Projeto: Avaliação de células CD30+ na gengivite de pacientes HIV-positivos.

Ref. CBS 1999/95

Data de Início: 18/10/97 Término: 17/10/97

Coordenador da Pesquisa: Ricardo Santiago Gomez

O projeto foi encerrado dentro dos prazos propostos e os resultados já foram publicados no Journal of Periodontology (1997;68:881-883). Os recursos obtidos com este projeto também contribuíram para as publicações e as dissertações listadas abaixo:

Trabalhos Científicos Publicados em Revistas Internacionais com Corpo Editorial


6) Barreto DC, Gomez RS, Carmo MAV. AgNOR in benign fibro osseous lesion. Medical Science Research 1998 (no prelo).

Observação: cópias dos trabalhos em anexo
UNIVERSIDADE FEDERAL DE MINAS GERAIS
Trabalhos Publicados em Revistas Nacionais com Corpo Editorial


Dissertações de mestrado defendidas

1) “Demonstração da expressão de citoqueratinas nos cistos e tumores odontogênicos” – Dissertação de mestrado defendida no Curso de Pós-Graduação em Patologia, área de Odontologia, da Faculdade de Medicina da UFMG. 
Aluna: Júlia Noronha Carvalhais 
Orientador: Ricardo Santiago Gomez 
Data: 13/3/97.

2) “Análise quantitativa de células de Langerhans em periodontites de início precoce e periodontite do adulto” – Dissertação de mestrado defendida no Pós-Graduação em Odontologia, área de concentração Periodontia, da Faculdade de Odontologia da UFMG. 
Aluna: Andréa Mara de Oliveira 
Orientador: Ricardo Santiago Gomez e José Eustáquio da Costa 
Data: 11/7/97.

3) “Estudo imuno-histoquímico das lesões ósseas de células gigantes” – Dissertação de mestrado do Curso de Pós-Graduação em Patologia Médica da Faculdade de Medicina da UFMG. 
Aluna: Júlia Filardi Jardim. 
Orientador: Ricardo Santiago Gomez 
Data: 5/12/97.

Prof. Ricardo Santiago Gomez
CD30+ Lymphocytes in Chronic Gingivitis From HIV-Positive Patients: A Pilot Study

Ricardo Santiago Gomez,* Paulo Eduardo Alencar de Souza,* José Eustáquio da Costa,* and Ney Soares Araújo*

Th2 type lymphocytes are characterized by high expression of CD30 glycoprotein. Increased serum levels of CD30 and Th2 IL-4 producing T-cells are found during AIDS progression. Since HIV-positive patients are more susceptible to periodontal disease, quantitative analysis of positive cells for the CD30 receptor in chronic gingivitis of both HIV-infected and non-infected patients (NSG) would help to clarify the immunoregulation of HIV-associated periodontal diseases. The purpose of this study was to evaluate CD30+ lymphocytes in gingival biopsies from sites exhibiting chronic gingivitis on HIV-positive patients (CG-HIV) and NSG. A biotin-streptavidin amplified system was used for identification of the CD30 receptor. The results demonstrated increased proportions of Th2 cells in CG-HIV as compared to NSG. Additional studies are necessary to understand the importance of these cells to the biological activity or inactivity of the disease. J Periodontol 1997;68:881–883.

Key Words: Acquired immunodeficiency syndrome; HIV infections; gingivitis/epidemiology; periodontal diseases/epidemiology; lymphocytes.

Mice and human CD4+ lymphocytes express functionally heterogeneous profiles of cytokine production.12 Mouse Th1 cells produce interleukin 2 (IL-2), interferon γ (IFN-γ) and lymphotoxin, whereas Th2 cells produce IL-4, IL-5, IL-6, IL-9, IL-10, and IL-13. Human Th1 and Th2 cells produce similar patterns, although IL-2, IL-6, IL-10, and IL-13 synthesis is not as restricted to a single subset. Until recently, these types of lymphocytes could only be discriminated by using immunoassays for cytokine identification. CD30 is a membrane-bound glycoprotein originally described as a surface molecule recognized by the Ki-l monoclonal antibody on Hodgkin’s and Reed-Sternberg cells in patients with Hodgkin’s disease.13 Under normal conditions CD30+ cells are not identified in peripheral blood.4 A strict association between CD30 expression and the production of Th2 type cytokines has been reported in the literature.2 Th2 dominant pattern of cytokine production and elevated serum levels of CD30 are observed in HIV-positive patients during progression to AIDS.2,6

Various forms of periodontal disease have been described in HIV-infected patients. Some of them, such as linear gingival erythema and necrotizing periodontitis, are very specific, while others, like chronic gingivitis, are clinically similar to conventional non-specific gingivitis (NSG). It is well known that HIV-positive patients are more susceptible to periodontal disease.13,8 Furthermore, Th1 and Th2 lymphocytes have not been evaluated on periodontal disease by CD30 expression. Therefore, quantitative analysis of positive cells for this receptor in chronic gingivitis of both HIV-infected and non-infected patients would help to clarify the immunoregulation of HIV-associated periodontal diseases. The purpose of this study is to compare relative populations of Th2 cells (CD30+) in gingival biopsies from sites exhibiting chronic gingivitis in HIV-positive patients (CG-HIV) and NSG.

MATERIALS AND METHODS

Five biopsies of CG-HIV were retrieved from the files of Minas Gerais Federal University Service of Oral Pathology. These biopsies were from 5 HIV-positive patients with clinical symptoms corresponding to acquired immune deficiency syndrome (AIDS). The age, sex, race, and location of the biopsy specimens are presented in Table 1. The intensity of inflammatory cell infiltrate was assessed by counting all the cells at 8 microscopic fields (1,000×) on stained sections. All of the CG-HIV showed severe inflammatory cell infiltration, i.e., more than 190 cells. Ten NSG biopsies of sex-, age-, and race-matched
HIV-negative patients were also retrieved from the same location as the CG-HIV cases. After matching NSG and CG-HIV cases by the intensity of the histological inflammatory cell infiltration, 5 biopsies of the former were selected and included in the study. All of these specimens had dense inflammatory cell infiltration. The average of total inflammatory cell counts in CG-HIV was 291 (ranged from 198 to 327) and in NSG was 258 (ranged from 190 to 289).

**Immunohistochemical Methods**

After matching both groups, 3 μm of formalin-fixed, paraffin-embedded tissue blocks of the samples selected were cut and subjected to the biotin-streptavidin amplified system. Since formalin fixation and paraffin-wax embedding interfere with immunocytochemical detection of CD30 antigen, a microwave stimulation as described by Shi et al. 10 and Cattoretti et al. 11 was carried out. The slides were submitted to a microwave/citrate buffer pre-treatment for 10 minutes. Sections were then immersed in 3% methanol-hydrogen peroxide solution for 10 minutes to block endogenous peroxidase activity and incubated with anti-Ki-1 antigen (CD30) diluted 1:20 in 5mM tris-HCl buffer for 18 hours at 4°C with 1% bovine serum albumin. After washing in tris-HCl buffer (pH 7.4), the sections were incubated at room temperature with: 1) biotinylated rabbit anti-mouse immunoglobulin diluted 1:400 in tris-HCl for 30 minutes; 2) washed with tris-HCl twice for 10 minutes; 3) incubated for 30 minutes with horseradish peroxidase-conjugated streptavidin prepared according to the manufacturer’s directions; 4) washed with tris-HCl; 5) incubated for 3 minutes with 0.03% diaminobenzidine tetrahydrochloride (DAB) and 0.3% H2O2 in 5mM tris-HCl buffer at pH 7.4; and 6) rinsed in distilled H2O for 10 minutes and counterstained with hematoxylin. Omission of the primary antibodies was performed for negative controls, and sections of tonsil were used for positive controls. One section of 5μm was used for the conventional H&E method.

**Morphometric Analysis**

Cell counting was performed in duplicate at 8 microscopic fields at the epithelium/connective tissue interface and 8 at the central lamina propria by two investigators at 1,000× magnification through a square micrometer (0.025 mm2). The definitions of the first microscopic field were established by adjusting the square micrometer at the apical interface of the junctional epithelium/connective tissue. The 8 microscopic fields of the central connective tissue were selected randomly. All specimens had more than 188 infiltrated cells on the 16 microscopic fields counted. The results were expressed as the percentage of positive cells.

**Statistical Analysis**

Since percentage values do not demonstrate a normal distribution, angular transformation (angle = arcsin √%) was applied as suggested by Snedecor and Cochran. 12 The mean values of the angles of both groups were compared by t-test. The error in cell counts was determined by 6 double counts in different areas and was below 0.8% of the cell-specific counts.

**RESULTS**

The percentage of CD30+ cells in CG-HIV and NSG cases is shown in Table 1. The mean ± SD percentage of CD30+ cells in CG-HIV (3.52 ± 1.14) was statistically greater than NSG (1.14 ± 0.60) (P = 0.0029) after angular transformation (Table 1). Although great variation of these cell distributions was observed, no positive intrapithelial cells were identified. All the samples exhibited positive cells for this antigen. Most of the Ki-1 positive cells were large lymphocytes (Fig. 1).

**DISCUSSION**

HIV infection has often been associated with oral diseases such as candidiasis, hairy leukoplakia, Kaposi's sarcoma, and severe periodontal disease. 13 Although many clinical and microbiologic studies of HIV-associated peri-
REFERENCES


Acknowledgments

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Peripheral dentinogenic ghost-cell tumor: A case report

Wagner Henriques Castro*/Maria Cássia Ferreira de Aguiar*/Ricardo Santiago Gomez*

Abstract  The dentinogenic ghost-cell tumor is a rare solid variant of the calcifying odontogenic cyst. Few peripheral cases of this tumor with clinical and radiographic documentation have been reported. A case of peripheral dentinogenic ghost-cell tumor is presented and the literature is reviewed.

Introduction
The dentinogenic ghost-cell tumor (DGCT) is an uncommon odontogenic neoplasm, regarded as a solid variant of the calcifying odontogenic cyst.1 This neoplasm occurs predominantly in later life and consists microscopically of ameloblastoma-like strands and islands of odontogenic epithelium with ghost cells and dentinoid material. Peripheral occurrence of DGCT is rare, and few reports with clinical, radiographic, and histologic documentation can be found in the English literature.2–3 The purpose of this report is to present a case of peripheral DGCT and review the literature.

Case report
An 83-year-old black woman was referred to the Surgery Service, Minas Gerais University, School of Dentistry, for evaluation of a painless, slow-growing tumoral lesion on the anterior ridge of an edentulous mandible. The polyoid sessile lesion had been noted 2 years earlier (Fig 1). Radiographic examination disclosed cortical resorption in a cup-shaped fashion and faint and diffuse opacities within the bulk of the lesion (Fig 2). The clinical diagnosis was peripheral ossifying fibroma. An excisional biopsy of the tissue lesion was performed, and the specimen was submitted for histopathologic evaluation.

The gross specimen consisted of a grayish piece of tissue, measuring 30 × 15 × 10 mm, and had a firm consistency. Histologic examination revealed a solid tumor with an overlying hyperplastic mucosal epithelium. The tumor mass was composed of ameloblastoma-like strands and islands of odontogenic epithelium with cuboidal to cylindrical basal cell layers and central stellate reticulum-like cells (Fig 3). These elements were associated with abundant eosinophilic ghost cells with shadowy nuclear outlines (Fig 4). Irregular foci of tissue resembling dentin were observed extruding from the islands, in direct connection with the connective tissue. A diagnosis of dentinogenic ghost-cell tumor was made. Postoperative healing was uneventful, and there were no signs of recurrence in 3 years of follow-up.

Discussion
Peripheral occurrence of DGCT is rare. Although Bhaskar4 reported two cases of peripheral odontogenic tumors that can be interpreted histologically as DGCT, bone involvement cannot be ruled out because no radiographs were presented. Some clinical reports published could also be interpreted as cases of peripheral DGCT, but because only histologic photographs were available in such studies, they were not included in the current review.5–10 Vuletin et al11

* Professor, Department of Oral Surgery and Pathology, University of Minas Gerais, Belo Horizonte, Brazil.
* Reprint requests: Dr Ricardo Santiago Gomez, Departamento de Clínica, Patologia e Cirurgia, Faculdade de Odontologia da Universidade Federal de Minas Gerais, Rua Conde de Líndares, 141, Cidade Jardim, Belo Horizonte, Brazil CEP 30380-030.
* This study was supported by Conselho Nacional de Pesquisa, Fundação de Amparo à Pesquisa do Estado de Minas Gerais, and Pró-Reitoria de Pesquisa da Universidade Federal de Minas Gerais.
reported a case of a peripheral odontogenic tumor with
ghost-cell keratinization, but because the stroma of the
tumor was composed of cellular myxoid fibroblastic
tissue, it was also excluded.

After a thorough review of the English literature,
only two cases of peripheral DGCT with radiographic
documentation were found.2,3 Günhan et al2 reported
a palatal fibrous swelling on the anterolateral portion
of the right maxilla of a 71-year-old woman, while
Raubenheimer et al3 described a slow-growing nodule
on the right alveolar ridge of an 82-year-old man. To
our knowledge, the present study shows the first
clinical documentation of this peripheral tumor.

Peripheral DGCT occurs as a nodular swelling on
the edentulous alveolar mucosa of denture wearers.

Because of the rarity of this odontogenic tumor and the
clinical aspect of the present lesion, it was initially
considered a fibrous hyperplasia. This clinical diagno-
sis was changed after occlusal radiographic examina-
tion. The cortical resorption and the presence of small
radiopacities within the lesion suggested a provisional
diagnosis of peripheral ossifying fibroma. The lesion
described by Günhan et al2 involved slight erosion of
the underlying bone.

Although central DGCTs have a high rate of
recurrence after surgery, the lack of recurrence of any
of the three peripheral tumors reported in the litera-
ture, including the present case, suggests a favorable
course for this peripheral variant.
References


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