Submucoal lymphoid aggregates of the lower lip in a 10-year-old boy

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Introduction
Lymphoid hyperplasia may affect the lymph nodes, the lymphoid tissue of Waldeyer’s ring, and the aggregates of lymphoid tissue that are scattered throughout the oral cavity, particularly in the oropharynx, the soft palate, the tongue, and the floor of the mouth. More diffuse lymphoid hyperplasia occasionally involves the hard palate and produces a slow-growing, non-tender swelling with an intact mucosal surface.

We report an unusual case of lymphoid hyperplasia in the lower lip of a 10-year-old boy.

Case report
A 10-year-old otherwise healthy boy presented with a painless soft-tissue swelling in the lower lip. His mother told us that she had noticed the swelling about a month ago and could remember no injury.

He had a 1 cm slightly bluish, soft-to-firm, non-tender circumscribed swelling on the left side of the lower lip (Fig. 1). We diagnosed a mucocele and excised the lesion under local anaesthesia.

Figure 1 A non-ulcerated soft-tissue swelling in the left lower lip.
Figure 2. Lymphoid infiltrate consisting of small darkly staining mature lymphocytes admixed with small cleaved and non-cleaved lymphoid cells. A residual salivary duct-like structure can be seen within the lymphoid infiltrate (haematoxylin and eosin, original magnification 100×).

Histological examination showed a well-demarcated aggregate of lymphoid cells arranged in a follicular pattern beneath mildly hyperplastic stratified squamous cell epithelium (Fig. 2). There were no mitoses. A few salivary duct-like structures were seen within the lymphoid infiltrate. Immunohistochemical studies showed both B cells and T cells. Among the B cells were both kappa-expressing and lambda-expressing cells. The histological picture was consistent with follicular lymphoid hyperplasia.

The wound healed without complications and we made extensive investigations that eliminated the possibility of extranodal non-Hodgkin’s lymphoma. We have followed the boy up for 2 years and there has been no recurrence of the initial lesion or the development of any other signs or symptoms.

Discussion

The clinicopathological features of lymphoid hyperplasia in the mouth were reported by Adkins in 1973. The palatal and lingual lesions that he reported were apparently clinically indistinguishable from lymphoma. Since then, about 20 cases of lymphoid hyperplasia in the oral mucosa have been reported, usually in the palate, in a few cases they were multicentric with or without associated lymphadenopathy. Lymphoid hyperplasia has not to our knowledge been described previously in the lower lip.

With rare exceptions, lymphomas are monoclonal, as shown by the presence of a single light-chain type among the lymphocytes, while reactive lymphoid infiltrates are polyclonal. The lesion in our case was polyclonal. The possibility of a benign lymphoepithelial lesion of a minor salivary gland, as in Sjogren’s syndrome, is unlikely, as such lesions usually occur in elderly women.

Lymphocytic replacement of salivary gland tissues is associated with a risk of malignant lymphoid transformation. However, our patient has been well for a long time, but we will continue to follow his progress.

References