Inflammatory complications and myasthenia gravis
An investigation of the role inflammatory complications play in triggering myasthenia gravis

Treatment planning for dental implants requires not only an evaluation of the patient's oral situation, but also an assessment of patient-related medical risk factors. While numerous risk factors have been documented, there are some autoimmune diseases which can lie dormant for years before they manifest themselves, and their onset cannot be predicted. Such diseases are referred to as 'sleeping syndromes' and may be awakened by some key factors, such as acute infection.

Myasthenia gravis (MG) is one such disease. MG is a chronic autoimmune disorder which affects neuromuscular transmission, resulting in muscle weakness. It presents several challenges for patients and clinicians. MG is no longer considered a terminal illness, and quality of life varies depending on the severity of symptoms and what initially triggered the disorder.

This case report describes a patient who had implants placed in the maxilla and developed MG two years after prosthetic rehabilitation. While the majority of scholarly literature concerning MG patients focuses on dental treatment, thus far, no correlation between implant placement and the occurrence of MG – as a direct consequence of implant-related inflammatory complications – has been reported.

Case report

A 54-year-old male patient was referred to the clinic from a regional ENT department with a diagnosis of acute maxillary sinusitis and ocular manifestations. An oral surgeon, ophthalmologist, neurologist and thoracic surgeon consulted and treated the patient.

Medical history

The upper left first molar had been extracted three years before the patient was referred. Oroantral communication arose after the tooth had been extracted and the oral mucosa was sutured closed. One year later, the patient received two implants in the region of the upper right first and second premolar which were restored with cement-retained splinted crowns following osseointegration of the implants. All treatment was performed by the same dentist.

Six months after prosthetic rehabilitation, the first rhinogenic symptoms appeared, including headache, blocked nose, feeling of nasal stuffiness, pressure and pain in the left frontal and maxillary sinuses. The patient was treated at an ENT department, where endonasal revision and drainage of the left maxillary sinus was performed. Necrotic tissue and pus indicative of acute sinusitis were both present at this stage. Adjunctive conservative treatment consisted of antibiotics, and anti-inflammatory and analgesic medication. After ten days the patient developed ptosis (drooping of the upper eye-lid) and diplopia (double vision) of the left eye. A second revision of the maxillary, ethmoid and frontal sinuses did not show any pathological changes. At this point, the patient was referred to our clinic.

An ophthalmic examination revealed that visual acuity was not disturbed. The only ophthalmic symptom was ptosis. Optical coherence tomography of the retina and optic nerve in both eyes showed no pathological changes (Figures 1–2).

Dental examination

A panoramic X-ray revealed crater-like peri-implant bone loss surrounding the implant in region 25 and the extrusion of root canal filling material in multiple teeth, as well as close proximity of the implant in the area of the upper right first premolar and the root of the upper right canine (Figure 3).

An intraoral examination revealed mobility of the splinted crowns replacing the upper left first and second premolar (note the gap between abutment and restoration in Figure 3) without any sign of pain or bleeding. Both the splinted crowns and the implant in the region of tooth 25 were

Figure 1: Optical coherence tomography of optic nerve.

Victor Palarie (Dr. Med. Dent.) received his postgraduate degree in oral and maxillofacial surgery and implantology at the Nicolae Testemiţanu State University of Medicine and Pharmacy, Moldova. He is director of the Centre of Regenerative Medicine and co-founder of the International Clinic of Diabetes, Nutrition and Metabolic Diseases. He is also a scientific researcher at Nicolae Testemiţanu State University, and part of the BiomatICS research group of the University of Mainz, Germany. His research interests include biomaterials, tissue engineering, prosthetic rehabilitation of maxillofacial defects, and dental treatment of patients with chronic and systemic diseases.

Co-authors: Igor Maxim, MD, PhD; Mihail Gavriliuc, MD, Dr. Habil. Med.; Natalia Palarie, MD
removed under local anaesthesia. Execution of the Valsalva manoeuvre confirmed that no oroantral communication was present.

A neurological examination found fatigable muscular weakness, with normal sensation and reflexes in the muscles. This resulted in the preliminary diagnosis of myasthenia gravis accompanying acute infection.

Specific laboratory and thoracic CT examinations confirmed the diagnosis of MG (Figure 4). The chest CT scan identified a thymoma as an anterior mediastinal mass. To differentiate MG from other neurological disorders, a cholinesterase inhibitor (prozerin) was administered, and electromyographic tests were carried out. Specifically, the performance of the patient’s rheumatoid factor, thyroid function and antinuclear antibodies were tested in order to exclude systemic lupus erythematosus, hyperthyroidism and rheumatoid arthritis.

**Surgical treatment and post-operative treatment**

Surgical resection is state of art in treatment for thymomas. A thoracoscopic approach was used in this case (Figure 5). To avoid having to cut the sternum or the ribs, the operation was performed endoscopically. Small incisions were made between the ribs on the right part of the chest, through which a camera and flexible thoracoscope accessed the thymus and removed the thymoma. An active tubular drainage system was used for two days after the operation.

A histological examination determined that the thymoma could be classed as type AB (Figure 6).

Morphologically, type AB thymomas exhibit foci with features of both type A and type B thymomas, combined with foci rich in non-neoplastic lymphocytes. Post-operative treatment involved the administration of acetylcholinesterase inhibitors and muscarinic receptor antagonists.

**Conclusion**

An acute infection which may not be localised in the mediastinum may trigger the onset of MG. As far as can be determined, this is the first report describing the presence of peri-implantitis, maxillary sinusitis and concomitant onset of MG. It is still not clear if peri-implantitis is a potential cause of thymoma. Greater numbers of patients with MG who undergo dental procedures must be evaluated in order to illuminate the clinical implications of dental treatment in the occurrence of myasthenia gravis.

**References**