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# Histopathological Findings of Acute Pulpitis in HIV$^{+ve}$ Patient

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**Abstract:** The purpose of the present investigation was to study the histological findings in a tooth diagnosed with acute pulpitis in a patient suffering from AIDS. Histological investigation of the pulp revealed that decay progress caused inflammatory response of the underlying pulp with different features compared to the inflammatory response found in the teeth of non-diseased individuals. The study revealed different kinds of chronic inflammatory cells and absence of polymorphonuclear leucocytes. Furthermore, widely dilated pulpal blood vessels and formation of zones, where the pulpal tissue was absent, were observed, as well as, traces of hard tissue of the osteotypic form. The inflammation was found to follow the Van Hassel model (expansion of the inflammatory reaction from the primary inflamed area towards the apices).

**Key words:** AIDS patient, acute pulpitis, histological findings

## INTRODUCTION

In a clinically, diagnosed pulpitis the histological findings are not expected to show similarities to the characteristics of an acute inflammation. However, in order to perform a study on the acute pulpitis, certain clinical criteria must be taken into account, such as, the production of secondary dentine, changes to the ground substance, vascular and cellular changes (Massler, 1967; Van Hassel, 1973; Matsaniotis, 1988).

This case report describes the histological findings of pulpitis in a diagnosed HIV positive patient. These microscopical features are discussed with a great interest on the possible synchronous correlation of the findings with the disease.

## CASE REPORT

Patient, 32 years old, from Sweden, suffered from AIDS since 1986. He was tested positive to the HIV virus in 1987. He was hospitalized several times and in 1988 started his medication. He was treated at times with Retrovir, Flagyl, Augumentin, Zidovondine, Reasec, Folic acid. His blood tests showed: lymphocyte count = 2.385 mm$^{-3}$, T4 cells = 13% or 310 mm$^{-3}$, T8 cells = 60% or 1431 mm$^{-3}$, therefore T4/T8 = 0.2. The diagnosis confirmed with ELISA immune reaction. The patient was examined in the oral pathology clinic of the dental school, where he was diagnosed with hairy leukoplakia and endodontic problems. He was, immediately, referred to the Endodontology clinic with intense pain. The examination showed acute pulpitis of the upper left second molar.


Fig. 1: Composite photomicrograph of pulp horn area next to carie (C). Irregular secondary dentine (SD) and destruction of odontoblastic layer and underlying pulp. Areas of pectic necrosis (PN)

During the removal of the carious dentine the pulpal tissue was exposed. At the patients request the tooth was extracted at the department of Dento-alveolar/Implant Surgery and Radiology of the dental school. In order for the tooth to be used in research, it was placed in a secure vial containing 10% formaldehyde solution and was kept in the vial for several days. Following that, several histological samples were prepared in the department of Oral pathology of the dental school.

Examination of the samples revealed irregular continuation of secondary dentine produced in the form

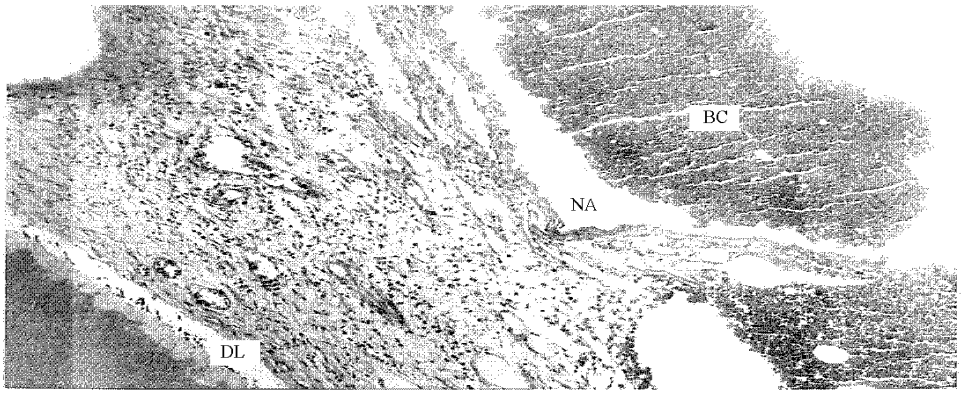


Fig. 2: Area of Fig. 1. Blood clot (BC), destruction of odontoblastic layer (OL) of the subpulpal wall of the pulp cavity. Low grade inflammatory infiltration of pulpal tissue. Necrotic areas (NA) lay the groundworks for expansion of inflammation until total pulpal necrosis

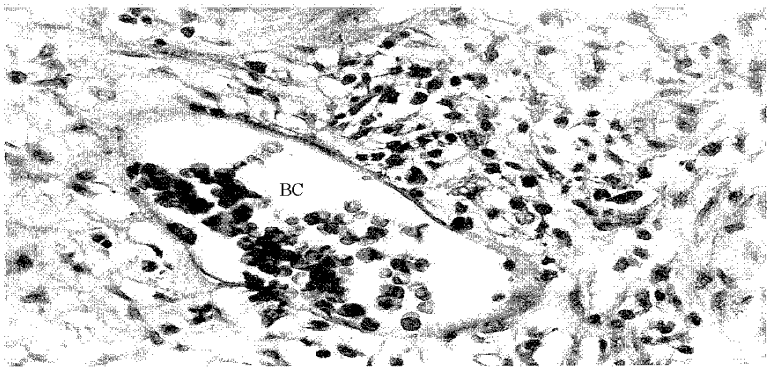


Fig. 3: Lympho-plasmo-hysticytic immune reaction against the AIDS virus. Lymphocytes, plasmocytes, hystiocytes. Dilated pulpal blood vessels full of red blood cells (BC) but no sign of margination. Note the absence of polymorphonuclear cells in the pulpal tissue

of osteodentine, masses of osteodentine within the pulp and pulpal tissue destruction with necrotic zones adjacent to the exposure site. The inflammatory infiltration of the underlying pulp was found to be of low grade (Fig. 1 and 2). In detail lymphocytes, plasmocytes and hysticytes were found. On the other hand, there was found no sign of polymorphonuclear leukocytes migration around the walls of the dilated pulpal vessels or any polymorphonuclear infiltration of the pulpal tissue. Finally, microabcesses formation was not observed (Fig. 3).

## DISCUSSION

The typical image of a patient suffering of acute inflammation of the pulp, due to a chronic effect of caries should be one that is predominated by polymorphonuclear leukocytes (Stasinopoulos, 1982). Despite the fact that the tissue infiltration was less than the one expected and that the polymorphonuclear eukocytes were completely absent, there was noted no formation of microabcesses. A cellular immuno-response was found that had grown against the HIV virus. The explanation to the white blood cells' absence is explained by the general immunosupression that AIDS causes. Numerical and functional shortage of the helper T-lymphocytes, the result of the disease, is what causes reduced signals to different cell groups (Matsianotis, 1988). AIDS results in less leukocytes and less lymphocytes produced, reduction of the count of T4-lymphocytes and of the T4/T8 proportion and reduction of the T-lymphocytes cytotoxic ability (Laskaris, 1986). Authors refer in their papers to the deduction in the chemotaxis and the phagocytic ability of the monocytes (Pinching *et al*., 1983; Mertzanos and Pararas, 1988; Oikonomidou, 1989). Damage of the T4-lymphocytes leads to functional abnormalities of other cells such as T8, NK B- lymphocytes and macrophages which lose their speciality and are driven in the overproduction of non-specific substances such as antibodies or monokines (Pavlatou, 1987).

Exposure of the pulp in long term decay results in a progressive inflammatory response characterized by the histopathological features of intense infiltration predominantly with polymorphonuclear leukocytes and of abscess formation (Stasinopoulos, 1982). The present histopathological findings in the exposed pulp of a patient diagnosed with Acquired Immunodeficiency Syndrome (AIDS) implied a limited inflammatory infiltration by lymphocytes, plasmocytes and histiocytes. The necrotic pulpal areas8 adjacent to the decayed dentine along with the normal basic architecture of the raticular pulp indicated the typical expansion process (Van Hassel, 1973). According to study, AIDS is characterized by incomplete chemotaxis and phagolytic capability of the monocytes (Pinching *et al*., 1983). The expected concentration of leukocytes in the defensive zone is impeded and therefore, the entrapment and destruction of the harmful elements is hindered. Reduced levels of the lymphosines inhibit the leukocytic migration while the disruption in the synthesis of IL-2 causes dysfunction of T-lymphocytes or macrophages or both (Murray *et al*., 1984; Bowen *et al*., 1985; Hauser *et al*., 1984; Cavailli-Coll *et al*., 1984). Existing knowledge of the limitations in the inflammatory response in AIDS patients confirm the outcome of this study. In general, HIV infection is not significantly associated with tooth loss if compared to healthy individuals (Engeland *et al*., 2008).

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