Plasma cell granuloma of the tongue. Report of a case

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Summary. A case of plasma cell granuloma of the tongue in an otherwise symptomless 48-year-old caucasian female is reported. The polyclonal nature of the plasmocytes was revealed by immunostaining of kappa and lambda light chains. Electron microscopic observations showed typical mature plasmocytes. A parasitic etiology of this type of lesion is suggested.

Key words: Plasma cell granuloma - Tongue - Human - Electron microscopy - Immunocytochemistry

Introduction

Plasma cell granuloma (PCG) is a peculiar tumor-like lesion whose etiology remains uncertain.

It is formed by aggregates of mature plasma cells intermixed with mesenchymal cells mostly of the fibroblast and histiocyte-type and arranged in a granulomatous pattern.

There are reports of PCG in different organs but to our best knowledge there is no reference in the literature concerning their presence in the tongue.

Materials and methods

A 48-year-old caucasian female presented for 4 months a slow growing hard nodule in the lateral margin of the tongue. No traumatism or inflammation of the oral cavity were noticed. There is no ulceration. There were no other nodules in the oral cavity. No systemic symptoms were observed and the laboratory investigation was normal.

For electron microscopic observations formalin-fixed material was washed in HCl-cacodilate buffer, pH 7.3, post-fixed in 1% osmium tetroxide and embedded in Epon-Araldite. Sections contrasted with uranyl acetate and lead citrate were observed in a Jeol 100C EM.

Results

The segment of the tongue resected showed a non-capsulated subepithelial nodule of 1 cm in diameter; its cut surface was whitish, firm and homogeneous.

Light microscopic examination revealed confluent aggregates of mature plasmocytes, some of them containing Russell bodies. They showed a predominant granulomatous arrangement. The granulomas also contained histiocytes and fibroblasts. They were

Fig. 1. Subepithelial dense inflammatory cell infiltration. H & E. x 100
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Fig. 2. Granulomatous arrangement of the inflammatory infiltrate, plasmocytes largely predominating. H & E. × 2.0

Fig. 3. A. Kappa light chain immunoreactivity present in the cytoplasm of the plasmocytes. PAP method with Harris hematoxylin contrast staining. × 1.050 B. Lambda light chain immunoreactivity in the cytoplasm of some plasmocytes in the same area of a consecutive section of that of Fig. 3A. PAP method with Harris hematoxylin contrast staining. × 1.050

Fig. 4. Semithin section in Epon Araldite embedded material. Mature plasmocytes, lymphocytes, histiocytes and small vessels. Alkaline toluidine blue staining. × 1.500

Fig. 5. Plasmocytes with well developed rough endoplasmic reticulum and without nuclear atypias. Uranyl acetate/ lead citrate staining. × 3.300

Discussion

The term "plasma cell granuloma" was coined by Bahadori and Liebow (1973) to describe a pseudotumoral lesion of the lung. It consists of a granulomatous arrangement of different cell types, the plasmocytes being largely predominant. This type of lesion is always well circumscribed although non-capsulated. Variable amounts of fibrous stroma are also present.
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The admixture of inflammatory cell distinguishes plasma cell granuloma from plasmocytomas which are constituted exclusively by an infiltration of neoplastic plasmocytes. Besides, neither atypical plasma cells nor mitotic figures are found in plasma cell granulomas. The presence of fibrous tissue inside the lesion and demarcating its outlines also helps on the differential diagnosis of PCG and solitary plasmocytoma.

In the present case multiple myeloma has been ruled out on the basis of conventional clinico-pathological investigations.

The polyclonal nature of the plasma cell infiltrate was documented by positive cytoplasmic staining for both kappa and lambda light chains. This distinguishes PCG from neoplastic plasma cells which constitutes a monoclonal cell proliferation (Isaacson et al., 1978).

The occurrence of PCG is rare, and its course is benign and non-recidivating upon surgical resection. Most cases of PCG have been reported in the lung of young patients (Bahadory and Liebow, 1973; Manson et al., 1982). Sporadic cases of extrapulmonary PCG have been reported in stomach (Soga et al., 1970; Isaacson et al., 1978), thyroid (Holek, 1980; Yapp et al., 1985) meninges (Eimoto et al., 1978), liver (Pack and Baker, 1953), pancreas (Abrebanel et al., 1984) and retroperitoneum (Wu et al., 1973). To our best knowledge only another case of this rare lesion was reported in the oral cavity, in the gingiva (Peison et al., 1982).

The etiology and the pathogenesis of PCG still remains unclear. PCG of the thyroid gland was suggested to be an unusual presentation of Hashimoto’s thyroiditis (Yapp et al., 1985) although typical epithelial changes of the thyroid follicles were absent. The association of PCG and hypergammaglobulinemia may be related to the existence of an intrinsic abnormality in plasma cell differentiation as was also suggested by Yapp et al. (1985).

It is difficult to find an etiological link between the cases reported so far. Nevertheless, it is interesting to note that most of the cases reported in the lung concerned people under 25 years (55 % on the series reported by Bahadory and Liebow, 1973). The morphology of the lesions strongly points to an immunologic reactive pathogenesis. We hypothesize a peculiar reaction stimulated by parasitic antigens. In fact some of the most common infestations (e.g. Ascaris) have a silent pulmonary phase of the parasite cycle. Most of the parasites infestating humans are able to evoke strong immunological reactions, acting as potent B-lymphocyte mitogens (Cohen and Warren, 1982). The persistence of parasitic remnants in the tissues due to abnormal cycles could elicit a local chronic granulomatous reaction rich in transformed B-cells, actively forming anti-parasitic immunoglobulins.

Future cases should be investigated for a past or coexistent parasitic infestation to support this hypothesis.

References


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