

Adenomatoid hyperplasia of palatal minor salivary glands

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Abstract

Adenomatoid hyperplasia of palatal minor mucous glands is rare but significant because the clinical appearance mimics malignant disease. The typical history of a painless, indolent palatal swelling, together with the histological picture of benign glandular hyperplasia and hypertrophy, are illustrated in this report.

Key words: Salivary gland diseases; Palate; Hyperplasia

Introduction

Adenomatoid hyperplasia of oral minor mucous salivary glands is an uncommon lesion which is most often encountered in the palate. Its importance lies in the clinical resemblance to more sinister disease. The principal histological features are hyperplasia and hypertrophy of mucous acini, changes which are of uncertain aetiology but which, like the clinical course, are entirely benign with adequate excision curative. Here we describe a case of adenomatoid hyperplasia which demonstrates the typical clinicopathological characteristics of the condition.

Case report

A 48-year-old Asian man was referred to the Department of Oral and Maxillofacial Surgery with a painless swelling in his palate which was first noted at a visit to a dental hygienist. The duration of the lesion was unknown, but the patient felt that the lump was slowly enlarging although there were no other symptoms. Review of his past medical history, physical examination, haematological

and biochemical investigations were unremarkable. He was a non-smoker, and drank alcohol only occasionally.

Examination of the head and neck demonstrated no



FIG. 1

Clinical photograph to show palatal swelling extending to the midline.

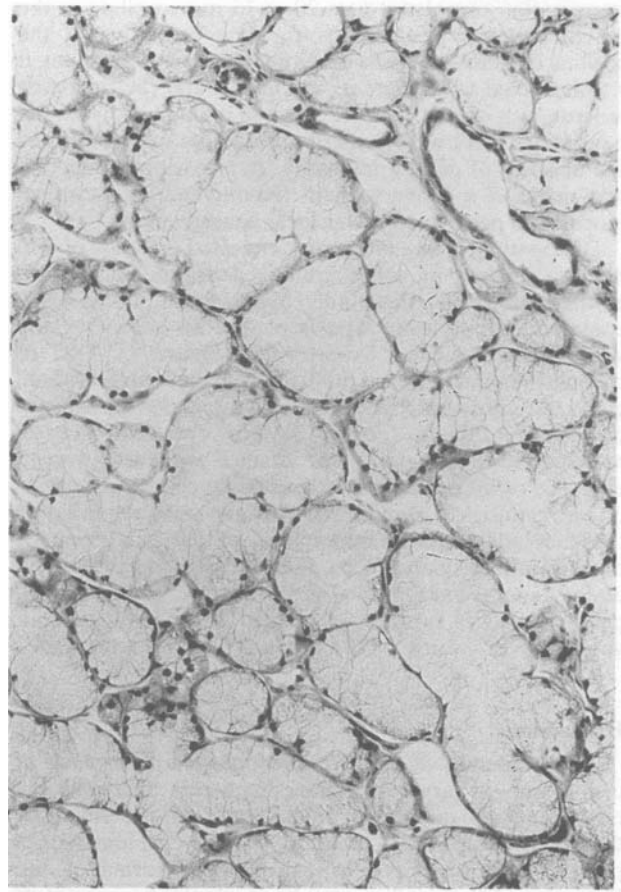


FIG. 2

Increased numbers of hypertrophic mucous acini showing nuclear displacement towards the basal pole. (H & E; $\times 40$).

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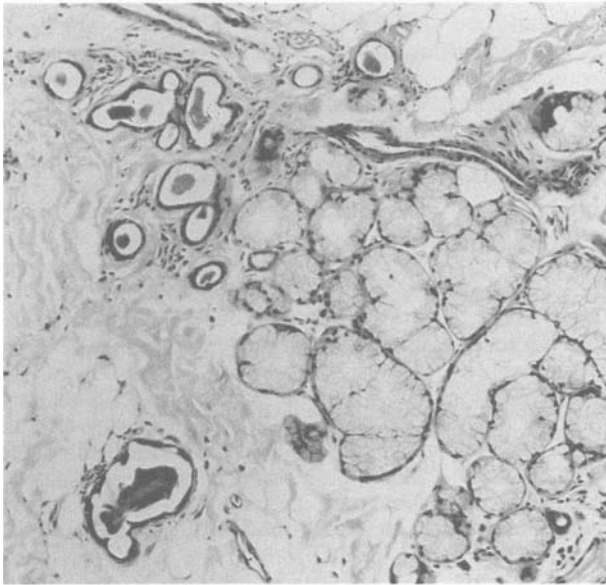


FIG. 3

The affected gland containing areas of glandular atrophy and fibrosis, with duct dilation and chronic inflammation. (H & E; $\times 25$).

cervical lymphadenopathy or facial asymmetry. Intra-oral examination revealed a firm 20×15 mm swelling at the junction of the hard and soft palate extending to the midline (Figure 1). This was neither tender nor fluctuant, it was not fixed to the underlying tissues and the overlying mucosa was intact and of normal colour. All of the permanent teeth were present and radiographs confirmed the absence of dental pathology. A provisional diagnosis was made of a salivary gland tumour and an incisional biopsy was performed under local anaesthetic.

Histopathological examination revealed a nodule of oral mucosa covered by orthokeratinized stratified squamous epithelium which was mildly hyperplastic. The corium contained hyperplastic lobules of mucous salivary gland which showed acinar hypertrophy (Figure 2). Foci of chronic inflammatory cell infiltration, mucus extravasation, gland fibrosis and atrophy were present, and some salivary ducts were dilated (Figure 3). However, the general architecture of the gland was retained and there was no evidence of invasion of the surrounding connective tissue, nor of cytological atypia. The features were regarded as those of adenomatoid hyperplasia of the palatal minor mucous salivary glands. Complete excision was performed under general anaesthesia, and the defect closed with a pack which was sutured into place. The patient made a good recovery, since when there has been no recurrence.

Discussion

Approximately 80 cases of adenomatoid hyperplasia of oral minor salivary glands have been reported since the condition was definitively identified and described by Giansanti *et al.* (1971). Patients typically present with a painless lump which is sessile, firm and of normal colour. Detection of the swelling may be incidental because of its indolent nature, and pain is usually due to repeated trauma with eventual ulceration (Barrett and Speight, 1995); one reported case where the mass was painful was situated close to carious teeth (DeLuke *et al.*, 1992). As in the present case, the individuals affected are usually middle-aged, though a wide age range may be affected with a slight male preponderance (Arafat *et al.*, 1981; Buchner

et al., 1991; Barrett and Speight, 1995). The site affected in the present case is also as expected from previous studies; the hard and soft palate are by far the areas most often involved, but isolated cases have been reported in the mandibular retromolar, buccal, labial or ventral lingual mucosa (Brannon *et al.*, 1985; Buchner *et al.*, 1991; Barrett and Speight, 1995). It is apparent, therefore, that any oral site where mucous salivary glands are found may be affected.

The aetiology of adenomatoid hyperplasia, however, remains conjectural. Local trauma has been proposed as a likely cause because several instances have occurred in wearers of maxillary dentures which lie in close proximity to palatal lesions, or which occlude against retromolar lesions (Devildos *et al.*, 1976; Scully *et al.*, 1992; Barrett and Speight, 1995). In one series, 14 out of 20 subjects either wore dentures or smoked tobacco and histological features were typically present that supported a traumatic origin, namely chronic inflammation of the affected glands, hyperplasia of the overlying mucosal epithelium, areas of mucus extravasation, glandular fibrosis and atrophy, ductal dilation (Figure 3) and hyperkeratosis of the orifice (Barrett and Speight, 1995). One series excluded those instances where mucus spillage and inflammation were prominent, but nevertheless noted inflammatory infiltrates in some of their remaining cases (Buchner *et al.*, 1991). The individual described in this case neither smoked nor wore a denture, and the cause of the lesion is not apparent. The factors involved in sialadenosis of the major salivary glands do not produce adenomatoid hyperplasia (Seifert *et al.*, 1986), which is restricted to minor glands. Whilst a hamartomatous element has been proposed (Arafat *et al.*, 1981), this is unlikely in patients in their fourth to sixth decades.

The largest series reported to date indicates a predilection for Caucasians (90 per cent), with black and Hispanic populations less commonly affected (Buchner *et al.*, 1991). Although the absence of Asian patients is highlighted in this series, a subsequent study (Barrett and Speight, 1995), and, of course, the present case, has shown Asian individuals may be affected on occasion. It might therefore be concluded that race is an insignificant feature, unless coincident environmental or social factors are instrumental in the aetiology of this lesion.

Adenomatoid hyperplasia has been described as 'a sheep in wolf's clothing' (Scully *et al.*, 1992) and the lesion's significance lies in the clinical resemblance to a minor salivary gland tumour. However, the histopathological features are specific and do not resemble benign or malignant salivary neoplasms. The high incidence of malignant salivary gland tumours in the palate nevertheless means that biopsy of soft palatal swellings is mandatory, but once a diagnosis of adenomatoid hyperplasia is established, conservative excision is all that is required and recurrence is exceptional.

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