

Medical Contraindications to Dental Implant therapy: A Review

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I. Introduction

Systemic conditions & habits influence dental implant survival to varying degrees. For a successful implant therapy, it is imperative to select patients who do not possess any local or systemic contraindications to implant therapy. 3 major causes for implant failure are; i) impaired host healing, ii) disruption of a weak bone-to-implant interface after abutment connection & iii) infection¹. The intrinsic adeptness of a patient to retain an implant relies on patient's health status. Despite, the functional & emotional toll edentulism engenders on an individual, implant therapy remains elective treatment.

Medical contraindications of dental implant therapy are of two types:

1. *Absolute contraindications*

2. *Relative contraindications*: The mere presence of a disease, however, does not necessarily preclude implant therapy or affect significantly long term outcomes. Certain disorders when controlled, or other situations allow implant survival rates that match with healthy patients.

Absolute contraindications ²	Relative contraindications ³
Recent Myocardial Infarction	Adolescence
Cerebrovascular accident	Aging
Valvular prosthesis surgery	Osteoporosis
Immunosuppression	Smoking
Bleeding disorders	Diabetes
Active treatment of malignancy	Positive interleukin-1 genotype
Drug abuse	Human Immunodeficiency virus positive
Psychiatric illness	Cardiovascular disease
Intravenous use of bisphosphonate	Hypothyroidism

(I) ABSOLUTE CONTRAINDICATIONS

1) *Recent myocardial infarction / cerebrovascular accident*

Ischemia to the heart or the brain generates necrosis and functional deficits for an ample amount of time. With intervention and an ameliorating period of roughly 6–12 months after preliminary care, patient stability occurs. Meanwhile, and for 3–6 months after initial stability, it is necessary to avoid any stress, including surgical, that could trigger post-ischemic complications. Due to the high risk of complications following a myocardial infarction or cerebrovascular accident, the dental provider must wait until preliminary stabilization. The patient may pursue elective dental care only if at least 6 months have passed since the ischemic incident and the patient obtains medical clearance. The health care professional must be aware of any anticoagulant or thrombolytic therapy administered and understand that the desire for oral implants does not necessarily justify interruption of a therapeutic international normalized ratio (INR)².

2) **Valvular Prosthesis placement**

Prosthetic valves restore function in patients with progressive congestive heart failure, systemic emboli, or endocarditis, but they are prone to microbial infections⁴. Three forms of prosthetic valve exist: bioprostheses, mechanical valves, and homografts or autografts. Except autograft all forms fall subject to endocarditis, as well as regurgitation, stenosis, and degeneration. The prevalence of prosthetic valve endocarditis hovers around 1% to 3%, and the greatest risk occurs within the first 3 months⁵. By 6 months, the prosthetic valve endocarditis rate drops to 0.4%. With prosthetic valve replacement, stability occurs at least 6 months to 1 year after cardiac surgery. Invasive periodontal procedures should be absolutely avoided in order to prevent bacteremia and possible subsequent valve loss. Depending on the type of valve used (mechanical or bioprosthesis [porcine]), the patient requires different drug regimens (anticoagulants or plasma volume elevators, respectively). Any dental treatment must take such medications into consideration².

3) **Bleeding**

If an appropriate hemostasis cannot occur, elective surgery must not take place. A loss of 500 mL of blood requires volume replacement⁶. Uncontrolled hemorrhage stems from a multitude of conditions, including platelet and clotting factor disorders, but often originates from drug therapy. Patients taking oral anticoagulants (e.g., aspirin, warfarin, clopidogrel, among others) for cardiovascular maladies must receive careful supervision of bleeding time and INR. Little risk of significant bleeding following dental surgical procedures in patients with a prothrombin time of 1.5–2 times is normal⁷. Fazio and Fang⁸ suggested an INR of 2.2 or lower for surgical procedures. Lack of platelets due to infection, idiopathic thrombocytopenia purpura, radiation therapy, myelosuppression, and leukemia may lead to bleeding disorders during or after surgery as well. The normal platelet count has a wide range, between 100,000 and 500,000/mm³. Mild thrombocytopenia, or 50,000–100,000/mm³, may produce abnormal postoperative bleeding. Levels below 50,000/mm³ lead to major postsurgical bleeding; spontaneous bleeding of mucous membranes occurs below 20,000 cells/mm³.⁹ Such patients often require transfusion before surgery. For most dental patients, the hematocrit is crucial to outpatient care only when values drop to roughly 60% of low normal range².

4) **Immunosuppression**

The aptness to revive an adequate immune response is crucial to wound healing. Oral surgery is typically contraindicated when the total white blood count falls below 1500–3000 cells/mm³, as the patient becomes susceptible to infection and compromised to repair or regeneration¹⁰. Despite a total white blood count within normal range (5000–10,000 cells/mm³), a grossly abnormal absolute neutrophil count, which includes polymorphonuclear neutrophils and bands, renders the patient unable to combat an immediate antigenic challenge. A normal absolute neutrophil count level lies between 3500 and 7000 cells/mm³. A person with levels between 1000 and 2000 cells/mm³ requires broadspectrum antibiotic coverage¹¹. Those with less than 1000 cells/mm³ require immediate medical consultation and cannot receive dental implantation. In order to sustain health and homeostasis, the normal CD4 T-cell count measures above 600 cells/mm³; values below 500 cells/mm³ are considered immunosuppressed.¹¹ The lower the CD4:CD8 ratio, which normally approximates 2.0, the more immunocompromised is the patient.¹¹

5) **Active Cancer Therapy**

While needed to destroy rapidly dividing malignant cells, both ionizing radiation and chemotherapy disrupt host defense mechanisms and hematopoiesis. Because the patient on such regimens cannot mount an appropriate response to wounding from surgery, implantation is prohibited. The total dose of ionizing radiation for cancer treatment ranges from 50 to 80 Gy.² A very limited number of investigations has been conducted on chemotherapeutic effects on implant survival. Case reports on subjects with dental implants who then undergo cancer chemotherapy show conflicting, though mostly adverse, results.¹²⁻¹⁴

6) **Psychiatric Disorders**

In a patient who will fail to comprehend and anticipate dental treatment logically, it is best not to place implants. Often, mental illnesses are undiagnosed or unreported. Blomberg¹⁵ identified several conditions as incongruous with implant placement. These include psychotic disorders (e.g., schizophrenia), severe character disorders (hysteroid and borderline personalities), dysmorphophobia, cerebral lesions, and presenile dementia, as well as alcohol and drug abuse. There exist no biological reasons for patients with most of the above disorders to lose implants (at least none that have been determined), but various case reports blame removal of osseointegrated fixtures on psychiatric factors.¹⁶ A patient who abuses alcohol or drugs may suffer from an inability not only to recognize or accept realistic treatment outcomes but also to heal.²

7) **Intravenous Bisphosphonate Treatment**

Recently, a number of clinicians published links between intravenous (IV) bisphosphonate use to osteonecrosis of the jaws. Bisphosphonates inhibit bone resorption, and, thus, treat osteoporosis, hypercalcemia of malignancy, and Paget's disease. They tend to dwell in the bone for long periods of time. There exist both oral and IV routes of administration for bisphosphonates.² With respect to oral bisphosphonate use, 1 case report links it to osteonecrosis of the jaw, and the American Dental Association does not suggest modification of treatment plans for most people on such drugs.¹⁷ If other risk factors (i.e., prolonged use, concomitant estrogen or glucocorticoid therapy, older age) exist, however, and the patient requires dental surgery that involves the periosteum or bone, he or she should be informed of potential complications.¹⁸ Surgery is not contraindicated with use of oral bisphosphonate, but the dental provider must exercise caution. In the case of IV bisphosphonates, on the other hand, elective surgery is not allowed.²

(II) RELATIVE CONTRAINDICATIONS

1) **Adolescence**

According to World Health Organization an adolescent is between the age of 10 to 19.¹⁹ In a growing individual, an implant may cause sequelae similar to that of an ankylosed tooth; this has been shown in a pig model.²⁰ Such teeth submerge during growth since they are unable to erupt to compensate for vertical growth of the alveolar process. Thus, a major concern in placing implants in adolescents is the possibility of relocation or displacement with time with respect to natural dentition. In addition, the placement of a rigid, implant-borne prosthesis may inhibit growth activity.³ Research suggests that the maxilla changes in all 3 planes of space; it is difficult to predict the behavior of implants in this dynamic situation.²¹ To prevent complications and enhance predictability, it is best to wait until cessation of growth before implant placement in a young person.

2) **Aging**

The elder individuals tend to have greater prevalence of local (xerostomia, ridge resorption) as well as systemic diseases (osteoporosis, diabetes), and more difficulty with muscular adaptation to prostheses. With age, alterations in mineral composition, collagen, bone morphogenetic protein content, and bone conformation take place. Delayed fracture healing and less tissue regeneration occur too.²² The majority of clinical investigations, however, do not correlate age with implant failure after adjustment for other factors. Smith²³ *et al.*, failed to associate age with implant failure in a retrospective study of 313 implants in 104 patients up to 88 years of age. Failure related to aging alone seems to happen only rarely, and various studies reflect this. Nevertheless, investigations observe comparable success rates between different age groups, and so aging, by itself, does not affect survival.^{24,25}

3) **Osteoporosis**

The prevalence of osteoporosis increases with age; epidemiological studies indicate that bone loss arises after the fourth or fifth decade in both men and women.²⁶ Postmenopausal women are at particular risk for bone loss. The major concern about osteoporosis with respect to implant placement is the possibility that the disease modifies bone quality, formation, or healing to an extent that osseointegration is unlikely or impossible. Osteoporotic bone in human histological studies exhibits reduced mechanical strength, alterations in trabecular architecture, decreased mineral content, increased crystallinity, and higher carbonate-to-phosphate ratios.²⁷ The exact clinical significance of these properties remains unclear. An examination of dental clinical studies on this osteoporosis reveals little effect of this disease on implant success, at least in the lower jaw. A retrospective analysis by August²⁸ *et al* determined that mandibular implant failure rates did not vary between premenopausal and postmenopausal women; in contrast, postmenopausal subjects had significantly more maxillary implants fail than their premenopausal counterparts. Friberg²⁹ *et al* placed 70 implants in the jaws of 14 patients with osteoporosis. This group achieved 97% success in both maxilla and mandible after 3-year follow-up. Taking the above mentioned reports into consideration, osteoporosis alone does not affect implant success.

4) **Smoking**

Cigarette byproducts such as nicotine, carbon monoxide, and hydrogen cyanide incite toxic biological responses.³⁰ With respect to clinical dentistry, smokers experience a number of problems. They have significantly reduced blood fill in extraction sockets, especially in the maxilla, which results in localized alveolar osteitis.³¹ Patients who smoke have less successful surgical results and may experience refractory periodontitis.^{32,33} During maintenance, pocket depths increase, and clinical attachment levels tend to deteriorate.³⁴ Several investigations implicate tobacco use in implant failure as well. More recent studies examine the effect of smoking in patients with treated periodontal disease. The results remain mixed. On the whole, smoking appears to reduce implant success in the maxilla, but smoking cessation prior to implant

rehabilitation appears to improve results. The use of surface-modified fixtures may decrease the risk of failure in smokers, though evidence is preliminary.³

5) **Diabetes**

Diabetes is the most common cause of blindness and non-trauma lower extremity amputation. Moreover, it is a major cause of end-stage renal disease. These pathological changes occur in response to insulin deficiency and/or dysfunction. In normal homeostasis, insulin stimulates directly osteoblastic matrix synthesis. It also induces hepatic output of insulinlike growth factor-1, which increases the number and up-regulates the activity of differentiated osteoblasts. Eventually, if glucose concentration remains high, protein interactions with glucose metabolites result in irreversible advanced glycosylation end products, which accumulate over time on macromolecules (e.g., proteins, lipids), and injure various cellular processes involved in tissue healing and bone formation. For instance, advanced glycosylation end products lower the quality and quantity of extracellular matrix components such as collagen, laminin, vitronectin, and osteocalcin. It is obvious then that diabetes may generate a less-than-ideal environment for implant placement.³

What is an acceptable level of control? Physicians measure hemoglobin A1C (HbA1C) levels every 3 months to estimate blood sugar level over preceding 2-3 months.³⁵ The American Diabetes Association recommends an A1C level of 7.0% in patients with type II diabetes; goals, however, must be individualized.³⁶ In those with frequent or severe hypoglycemic episodes, a less stringent HbA1C level is acceptable. On the other hand, the stricter the control (i.e., 6.0%), the lesser the risk for organ level complications. Certain subsets of patients (e.g., type I diabetes sufferers) must chance hypoglycemia to attain microvascular and neurological stability.

Although used as the gold standard to assess glycemic control, HbA1C evaluates past levels. What appraises current management? Preprandial and peak postprandial capillary plasma glucose levels help to check current progress (Table 1). In combination with blood pressure and lipid supervision, these values allow the patient as well as health care professional to evaluate control and disease prognosis. If glycemic control is adequate, according to the HbA1c level, diabetes does not compromise implant success.³

Glycemic control	
A1C	<7.0%
Preprandial plasma glucose	90-130mg/dL
Peak postprandial plasma glucose	<180mg/dL
Blood pressure	<130/80
Lipids low density lipoprotein	<100 mg/dL
Triglycerides	<150mg/dL
High density lipoprotein	>40mg/dL

6) **Interleukin-1 genotype**

The interleukin (IL)-1 composite genotype appeared to associate with periodontal disease in the literature of the last century.³⁷⁻⁴⁰ First analyzed by Kornman³⁸ *et al.*, who later developed the Periodontal Susceptibility Test for IL-1 genotype detection (Interleukin Genetics, Waltham, MA), in a Northern European population, IL-1 composite positivity consists of at least 1 copy of allele 2 at each of the 2 specific polymorphisms of IL-1 gene clusters on chromosome 2: allele 2 at the IL-1A (889) locus plus allele 2 at the IL-1B (3954) locus. As another, easier-assayed polymorphism at IL-1A (4845) is in 99% linkage disequilibrium with IL-1A (889), it is used to test for the variant IL-1 allele A homozygous allele 2 at the IL-1B (889) position appeared to increase the IL-1B response to inflammation.⁴¹ In nonsmokers, it appears to create a high odds ratio (OR) for moderate-to-severe periodontitis as well as tooth loss.

Could genotype positivity affect implant survival? Most current reports show no influence.⁴²⁻⁴⁴ Interestingly, a Japanese group coupled a nontraditional IL-1 polymorphism (at IL-1B-511) to early marginal bone loss.⁴⁵ At this point, there is not enough evidence to verify that IL-1 composite genotype positivity generates implant loss.

7) **Human Immunodeficiency Virus**

Very few studies on HIV and implantology exist. Harrison⁴⁶ *et al.* conducted a prospective, blind, controlled study on wound infection and orthopedic implants in HIV-positive and negative subjects. With no preoperative contamination, the incidence of wound infection failed to differ between patient groups. In regard to intraoral implants, Fielding *et al.*⁴⁷ presented successful osseointegration and function after 4 years in HIV-positive patients with CD4+ counts of >200 cells/mm³ at the UCLA Symposium on Implants in the Partially Edentulous Patient. A case report demonstrated 18-month functional success of an immediate dental implant in

an HIV-positive patient with CD4+ counts less than 200 cells/mm³; a regimen of amoxicillin was given postoperatively.⁴⁸ Without the presence of severe immunosuppression or bleeding disorders, HIV status does not lower implant success. Use antibiotics as needed.³

8) **Cardiovascular disease**

Five forms of cardiovascular disease (hypertension, atherosclerosis, vascular stenosis, coronary artery disease, and congestive heart failure) may impair the healing process, which depends on oxygen supply delivered by a normal blood flow.³ Despite causing physiological alterations, cardiovascular disease seems not to affect clinical implant success. Khadivi *et al.*⁴⁹ explored cardiovascular disease with respect to initial osseointegration in a retrospective analysis that involved 246 patients, 39 with cardiovascular disease of interest. Their retrospective study demonstrated 13% failure in both the cardiovascular disease and control groups. Studies with implants in function are needed, but it appears that cardiovascular disease does not diminish initial implant survival.³

9) **Hypothyroidism**

Thyroid disorders affect bone metabolism. Thyroxine (T4) and, to a lesser extent, T3 regulate several homeostatic processes. In soft tissue and bone fractures, these hormones manage wound healing. Hypothyroidism decreases recruitment, maturation, and activity of bone cells, possibly by reducing circulating levels of insulinlike growth factor-1; this suppresses bone formation as well as resorption. Fracture healing is therefore inhibited. From this information, one may deduce that hypothyroid states lead to greater failures in implant osseointegration.³

The few studies on this subject, however, do not mirror this idea. Attard and Zarb⁵⁰ performed a retrospective study on 27 female patients (82 fixtures) with hypothyroidism on replacement medications and matched them to 29 controls (81 fixtures). Cumulative success rates after more than 1 year of function were 95% and 97%, respectively. Likewise, another group found no correlation between thyroid status and implant failure, though the patient pool was extremely small. Thus, in a controlled patient, hypothyroidism fails to influence implant survival.³

II. Conclusion

Proper case selection is needed for a successful implant therapy. For a successful implant case, an appropriate healing response is required. Not all edentulous patients are candidates for implant therapy. Absolute medical contraindications exist and must be adhered to. In order to avoid any infection, implant failure, or even patient death. But then there are conditions, if stabilized, they do not interfere with healing; those are relative contraindications to elective oral surgery. The careful practitioner understands the nature of a number of diseases assess evidence regarding implant therapy in such patients and picks his or her cases based on this knowledge. It is an informed choice that we make, and if we choose properly, predictability results.

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