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Dental Implant Complications:

Opportunities for progress and excellence

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Peri-implantitis: Biologic complications to biomaterials

By Binnaz Leblebicioglu, DDS, MS, PhD
Academy News *Guest Contributor*

Peri-implant diseases are "plaque-associated pathological conditions occurring in tissues around dental implants, characterized by inflammation in the peri-implant mucosa and subsequent progressive loss of supporting bone."[1] Peri-implantitis is reported in 20-40% of individuals with implant restorations and it's estimated to involve about 10% of all dental implants.[2,3]

Clinical signs of peri-implantitis may include suppuration, bleeding on probing, increased probing depths, recession of the mucosal margin, and radiographic bone loss.[1] Diagnosis of peri-implantitis by using periodontal clinical parameters, bite-wing radiographs without baseline documentation has been challenging.[4] Detecting exposed implant threads or bone loss can be illusive. Thus, most peri-implantitis cases are diagnosed later in disease progression. Clinical detection of peri-implantitis and tissue breakdown is time dependent with a threshold point for more severe bone loss reported five years after implants were in function.[5,6]

The primary etiological factor for peri-implant diseases is bacterial pathogens.[7] Peri-implant subgingival biofilm is different in partial vs. complete edentulism.[8] This has been traditionally explained by possible contamination from tooth sites.[9] However, more recent reports indicate "peri-implant and periodontal microbiomes represent microbiologically distinct ecosystems" with significant differences during "health and disease in each ecosystem."[10]

History of periodontal disease is considered a risk factor for peri-implantitis.[11] However, this may not be due to similarities in bacterial pathogens but may be associated with susceptibility of the host to tissue breakdown.[12] Progression of peri-implantitis can involve a fast-track inflammatory process and rapid bone loss in the presence of minimal dental plaque. It can be regarded as a process similar to connective tissue capsulation of the biomaterial.[13] Thus, the host response in relation to distinct differences at peri-implant and periodontal interfaces should be considered as we seek to understand implant specific biologic complications.[14] In addition to systemic diseases and habits that may affect host response (e.g., diabetes and smoking), certain medications are currently

▲ *Dr. Leblebicioglu is a board certified periodontist from Ohio State University and recipient of the Osseointegration Foundation's 2017-2018 Applied Science Grant on "Identification of Host Related Risk Factors for Peri-implant Tissue Loss."*

investigated as possible risk indicators/mediators for peri-implantitis.[15] Most recently, the presence of titanium particles within peri-implant tissues, bacterial plaque and the associated host response has been explored.[16,17] The effect of titanium particles on subgingival biofilm complexity as well as on host response will likely be the subject of more in-depth investigations.

On the basis of currently available RCTs, there is insufficient evidence to support that any particular non-surgical treatment for peri-implantitis showed better performance than debridement alone.[18] Recent systematic reviews report that there is no evidence to support one surgical peri-implantitis treatment modality approach to another since short- and long-term outcomes are not predictable.[19, 20] In addition, titanium surface decontamination through various mechanical and chemical methods, and its effect on material integrity requires clarification.[21]

In summary, peri-implantitis is different from periodontitis and it should not necessarily be managed in the same way. Challenges in diagnosing and treating this disease are overwhelming due to complexity of biologic complications around an implant device. However, it's clear that prevention and maintenance in the form of supportive implant therapy may play important roles for successful long-term outcomes.[22]

Please scan the QR code to view references for this article.

Dr. Tara L. Aghaloo elected AO President; new officer, director also selected

Tara L. Aghaloo, DDS, MD, PhD, professor in Oral and Maxillofacial Surgery and assistant dean for clinical research at the UCLA School of Dentistry, was elected the Academy's 35th president at its Annual Business Meeting. One new AO at-large director and secretary were also among those elected.

"In the coming year, I would like to see us getting back to normal, but obviously with a new sense of normal. We have really seen what it is like to be a community of like-minded professionals and have connected on so many new levels never thought possible. Let's use this community we have strengthened through 2021 into a new 2022," stated Dr. Aghaloo during her acceptance remarks.

Also elected also were AO Fellows **Stephen L. Jacobs**, BDS (Glasgow, United Kingdom) as a new at-large director and **Robert C. Vogel**, DDS (Palm Beach Gardens, FL) as secretary, who has served as a director since 2017.

A member of the Academy since 2002, Dr. Jacobs' involvement with the Academy currently includes serving as a member of the Fellowship & Certificate and Global Education committees, where he also served as chair, and has also served as a member on the *Academy News*, William R. Laney Award, and Clinical Innovations committees.

"Being elected to the AO Board is a true career highlight, one that I am very proud of and one that I know my late wife, Lucy, would have also been proud of, and I dedicate this appointment to her. I cannot wait to get to work, where the focus is naturally going to be the progression of the Academy in a 'post-COVID' world. This next phase is going to be both exciting and challenging," said Dr. Jacobs.

The 2021 - 2022 AO slate of officers and directors includes:

Officers:

President - AO Fellow **Tara L. Aghaloo**, DDS, MD, PhD, oral and maxillofacial surgeon, Los Angeles, CA

President-elect - AO Fellow **Amerian D. Sones**, DMD, MS, prosthodontist, Dallas, TX

Vice President - AO Fellow **Hom-Lay Wang**, DDS, MSD, PhD, periodontist, Ann Arbor, MI

Secretary - AO Fellow **Robert C. Vogel**, DDS, general practitioner, Palm Beach Gardens, FL

Treasurer - AO Fellow **Joerg Neugebauer**, DDS, PhD, oral surgeon, Landsberg am Lech, Germany

Immediate Past President - AO Fellow **Clark M. Stanford**, DDS, PhD, MHA, prosthodontist, Chicago, IL

Directors:

AO Fellow **Joseph Y. K. Kan**, DDS, MS, prosthodontist, Loma Linda, CA

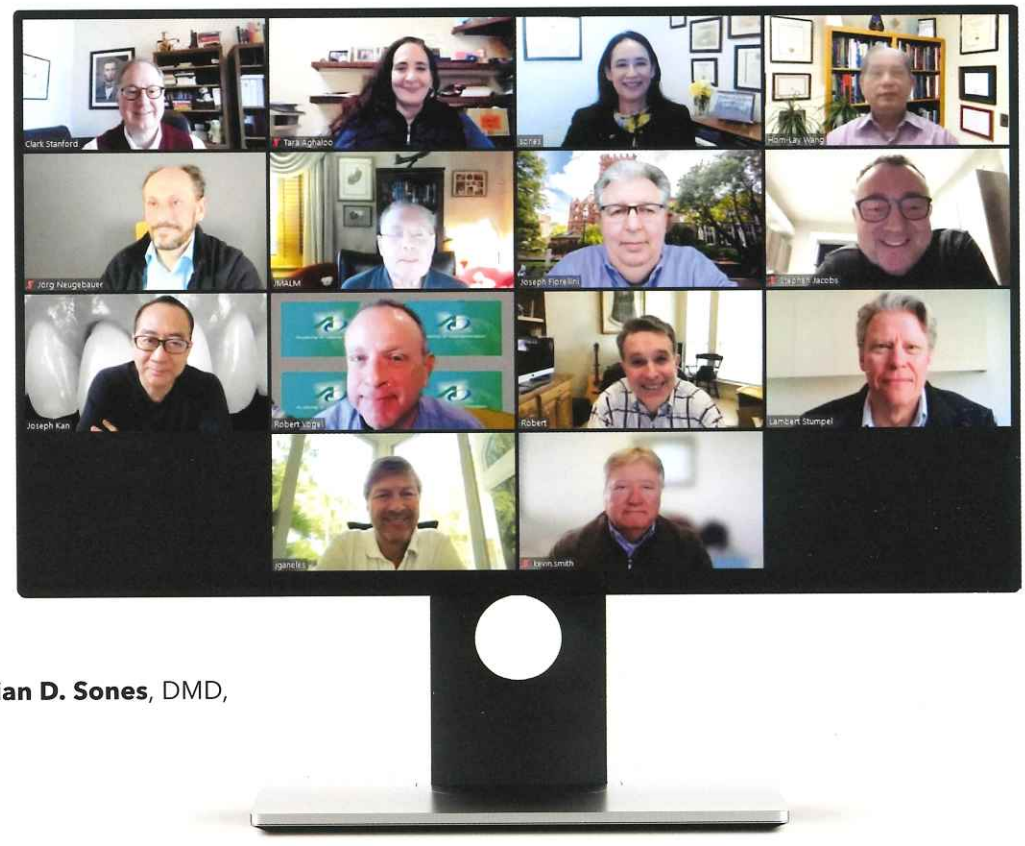
AO Fellow **Joseph P. Fiorellini**, DMD, DMSc, periodontist, Philadelphia, PA

AO Fellow **Jeffrey Ganeles**, DMD, periodontist, Boca Raton, FL

Robert R. Lemke, DDS, MD, oral and maxillofacial surgeon, San Antonio, TX

AO Fellow **Lambert J. Stumpel**, DDS, general practitioner, San Francisco, CA

AO Fellow **Stephen L. Jacobs**, BDS, general practitioner, Glasgow, United Kingdom



Are you prepared? – A simple implant turned emergency tracheostomy

By Robert R. Lemke, DDS, MD, Academy News *Guest Contributor*

We learn from the knowledge and experience of others. Experiences are harder to teach. The AO DocMatter Community is a new online platform for members to pass on not just knowledge, but also our experiences. Here is an experience I recently shared on the forum:

It was over 12 years ago. My OMS practice at that time did not have a CT machine. The day began by treating a kind gentleman who is a surgeon himself. I had placed several implants. One required a soft tissue graft.

I noticed the implant recently placed at #28 demonstrated bone loss and mobility. I decided to replace it with a new implant placed slightly deeper. The culprit was an abscess from the neighboring #27. I decided to extract the non-restorable canine and graft it.

During this time, our patient had spikes of elevated blood pressure, which I controlled with IV Labetalol. This worked for about five to 10 minutes at a time. When I drilled to place the new implant, I slightly perforated through the lingual cortex. There was no immediate bleeding, but within 20 minutes, a sublingual hematoma grew, pushing his tongue to the roof of his mouth. To remove the pool of blood, I had to access the floor of his mouth. I opened the lingual tissue away from the mandibular body expecting to find a little gush of blood, I found nothing.

Finishing, I then noted significant swelling in the sub-mental area. I used liposuction cannulas to attempt evacuation of blood in the sub-platysmal space. Nothing came. To use gravity to help drainage, I connected the floor of the mouth to the outside of his face through the sub-mental space. Still nothing came. I placed extra-oral drains and packed the floor of the mouth with gauze with no apparent bleeding.

During his post-operative recovery, he was awake and stable. After some time, I removed the intra-oral gauze and the site was perfectly dry. Just before I could place a collagen tape, our patient coughed. Then all heck occurred. He began to bleed profusely. Pressure would not stop it. Blood was filling his mouth. He started to gag, desaturate, and gag more. A yanker suction was not enough.

I could either apply pressure intra-oral to control bleeding or ventilate with an ambu/mask. If I used an ambu bag to keep his oxygen up, he would choke on the blood that was welling intra-orally. I could not intubate him as his tongue was at the roof of his mouth from the swelling. After about three minutes of alternating, I called 911. His oxygen level dropped to the 70's. He became diaphoretic with beads of sweat all over his forehead. Within another minute, his saturation dropped to 50's. He turned slightly pale, then blue and his arm dropped limp by his side.

I knew in about five minutes I could have a brain-dead patient. I told my assistant: "We need to do a trach" (tracheostomy). I asked for local anesthesia and then thought "why on earth do I need local? He's non-responsive!" I took a breath and performed the tracheostomy placing the ET tube into his neck.

The EMT's arrived. I went to the hospital in the EMT van and assisted an ENT in the OR to convert my emergency cricothyrotomy to tracheostomy. The ENT, Dr. **Nathan Marshall**, that day gave up his own cancer therapy to help my patient (As a side note, Dr. Marshall eventually succumbed to his cancer, but not after dedicating his life helping others). The next day, I went to visit the patient. Intubated through his tracheostomy, he rested in the SICU. I was worried he was brain dead.

He opened his eyes. I said, "Thank you God" (internally). He asked for a clipboard and wrote "What the --- happened?" (just like that).

I told him. He was a general surgeon himself. He took my hand, gently squeezed it and then wrote on the clipboard "Will you still be my doctor?" Now, many years later, I still get teary eyed thinking about this wonderful gentleman. I learned more from his compassion as a patient than from any other "lesson" over my career.

Subsequently, I learned that the literature has numerous reports of patients dying from hematoma associated with "routine" placement of a mandibular implant.

Do you have an emergency story to share with others? Please do so on DocMatter. Challenges occur through each of our lives. Overcoming them is what makes life meaningful.

Editor's note: The AO DocMatter Community is a new, secure and confidential online forum for exchange of clinical and scientific information, and the newest benefit of membership in the Academy. The terms of use are strict and disclosure outside the forum is prohibited. This narrative was published by the author with special permissions.

Dr. Lemke is a board certified oral & maxillofacial surgeon in private practice in San Antonio, Texas and member of Academy's board of directors.