Osteomyelitis from Chronic Periodontal Infection in a Patient with Uncontrolled Diabetes Mellitus: Case Report

Kronik Periodontal Enfeksiyonu Olan Kontrolsüz Diyabet Hastasında Gelişen Osteomiyelit

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Geliş Tarihi/*Received:* 28.06.2008
Kabul Tarihi/*Accepted:* 26.11.2008

This case report was presented by Dr. Esra Guzeldemir in SCOR 2006, Minia, Egypt.

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ABSTRACT We aimed to present the effects of uncontrolled Type II diabetes mellitus on chronic periodontitis and complications of DM. A 45-year-old-male patient applied for treatment of periodontal abscess and pain. During periodontal surgery, a deep infrabony defect between teeth #33 and #34 which encircled the teeth along the root and reached the mental foramen and mandibular canal was seen. Teeth were extracted and the tissue specimens were sent for histopathologic examination. Histological examination indicated osteomyelitis. He was referred to a physician for investigation of his medical health and identification of the cause of osteomyelitis. The patient was diagnosed as DM. Wound healing was uneventful. Uncontrolled/poor glycemic condition is a risk factor for periodontal disease and periodontal disease may affect the diabetic condition. Patients with uncontrolled DM and severe periodontitis have been shown to have a much greater incidence of macrovascular complications and secondary infections.

Key Words: Diabetes mellitus, periodontitis, osteomyelitis

ÖZET Bu olgu sunumu ile kontrolsüz Tip II diabetes mellitusun, kronik periodontitis üzerine etkileri ve neden olduğu komplikasyonların sunulması amaçlanmaktadır. Kırk beş yaşında erkek hasta periodontal absenin ve ağrılarının giderilmesi isteği ile kliniğimize başvurmuştur. Periodontal cerrahi sırasında, 33 ve 34 no'lu dişlerin kökleri arasından mental foramen ve mandibüler kanala ulaşan kemik içi defekt görüldü. Bu dişler çekildi, bölgeden toplanan doku ve kemik örnekleri histopatolojik incelemeye gönderildi. Değerlendirme sonucu osteomiyeliti göstermekteydi. Hasta, osteomiyelit gelişiminin nedeninin anlaşılabilmesi amacıyla, ileri medikal değerlendirme için yönlendirildi. İnceleme sonucunda hastanın Tip II diabetes mellitusu olduğu anlaşıldı. Bu olgu, herhangi bir komplikasyon görülmeden iyileşti. Kontrolsüz veya yetersiz glisemik durum, periodontal hastalık oluşumu açısından risk oluşturduğu gibi, periodontal hastalıklar da diyabetik kontrolü etkileyebilmektedir. Kontrolsüz diabetes mellitus ve ileri periodontitis hastaları, makrovasküler ve ikincil enfeksiyonlara daha yatkındır.

Anahtar Kelimeler: Diyabet, periodontitis, osteomiyelit

Turkiye Klinikleri J Dental Sci 2009;15(2):144-50

Periodontal disease, characterized by a destructive inflammatory process that affects the supporting tissues of the teeth, affects many people worldwide.[1-5] The progression and severity of periodontal disease are determined by risk factors (immunologic, microbiologic, environmental, and genetic factors) and by the patient's age, sex, race, smoking history. There are also some additional factors, each having an effect on the

other diabetes mellitus and age, cardiovascular diseases and age, and smoking and heart disease.[2,6-12]

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by an abnormal regulation of glucose metabolism. Diabetes-related morbidity and premature mortality impose a sizeable burden on individuals with this disease and on society, representing a major public health concern.[13] DM affects 9% of the adult American population and 6 million of these individuals have the disease but undiagnosed.[14,15] DM and impaired glucose tolerance are also common in Turkey.[16,17] The prevalence of diabetes in the Turkish population is 7.2%.[13]

Multiple periodontal abscesses that lead to rapid destruction of periodontal support are often identified in people with undiagnosed or poorly controlled DM.[18] Uncontrolled DM is associated with several chronic complications including retinopathy,[19] nephropathy, neuropathy, cerebrovascular and cardiovascular diseases,[20] an increased susceptibility to infection,[21] and altered wound healing. DM is a modifiable risk factor for periodontal disease, which is the sixth complication of DM. The prevalence of DM is increasing worldwide; thus, the number of complications of this disease is increasing as well.[22] A review of the literature by Kinane and Chessnutt showed considerable evidence to suggest that DM and periodontitis have a direct relationship.[23] In a review by Van Dyke and Sheilesh, the association between poor glycemic control and periodontal disease was well summarized.[24] The American Diabetes Association has officially stated that periodontal disease is common in patients with diabetes, and the standards of care established by the organization included obtaining each diabetic patient's history of current or past dental infections as a part of the clinical examination.

Periodontitis has been shown to precede the worsening of glycemic control. Ueta and coworkers stated that DM is a predisposing factor for periodontal disease.[25]

Periodontal infection in individuals with a systemic disease could be a trigger for osteomyelitis, which involves inflammation of the bone and bone marrow, and sclerosing intramedullary bone infection caused by oral anaerobes such as an *Actinomyces* species and *Eikenella corrodens*.[26] The hallmark of mandibular osteomyelitis is persistent, intense pain that waxes and wanes, mild expansion of the mandible, and occasional soft tissue swelling during exacerbations of the disease. Mandibular regions-mostly in the body then in the angle, and ramus-are involved. Maxillary osteomyelitis has also been noted, but in a much lower incidence than the mandibular form. In patients with mandibular osteomyelitis, the mandible may be tender to palpation particularly at the buccal cortex. No suppuration or drainage can be noted and the radiographic image is mottled. Sclerosis of the tissue typically occurs over time. The clinical progression of orofacial osteomyelitis varies according to the patient's resistance to and the virulence of the microorganism involved.

In this case report, we describe a 45-year-old diabetic man with mandibular osteomyelitis caused by periodontal infection.

CASE REPORT

A 45-year-old man was admitted to the Baskent University Adana Medical and Research Center Oral and Dental Health Clinics in 2005 because of a periodontal abscess and pain while chewing. According to his self-reported medical history, he had no medical disorder. After his detailed dental examination and obtaining informed consent from patient, root canal therapy to the left lower canine (#33) and the first premolar (#34) were performed. Then he was referred to periodontal treatment. He described pain around teeth #33 and #34. The panoramic radiograph revealed common vertical bone resorption with radiolucent areas (Figure 1) that were between the mental foramen and the mesial root of the first molar bilaterally. During the patient's initial periodontal examination, 10 mm periodontal pockets were detected in the regions of teeth #33 and #34. The clinical diagnosis was chronic periodontitis. Subgingival curettage was performed during the patient's initial periodontal visit. To eliminate local pathogenic factors and the deep periodontal pocket, periodontal surgery was planned.

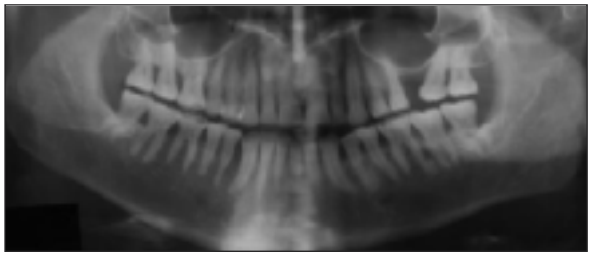

FIGURE 1: Preoperative panoramic radiograph.

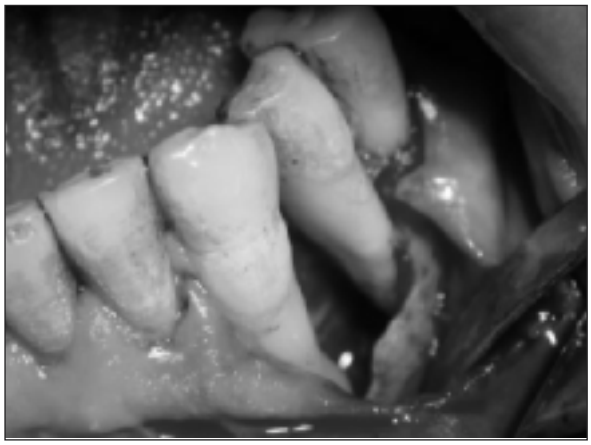

FIGURE 2: Reflected periodontal flap.

Three months after the initial treatment, periodontal surgery was performed. Following adequate local anesthetic administration (lidocaine 2% with 1:100.000 epinephrine), a sulcular incision was made and the periodontal flap was reflected from the mesial side of tooth #33 to the distal side of #34 (Figure 2). There was a deep infrabony combined defect between teeth #33 and #34 which encircled the teeth along the root periapically and reached the mental foramen and mandibular canal between the teeth. Teeth #33 and #34 were extracted, and the granulation tissue was removed (Figure 3). Specimens of granulation tissue and bone were sent for histopathological examination. The defect was expanded to the upper border of the mandibular canal and the mental foramen, and the mandibular and mental nerves were exposed. The defect, which was bordered with buccal and lingual cortical bone, continued to the posterior mandibular side under the periapical sides of the second premolar (#35) and the first molar (#36). Teeth #35 and #36 were also extracted; the granulation tissue was removed (Figure 4), and tissue specimens were obtained from that region for histological examination.

To enable haemostasis and infection control, an absorbable haemostatic agent (Surgicel. Oxidised Regenerated Cellulose 2005 Johnson&Johnson, North Yorkshire BD23 3RX, UK) was placed around teeth #35 and #36. To protect the mandible from fracture and to ensure alveolar bone volume for future prosthesis construction, the region around teeth #33 and #34 was augmented with a demineralized bone matrix (Grafton® DBM Putty,

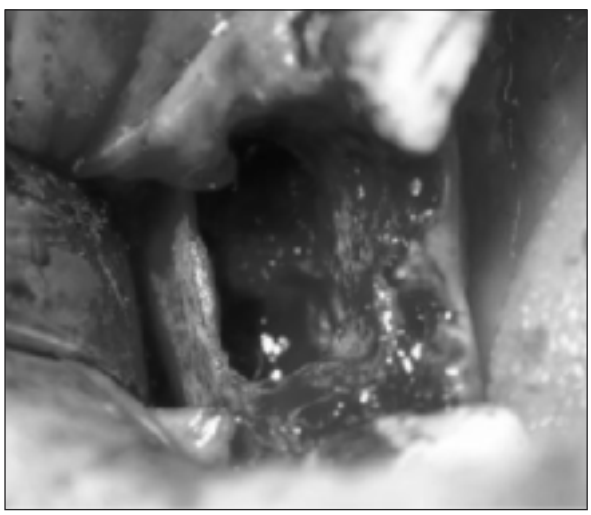

FIGURE 3: After the extraction of teeth #33 and #34, the granulation tissue was removed.

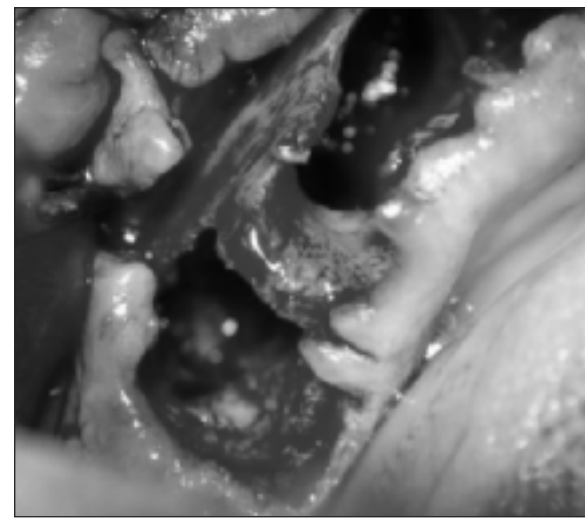

FIGURE 4: After the extraction of teeth #35 and #36, the granulation tissue was removed; cavity border was reached to the mandibular canal.

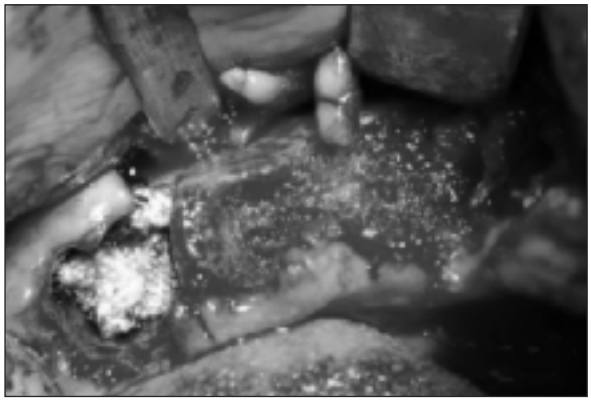

FIGURE 5: The absorbable hemostat around teeth #35 and #36 and augmentation with demineralized bone matrix around teeth #33 and #34.

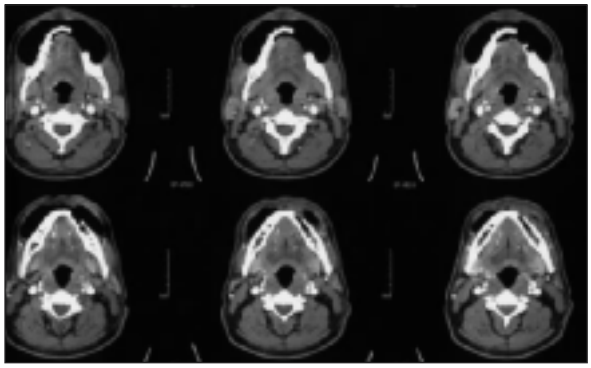

FIGURE 6: Post-operative computer tomography image.

Osteotech Inc. Cedec2-France). The complete flap closure was performed with continuous sutures with 4/0 silk sutures (Doğsan, Turkey) (Figure 5). Chemical plaque control with a 0.2% chlorhexidine gluconate (Klorhex Gargara, Drogsan, Turkey) was performed for 2 weeks. Postsurgical medications included amoxicillin 500 mg (Largopen 500 mg, Bilim, Turkey) twice daily for 7 days and flurbiprofen 100 mg (Majezik 100 mg, Sanovel, Turkey) twice daily.

Two days after the surgery, the patient was referred to the radiology department to undergo postsurgical evaluation with computerized tomography and magnetic resonance image scanning. The findings of histological and radiological studies indicated osteomyelitis (Figure 6-10).

This clinical course of the disease and the presentation of the lesion identified in our patient described, are not common. To our knowledge, there are no published reports of chronic periodontal inflammation resulting with osteomyelitis.

Chronic periodontitis is a very common disease; however we thought that clinical presentation of this case without an underlining medical disorder would be unexpected. Although he claimed that he had no medical problems, he was referred to a general physician for investigation of his medical health in detail so that the cause of osteomyelitis could be identified.

All sutures were removed after 10 days. The patient was then placed on a routine maintenance schedule to his individual needs. Healing of the extraction sockets was uneventfully.

Ten days after the surgery, the biochemical examination revealed that the patient's blood glucose level was 420 mg/dL. There are no data about his past glucose levels and his medical condition. The treatment of DM began immediately and three months later his blood glucose level was under control (278 mg/dL). An oral examination showed that there was no pathologic tissue formation in the operation area (Figure 11).

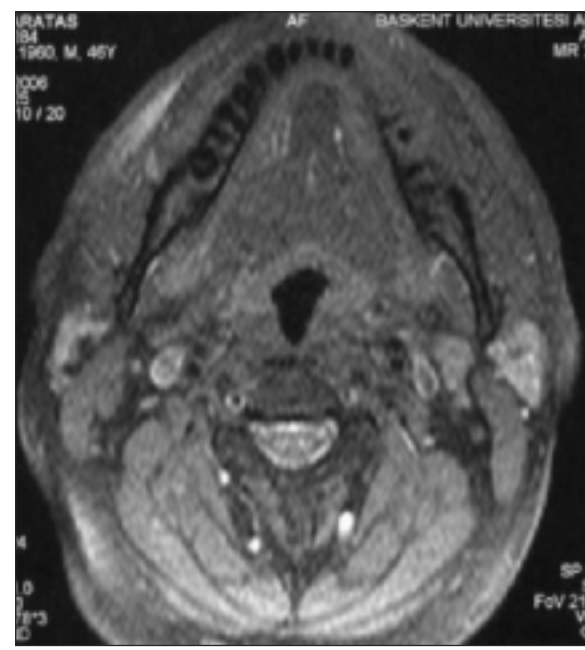

FIGURE 7: Post-operative magnetic resonance imaging.

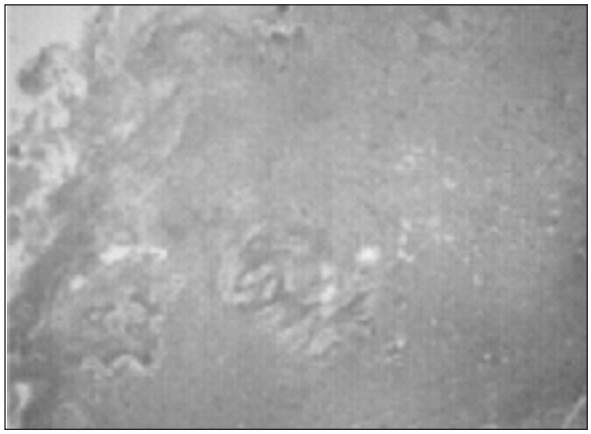

FIGURE 8: Histologic view of fibrous tissue between the bone specules (H&E×100).

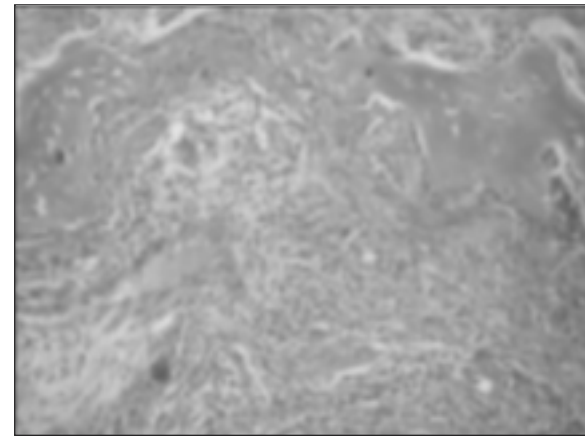

FIGURE 9: Histologic view of mixed inflammatory cell infiltration includes PMN between bone specules (H&E×200).

DISCUSSION

This case illustrates the importance of the accurate assessment of a patient's systemic health, the unexpected medical situations that face physicians in clinical practice, and the limitations of 2-dimensional dental radiographs. Another aim of this case report is to point the possible destructive effects even resulting with jaw fractures, of this common metabolic disease on periodontal status and tooth loss.

DM increases the risk and severity of periodontal disease.[27] Hyperglycemia is the primary factor responsible for the development of diabetic complications. An increased level of glucose in the gingival crevicular fluid could modify the quantity and species of the bacteria in the oral microflora. DM does not result in gingivitis or periodontal defects, per se, but it alters the response of the periodontal tissues to local pathogenic factors, causing a reduction in defense mechanisms, and an increased susceptibility to infection. It can be suggested that the metabolic control may affect the status of periodontal disease.

Although some investigators suggested that the periodontal treatment have no effect on metabolic control,[28,29] many studies affirmed that the state of inflammation may influence the state of insulin resistance and this result conduce the risk of poor glycemic control in diabetic patients.[30,31]

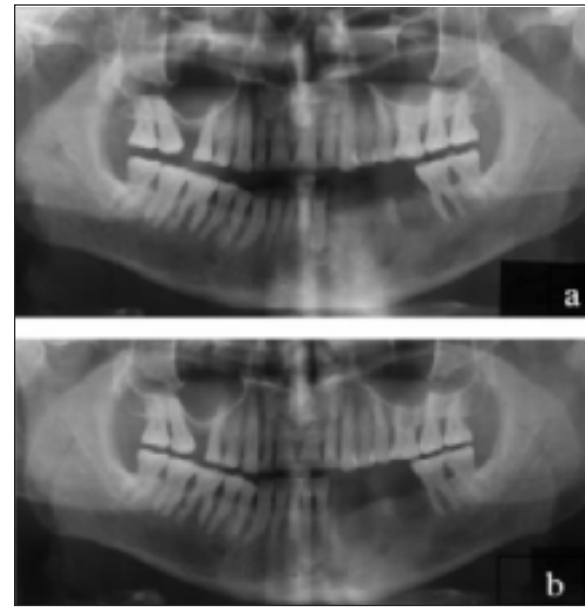

FIGURE 10: After the surgery (a) and 4 months later (b), in panoramic radiographs.

Studies have shown that patients with DM are at greater risk for ongoing periodontal destruction than are non-diabetic individuals.[32] Although people with poorly controlled or uncontrolled DM respond less successfully to periodontal therapy than do those with well-controlled DM or non-diabetic individuals,[24,32,33] Promsudthi and coworkers showed that mechanical periodontal therapy with adjunctive systemic antimicrobial treatment improved the periodontal status of the patients with un-

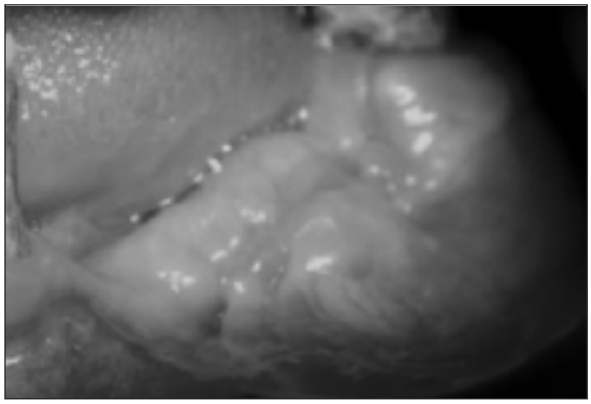

FIGURE 11: Intraoral view 4 months after the operation.

controlled DM.[34] Kiran and coworkers demonstrated that nonsurgical periodontal treatment was associated with improved glycemic control in individuals with DM;[31] however, Promsudthi and colleagues found no such correlation.[30] Ueta and colleagues showed that DM is a predisposing factor for periodontal and periapical abscess formation because neutrophil function is suppressed in diabetic individuals.[25] Preferansow et al. reported that uncontrolled diabetes was the crucial cause of periodontal changes and, to a large extent, influenced the function of the masticatory organ in patient.[35] The effects of DM on the host response of DM (and in particular neutrophil function) may account for this finding.

In our patient, to eliminate the risk of infection caused by endodontic infection, root canal treatments were performed before periodontal treatment. Because of the labial position of the canine outside the dental arch in our patient, impacted food might have caused a chronic periodontal infection and the subsequent formation of a periodontal abscess. We concluded that our patient demonstrated a combined abscess (periapical and periodontal) formation. Treatment of mandibular osteomyelitis involves removal of the diseased tooth (or teeth) and root along with debridement of the involved mandibular bone.[33]

Clinicians must remember that most of the systemic disorders have oral manifestations and patients with such disorders may not be aware of their medical conditions. DM is a modifiable risk factor for (but not a cause of) periodontal disease. Although DM cannot be cured, it can be controlled. Uncontrolled systemic diseases often contribute to the development of more complicated and advanced disorders that affect the patient's quality and duration of life.

Conflict of interest and source of funding statement: The authors declare that they have no conflict of interests. This paper was prepared without any sources of financial support.

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