

Journal section: *Odontostomatology for the disabled or special patients*  
Publication Types: *Case Report*

doi:10.4317/jced.3.e408

## **Necrotizing periodontitis in the context of Takayasu's disease: Report of a case.**

**Javier Silvestre-Rangil<sup>1</sup>, Francisco Javier Silvestre-Donat<sup>2</sup>, Fernando Llambés-Arenas<sup>3</sup>, Angel Puente-Sandoval<sup>1</sup>**

<sup>1</sup> Collaborating dental surgeon of the Stomatology Unit of Dr. Peset University Hospital (Valencia, Spain).

<sup>2</sup> Assistant Professor of the Department of Stomatology of the University of Valencia. Head of the Stomatology Unit of Dr. Peset University Hospital (Valencia, Spain).

<sup>3</sup> Periodontist. Private Practice, Valencia, Spain.

*Correspondence:*  
*Hospital Universitario Dr. Peset*  
*Consultas Externas*  
*C/ Juan de Garay s/n*  
*46017 – Valencia (Spain)*

Received: 13/06/2011  
Accepted: 03/08/2011

Silvestre-Rangil J, Silvestre-Donat FJ, Llambés-Arenas F, Puente-Sandoval A. Necrotizing periodontitis in the context of Takayasu's disease: Report of a case. *J Clin Exp Dent.* 2011;3(Suppl1):e408-11.  
<http://www.medicinaoral.com/odo/volumenes/v3iSuppl1/jcedv3iSuppl1p408.pdf>

Article Number: 50604 <http://www.medicinaoral.com/odo/indice.htm>  
© Medicina Oral S. L. C.I.F. B 96689336 - eISSN: 1989-5488  
eMail: [jced@jced.es](mailto:jced@jced.es)

### **Abstract**

Necrotizing periodontitis is characterized by gingival necrosis with bleeding and pain, and alveolar bone attachment loss. The disease is associated to spirochete and *Prevotella intermedia* infection, as well as to a series of bacterial infections, with an altered local host immune response and tissue destruction.

We present the case of a 40-year-old woman with inflammatory vasculitis of the aorta and its main branches, known as Takayasu's disease. In the course of treatment of the latter, she developed necrotizing periodontitis. In this case local factors such as abundant bacterial plaque accumulation secondary to a lack of tooth brushing, and the administration of methotrexate, may have played a role. The clinical condition improved notoriously after emergency treatment with local antiseptics and tartar removal.

**Key words:** *Necrotizing periodontal disease, necrotizing ulcerative periodontitis, diagnosis, treatment, Takayasu's disease.*

## Introduction

Necrotizing periodontitis (NP) is a destructive periodontal disorder found in the context of diseases that are little understood, or which are characterized by host immune deficiency. NP manifests with the characteristics of necrotizing gingivitis (NG), with acute episodes of gingival necrosis at papillary level, spontaneous bleeding and intense pain, though it moreover also presents clinical and alveolar bone attachment loss. NP may be the evolutive consequence of various NG episodes, or result from complications of prior periodontitis in the affected zone (1-3).

Immune system deficiencies may be implicated in these processes when associated to the presence of periodontal pathogenic flora such as certain constant species of *Treponema* or *Selenomonas*, *Fusobacterium nucleatum*, *Prevotella intermedia*, or a variable group of heterogeneous bacteria. The accumulation of gramnegative bacteria results in the release of endotoxins that can directly or indirectly cause tissue destruction through modification of the host tissue response, affecting polymorphonuclear cell chemotaxis and phagocytosis (4-6).

Such immune deficiency would explain the occasionally aggressive nature of NP in HIV-infected individuals with CD4+ cell counts under 200 cells/mm<sup>3</sup>, or in situations of severe malnutrition. A clinical characteristic of such infection is tissue invasion by the causal microorganisms. However, this has also been seen in patients with very low CD4+ cell counts who do not develop NP; as a result, other factors in addition to immune depression have been suggested to play a role (7,8).

Classically, NP has been associated to weakening situations and intense stress. The disease is characterized by the presence of ulcerated punch-like or cratered lesions in the crests of the interproximal papillae that can spread towards the marginal gingival tissue. These craters are covered by a pseudomembrane surrounded by an erythematous halo, though the membrane is sometimes missing. The lesion gradually destroys the gingival margin and the underlying periodontal tissues. Necrosis in turn produces a foul odor and metallic taste. The anterior lower and upper sectors are the most commonly affected zones. Local adenopathies are sometimes observed. The lesions tend to progress indefinitely or can show spontaneous limitation, though recurrences are common (9). Takayasu's disease is a granulomatous vasculitis of unknown origin that affects the aorta and/or its main branches, though the pulmonary, femoral, renal, mesenteric and vertebral arteries can also be affected (10).

The present study describes a very aggressive case of NP in a woman with Takayasu's disease secondary to immunosuppressor treatment.

## Clinical Case

We present the case of a 40-year-old woman diagnosed

with Takayasu's disease and with a history of arterial hypertension, hypercholesterolemia and ischemic heart disease. She had been treated for vascular lesions related to Takayasu's disease in the *arteria intermedia*, with placement of a stent in the right subclavian artery, and had undergone surgical clipping due to a saccular aneurysm of the anterior communicating artery.

The patient presented no toxic habits and was on a low-salt and lipid-lowering diet. She was receiving omeprazole 20 mg (1 tablet/day), antiplatelet medication such as aspirin 100 mg (1 tablet/day) and clopidogrel 75 mg (1 tablet/day), antihypertensive agents such as ramipril (tablet/day) and atenolol (1 tablet/12 hours), and immunosuppressive therapy with methotrexate (4 tablets/week, though she had suspended this treatment two weeks before the visit) and one polinic acid tablet/week.

The patient was referred to the Stomatology Unit of Dr. Peset University Hospital (Valencia, Spain) for the evaluation of oral lesions. At exploration, she presented ulcerative gingival lesions with a necrotic crater, surrounded by an erythematous halo, and which in some cases extended to remnant zones of swollen and intensely erythematous papillae that showed spontaneous bleeding (Fig. 1).



Fig. 1. Clinical view of gingival lesions.

The lesions were more manifest in the anterior sector of both arches (Fig. 2). In the region of the lower incisors the periodontal destruction was very evident. Clinically,



Fig. 2. Magnified detail of anterior sector.

the lesions were associated with intense pain and a foul odor. The patient presented abundant bacterial plaque and reported poor oral hygiene. No adenopathies were detected upon palpation.

A panoramic X-ray study revealed generalized horizontal loss of alveolar bone, particularly marked in the region of the lower incisors (Fig. 3).



**Fig. 3.** View of panoramic X-ray with a generalized loss of alveolar bone.

A small gingival tissue sample of posterior area was obtained to evaluate possible microvasculitic lesions of the gingival blood vessels, in consonance with very infrequent lesions that have been reported in the skin of patients with Takayasu's disease. The histological findings discarded the presence of vasculitis in this location. However, an important nonspecific infiltrate was observed over the entire chorion. Such an infiltrate can also be seen in the lesions associated with necrotizing periodontal diseases (NG and NP), exhibiting an inflammatory exudate with areas of necrosis, fibrin pseudomembranes with fragments of necrotic epithelium, neutrophils and polymorphonuclear cells, as well as the presence of microorganisms (12). The vessels of the chorion were dilated and densely populated by polymorphonuclear cells and rounded plasmocyte-type cells.

A dental prophylaxis was performed initially, 3% hydrogen peroxide was associated as a rinse (twice a day, 10 days) and metronidazol was prescribed systemically (500 mg/8 h, 10 days). Acute pain disappeared rapidly and gingival inflammation was reduced significantly in the first week. Detailed scaling and root planning was done 7 days after the initial treatment and this improved the periodontal clinical results. Gingival contour remained affected but without inflammation. Patient was sent for periodontal maintenance every 4 months.

## Discussion

Takayasu's disease is an infrequent chronic disorder characterized by inflammatory vasculitis affecting mainly the aorta and its main branches, as well as the pulmonary and coronary arteries. The mean age at onset of the

disease is 35-40 years, and most patients are women. The diagnostic criteria of Takayasu's disease include the presence of lesions in at least one of the two subclavian arteries, and characteristic signs and symptoms for a period of at least one month. These signs comprise limb claudication, the loss of pulse or differences in pulse between limbs, transient amaurosis, blurry vision, dyspnea, palpitations or syncope. There is a relationship between the anatomical location of the lesions and the clinical manifestations of the disease (10,11).

Of note in our patient was the severity of the lesions, which had been present for at least two weeks, with a predominance of necrotic periodontal destruction.

The fact that she had been receiving methotrexate immunosuppressive therapy for the control of her vasculitis may have facilitated the development of the lesions, triggered by local factors such as the presence of abundant bacterial plaque and previous chronic periodontal lesions. However, the patient neither smoked nor consumed alcohol, and presented no evident psychological stress.

GN and NP diagnostic is based on the typical clinical signs and symptoms, the histopathology does not help for diagnostic purposes and biopsy shouldn't be performed in necrotic and infected gingival areas. Patient had a dental history of chronic periodontitis and both periodontal pathologies could coexist in this case. The differential diagnosis was established with systemic hematological processes that can also involve immune deficiencies, such as neutropenia or agranulocytosis and leukemia (13).

Emergency treatment in these cases should include control of the local factors, with very careful tartar removal to eliminate the plaque and dental calculus (14,15). Likewise, gentle tooth brushing should be recommended, with chemical plaque control and antibiotics as amoxicillin / clavulanic acid (875 / 125 mg every 8 hours for one week) or metronidazol (500 mg every 8 hours) can also be prescribed. However, in immune depressed patients this may favor the appearance of candidiasis; as a result, an antimycotic such as nystatin is advised. Some studies have reported complex cases in which NP proves refractory to treatment.

Although the systemic conditions are important in necrotizing periodontal disease, local factors such as deficient oral hygiene, smoking or previous gingival irritation are also very important (1,3). The microorganisms of dental plaque are known to initiate periodontal alteration, and its progression is decisively influenced by the host immune condition – to the point where its clinical appearance has been regarded as a marker of immune deterioration (15). Thus, both the systemic alterations and the use of immunosuppressor drugs can influence the development, frequency and severity of the disorder. Likewise, the appearance of NP may be an early sign of

imminent serious disease.

## References

1. Novak MJ. Necrotizing ulcerative periodontitis. *Ann periodontol.* 1999; 4:74-8.
2. Barr CE, Robbins MR. Clinical and radiographic presentations of HIV-1 necrotizing ulcerative periodontitis. *Spec Care Dentist.* 1996; 16:237-41.
3. Bermejo-Fenoll A, Sánchez-Pérez A. Necrotising periodontal diseases. *Med Oral Patol Oral Cir Bucal.* 2004; 9 Suppl:114-9;108-14.
4. Mikx FMH, Van Campen GJ. Preliminary evaluation of the microflora in spontaneous and induced necrotizing ulcerative gingivitis in the beagle dog. *J Periodontal Res.* 1982; 17:460-1.
5. Rowland RW, Mestecky J, Gunsolley JC. Serum IgG and IgM levels to bacterial antigens in necrotizing ulcerative gingivitis. *J Periodontol.* 1993; 64:195-201.
6. Brook I. Microbiology and management of periodontal infections. *Gen Dent.* 2003; 51:424-8.
7. Phiri R, Feller L, Blignaut E. The severity, extend and recurrence of necrotizing periodontal disease in relation to HIV status and CD4+ T cell count. *J Int Acad Periodontol.* 2010; 12:98-103.
8. Jiménez LM, Duque FL, Baer PN, Jiménez SB. Necrotizing ulcerative periodontal disease in children and young adults in Medellín, Colombia 1965-2000. *J Int Acad Periodontol.* 2005; 7:55-63.
9. Horning GM, Cohen ME. Necrotizing ulcerative gingivitis, periodontitis, and stomatitis: clinical staging and predisposing factors. *J Periodontol.* 1995; 66:990-8.
10. Kurien M, Bottomley JR, McGrath EE. Takayasu arteritis. *Eur J Cardiothorac Surg.* 2011 May 4(Epub ahead of print).
11. Seyahi E, Ugurlu S, Cumali R, Balci H, Seyahi N, Yurdakul S, Yazici H. Atherosclerosis in Takayasu arteritis. *Ann Rheum Dis.* 2006; 65:1202-7.
12. Guvenc D, Gokbuget AY, Cintan S, Kara G, Cifcibasi EY, Unuvar E et al. An atypical form of necrotizing periodontitis. *J Periodontol.* 2009;80:1548-53.
13. Horning GM. Necrotizing gingivostomatitis: NUG to noma. *Compend Contin Edu Dent.* 1996; 17:951-4, 956, 957-8.
14. Umezudike KA, Savage KO, Ayanbadejo PO, Akanmu SA. Severe presentation of necrotizing ulcerative periodontitis in a Nigerian HIV-Positive: A Case Report. *Med Princ Pract.* 2011; 20:374-6.
15. Batista EL Jr, Novaes AB Jr, Calvano LM, do Prado EA, Goudouris ES, Batista FC. Necrotizing ulcerative periodontitis associated with severe congenital immunodeficiency in a prepubescent subject: clinical findings and response to intravenous immunoglobulin treatment. *J Clin Periodontol.* 1999; 26:499-504.