Silent inflammation in the jaw and neurological dysregulation

Part one of a two-part article series

Literature

Dr Johann Lechner, Germany

Editorial note: This is the first part of a two-part article series. The second part will be published in issue 1/21 of ceramic implants *and will cover further aspects of fatty degenerative osteolysis of the jawbone in relation to a clinical case that is described in the first part.*

The transition from acute local inflammation after surgical removal of third molars to a chronic stage of silent inflammation could be a neglected cause of inexplicable medical symptoms. In this case report, we present an unusual case of recurrent syncope in a 19-year-old woman, whose 12-month treatment in various clinics, including a wide range of medications, did not lead to an improvement in her condition.

Background

Autonomous dysregulation with poorly defined multisystem disorders often represents a challenging clinical situation for doctors.[1,2] With no apparent cause and insufficient research on this increasingly common phenomenon, many representations of this form of idiopathic multi-morbidity are often assumed to be of psychogenic origin and pharmacological interventions in the form of psychoactive substances are common.[3,4] A case study from our clinic in Munich in Germany calls for a broader pathogenetic perspective in neurological dysregulation. Here we describe the case of recurrent syncope in an adolescent patient. In view of the unclear aetiology, we connect this case with the phenomenon of avascular and aseptic osteolysis in the jaw, which is also known collectively as "silent inflammation".

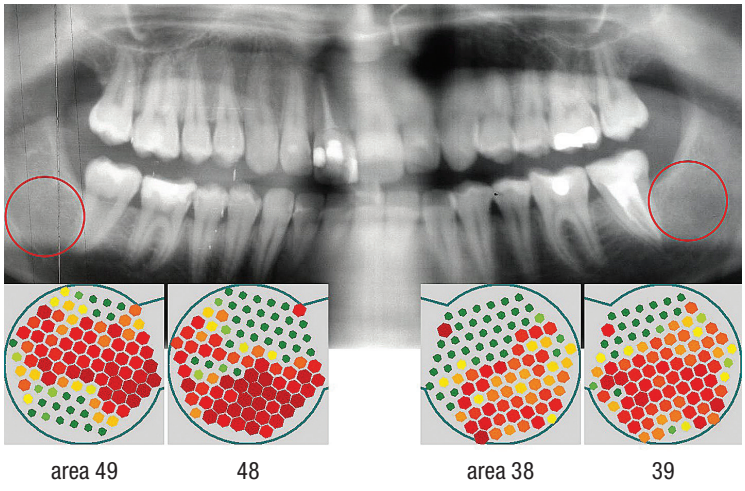


Fig. 1: Inconspicuous dental panoramic tomogram from December 2009 with no radiological abnormalities in the areas of the surgically removed teeth #18, 28, 38 and 48. The lower line shows the through-transmission alveolar ultrasonography scan; green corresponds to firm, that is, healthy jawbone; red indicates softened jawbone with inflammation potential.

Case report

The 19-year-old patient lost consciousness between two third molar extractions in October and December 2008. In the months that followed, the number of syncope incidents increased. After admission to a clinic, she was diagnosed with postural orthostatic tachycardia syndrome (POTS) and a disorder of the autonomic nervous system. Numerous drugs (for treating adrenal insufficiency, high blood pressure and paroxysmal tachycardia) brought no improvement. After further tests, the patient was discharged as a psychological case after one year. After that, it was "normal" for the patient to lose consciousness several times a day. She could no longer leave the house unattended, and the syncope resulted in daily falls.

From June 2009 to January 2010, a total of ten internal, neurological and psychiatric evaluations were carried out. The diagnosis of POTS and orthostatic hypotension was made repeatedly. In November 2009, the patient was last examined by a specialist in psychiatry and psychotherapeutic medicine, who noted "recurrent falls due to dissociation" and noted that the previous diagnosis of POTS from a clinical historical perspective and epileptic events was extremely unlikely or atypical. He recommended the rigorous discussion of the psychosomatic connections and the continuation of the accompanying psychotherapy consultations. Socio-therapeutic care was considered necessary for the 19-year-old because her mobility and safety outdoors and in traffic were severely restricted owing to her frequent syncope episodes.

Materials and methods

Diagnosis of silent inflammation in the jaw

After the hospitalisation period mentioned, the patient brought a 2D dental panoramic tomogram to our clinic to assess the possibility of chronic inflammatory processes in the tooth and jaw area. This showed no anomalies or indications of silent inflammation or osteolysis in the third molar areas (Fig. 1). In previous publications, we pointed out the insufficient diagnostic presentation of silent inflammation in the form of fatty degenerative osteolysis of the jawbone (FDOJ).[5] 2D radiographic technology alone is not suitable for a reliable diagnosis to rule out FDOJ. However, complementary through-transmission alveolar ultrasonography of bone density is suitable for diagnostic imaging of FDOJ.[6] Such ultrasonic imaging enables targeted detection and, on this basis, treatment of osteonecrotic and ischemic areas of the medullary alveolar bone.[7]

Morphology of FDOJ

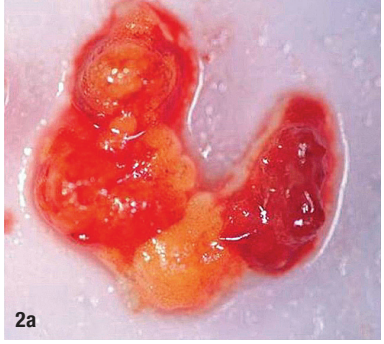
In the FDOJ areas, there is an irregular bone structure with thinned and hollowed cancellous and marrow cavities. Clinically and macroscopically, FDOJ often presents as a fatty lump. The almost complete absence of trabecular cancellous bone structure is remarkable. Figure 2 shows an intraoperative FDOJ tissue sample with predominantly fatty transformation and the extent of this FDOJ in the mandibular right retromolar area.

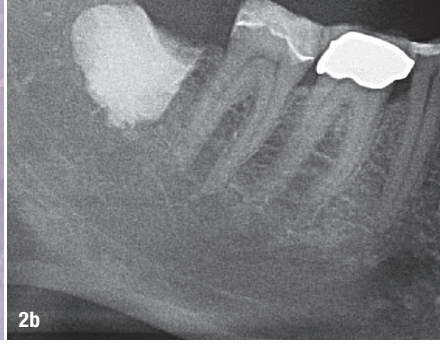
Over-activated immune mediators in the osteolytic jawbone

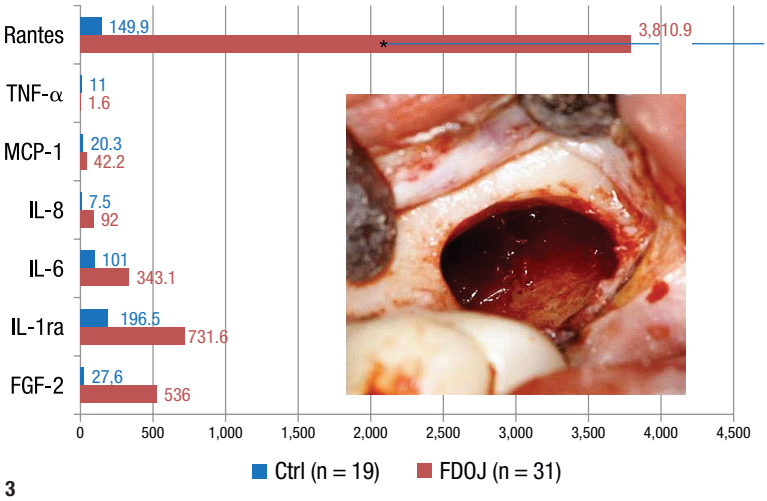
In previous studies, we were able to determine a 21-fold increase in RANTES/CCL5 (R/C) expression in 31 FDOJ samples compared with normal control samples (Fig. 3).[8] The pathologically altered FDOJ samples from 31 jawbones were obtained from the third molar and retromolar areas. A total of seven cytokines were measured in FDOJ tissue from the FDOJ group (n = 31). The distribution of immune mediators shows the clear prevalence of R/C. The mean value of R/C in the FDOJ samples was 3,810.90 pg/ml. These high R/C values in the FDOJ tissue were observed in all 31 samples. Figure 3 compares the medians of seven cytokines in 19 healthy bone samples (149.9 pg/ml for R/C) with those in 31 FDOJ samples. The fact that the acute cytokines tumour necrosis factor-α and interleukin-6 are almost absent in the FDOJ samples proves that FDOJ is a chronic, subliminal inflammatory process. FDOJ areas can be defined as osteolytic areas of the jaw with a non-acute but chronic inflammatory burden.

Histology of the FDOJ areas

The histological evaluation of this patient reads: "Vital, irregular cancellous bone tissue with no signs of active bone reconstruction. In the medullary spaces, there is not only internal bleeding but also moderate, chronic osteitis. No florid inflammation, no osteomyelitis. The fat cells partially show a myxoid transformation or a vacuolar degeneration of the cytoplasm, which is consistent with trophic disorders." The signs of FDOJ are present, but there is only a moderate inflammatory tendency without the clinical development of osteomyelitis. This histology with the long-term transition from an acute infectious wound to chronic inflammation is characteristic of FDOJ.[9]







3

Fig. 2: a) Typical fatty degenerative structure of FDOJ. **b)** Documentation of the extent of FDOJ in the mandibular right retromolar area with contrast medium intraoperatively, comparable to the case described here. **Fig. 3:** Comparison of the medians of seven cytokines in 31 FDOJ samples (red) and in 19 samples from healthy jawbone (blue); the inset shows the mandibular left retromolar area after the FDOJ parts had been removed from the medullary canal.

TNF-α = tumour necrosis factor-α; MCP-1 = monocyte chemoattractant protein-1; IL-8 = interleukin-8; IL-6 = interleukin-6; IL-1ra = interleukin-1 receptor antagonist; FGF-2 = fibroblast growth factor-2.

about the author

Since 1980, **Dr Johann Lechner** has been the head of a clinic for holistic dental medicine in Munich. He is a guest lecturer at Capital University of Washington DC, USA, and Beijing University Dental Clinic, China. He holds several medical patents for holistic systemic diagnostics and ultrasonic application in the jaw area.

contact

Dr Johann Lechner
Grünwalder Straße 10 A
81547 Munich
Germany
Phone: +49 89 6970129
drlechner@aol.com

Author details