

Necrotizing Periodontal Disease in a Patient with Leukocytoclastic Vasculitis

Andrea Nelken, Matteo Basso, Luca Francetti, Massimo Del Fabbro, Tiziano Testori, Roberto L. Weinstein

The vasculitides are a group of syndromes marked by inflammation and necrosis of blood vessels of various calibres, with luminal modifications and ischemia-induced changes in one or more organs supplied. They are primarily the expression of the tissue ischemia provoked by the damaged vessel. Their etiology is still uncertain, although viral infections are thought to be responsible for some forms.

Periodontal tissues may be involved by the vessel-degenerative process. This article describes a case of leukocytoclastic vasculitis in a 23-year-old Caucasian male patient with severe necrotizing periodontitis: an average 4.7 mm full-mouth attachment loss measured involvement of the furcations and alveolar bone loss. It provides an illustration of how systemic diseases may predispose to necrotizing periodontal disease, in this case caused by tissue ischemia, and how clinicians can provide appropriate dental management of damaged oral tissues.

Key words: vasculitis, necrotizing periodontitis, periodontal disease

INTRODUCTION

Vasculitides are a group of syndromes marked by inflammation and necrosis of blood vessels of various calibres, with luminal impairment and ischemia-induced changes in the tissues supplied (Hunder et al, 1996; Rao et al, 1998; Jennette et al, 1994). Vasculitis was first recognised as a disease in 1801 when Heberden described a case in a five-year-old child. Further descriptions were provided by Schönlein in 1837 and Henoch in 1874 (Hunder et al, 1996).

Arteries, arterioles, capillaries, venules and in some cases the major veins may be involved in wall inflammation. Damage to internal organs, especially the kidneys and the heart, is very frequent, along with typical skin lesions with deposition of red cells (purpura) (Hunder et al, 1996; Braunwald et al, 2001, Tierney et al, 2003) However, sometimes all body areas may be involved, including oral and periodontal tissues.

The etiology of vasculitis is not entirely clear. It is probably the expression of an autoimmune mechanism whereby damage is due to immune complex-

es generated by exposure to a specific antigen, such as an infectious agent, a drug or other endogenous and exogenous substances (Braunwald et al, 2001, Tierney et al, 2003) The mechanisms of tissue damages induced by immunocomplexes are similar to those typical of serum disease (Kadison and Haynes, 1988). Complexes of antigen-antibody, formed in presence of antigen excess, deposit into the vessel walls where the permeability is augmented by release of vasoactive substances (such as histamine or leucotriens) from platelets or IgE-mediated mast-cells degranulation. The deposition of complexes determines complement activation and, above all, C5a formation, which detains high chemotactic activity on neutrophils. Then, neutrophils infiltrate vessel walls and phagocytize complexes, releasing intracytoplasmatic enzymes that determine vascular damage. When the process becomes subacute or chronic, mononucleated cells also infiltrate the vessel walls. As a consequence of this, luminal impairments appear with ischemic alterations at various tissues. Other immunological mechanisms may cause vascular damage. Among these, the most important

Systematic necrotizing vasculitis Polyarteritis nodosa Churg-Strauss syndrome Polyangiitis overlap syndrome
Hypersensitivity vasculitis Exogenous stimuli proved or suspected: Henoch-Schönlein purpura Leukocytoclastic vasculitis Serum sickness and serum sickness-like reactions Other drug-induced vasculitides Vasculitis associated with infective diseases Endogenous antigens likely to be involved: Neoplasms-related vasculitis Connective tissue disorders-related vasculitis Vasculitis associated with other underlying diseases Vasculitis associated with complementing congenital dysfunctions
Wegener`s granulomatosis
Giant cell arteritis Giant cell (temporal) arteritis Takayasu`s arteritis
Other vasculitis syndromes Kawasaki`s disease Buerger`s disease Behcet`s syndrome Other vasculitides

Table 1 Vasculitis classification (Braunwald et al, 2001)

Exam	January 1999 results	May 2003 results
pANCA (perinuclear)	Absent	Absent
cANCA (cytoplasmatic)	Absent	Absent
Leukocytes (normal: 4–10 ³ µl)	14.4	8.2
HBV Ag and Ab	Negative	Negative
HCV Ab	Negative	Negative

Table 2 Laboratory tests

are represented by retarded hypersensitivity or by immunological cell-mediated damages, typical of granulomatosis vasculitis. Furthermore, in Wegener`s granulomatosis, antibodies against cytoplasmatic elements of neutrophils have been described, but their role, if it does exist, is not clearly defined. Lastly, genetic features cannot be excluded in describing vasculitis patogenesis, because genetics may explain why the same conditions cause vasculitis disease in certain patients and not in others, but genetic involvement has not yet been identified.

Different aspects of vessel and organ involvement give high heterogeneity to this family of diseases. Differentiation criteria have also been defined by

several authors (Hunder et al, 1990; Rao et al, 1998; Braunwald et al,2001; Tierney et al, 2003). A common classification, made on clinical and laboratory findings, is reported in Table 1. Hypersensitivity vasculitides are the most common. They form a heterogeneous group of disorders whose common feature is the involvement of small vessels, usually the postcapillary venules. The internal organs may be involved, but their involvement is generally less severe in hypersensitivity vasculitides than in other systemic vasculitides. Skin purpura is commonly present.



Fig. 1 Necrotizing periodontitis in a patient with leukocytoclastic vasculitis. Note the irregular morphology of the gingival margin as determined by the progressive loss of the interdental papillae. Texture alterations of the gingiva, gingival recessions (in the frontal regions) and supragingival calculus deposits are also evident. was root-amputated, thereby conserving the crown.



Fig. 2 Six months after treatment. The result of treatment is satisfactory, oral hygiene is improved. Patient is satisfied.

CASE DESCRIPTION

A 23-years-old Caucasian male was seen in December 2003 for severe periodontal disease at the Department of Odontology, Section of Periodontology, University of Milan at the Galeazzi Orthopaedic Institute, Italy.

The anamnesis collected before treatment disclosed hospitalisation in December 1998 owing to the appearance of lower-limb hemorrhagic lesions and swelling of the large joints (palpable purpura). Different laboratory analysis were performed in order to identify the disease, such as blood tests, research for antibodies against neutrophils cytoplasm components (ANCA tests) (Gross et al, 1993), and antinucleus antibodies (ANA test) and a skin biopsy were employed to properly identify the pathology (Gross et al, 1993). The most important laboratory tests results are reported in Table 2. From the clinical aspects and laboratory findings the final diagnosis of small-vessel, leukocytoclastic vasculitis was arrived at. Since this vasculitis is classified as a hypersensitivity vasculitis, corticosteroid therapy (methylprednisolone), sucralofate and mysoprostole were administered by specialists immediately after diagnosis to control the first acute phase. Initial clinical manifestations regressed with described corticosteroids therapy, while complete vasculitis symptomology regressed after six months with all typical clinical aspects of

purpura and joint pain. Blood analysis was performed after six months and repeated after one, two, three and four years in order to control general parameters.

ORAL FINDINGS

At the time of seeing the patient for periodontal treatment, in December 2003, there was no evidence of any general sign of vasculitis. Anamnesis did not reveal other local or systemic disease. His blood pressure appeared normal. His cigarette consumption was quite light – about five cigarettes a day. No regular consumption of drugs for any therapies was revealed in anamnesis. The patient's family history was negative for periodontitis and vasculitis. Anamnesis revealed absence of periodic professional dental attention. The intraoral examination showed poor dental care and supra- and subgingival mineralized and non-mineralized plaque deposits (Fig 1). There were pronounced upper and lower frontal gingival recessions, with signs of necrotizing periodontitis (Fig 3). The clinical picture also included increased mobility of some teeth, foetor ex ore, root dentin sensitivity and severe pain. Periodontal charting (Nyman and Lindhe, 2003) identified attachment loss on all teeth with average periodontal attachment loss (PAL) of 4.71 mm, while the average probing



Fig. 3 Elements 12 and 13 before treatment.



Fig. 4 Elements 12 and 13 six months after treatment.



Fig. 5 Orthopantomography (OPT).

pocket depth (PPD) was 3.4 mm, with involvement of several furcations. The full mouth plaque score (FMPS) was 45% and the full mouth bleeding score (FMBS) was 36%.

The patient brought his orthopantomographic rx full-arch exam (Fig 5) and the radiographic dental status (Gross et al, 1993) (Fig 6) revealed horizontal bone resorption and inclusion of 18, 28, 37, 38 and 48. Thirty-five and 45 were missing, and symptomatic decay of the mesial surface of 17 was also evident.

Necrotizing periodontitis associated with systemic disease (Armitage, 1999; Page and Schroeder, 1976) was diagnosed. The possible association between necrotizing periodontitis and vasculitis was immediately suspected because the patient did not declare any dental involvement and, above all, he did not experience bleeding and severe pain before the vasculitis emerged.

Before any periodontal procedure was started, oral hygiene instructions (Dahlen et al, 1992) were given. This was particularly difficult to perform at the beginning of treatment owing to severe pain. Composite resin was used to splint the anterior mandibular teeth from tooth 33 to tooth 43 and element 12 with 22. Extraction of teeth 26, 27 and 36 was planned, but the patient wanted to maintain those molar teeth. He was informed about all risks of maintaining periodontal compromised teeth. The authors preferred to evaluate the patient's ability in cleaning teeth before deciding with him on the future of his molars. Element 17 was treated endodontically and restored.

Since necrotizing periodontitis was not in the acute phase, long-term antibiotic therapy was avoided. The patient was instructed to rinse his mouth with a 0.2% chlorhexidine (CHX) solution twice a day for two weeks (Francetti et al, 2000). Due to the duration of CHX therapy the authors decided to prescribe a solution with an anti-discoloration system in order to avoid teeth pigmentation (Addy et al, 1991; Bernardi et al, 2004) (Curasept CHX 0.2% with Anti Discoloration System (ADS®), Curaden, Italy) and two grams of amoxicillin plus clavulanic acid (Augmentin tablets one gram, Smithkline & Beecham, Italy) were prescribed as prophylaxis one hour before each of two supragingival scaling sessions (Cobb, 1996). Subsequently, ultrasonic subgingival scaling and debridement sessions were conducted under local anaesthesia (Mepivacain 2% with adrenalin 1:100.000, Astra Zeneca, Sweden) and the same antibiotic cover

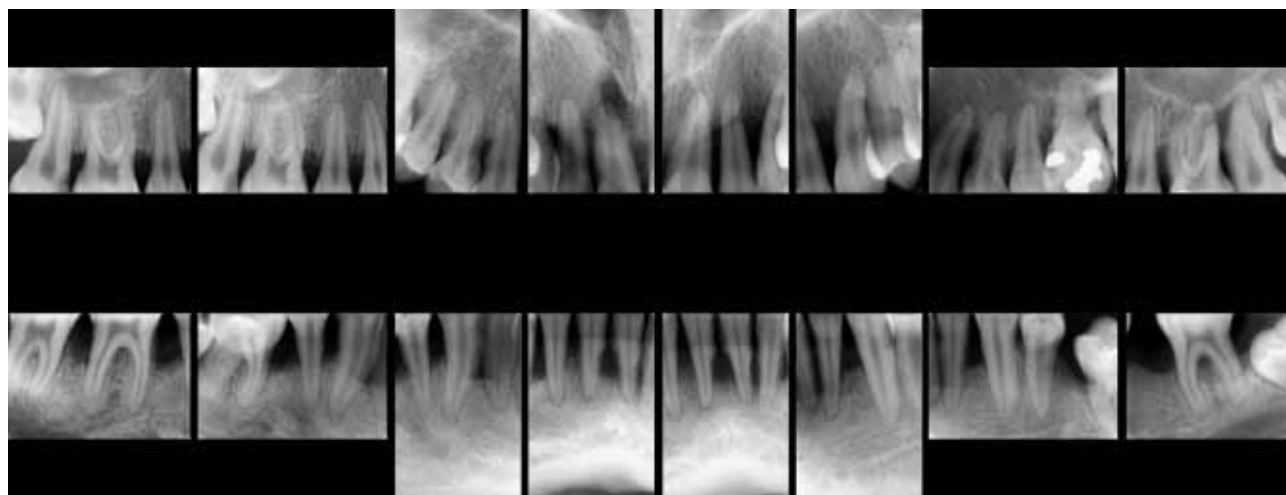


Fig. 6 Periodontal rx status executed before periodontal treatment in 2003

described for supragingival therapy. Polishing was then performed with fluoride light-abrasive paste and a rubber cap (Cobb, 1996; Badersten, 1984).

In view of the attachment loss, the medium pocket depth and the need for antibiotic cover, it was decided to conduct only two sessions of subgingival periodontal therapy (one for each jaw) over the course of a few days. No post-operative antibiotics were administered, while further oral hygiene instructions were reinforced. Dental care with toothbrush, dental floss and interdental brush was prescribed and recommended.

The patient was recalled monthly for professional tooth cleaning and reinforcement of his own oral hygiene measures. Instruction in gentle but effective brushing is essential. In cases like this, tissue destruction leads to residual soft-tissue defects that are difficult for the patient to keep clean.

The patient was re-evaluated (Rosling et al, 2001; Ramfjord, 1987) with periodontal charting after three months, when he showed diminished pain and root dentin sensitivity. Pain on the treated tooth 17 vanished. The patient was not motivated to carry out proper oral hygiene, and the FMPS was 67%. Poor oral hygiene habits are frequently reported in patients with necrotizing periodontitis, which may be averse to dental treatment in general. Periodontal treatment success was, however, confirmed by the improved FMBS (4% of total surfaces) and the average PPD was reduced at three mm. Further oral hygiene instructions were again given. Further control was finally performed at six months after therapy. This time the patient showed satis-

factory oral hygiene (Fig 2), showing FMPS of 20 % and FMBS of 4%. Plaque deposits were mainly found in recessions and furcations of those molar elements that we had previously recommended for extraction. The need for extraction was emphasized once more, but the patient again decided to keep his teeth. The gingival anatomy appeared heavily affected by the periodontal pathology (Fig 4) but there was reduced inflammation, pain and sensitivity. The patient reported improved social relationships, especially due to the disappearance of halitosis (foetor ex ore) and pain during mastication, changing his quality of life.

DISCUSSION

The etiology of vasculitis is still unclear. Diagnosis is often made difficult by the non-specificity of the symptoms and/or the absence of an explanation for the altered function of one or more organs. It is rarely clear from the start that vasculitis is responsible for the symptomology. Differential diagnosis, in fact, is directed at determining its presence and, wherever possible, its precise definition, since some forms, such as Schönlein-Henoch purpura and the hypersensitivity vasculitides are benign or self-limiting.

A detailed case history is needed to find out whether a patient has recently been on drugs (hypersensitivity vasculitides), suffered from viral diseases such as hepatitis B or C or from diseases frequently concomitant with vasculitis, such as lupus. In the absence of specific tests, the diagnosis of in-

dividual forms of vasculitis must still be based on the type and size of the vessels involved, the clinical picture and the laboratory findings. C-ANCA (cytoplasmatic antibodies test), for example, identifies about 90% of cases of Wegener's granulomatosis, while P-ANCA (perinuclear antibodies test), if positive, is used to diagnose polyarteritis nodosa and the necrotizing vasculitides. A biopsy is, however essential, to clinch the diagnosis.

The interest in this case lies in the association of necrotizing periodontitis with vasculitis. It seems, in fact, that the latter may result in increased susceptibility to generalized periodontal tissue breakdown. International literature is really poor as far as descriptions on the relationship between vasculitis and periodontal disease are concerned, even if the afflicted structures (vessels walls) may also involve periodontal tissues. Some specific vasculitic syndromes, such as Behçet syndrome, are characterized by oral involvement, typically mucous membrane ulcerations, confirming the possible relationship with the oral manifestations described in this case report. Furthermore, in this case the patient declared severe oral involvement, with pain and bleeding, only after vasculitis-related symptoms appeared.

Treatment of this kind of patient should obviously be medical as well as dental. Many cases of hypersensitivity vasculitis subside spontaneously, whereas the course of other forms, such as Schönlein-Henoch purpura, is marked by regression and recurrence until complete disappearance. Treatment is directed to the presumed triggering factor, if existing or identified (infections or a concomitant disorder). Alternatively, corticosteroids are commonly used. Cytotoxic drugs such as methotrexate are administered if no response is obtained, but this kind of therapy, following general guidelines for vasculitis, should be avoided when possible because of possible adverse systemic reactions.

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Reprint requests:

Andrea Nelken
 University of Mailand, Dept. of Odontology
 Galeazzi Institute
 Via R. Galeazzi 4
 20161 Milano (Italy)
 Fax: +39 02 50319960
 E-mail: nelken2@libero.it