implant complications: Etiology, prevention, and treatment (1st ed). Blackwell publishing, 2010:110-18.
30. Pi-Anfruns J. Complications in implant dentistry. Alpha Omegan. 2014 Spring;107(1):8-12.
31. Paquette D, Brodala N, Williams N. Risk Factors for Endosseous Dental Implant Failure. Dent Clin N Am 2006;50(3):361-74.
32. Pjetursson BE, Brägger U, Lang NP, Zwahlen M. mparison of survival and complication rates of tooth-supported fixed dental prostheses (FDPs) and implant-supported FDPs and single crowns (SCs). Clin Oral Implants Res. 2007 Jun;18 Suppl 3:97-113.
33. Rangert B, Krogh PH, Langer B, Van Roekel N. Bending overload and implant fracture: A retrospective clinical analysis. Int J Oral Maxillofac Implants. 1995;10:326–34.
34. Meijer HJ, Stellingsma K, Meijndert L, Raghoebar GM.A new index for rating aesthetics of implant-supported single crowns and adjacent soft tissues--the Implant Crown Aesthetic Index. Clin Oral Implants Res. 2005 Dec;16(6):645-9.
35. Henry PJ, Laney WR, Jemt T, Harris D, Krogh PH, Polizzi G, Zarb GA, Herrmann I.Osseointegrated implants for single-tooth replacement: a prospective 5-year multicenter study. Int J Oral Maxillofac Implants. 1996 Jul-Aug;11(4):450-5.
36. Berglundh T, Persson L, Klinge B.A systematic review of the incidence of biological and technical complications in implant dentistry reported in prospective longitudinal studies of at least 5 years. J Clin Periodontol. 2002;29 Suppl 3:197-212; discussion 232-3.
37. Whitney JD. The influence of tissue oxygen and perfusion on wound healing. AACN Clin Issues Crit Care Nurs. 1990 Nov;1(3):578-84.
38. Rabkin JM, Hunt TK. Infection and oxygen. In Davis JC, Hunt TK, eds. Problem Wounds: The Role of Oxygen, New York, Elsevier, 1988: 1–16.
39. Rees TD, Mealey BL. Periodontal treatment of the medically compromised patient. In: Periodontics, medicine, surgery, and implants. St. Louis, MO: Mosby, 2004.
40. Mori H, Manabe M, Kurachi Y, Nagumo M. Osseointegration of dental implants in rabbit bone with low mineral density. J Oral Maxillofac Surg. 1997 Apr;55(4):351-61.
41. Heersche JN, Bellows CG, Ishida Y. The decrease in bone mass associated with aging and menopause. J Prosthet Dent. 1998 Jan;79(1):14-6.
42. Dao TT, Anderson JD, Zarb GA. Is osteoporosis a risk factor for osseointegration of dental implants? Int J Oral Maxillofac Implants. 1993;8(2):137-44.
43. Beiker T, Flemmig T. Implants in the medically compromised patient. Crit Rev Oral Biol Med 2003;14:305-16.
44. Dowell S, Oates TW, Robinson M. Implant success in people with type 2 diabetes mellitus with varying glycemic control: a pilot study. J Am Dent Assoc. 2007 Mar;138(3):355-61.
45. Johnson GK, Guthmiller JM. The impact of cigarette smoking on periodontal disease and treatment. Periodontol 2000. 2007;44:178-94.

46. Safkan B, Knuuttila M. Corticosteroid therapy and periodontal disease. J Clin Periodontol. 1984 Sep;11(8):515-22.
47. Heinemann DF. Osteoporosis. An overview of the National Osteoporosis Foundation clinical practice guide. Geriatrics. 2000 May;55(5):31-6.
48. Ruggiero SL, Drew SJ. Osteonecrosis of the jaws and bisphosphonate therapy. J Dent Res. 2007 Nov;86(11):1013-21.
49. Marx RE, Sawatari Y, Fortin M, Broumand V. Bisphosphonate-induced exposed bone (osteonecrosis/osteopetrosis) of the jaws: risk factors, recognition, prevention, and treatment. J Oral Maxillofac Surg. 2005 Nov;63(11):1567-75.
50. Schardt-Sacco D. Update on coagulopathies. Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 2000 Nov;90(5):559-63.
51. Grines CL, Bonow RO, Casey DE Jr, Gardner TJ, Lockhart PB, Moliterno DJ, O'Gara P, Whitlow P. Prevention of premature discontinuation of dual antiplatelet therapy in patients with coronary artery stents: a science advisory from the American Heart Association, American College of Cardiology, Society for Cardiovascular Angiography and Interventions, American College of Surgeons, and American Dental Association, with representation from the American College of Physicians. J Am Coll Cardiol. 2007 Feb 13;49(6):734-9.
52. Misch CE, Resnik RR. Contemporary Implant Dentistry. 3rd ed. St. Louis: Mosby; 2008. 438 p.
53. Vadiya S, Khalikar A, Dange SP, Desai R. Complications and their management in implantology. Int J Prosthodont Dent 2012;2(4):150-55.
54. Froum SJ. Dental Implant complications: Etiology, prevention, and treatment (1st ed). Blackwell publishing, 2010:65-72.
55. Yepes JF, Al-Sabbagh M. Use of cone-beam computed tomography in early detection of implant failure. Dent Clin North Am. 2015 Jan;59(1):41-56.
56. Annibali S, Ripari M, La Monaca G, Tonoli F, Cristalli MP. Local accidents in dental implant surgery: prevention and treatment. Int J Periodontics Restorative Dent. 2009 Jun;29(3):325-31.
57. Kalpidis CD, Setayesh RM. Hemorrhaging associated with endosseous implant placement in the anterior mandible: a review of the literature. J Periodontol. 2004 May;75(5):631-45.
58. Isaacson TJ. Sublingual hematoma formation during immediate placement of mandibular endosseous implants. J Am Dent Assoc. 2004 Feb;135(2):168-72.
59. Park SH, Wang HL. Implant reversible complications: classification and treatments. Implant Dent. 2005 Sep;14(3):211-20.
60. Misch, K.; Wang, HL. (2008). Implant surgery complications: etiology and treatment. Implant Dent 2008;17(2):159-68.
61. Kraut RA, Chahal O. Management of patients with trigeminal nerve injuries after mandibular implant placement. J Am Dent Assoc. 2002 Oct;133(10):1351-4.
62. Worthington P. Injury to the inferior alveolar nerve during implant placement: a formula for protection of the patient and clinician. Int J Oral Maxillofac Implants. 2004 Sep-Oct;19(5):731-4.
63. Zijderveld SA, van den Bergh JP, Schulten EA, ten Bruggenkate CM. Anatomical and surgical findings and complications in 100 consecutive maxillary sinus floor elevation procedures. J Oral Maxillofac Surg. 2008 Jul;66(7):1426-38.
64. Ward BB, Terrell JE, Collins JK. Methicillin-resistant Staphylococcus aureus sinusitis associated with sinus lift bone grafting and dental implants: a case report. J Oral Maxillofac Surg. 2008 Feb;66(2):231-4.
65. Chanavaz M. Maxillary sinus: anatomy, physiology, surgery, and bone grafting related to implantology--eleven years of surgical experience (1979-1990). J Oral Implantol. 1990;16(3):199-209.
66. van den Bergh JP, ten Bruggenkate CM, Disch FJ, Tuinzing DB. Anatomical aspects of sinus floor elevations. Clin Oral Implants Res. 2000 Jun;11(3):256-65.
67. Ardekian L, Oved-Peleg E, Mactei EE, Peled M. The clinical significance of sinus membrane perforation during augmentation of the maxillary sinus. J Oral Maxillofac Surg. 2006 Feb;64(2):277-82.
68. Reiser GM, Rabinovitz Z, Bruno J, Damoulis PD, Griffin TJ. Evaluation of maxillary sinus membrane response following elevation with the crestal osteotome technique in human cadavers. Int J Oral Maxillofac Implants. 2001 Nov-Dec;16(6):833-40.
69. Galindo P, Sánchez-Fernández E, Avila G, Cutando A, Fernandez JE. Migration of implants into the maxillary sinus: two clinical cases. Int J Oral Maxillofac Implants. 2005 Mar-Apr;20(2):291-5.
70. Hunter WL,Bradrick JP, Houser SM, Patel JB, Sawady J. Maxillary sinusitis resulting from ostium plugging by dislodged bone graft: case report. J Oral Maxillofac Surg. 2009 Jul;67(7):1495-8.
71. Peleg M, Garg AK, Mazor Z. Predictability of simultaneous implant placement in the severely atrophic posterior maxilla: A 9-year longitudinal experience study of 2132 implants placed into 731 human sinus grafts. Int J Oral Maxillofac Implants. 2006 Jan-Feb;21(1):94-102.
72. Elian N, Wallace S, Cho SC, Jalbout ZN, Froum S. Distribution of the maxillary artery as it relates to sinus floor augmentation. Int J Oral Maxillofac Implants. 2005 Sep-Oct;20(5):784-7.
73. Ueda M, Kaneda T. Maxillary sinusitis caused by dental implants: report of two cases. J Oral Maxillofac Surg. 1992 Mar;50(3):285-7.
74. Steiner M, Ramp WK. Short-term storage of freshly harvested bone. J Oral Maxillofac Surg. 1988 Oct;46(10):868-71.
75. Field EA. The use of powdered gloves in dental practice: a cause for concern? J Dent. 1997 May-Jul;25(3-4):209-14.
76. Hooe W, Steinberg B. Management of contaminated bone grafts: an experimental in vitro study. Oral Surg Oral Med Oral Pathol Oral RadiolEndod.1996 Jul;82(1):34-7.
77. Misch CE *et al*. Steroids and the Reduction of Pain, Edema and Dysfunction in Implant Dentistry. Int J Oral Implantol1989;6(1):27-31.
78. Carlson ER, Monteleone K. An analysis of inadvertent perforations of mucosa and skin concurrent with mandibular reconstruction. J Oral Maxillofac Surg. 2004 Sep;62(9):1103-7.
79. Lindeboom JA, van den Akker HP. A prospective placebo-controlled double-blind trial of antibiotic prophylaxis in intraoral bone grafting procedures: a pilot study. Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 2003 Dec;96(6):669-72.
80. Scharf DR, Tarnow DP. Success rates of osseointegration for implants placed under sterile versus clean conditions. J Periodontol. 1993 Oct;64(10):954-6.
81. Young MP, Korachi M, Carter DH, Worthington HV, McCord JF, Drucker DB. The effects of an immediately pre-surgical chlorhexidine oral rinse on the bacterial contaminants of bone debris collected during dental implant surgery. Clin Oral Implants Res. 2002 Feb;13(1):20-9.
82. Branemark PI, Grondahl K, Worthington P. Osseointegration and autogenous onlay bone grafts: reconstruction of the edentulous atrophic maxilla. Chicago, IL: Quintessence, 2001:112-34.
83. Burchardt H. The biology of bone graft repair. Clin OrthopRelat Res. 1983 Apr;(174):28-42.
84. Smith JD, Abramson M. Membranous vs endochondrial bone autografts. Arch Otolaryngol. 1974 Mar;99(3):203-5.
85. Ozaki W, Buchman SR. Volume maintenance of onlay bone grafts in the craniofacial skeleton: micro-architecture versus

embryologic origin. PlastReconstr Surg. 1998 Aug;102(2):291-9.
86. Misch CM, Misch CE, Resnik RR, Ismail YH. Reconstruction of maxillary alveolar defects with mandibular symphysis grafts for dental implants: a preliminary procedural report. Int J Oral Maxillofac Implants. 1992 Fall;7(3):360-6.
87. Nystrom E, Ahlqvist J, Kahnberg KE, Rosenquist JB. Autogenous onlay bone grafts fixed with screw implants for the treatment of severely resorbed maxillae. Radiographic evaluation of preoperative bone dimensions, postoperative bone loss, and changes in soft-tissue profile. Int J Oral Maxillofac Surg. 1996 Oct;25(5):351-9.
88. de Carvalho PS, Vasconcellos LW, Pi J. Influence of bed preparation on the incorporation of autogenous bone grafts: a study in dogs. Int J Oral Maxillofac Implants. 2000 Jul-Aug;15(4):565-70.
89. Verardi S, Simion M. Management of the exposure of e-PTFE membranes in guided bone regeneration. PractProcedAesthet Dent. 2007 Mar;19(2):111-7.
90. 90.Buser D, Dula K, Belser UC, Hirt HP, Berthold H. Localized ridge augmentation using guided bone regeneration. II. Surgical procedure in the mandible. Int J Periodontics Restorative Dent. 1995 Feb;15(1):10-29.
91. Jovanovic SA, Spiekermann H, Richter EJ. Bone regeneration around titanium dental implants in dehisced defect sites: a clinical study. Int J Oral Maxillofac Implants. 1992 Summer;7(2):233-45.
92. Simion M, Trisi P, Piattelli A. Vertical ridge augmentation using a membrane technique associated with osseointegrated implants. Int J Periodontics Restorative Dent. 1994 Dec;14(6):496-511.
93. Rangert B, Jemt T, Jorneus L. Forces and moments on Branemark implants. Int J Oral Maxillofac Implants. 1989 Fall;4(3):241-7.
94. Eckert SE, Meraw SJ, Cal E, Ow RK. Analysis of incidence and associated factors with fractured implants: a retrospective study. Int J Oral Maxillofac Implants. 2000 Sep-Oct;15(5):662-7.
95. Adell R, Lekholm U, Rockler B, Branemark PI. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. Int J Oral Surg. 1981 Dec;10(6):387-416.
96. Kong L, Hu K, Li D, Song Y, Yang J, Wu Z, Liu B. Evaluation of the cylinder implant thread height and width: a 3-dimensional finite element analysis. Int J Oral Maxillofac Implants. 2008 Jan-Feb;23(1):65-74.
97. Craig R. Craig's restorative dental materials. St Louis, MO: Mosby-Elsevier, 2006.
98. Jemt T, Carlsson L, Boss A, Jorneus L. In vivo load measurements on osseointegrated implants supporting fixed or removable prostheses: a comparative pilot study. Int J Oral Maxillofac Implants 1991; 10:413-17.
99. Mericske-Stern R, Venetz E, Fahrländer F, Bürgin W. In vivo force measurements on maxillary implants supporting a fixed prosthesis or an overdenture: a pilot study. J Prosthet Dent. 2000 Nov;84(5):535-47.
100. Weinberg L. Atlas of implant and tooth supported prosthodontics. Chicago, IL: Quintessence, 2003.
101. Wiskott HW, Belser UC. A rationale for a simplified occlusal design in restorative dentistry: historical review and clinical guidelines. J Prosthet Dent. 1995 Feb;73(2):169-83.
102. Eriksson RA, Adell R. Temperatures during drilling for the placement of implants using the osseointegration technique. J Oral Maxillofac Surg. 1986 Jan;44(1):4-7.
103. Piattelli A, Piattelli M, Mangano C, Scarano A. A histologic evaluation of eight cases of failed dental implants: is bone overheating the most probable cause? Biomaterials. 1998 Apr-May;19(7-9):683-90.
104. Lambert PM, Morris HF, Ochi S. Positive effect of surgical experience with implants on second-stage implant survival. J Oral Maxillofac Surg. 1997 Dec;55(12 Suppl 5):12-8.
105. Jemt T, Hager P. Early complete failures of fixed implant-supported prostheses in the edentulous maxilla: a 3-year analysis of 17 consecutive cluster failure patients. Clin Implant Dent Relat Res. 2006;8(2):77-86.
106. Berglundh T, Lindhe J. Dimension of the periimplant mucosa. Biological width revisited. J Clin Periodontol. 1996 Oct;23(10):971-3.
107. Kan JY, Rungcharassaeng K, Umezu K, Kois JC. Dimensions of peri-implant mucosa: an evaluation of maxillary anterior single implants in humans. J Periodontol. 2003 Apr;74(4):557-62.
108. Buser D, Martin W, Belser UC. Optimizing esthetics for implant restorations in the anterior maxilla: anatomic and surgical considerations. Int J Oral Maxillofac Implants. 2004;19 Suppl:43-61.
109. Esposito M, Ekestubbe A, Grondahl K. Radiological evaluation of marginal bone loss at tooth surfaces facing single Brånemark implants. Clin Oral Implants Res. 1993 Sep;4(3):151-7.
110. Parel SM, Sullivan DY. Esthetics and osseointegration. University of Texas Health Science, 1989.
111. Small PN, Tarnow DP. Gingival recession around implants: a 1-year longitudinal prospective study. Int J Oral Maxillofac Implants. 2000 Jul-Aug;15(4):527-32.
112. Chen ST, Darby IB, Reynolds EC. A prospective clinical study of non-submerged immediate implants: clinical outcomes and esthetic results. Clin Oral Implants Res. 2007 Oct;18(5):552-62.
113. Lindeboom JA, Tjiook Y, Kroon FH. Immediate placement of implants in periapical infected sites: a prospective randomized study in 50 patients. Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 2006 Jun;101(6):705-10.
114. Evans CD, Chen ST. Esthetic outcomes of immediate implant placements. Clin Oral Implants Res. 2008 Jan;19(1):73-80.
115. Kassolis JD, Baer ML, Reynolds MA.The segmental osteotomy in the management of malposed implants: a case report and literature review. J Periodontol. 2003 Apr;74(4):529-36.
116. Dawson T, Chen ST. The SAC classification in implant dentistry. Berlin: Quintessence, 2007:11-20.
117. Martin WC, Morton D, Byser D. Diagnostic factors for esthetic risk assessment. In: Buser D, Belser U, Wismeijer D, eds. ITI treatment guide. Vol. 1: Implant therapy in the esthetic zone - single-tooth replacements. Berlin: Quintessence, 2007:11-20.
118. Spray JR, Black CG, Morris HF, Ochi S. The influence of bone thickness on facial marginal bone response: stage 1 placement through stage 2 uncovering. Ann Periodontol. 2000 Dec;5(1):119-28.
119. Chen ST, Buser D. Clinical and esthetic outcomes of implants placed in postextraction sites. Int J Oral Maxillofac Implants. 2009;24 Suppl:186-217.
120. Buser D, Halbritter S, Hart C, Bornstein MM, Grütter L, Chappuis V, Belser UC. Early implant placement with simultaneous guided bone regeneration following single-tooth extraction in the esthetic zone: 12-month results of a prospective study with 20 consecutive patients. J Periodontol. 2009 Jan;80(1):152-62.
121. von Arx T, Buser D. Horizontal ridge augmentation using autogenous block grafts and the guided bone regeneration technique with collagen membranes: a clinical study with 42 patients. Clin Oral Implants Res. 2006 Aug;17(4):359-66.
122. Boyne PJ, Herford AS. An algorithm for reconstruction of alveolar defects before implant placement. In: Boyne PJ, guest ed. Oral and maxillofacial Surgery Clinics of North America. Alveolar ridge reconstruction/guided bone regeneration and bone grafting. Philadelhia, PA: WA Sanders, 2001;13(3):533-541.
123. Lazzara, RJ. Esthetic and Restorative Benefits of Non-Axially Loaded Implants. Implant Dentistry: Winter 1995;4(4):283.

124. Williamson RT, Robinson FG. Retrieval technique for fractured implant screws. J Prosthet Dent. 2001 Nov;86(5):549-50.
125. Balshi TJ. Preventing and resolving complications with osseointegrated implants. Dent Clin North Am. 1989 Oct;33(4):821-68.
126. Jemt T, Book K, Linden B, Urde G. Failures and complications in 92 consecutively inserted overdentures supported by Branemark implants in severely resorbed edentulous maxillae: a study from prosthetic treatment to first annual check-up. Int J Oral Maxillofac Implants. 1992 Summer;7(2):162-7.
127. Naert I, De Clercq M, Theuniers G, Schepers E. Overdentures supported by osseointegrated fixtures for the edentulous mandible: a 2.5-year report. Int J Oral Maxillofac Implants. 1988 Fall;3(3):191-6.
128. Pauletto N, Lahiffe BJ, Walton JN. Complications associated with excess cement around crowns on osseointegrated implants: a clinical report. Int J Oral Maxillofac Implants. 1999 Nov-Dec;14(6):865-8.
129. Agar JR, Cameron SM, Hughbanks JC, Parker MH. Cement removal from restorations luted to titanium abutments with simulated subgingival margins. J Prosthet Dent. 1997 Jul;78(1):43-7.
130. Mombelli A. Microbiology and antimicrobial therapy of peri-implantitis. Periodontol 2000. 2002;28:177-89.
131. 131.Smeets R, Henningsen A, Jung O, Heiland M, Hammächer C, Stein JM. Definition, etiology, prevention and treatment of peri-implantitis--a review. Head Face Med. 2014 Sep 3;10:34.
132. Luterbacher S, Mayfield L, Brägger U, Lang NP. Diagnostic characteristics of clinical and microbiological tests for monitoring periodontal and peri-implant mucosal tissue conditions during supportive periodontal therapy (SPT). Clin Oral Implants Res. 2000 Dec;11(6):521-9.
133. Weber HP, Buser D, Fiorellini JP, Williams RC. Radiographic evaluation of crestal bone levels adjacent to nonsubmerged titanium implants. Clin Oral Implants Res. 1992 Dec;3(4):181-8.
134. Loe h, Theilade e, Jensen sb. Experimental gingivitis in man. J Periodontol. 1965 May-Jun;36:177-87.
135. Lang NP, Kiel RA, Anderhalden K. Clinical and microbiological effects of subgingival restorations with overhanging or clinically perfect margins. *J Clin Periodontol*. 1983 Nov;10(6):563-78.
136. Lang NP, Berglundh T, Heitz-Mayfield LJ, Pjetursson BE, Salvi GE, Sanz M. Consensus statements and recommended clinical procedures regarding implant survival and complications. *Int J Oral Maxillofac Implants*. 2004;19 Suppl:150-4.
137. Lang NP, Brecx M. Chlorhexidine digluconate–an agent for chemical plaque control and prevention of gingival inflammation. J Periodont Res 1986;21:(suppl 18):74-89.
138. Roos-Jansaker AM, Renvert S, EgelbergJ.Treatment of peri-implant infections: a literature review. *J Clin Periodontol.* 2003 Jun;30(6):467-85.
139. Schou S, Berglundh T, Lang NP. Surgical treatment of peri-implantitis. *Int J Oral Maxillofac Implants*. 2004;19 Suppl: 140-9.
140. Ashnagar S, Nowzari H, Nokhbatolfoghahaei H, Zadeh BY, Chiniforush N, Zadeh NC. Laser Treatment of Peri-Implantitis: A Literature Review. J Lasers Med Sci. 2014 Autumn; 5(4): 153–162.
141. Yamamoto A, Tanabe T. Treatment of peri-implantitis around TiUnite-surface implants using Er:YAG laser microexplosions. Int J Periodontics Restorative Dent. 2013 Jan-Feb;33(1):21-30.
142. Meffert RM. Maintenance and treatment of the ailing and failing implant. J Indiana Dent Assoc. 1994 Fall;73(3):22-4.
143. Gelb DA. Immediate implant surgery: three-year retrospective evaluation of 50 consecutive cases. Int J Oral Maxillofac Implants. 1993;8(4):388-99.
144. Mensdorff-Pouilly N, Haas R, Mailath G, Watzek G. The Immediate Implant: A Retrospective Study Comparing the Different Types of Immediate Implantation. Int J Oral Maxillofac Implants 1994;9:571-8.
145. Oates TW, Valderrama P, Bischof M, Nedir R, Jones A, Simpson J, Toutenburg H, Cochran DL. Enhanced implant stability with a chemically modified SLA surface: a randomized pilot study. I*nt J Oral Maxillofac Implants*. 2007 Sep-Oct;22(5):755-60.
146. Lindh T, Gunne J, Tillberg A, Molin M. A meta-analysis of implants in partial edentulism. Clin Oral Implants Res. 1998 Apr;9(2):80-90.
147. Attard NJ, Zarb GA. Immediate and early implant loading protocols: a literature review of clinical studies. *J Prosthet Dent*. 2005 Sep;94(3):242-58.
148. Cranin AN, DeGrado J, Kaufman M, Baraoidan M, DiGregorio R, Batgitis G, Lee Z. Evaluation of the Periotest as a diagnostic tool for dental implants*. J Oral Implantol.* 1998;24(3):139-46.
149. Sennerby L, Meredith N. Implant stability measurements using resonance frequency analysis: biological and biomechanical aspects and clinical implications. Periodontol 2000. 2008;47:51-66.
150. Kan JY, Rungcharassaeng K, Lozada J. Immediate placement and provisionalization of maxillary anterior single implants: 1-year prospective study. Int J Oral Maxillofac Implants. 2003 Jan-Feb;18(1):31-9.
151. Baxter C. Malpractice survey: a survey of 242 dental negligence cases. www.experts.com,2008.
152. Albrektsson T. Is surgical skill more important for clinical success than changes in implant hardware? Clin Implant Dent Relat Res. 2001;3(4):174-5.
153. Regenbogen SE, Greenberg CC, Studdert DM, Lipsitz SR, Zinner MJ, Gawande AA. Patterns of technical error among surgical malpractice claims: an analysis of strategies to prevent injury to surgical patients. Ann Surg. 2007 Nov;246(5):705-11.
154. Federal Judicial Center. Reference manual on scientific evidence, 2nd edn, 2000.
155. Sfikas P. A duty to disclose. *J Am Dent Assoc* 2003; 134:1329-33.

How to cite this article:

Kavyashree PM and Nagarathna DV (2021) 'Oral Implant Complications and Its Management', *International Journal of Current Advanced Research,* 10(02), pp. 23778-23799. DOI: http://dx.doi.org/10.24327/ijcar.2021.23799.4713

****