Oral Bisphosphonate Therapy and Osteonecrosis of the Jaw: What to Tell the Concerned Patient

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Bisphosphonate-associated osteonecrosis of the jaw (BONJ) is encountered predominantly in cancer populations being treated with high-dose intravenous bisphosphonates for skeletal complications such as bone metastases and secondary fracture risk. A minority of BONJ lesions have been observed in patients receiving oral bisphosphonates for management of osteoporosis or osteopenia. In this paper, the current knowledge pertaining to the incidence, definition, and signs and symptoms of BONJ is presented, followed by a discussion of the incidence and consequences of osteoporotic skeletal fracture and the use of oral bisphosphonates to mitigate fracture. The risk of BONJ appears to be very small in patients taking oral bisphosphonates. In addition, the consequences of osteoporotic fracture likely have significantly greater mortality and morbidity than BONJ. Within this context, management concepts and guidelines are presented to help the dental clinician allay concerns about BONJ expressed by patients receiving oral bisphosphonate therapy. *Int J Prosthodont 2007;20:115–122.*

Bisphosphonate-associated osteonecrosis of the jaw (BONJ) is a debilitating maxillofacial condition recently described in numerous published case series and case reports.[1–6] These reports indicate that the significant majority of BONJ cases are observed in patients who suffer from cancers with ensuing skeletal-related complications that require mitigation by intravenous bisphosphonate therapy. Only a small number of cases have been reported in patients receiving oral bisphosphonate therapy for osteoporosis.[1,2,7] The risk of developing BONJ in postmenopausal osteoporotic patients treated with oral bisphosphonates such as alendronate, risedronate, and ibandronate is not known, as only a small number of cases have been reported. In a narrative review of 368 cases of BONJ, 18 were associated with oral alendronate, and 13 of these cases occurred in osteoporotic patients without cancer.[7] Almost all of the remaining cases were associated with cancer therapy. The same review described 1 case of BONJ associated with risedronate and 1 case associated with oral ibandronate, both of which occurred in osteoporotic patients without cancer. The relatively few cases of BONJ seen in this latter population is comforting given the large number of women and men currently receiving oral bisphosphonate therapy, and indicates that the risk of BONJ in this population is likely to be very low.

It is important to understand that no causal relationship between bisphosphonate therapy and osteonecrosis of the jaw has been established. To date, only an association has been recognized. Hence, the

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Table 1 Oral Bisphosphonates

Drug compound	Trade name	Manufacturer
Alendronate	Fosamax	Merck
Etidronate*	Didronel	Procter & Gamble
Ibandronate	Boniva	Roche
Risedronate	Actonel	Procter & Gamble
Tiludronate*	Skelid	Sanofi-Aventis

*Approved in the United States for the management of Paget's disease, not osteoporosis.

designation of BONJ neither infers nor suggests that bisphosphonates induce or cause osteonecrosis of the jaw. Instead, use of the term BONJ merely acknowledges an association of bisphosphonate therapy with development of osteonecrosis of the jaw. Clearly, in this early period of investigation of BONJ and with only a small number of cases to study, the requisite for a cofactor or cofactors to lead to development of this condition must be recognized.

Regardless of the few cases of BONJ reported in oral bisphosphonate users, the large number of individuals currently taking oral bisphosphonates to manage risk for osteoporosis has led to concern that the frequency of BONJ will increase. The difficulty in successfully managing established BONJ is cause for anxiety when providing for the medical and dental needs of patients taking these bisphosphonates. In addition, newspaper articles and reports of litigation aimed at pharmaceutical companies who manufacture oral bisphosphonates (Table 1) have enhanced public awareness and created an air of unease, as clinicians are questioned by apprehensive patients whether they should discontinue osteoporosis-related bisphosphonate therapy.

This article proposes a conceptual framework that practitioners may choose to consider when confronted either with the risk of BONJ or with addressing the clinical manifestations of BONJ in patients taking oral bisphosphonates. The authors concur with the panels of experts representing the American Dental Association, the American Association of Oral Surgeons (AAOMS), and the American Society for Bone and Mineral Research (ASBMR) that the exact incidence and risk of BONJ are unknown, and that there are no specific guidelines for the management of patients taking oral bisphosphonates. Hence, the framework proposed here is derived from an assessment of the risks and benefits of bisphosphonate therapy in patients with osteoporosis, as well as the risks of BONJ in each patient. Furthermore, we acknowledge that the framework proposed will necessarily evolve as new information regarding the incidence and management of BONJ becomes available.

Osteonecrosis of the Jaw and Bisphosphonate Therapy

What Is BONJ?

In a forthcoming paper, the ASBMR has defined 2 forms of BONJ (Shane E, personal communication, 2007). A *confirmed* case of BONJ is defined as an area of exposed bone in the maxillofacial region that does not heal within 8 weeks after identification by a health care provider in a patient who is currently receiving or has been exposed to a bisphosphonate and has not had radiation therapy to the craniofacial region. A *suspected* case of BONJ includes an area of exposed bone in the maxillofacial region that has been recognized by a health care provider as being present for less than 8 weeks in a patient who is currently receiving or has been exposed to a bisphosphonate and has not had radiation therapy to the craniofacial region.

Clarifications have also been made to aid clinicians in formulating a differential diagnosis. Any of the following additional signs and symptoms may or may not be present in BONJ: pain, swelling, paresthesia, suppuration, soft tissue ulceration, intra- or extraoral sinus tracts, and radiographic variability (ranging from no radiographic alterations to varying radiolucencies or opacities). However, in the absence of exposed bone as defined above, these signs and symptoms are neither individually nor collectively sufficient for a diagnosis of BONJ. Differential diagnoses should specifically exclude the following common intraoral conditions which, in the absence of exposed bone as defined above, are not necessarily cases of BONJ: periodontal disease, gingivitis or mucositis, temporomandibular joint disease, sinusitis, and periapical pathology caused by a carious infection. In addition, BONJ does not include these other conditions that may present with exposed bone without a history of bisphosphonate use: Herpes zoster infection–associated osteonecrosis, benign sequestration of the lingual plate, and Human Immunodeficiency Virus–associated necrotizing ulcerative periodontitis.

Clinically, BONJ typically appears as an intraoral lesion of exposed yellow-white hard bone with smooth or ragged borders, sometimes with associated extraoral or intraoral sinus tracts. Painful ulcers may be present in the soft tissues adjacent to the ragged bony margins of the lesion. The AAOMS[8] has classified risk factors for BONJ into different categories: drug-related factors, local factors, and medical/systemic factors.

1. Drug-related factors are type of drug (in general, intravenous (IV) bisphosphonates are far more potent than oral bisphosphonates) and duration of therapy.

2. Local factors, including dentoalveolar surgery such as extractions, periapical surgery, periodontal surgery, and implant placement. Additional local risk factors are periodontal disease and caries that necessitate tooth extraction, and local anatomy with inherently poorer blood supply and less robust healing potential (mylohyoid ridge, tori). As a result BONJ has been observed in edentulous patients whose mandibular denture has traumatized the mylohyoid ridge.
3. Principal systemic or medical risk factors are cancer with and without osteoporosis/osteopenia resulting in frequent infusions of nitrogen-containing bisphosphonates.

However, it is unclear whether cancer is truly a risk factor for BONJ, since patients without cancer do not receive IV bisphosphonate therapy in the doses given to cancer patients.

What Is the Incidence of BONJ?

In a recent narrative review of 368 cases of BONJ published between 1966 and 2006, Woo et al reported that 65% of cases affected the mandible only, 26% the maxilla only, and 9% both.[7] One-third of these lesions were painless at diagnosis, and 60% of cases occurred in women. Multifocal or bilateral involvement was more common in the maxilla than the mandible, with most lesions occurring on the posterior lingual mandible near the mylohyoid ridge. Sixty percent of cases occurred after oral surgery for dental extraction or other dentoalveolar surgery, whereas the remainder occurred spontaneously, often in patients wearing dentures. Most cases (94%) occurred in patients treated with intravenous bisphosphonates, and most (85%) had multiple myeloma or breast cancer metastatic to the skeleton. Patients receiving intravenous bisphosphonates for cancer were most often treated with 1 or more of the potent nitrogen-containing intravenous bisphosphonates, typically once a month for several years.

Clearly, these descriptive data shed light on the type of patient who is most likely to experience BONJ. However, only 2 studies have evaluated the incidence of BONJ with some degree of rigor. Bamias et al evaluated the incidence of BONJ in a population of 252 cancer patients prospectively since July 2003 who had received bisphosphonates since April 1995.[9] Seventeen patients (6.7%) developed BONJ: 11 of 111 (9.9%) with multiple myeloma, 2 of 70 (2.9%) with breast cancer, 3 of 46 (6.5%) with prostate cancer, and 1 of 25 (4%) with other neoplasms ($P = .289$). The median number of treatment cycles and time of exposure to bisphosphonates were 35 infusions and 39.3 months for patients with BONJ compared with 15 infusions ($P < .001$) and 19 months ($P = .001$) for patients with no BONJ. The incidence of BONJ increased with time of exposure from 1.5% among patients treated for 4 to 12 months to 7.7% for treatment of 37 to 48 months. The cumulative hazard was significantly higher with zoledronic acid than with pamidronate alone or pamidronate and zoledronic acid sequentially ($P < .001$). Also of significance, all but 2 patients with BONJ had a history of either dental procedures within the last year or use of dentures.

Dimopoulos et al assessed BONJ prospectively in 202 patients (some overlap with the Bamias et al population) with multiple myeloma who had received bisphosphonates between April 1995 and July 2003.[10] Fifteen patients (7.4%) developed BONJ. The median time of exposure to bisphosphonates was 39 months for patients with BONJ, compared to 28 months ($P = .048$) for patients with no BONJ. The cumulative hazard of developing BONJ was significantly higher in patients treated with zoledronic acid alone than in those treated with pamidronate alone, pamidronate followed by zoledronic acid, or zoledronic acid followed by ibandronate (1% at 1 year and 15% at 4 years vs 0% and 5%, respectively; $P = .003$). Although this supports previous data by quantifying the risk of BONJ in patients receiving IV bisphosphonate therapy and indicates that the duration of therapy and number of infusions correlate to BONJ risk, the information for those patients taking oral bisphosphonates is scant. Recently published data from Felsenberg et al indicates an extremely low BONJ incidence in oral bisphosphonate users. After a clinical examination of patients reported to have a BONJ lesion a review of each putative-BONJ patient's medication history, the incidence of BONJ in oral bisphosphonate users appears to be less than 4 in a million users (0.00038%); clearly an encouragingly low incidence.[11] Jeffcoat recently published results from a prospective study of dental patients randomized to placebo or alendronate.[12] Of the 385 subjects followed for 2 to 3 years prospectively in this study (both placebo and alendronate), none developed BONJ. These encouraging data were collected over a relatively short observation period, however. Although no BONJ was observed in the 2- to 3-year trial period, duration of therapy may be an important factor for BONJ.

Which Type of Bisphosphonate Is Associated with BONJ?

Most reported cases of BONJ occurred with IV zoledronic acid and pamidronate given as an adjunct in cancer therapy. An ever-strengthening body of evidence demonstrates that the standard regimen of IV zoledronic acid used in cancer therapy is associated with a higher incidence of BONJ than the standard regimen of pamidronate.[2,9,10] This led the Mayo Clinic

Table 2 WHO Classification System for Osteoporosis Based on Bone Mineral Density (BMD)

Normal	BMD value within 1 SD of the young adult mean value (T-score between 0 and −1)
Osteopenia	BMD value between 1 and 2.5 SDs below the young adult mean value (T-score between −1.0 and −2.5)
Osteoporosis	BMD value more than 2.5 SDs below the young adult mean value (T-score < −2.5)

Multiple Myeloma group to develop guidelines curtailing bisphosphonate use after 24 months of therapy.[13]

The risk of BONJ in postmenopausal osteoporotic patients treated with oral bisphosphonates such as alendronate, risedronate, and ibandronate is unknown, as only a small number of cases have been reported. Most of these cases are associated with alendronate therapy, likely a result of its wider use compared to other oral bisphosphonates. The review by Woo et al reported that only 15 patients without cancer were treated for osteoporosis with any bisphosphonate.[7]

What Is the Mechanism of BONJ Formation?

There is no established pathophysiologic mechanism by which oral or IV bisphosphonates are associated with BONJ. One hypothesis is that the since the bone turnover rate of the jaws is relatively high, the concentration of bisphosphonates in jawbone is higher than in extraoral sites. As a result, there may be a more profound effect of bisphosphonates on bone wound healing. Another consideration is the likelihood of a diminished healing response resulting from a compromised blood supply. This etiology is consistent with BONJ observed at sites such as the mylohyoid ridge and tori. Indirect support also comes from successful surgical procedures that reinstitute a good blood supply to confirmed BONJ sites.[14] Trauma or infection increases the demand for bone microdamage repair, which may lead to localized osteonecrosis, although it is not yet clear how exactly this may occur. Clearly, there are many factors that may influence the development of BONJ, and research to develop an animal model to understand how and why incipient BONJ lesions arise, take hold, and expand is needed.

What Are the Dental Benefits of Oral Bisphosphonate Therapy?

A reasonable body of evidence exists indicating that the systemic bone-sparing effects of oral bisphosphonates also manifest in the jawbones. In the study by Jeffcoat, 335 subjects with moderate to advanced periodontal disease were randomized to placebo or alendronate (70 mg once weekly).[12] A significant gain in alveolar bone height was seen in the alendronate-treated group (4.16 ± 0.11 mm baseline, 3.75 ± 0.18 mm 2 years) relative to the placebo group (4.22 ± 0.13 mm baseline, 4.61 ± 0.23 mm 2 years) ($P < .001$) in patients with low mandibular bone mineral density (BMD) at baseline. This significant difference was not observed in alendronate-treated patients with normal BMD at baseline (4.33 ± 0.13 mm baseline, 4.49 ± 0.21 mm 2 years) compared with placebo-treated subjects (4.32 ± 0.11 mm baseline, 4.31 ± 0.18 mm 2 years). These data expand those reported by Lane et al, who observed that in 70 patients who were randomized (43 to the bisphosphonate group and 27 to the placebo group), bisphosphonate therapy significantly improved clinical attachment loss (CAL), probing depth (PD), and bleeding on probing (BOP) relative to the placebo group during the 6- to 12-month period (CAL, $P = .0002$; PD, $P = .0156$; BOP, $P = .0079$).[15] However, there was no difference in the change in periodontal bone mass between the bisphosphonate and placebo groups as measured by fractal analysis and digital subtraction radiography. Taken together, these data and those from animal models suggest that oral bisphosphonate therapy benefits patients with periodontal disease.[16–20]

Osteoporosis and Bisphosphonate Therapy

Osteoporosis, as defined by the World Health Organization (WHO) (Table 2), is an important and growing public health problem.[21] Ten million Americans already have osteoporosis, and almost 34 million more are at risk with low bone mass (osteopenia).[22] The lifetime risk of a hip, spine, or distal forearm fracture is about 40% for women and 13% for men.[23] Osteoporosis results in health care costs of $20 billion/year (2004 dollars).[24] Of all fractures, hip fractures account for the greatest morbidity and mortality,[25] and the 1-year mortality rate after hip fracture is estimated to be 31% for men and 17% for women.[25] Up to 50% of individuals will need institutionalized care after hip fracture, and many who do return home are unable to regain their level of function prior to fracture.[26,27] While the medical and economic consequences are most evident for hip fractures, vertebral and distal forearm fractures also have a major impact.[25] In the United States, vertebral fractures generate about 66,000 physician office visits[24] and 70,000 hospital admissions annually.[28] Both men and women with clinically evident vertebral fractures will experience chronic pain and/or height loss,[29,30] which are difficult to alleviate without

expensive interventions such as kyphoplasty.[31] Forearm fractures account for more than 530,000 physician visits each year,[24] and a substantial number result in poor hand function.[32] The only feasible means of avoiding these adverse outcomes is to detect fracture risk early and institute preventive measures.

Bisphosphonates are currently the most potent oral antiresorptive agents available to prevent or treat osteoporosis. More than 192 million prescriptions have been written for oral bisphosphonates since 1996, when alendronate became available, with 77% of these being for alendronate. Oral bisphosphonates available in the United States include alendronate, risedronate, ibandronate, etidronate, and tiludronate, although the latter 2 are approved in the United States for the management of Paget's disease, not osteoporosis. Bisphosphonates incorporate into bone and block bone resorption by inhibiting osteoclast activity via several mechanisms. Oral alendronate and risedronate are proven to decrease bone turnover and reduce vertebral and hip fractures by 50% to 60% in postmenopausal women,[33–35] whereas oral ibandronate reduces vertebral fractures by 50% to 60% but not nonvertebral fractures.[36,37] Oral or IV etidronate and IV pamidronate have not yet been proven to decrease fractures in patients with postmenopausal osteoporosis. IV ibandronate prevents bone loss.[38] IV zoledronic acid has been proven to reduce vertebral fractures by 70% and hip fractures by 40% in postmenopausal women in a recently completed trial.[39]

Alendronate is approved in the United States for prevention and treatment of postmenopausal osteoporosis,[39] and for treatment of glucocorticoid-induced osteoporosis and male osteoporosis.[40–42] Risedronate is approved for prevention and treatment of postmenopausal osteoporosis[33,43–45] and glucocorticoid-induced osteoporosis[46] and treatment of male osteoporosis.[47] Oral and IV ibandronate are approved for the management of postmenopausal osteoporosis. Etidronate has been shown to prevent glucocorticoid-induced bone loss.[48,49]

Patients who stop alendronate after several years of therapy begin to lose bone about 6 months after cessation, but the rate of bone loss is slower than in controls who had never taken alendronate.[50] Alendronate appears to be safe for up to 10 years of continuous therapy, although the incidence of BONJ was not specifically documented as part of the study design.[51] Similar information for the other bisphosphonates approved for osteoporosis management is currently unavailable. Patients with hypocalcemia, hypersensitivity to medication, renal insufficiency with creatinine clearance $<$ 35 mL/min (alendronate) or $<$ 30 mL/min (risedronate and ibandronate), or esophageal irritation or strictures should avoid oral bisphosphonates.

Management Concepts

Two clinical conditions necessarily intertwine when contemplating the risk of BONJ in patients using oral bisphosphonates. We propose that management guidelines be framed in the context of the risks and sequelae of osteoporosis and osteonecrosis of the jaw. It is imperative that dental and medical professionals interact closely to properly identify the risk status for each clinical condition, as well as to coordinate care for each patient and properly assess changes in risk status.

Men and postmenopausal women with low BMD (Table 2) and/or high bone turnover are candidates for oral bisphosphonate therapy. The determination of these systemic skeletal conditions can only be made by a physician. The decision to begin oral bisphosphonate therapy is usually made by the patient after he/she has been informed as to the risks and benefits of therapy relative to the risks and benefits of not beginning therapy.

The principal risk factors for BONJ are *(1)* invasive dental surgeries that result in exposed bone and *(2)* trauma to gingiva or oral mucosa leading to exposed bone uncovered by soft tissue. Bone that is unable to heal and remains exposed for 8 weeks would then meet the ASBMR-sanctioned definition of BONJ. Oral conditions that predispose to tooth extraction, such as advanced caries, moderate to advanced periodontitis, and xerostomia, are indirect risk factors for BONJ. Factors that predispose to traumatic exposure of bone, such as ill-fitting denture bases or tori with thin mucosal coverings, are also indirect risk factors for BONJ. There is little rigorously obtained scientific information regarding dental implant placement in men and women taking oral bisphosphonates. Based on the results of a single study, dental implant placement does not appear to be a risk factor for BONJ.[12]

Management Guidelines

When considering individual patient-management options, it is imperative to allay patient fears regarding oral bisphosphonate therapy and BONJ. Although an association between bisphosphonate therapy and BONJ has been reported, patients should be informed that BONJ is seen predominantly in those who have cancer and are receiving high-dose IV bisphosphonate therapy. Clearly, given the enormous number of women and men taking oral bisphosphonates and given the small number of BONJ cases reported in this group, the risk of developing BONJ while on oral bisphosphonate therapy is low. However, further research is needed to establish the true incidence of BONJ in patients on oral bisphosphonate therapy, with a special emphasis on determining whether risk is modified by the type, dosage, or duration of bisphosphonate therapy, or by

other cofactors, eg, medications, genetic factors, or other therapies for BONJ development.

All patients should be encouraged to receive routine dental and medical care. At minimum, annual exams are desirable, as are dental prophylaxes every 6 months, since there is no evidence to date that performing dental prophylaxis increases the risk of BONJ. Concerned patients should be evaluated further to establish the likelihood for needing a dental extraction in the near or intermediate future. Patients with good oral hygiene, a low caries rate, and only limited periodontal bone loss are unlikely to need a dental extraction. These patients should be informed that their risk of developing BONJ is extremely low. Patients with advanced caries or advanced periodontal disease should be advised that there may be a small risk of developing BONJ if a dental extraction is necessary. However, until future research indicates otherwise, patients should be informed that this risk is low because so few cases of BONJ have been observed.

It has been proposed that stopping oral bisphosphonate therapy prior to a dental extraction may be advantageous. Given that the half-life of oral bisphosphonates is fairly long (up to 10 years in the case of alendronate), the effect of stopping therapy on fracture risk is likely small. Indeed, evidence indicates that no significant increase in bone turnover is noted in patients who stop alendronate therapy for up to 6 months, at which time osteoclast function begins to recover. More importantly, how much increase in bone turnover is necessary to lower BONJ risk to a significant degree is unknown. Therefore, although stopping alendronate therapy for up to 6 months could be considered, the decision to stop or continue should be made with the following factors in mind. First, although future publications may offer clarification, currently there is no published scientific evidence showing a benefit to stopping therapy for any defined period of time. Second, in many instances, delaying a dental extraction is not possible because of pain and/or disease management issues. In addition, any discussion with a patient about stopping or continuing bisphosphonate therapy must include the patient's entire medical team. It is imperative to recognize that although BONJ is a debilitating condition, it is rare compared to the occurrence of hip, wrist, and spine fractures in osteoporotic women and men. The mortality associated with these fractures is often significant, and the degree of debilitation far greater in terms of morbidity than that observed with BONJ. Therefore, ceteris paribus, preventing skeletal fractures is more compelling than preventing BONJ.

Edentulous or partially edentulous patients who wear removable dental prostheses should be carefully evaluated to see if the denture base fits sufficiently well. Some cases of BONJ are located in the mylohyoid ridge region, and the putative etiology in these cases is trauma secondary to ill-fitting denture bases. In addition, since the oral mucosa covering a torus is often thin and easily traumatized to expose underlying bone, patients with a torus should be counseled about their susceptibility to trauma and encouraged to eat foods that will lower the risk of trauma. In both the removable prosthesis–wearing population and the torus population, patients should be encouraged to seek a dental exam if they are concerned that they have traumatized mucosa and exposed maxillary or mandibular bone.

Summary

Given the recent concern regarding BONJ, the following information should be discussed with patients who are about to start or are currently on oral bisphosphonate therapy to arrest or slow bone turnover:

1. The large majority of BONJ cases have been observed in cancer patients receiving high-dose IV bisphosphonate therapy.
2. The risk of developing BONJ as a consequence of oral bisphosphonates used for osteoporosis is very low.
3. Most cases of BONJ are seen in patients who have had a recent dental extraction.
4. Continuing routine dental care, including dental prophylaxis and restorative dentistry, is recommended.
5. Concerned patients may opt to have a dental exam more frequently than a standard annual exam. However, there is no evidence to indicate that such a strategy aids in preventing or managing BONJ.
6. Concerned patients who are taking oral bisphosphonates and who need a dental extraction may consider temporarily stopping bisphosphonate therapy (for up to 6 months in the case of alendronate) prior to extraction. However, there is no evidence to indicate that such a strategy aids in preventing or managing BONJ.
7. Although it is not an indication for bisphosphonate therapy, patients should be informed that taking oral bisphosphonates may benefit the periodontium.
8. Patients with established BONJ, regardless of whether they are receiving or have received IV and/or oral bisphosphonate therapy, should be managed in accordance with the guidelines proposed by the AAOMS[8] and Ruggiero et al.[52]

These suggestions, derived from the currently available combination of low stringency studies and expert opinion, aim to offer the clinician a conceptual foundation on which to build discussion with concerned patients, as well as to develop a strategy for shared clinical decision making.

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Literature Abstract

Retention forces and seating discrepancies of implant-retained castings after cementation

This study evaluated the influence of 5 different cements (eugenol-free zinc oxide, zinc phosphate, glass ionomer, polycarboxylate, and self-adhesive resin), and the application technique (complete or half-coating of the intaglio surface) on seating discrepancies and retention forces. Noble alloy castings cemented on titanium abutments were evaluated. The surface of the titanium abutments were left as machined for the first part and air-abraded for the second part using only the complete-coating technique to evaluate the influence of air-abrasion on retention. The discrepancies were measured at 3 marked indentations on the surface of the castings, using 10X magnification before and immediately after cementation. Tensile tests were performed with a universal testing machine. Kruskal-Wallis test and Wilcoxon rank sum test adjusted with Bonferroni-Holm were used for multiple comparisons. Discrepancies ranged from 0 to 140 µm with no statistically significant differences between the 2 application techniques. Eugenol-free zinc oxide had the smallest discrepancy. Half-coating applications did not reduce the retention but did improve the fit. Polycarboxylate cement had the highest retention value (813 N), followed by resin (653 N), glass ionomer (469 N), zinc phosphate (346 N), and eugenol-free zinc oxide (177 N). Air-abrasion of the abutments with aluminum oxide enhanced the retention value of zinc phosphate, glass-ionomer, and self-adhesive resin cements. The results of the study support the application of a reduced amount of cement clinically to improve the marginal fit without a decrease in retention; furthermore, this application decreases the amount of excess cement.

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