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Chronic atrophic rhinitis with fetor (ozena): a histopathologic tretise

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SUMMARY

Biopsy material from 50 patients with a known clinical diagnosis of ozena was examined histopathologically. Certain features were invariably seen, including mucosal atrophy, squamous metaplasia, and chronic inflammatory cell infiltrate which suggests a humoral mediated immune process. Histopathologic features allow ozena to be distinguished from chronic non-specific hypertrophic rhinitis, which may have a cell-mediated immune basis underlying its pathogenesis.

INTRODUCTION

Ozena, a specific clinical entity characterized by an atrophic chronic rhinitis with a dry, crusted appearance and the emission of a fetid odor, has a characteristic histopathologic appearance. Although the exact etiopathogenesis is obscure, infectious agents acting with constitutional (including hormonal) and environmental factors certainly play a role (Barton et al., 1980; Han-Sen, 1982). In this tretise, a detailed description of the histopathologic changes found in ozena will be discussed. These findings will be contrasted with the histopathologic findings described in chronic, non-specific, hypertrophic rhinitis.

MATERIALS AND METHODS

Fifty patients with a clinically diagnosis of ozena were selected for this study. The age of the patients ranged from 14 to 35 years; with a mediam age of 25 years. Twenty-four were males and 26 were females.

Nasal biopsies were taken under local anaesthesia (2 % xylocaine) from the inferior turbinate, one cm from the anterior end. Each specimen was divided into two parts; the first was fixed in 10 % neutral formalin and stained with hematoxylin and eosin (method of Carleton and Drury, 1957) for light microscopic examination. The second specimen (1 mm x 1 mm) was immediately fixed in 2 % buffered gluteraldehyde at pH 7.4 (as per Sabatini et al., 1963) and prepared for electron microscopic examination. These findings will be reported in a follow-up article.

HISTOPATHOLOGIC FINDINGS

Although there is some variability from one area the another, as well as from case to

case, specific histopathologic changes are invariably found in ozena. Striking is the marked atrophy of the mucosa. The normal mucosa consists of a single layer of cells which is relatively thick because of their columellar shape. Mucosa of afflicted patients have focal areas which despite being several cells thick are very thin because of the cuboidal or plate-like shape of the cells. (Figure 1). Concomittent with mucosal atrophy is squamous metaplasia; usually of the non-keratinizing variety. This metaplasia often extends into the submucosal glands (Figure 2). The combination of atrophy-metaplasia is thought to be the histological counterpart of the "crusting" seen on gross examination of the nasal mucosal regions of patients with this condition. Other important features include a marked reduction or complete disappearance seen in some material, of the mucus (goblet) cells. This is due to the atrophy and metaplasia mentioned previously and it too is contributory to crusting. In addition, the reduction or loss of goblet cells removes a defence barrier against microorganism invasion, partially explaining why these patients are susceptible to recurrent infection. Other changes include thickening of the basement membrane, a characteristic response of respiratory mucosae to chronic insult, increased vascularity within

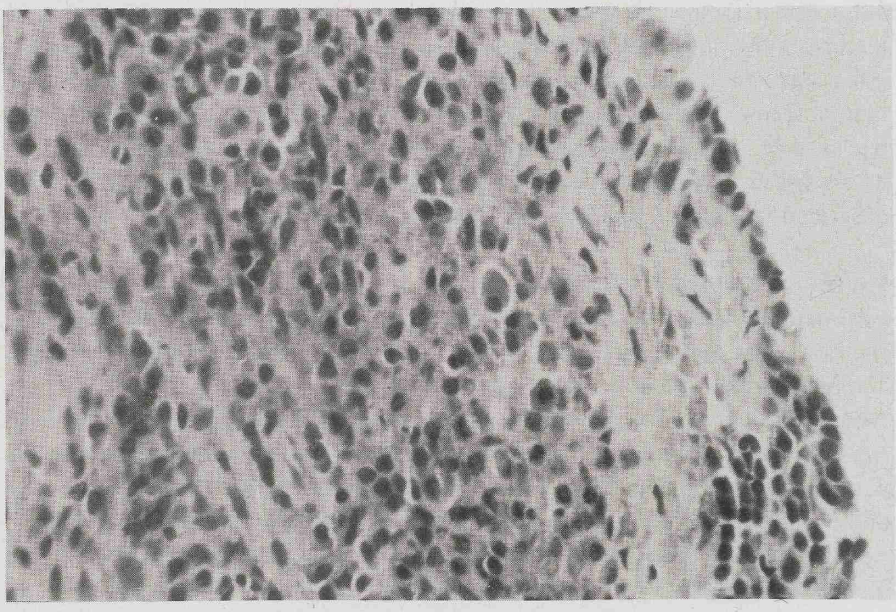

Figure 1. Nasal mucosal biopsy of a patient with ozena. Note the two layers of epithelium (top, left), the virtual disappearance of goblet cells, the increased vascularity and dense inflammatory infiltrate consisting of an approximate equal mixture of lymphocytes and plasma cells.

Hematoxylin and Eosin X 200

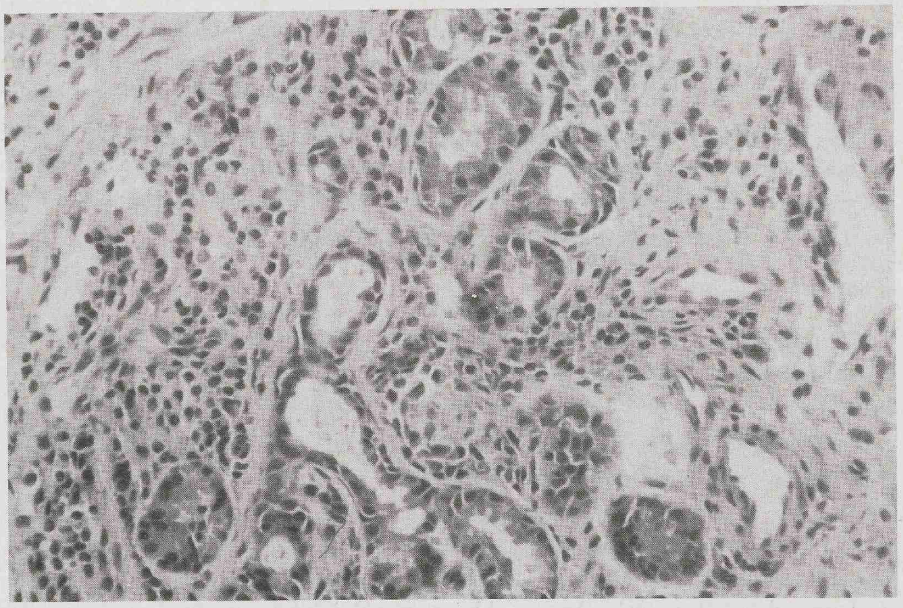

Figure 2. Submucosal glands demonstrating non-keratinizing squamous metaplasia (center, right) and chronic inflammatory infiltrate similar to previous photomicrograph.

Hematoxylin and Eosin X 100

the lamina propria – consisting of both dilated, pre-existing vessels as well as neoformed vascularity. Consistently found in all our material was a marked inflammatory infiltrate, comprised predominently of lymphocytes and plasma cells. In addition, many cells undergoing lymphoplasmacytoid transformation were identified, suggesting a B (humoral) rather than a T (cell-mediated) immune reaction underlying the process. The inflammatory infiltrate was present underlying the mucosal epithelium as well as the submucosal glands (Figures 1 and 2). In none of our case studied was there evidence of granulomata formation.

The histopathology described above contrasts markedly with chronic non-specific hypertrophic rhinitis, in which one consistently observes mucosal gland hyperplasia and hypertrophy (up to seven layers in thickness); often with an increase in the number of goblet cells. In addition, there is often fibrosis of the mucosal and submucosal tissues. Strikingly, the inflammatory cellular response in the latter condition consists predominently of lymphocytes in the form of follicles (not diffuse sheets of cells as is often the case in ozena) demonstrating germinal center formation; suggesting a cell mediated immune bases. Plasma cells are seen, but not to the extent seen in ozena.

CONCLUSION

Ozena, a specific clinical entity, has a characteristic histopathologic appearance. Many of these alterations can be correlated with the clinical symptomatologies of the afflicted individual (Holopainen, 1967). In addition, the inflammatory response tends to suggest a humoral, as opposed to a cell mediated immune process is involved, although the true etiology of this condition remains uncertain (Mygind et al., 1974; Fouad et al., 1980). Histopathology also distinguishes this entity from chronic non-specific hypertrophic rhinitis.

RÉSUMÉ

Les trois auteurs ont étudié les biopsies nasalses de cinquante patients souffrant d'ozène. Dans la muqueuse on trouve toujours des modifications significatives: athrophie de la muqueuse et des cellules inflammatoires. Les auteurs ont présumé qu'il s'agit probablement d'une résponse immunologiques du type humoral qui est importante pour la pathogenèse de cette entité.

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