

Migration of Implants into the Maxillary Sinus: Two Clinical Cases

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Invasion of the maxillary sinus is a relatively frequent complication in dental implant treatment of patients with inadequate bone height in the posterior maxilla. This event usually occurs during surgery and sometimes produces sinusitis. There is a paucity of reports in the literature of implants migrating into the sinus cavity after a period of function. In the 2 clinical cases presented, an intraosseous apical movement of the implants was produced several years after placement of the implants. Hypotheses and possible mechanisms by which an implant may migrate into the maxillary sinus are described.
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The failure rate of endosseous implants is relatively low but is generally greater in the maxilla than in the mandible. The anatomy of the maxilla, particularly in the posterior sectors, and the low density and poor quality of the bone may be responsible for this lesser capacity for osseointegration.¹ The distal segments of the maxilla generally show trabeculated

bone of low density and quality, with very thin cortical bone. The roots of the maxillary teeth approximate the maxillary sinuses and, in some cases, can even be found within them.

When individuals lose their teeth, the stimulus that maintains bone shape and density disappears. As a result resorption of the alveolar process begins, reducing height in the posterior maxilla. In addition, the osteoclastic capacity of the periosteum adjacent to the sinus membrane is activated after dental loss, thereby producing a pneumatization of the sinus through a centrifugal bone resorption process. For these reasons, prosthetic rehabilitation with implants in edentulous segments of the posterior maxilla can be complex.

Despite these limitations, residual bone between the alveolar process and the sinus floor can be adequate for conventional implant placement. However, this may not always be possible, and in some cases, the maxillary sinus floor must be surgically elevated with the use of bone grafts, as first described by Boyne in 1980.²

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Fig 1a Radiograph of the patient before prosthetic treatment (case 1).



Fig 1b Panoramic radiograph of the patient after removal of the prosthesis. The implant had migrated into the maxillary sinus. The patient had also lost an implant on the right side.

Most published reports of complications derived from maxillary sinus invasion refer to infectious complications as a consequence of the close contact of the implants with the mucosa of the sinus interior. However, there are few reports of actual displacement of implants in function into the maxillary sinus. Two clinical cases of implants that migrated toward the interior of the maxillary sinus after their placement are described.

CASES

Case 1

A 42-year-old man made a first visit for the placement of a fixed prosthesis on an implant placed 8 months earlier in the left maxilla. After clinical examination, a panoramic radiograph was taken. The radiographs showed a short well-integrated implant at the site of the maxillary left canine (Fig 1a). A tooth-supported fixed prosthesis was placed with an intermediate precision attachment from the maxillary left canine to second premolar. The patient was followed annually and presented no anomalies for the first

few years. Nearly 4 years after loading, before his 4-year follow-up, he presented at the clinic with mobility of the prosthesis at its distal end. Periapical radiography of the area failed to show the implant. A subsequent panoramic radiograph revealed the implant within the superior-external region of the maxillary sinus (Fig 1b). The patient presented no sinus symptomatology or discomfort on palpation. The prosthesis was removed, maintaining the crown of the maxillary left canine, and surgery to remove the implant from the maxillary sinus was proposed. The patient refused to undergo this operation and remained asymptomatic for two years after the migration of the implant. The latest radiograph showed that it has remained in the same location, encapsulated in the upper part of the maxillary sinus, which remains completely pneumatized, ie, without signs or symptoms of sinusitis.

Case 2

A 52-year-old man presented for the rehabilitation of occlusal function. He underwent surgery for immediate implant placement in both posterior sectors of the maxilla. After 6 months, during the second surgi-



Fig 2a Radiograph of the patient (case 2). The implant was displaced 5 mm apically.



Fig 2b Radiograph of patient after prosthetic rehabilitation. The implant is within the maxillary sinus.

Fig 2c Radiograph of the patient after implant removal.



cal phase, the implant at the site of the maxillary left first molar was not observed. Radiographic examination revealed an apical displacement of the implant toward the sinus (Fig 2a). It was decided to not use this implant in the prosthetic rehabilitation. Three years later, a follow-up radiographic examination showed that the implant had migrated even further and was in the maxillary sinus (Fig 2b). The patient consented to surgical intervention for implant removal from this localization, although he had remained symptom-free throughout this period of surgical and prosthetic treatment (Fig 2c).

DISCUSSION

There is evidence that contact between the maxillary sinuses and osseointegrated implants may produce complications. Local infection of tissue around the implant is the most frequent adverse effect and may be associated with extensive resorption of surrounding bone. For this reason, implants placed very close to the maxillary sinus may offer a route for infection from the oral cavity to the sinus. Thus, sinusitis can

readily result from peri-implantitis. A further cause of maxillary sinusitis may be the displacement of an implant into the maxillary sinus, which acts as a foreign body and produces chronic infection.

There are few reports in the literature on the migration of implants into the sinus. This migration may cause a sinus disorder, as in the cases described by Regev and associates,³ Ueda and Kaneda,⁴ and Quiney and colleagues,⁵ or the patient may remain asymptomatic, as in the present cases and that reported by Iida and coworkers.⁶

The reasons for migration of an implant from its initial position to the maxillary sinus are unknown. The scant thickness and density of the edentulous maxillary segment have been proposed as an explanation for inadequate implant anchorage and, therefore, a lack of primary stability. However, this may be simply a technical issue of inadequate preparation, milling, or placement of the implant. Migration reported at 2 weeks⁵ and 2 months⁶ after the implant placement may be caused by a problem in the surgical technique or even by the presence of bone that had previously suffered an alveolar infection and consequent bone destruction. The migration may

also be the result of a particular weakness of the bone, such as osteoporosis or osteopenia.

However, the migration mechanism of an implant into the maxillary sinus after several years of adequate function is less easy to understand. This was the case for the patient presented by Iida and colleagues,⁶ whose implant migrated after 10 years of placement; for the patient described by Ueda and Kaneda,⁴ who experienced migration after 5 years of implantation; and for the 2 present patients. The fact that an implant remains in direct contact with the floor of the maxillary sinus after its placement does not imply that an upward migration of the implant will result. Authors such as Boyne and James² have suggested that an implant can be introduced 2 to 4 mm into the maxillary sinus, elevating the membrane in a nontraumatic manner. Moreover, Adell and associates¹ reported an absence of maxillary sinusitis or other related complications in a series of 101 implants placed 2 to 4 mm within the maxillary sinus after a 15-year follow-up.

Various mechanisms have been proposed to explain the migration of an implant into the maxillary sinus, and these fall under 3 main headings: changes in intrasinal and nasal pressures; autoimmune reaction to the implant, causing peri-implant bone destruction and compromising osseointegration; and resorption produced by an incorrect distribution of occlusal forces, as proposed by Regev and coworkers.³

Changes in Intrasinal and Nasal Pressures

According to this hypothesis, changes in intrasinal and nasal air pressures produce a suction effect because of the negative pressure exerted by these cavities.³ In case 1, observation of the implant within the sinus cavity was a chance finding after the patient noted mobility of the prosthesis. It can be deduced that the implant had suffered a loss of integration months earlier without symptomatology and it was hypothesized that, being only 10 mm in length, the implant had readily suffered from suction related to intrasinal pressure. This is similar to the case presented by Iida and colleagues,⁶ except that the patient in that study showed symptoms derived from mobility of the implant 5 years after its placement, although migration was not diagnosed until another 5 years had passed.

In case 2, an implant placed equidistant from 2 other implants that had acceptable primary stability progressively migrated into the maxillary sinus. The fact that the implant was placed immediately after dental extraction may explain why, despite the relative primary stability obtained during surgery, the bone around the implant was resorbed, causing the

implant to gradually lose its primary retention and facilitating implant mobility. In this case, the implant migrated apically toward the sinus and was not exfoliated into the oral cavity, as can occur in implant failure. Although the available bone height was more than 10 mm and the implant was 13 mm long, the implant migrated apically to the anterior wall of the maxillary sinus. This could be explained by the centrifugal expansion of the sinus, which may have removed bone apically around the implant and produced the aforementioned suction effect.

Autoimmune Reaction to the Implant

A second possible explanation for implant migration into the maxillary sinus would be bone destruction secondary to infections at the implant site either before or after implantation. An example would be the previous presence of apical foci involving the teeth, producing osteitis and bone weakening with resorption of certain parts of the maxilla. Another would be peri-implantitis that produced progressive resorption of bone around the implant, permitting its mobility, communication of the oral flora with the sinus flora, and the production of associated sinusitis.⁵ If there were no oral-sinus communication, there might be no infection, and the implant could remain in the interior of the maxillary sinus in an aseptic and asymptomatic manner, as in case 1.

Most published studies on implant migration report the presence of associated symptomatology, such as pain and inflammation.⁵ However, the concept of migration based on peri-implant inflammation is not applicable to Regev and associates' first case,³ the case presented by Iida and coworkers,⁶ or the present 2 cases, because none of these patients presented symptoms, and the observation was a chance finding discovered during follow-up evaluation of the implants. Moreover, none showed signs of disease in the peri-implant area, only an absence of osseointegration.

Incorrect Distribution of Occlusal Forces

Finally, it has been proposed that implant migration to the sinus may result from incorrect occlusal forces produced by prosthetic restoration. Differentiation should be made between occlusal forces applied early to the implants and the definitive forces applied to the implants. Immediate or early loaded implants are currently being advocated on the grounds that a progressive loading of the implants allows little-mineralized bone to be transformed into better, more mature bone that can withstand masticatory loads. Hall and McKenna⁷ studied 70 implants placed in sinus grafts after elevation of the sinus and

reported that the prosthetic restorations had been attached early (within 3 weeks of implant placement) in all 7 cases of implant failure (90% success rate). The occlusion of the patient treated by Regev and coworkers³ had been relieved with the placement of a temporary prosthesis. It is undeniable that early prosthetic loads on maxillary bone can produce crestal resorption. Whether or not this resorption affects implant stability will remain controversial until studies can determine whether the immediate loading of implants interferes with long-term osseointegration.

This mechanism may explain the loss of anchorage by implants subjected to early loading and their movement into the maxillary sinus, as occurred in the cases of early migration.³ However, it is difficult to understand how definitive occlusal loads could cause intrasinus displacement of implants after years of function, as in case 1 and in the patients reported by Iida and colleagues⁶ and by Ueda and Kaneda.⁴ It seems an unlikely explanation in the case presented by Iida and colleagues, since the implant became dysfunctional beneath a cantilevered prosthesis.

In case 2, the implant migrated without being subjected to occlusal forces, given that the bone depth prevented integration of the implant after second-stage surgery. Implant migration was late in both cases, suggesting that occlusal forces were not involved.

CONCLUSIONS

The migration of an implant into the maxillary sinus is an uncommon finding, and the determining factors remain unknown. Because of the impossibility of predicting this event, it would not be appropriate to withhold implantation in any segments. However, it is important to consider the advisability of placing implants near these natural cavities and to take account of the specific characteristics of the patient and receptor site. The intention should be to secure an acceptable primary stability of the implant to obtain predictable outcomes.

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