

Lymphoepithelial sialadenitis vs lymphoma - A case report

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INTRODUCTION

Lymphoepithelial sialadenitis (LESA), is a benign disorder, in which there is a lymphoid infiltration throughout the salivary gland with atrophy of parenchyma, ductal hyperplasia, and presence of lymphoepithelial lesions. It mainly affects the parotid and submandibular glands. LESA is a risk factor for the development of extranodal marginal zone B-cell lymphoma, up to 44-fold. Differentiation of LESA from marginal B-cell lymphoma is imperative, and it frequently presents a challenge due to histological changes in lymphoid tissue associated with HIV infection (persistent generalized lymphadenopathy).

MATERIALS

We present a case of a 55 year-old, HIV (+) male who presents with bilateral submandibular enlargement for the past few months. He had several abscesses in previous months in his lower extremities and face, which were surgically drained previous to the development of this lesion. Excisional biopsy was performed.

RESULTS

The biopsy specimen consisted of a 3cm, well circumscribed, soft tissue mass. Cross sections revealed a gray-brown cut surface.

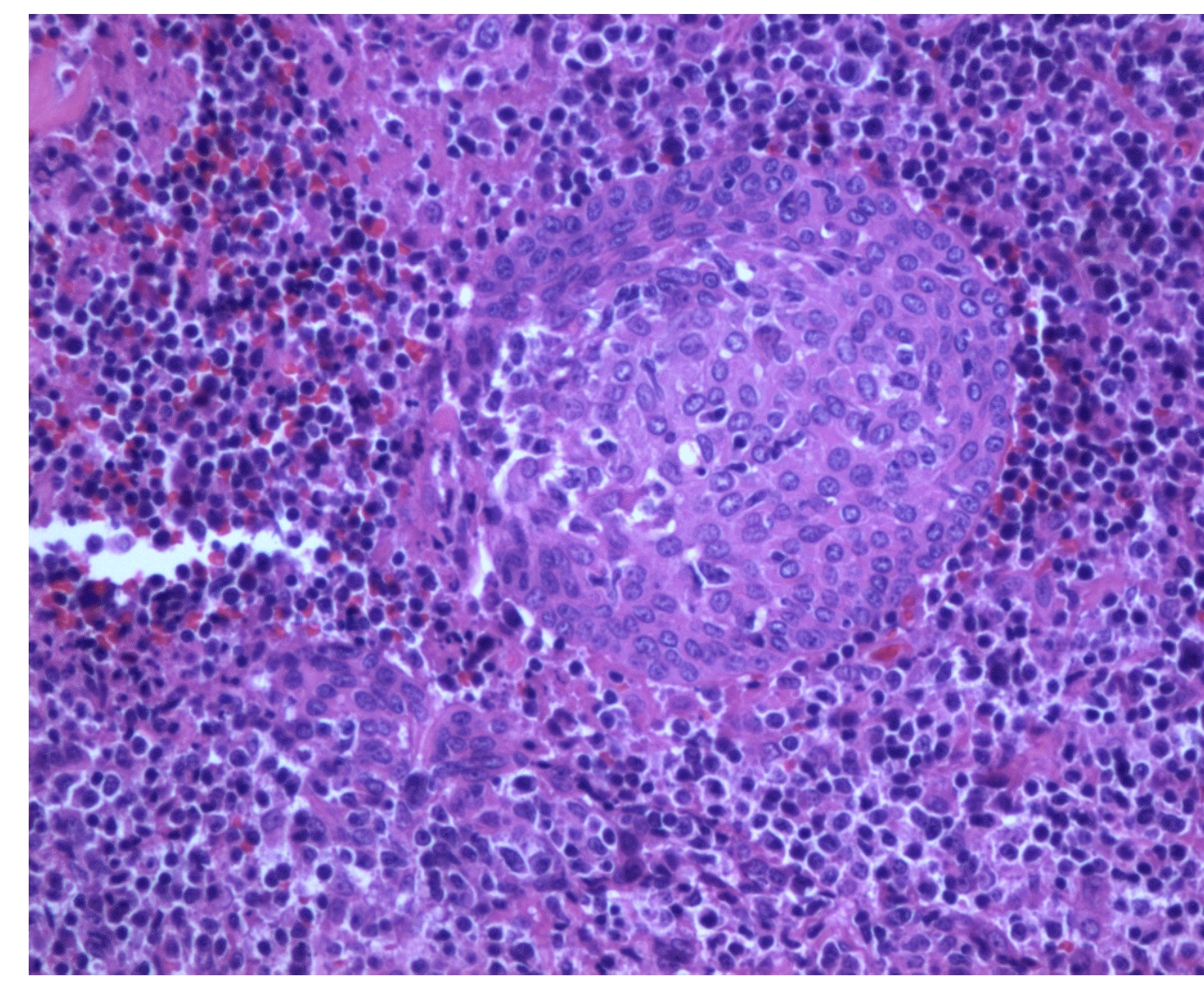


Figure 1.

Microscopic examination demonstrated a diffuse lesion with islands of epithelial cells with squamous differentiation. Basement membrane-like material was identified within the islands, as well as intraepithelial lymphocytes, H&E 10x.

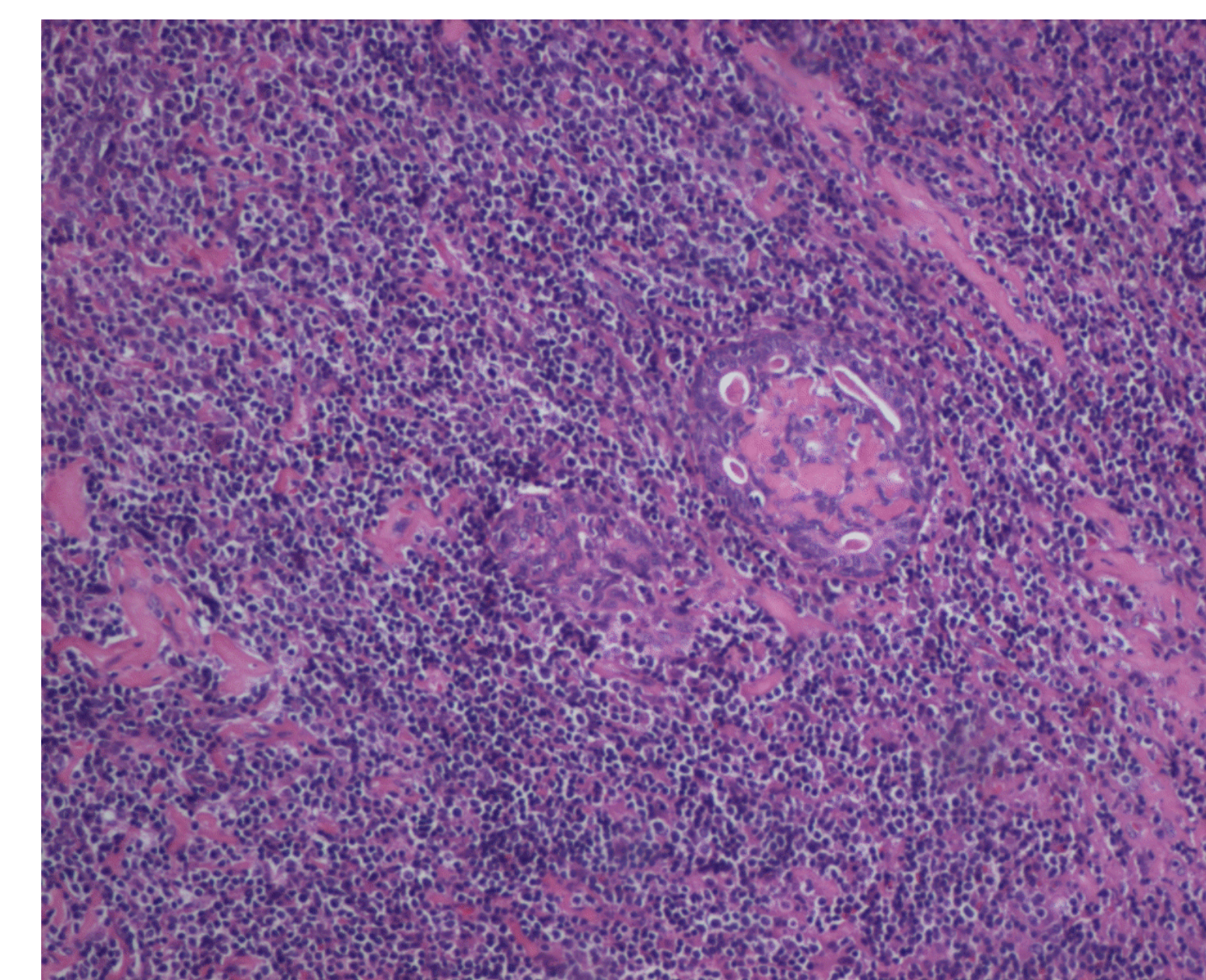


Figure 2.

A dense lymphoid background surrounded the epithelial islands with ill-defined follicular pattern, folliculolysis, attenuated mantle zone, and marked increase in vascular proliferation, H&E 10x.

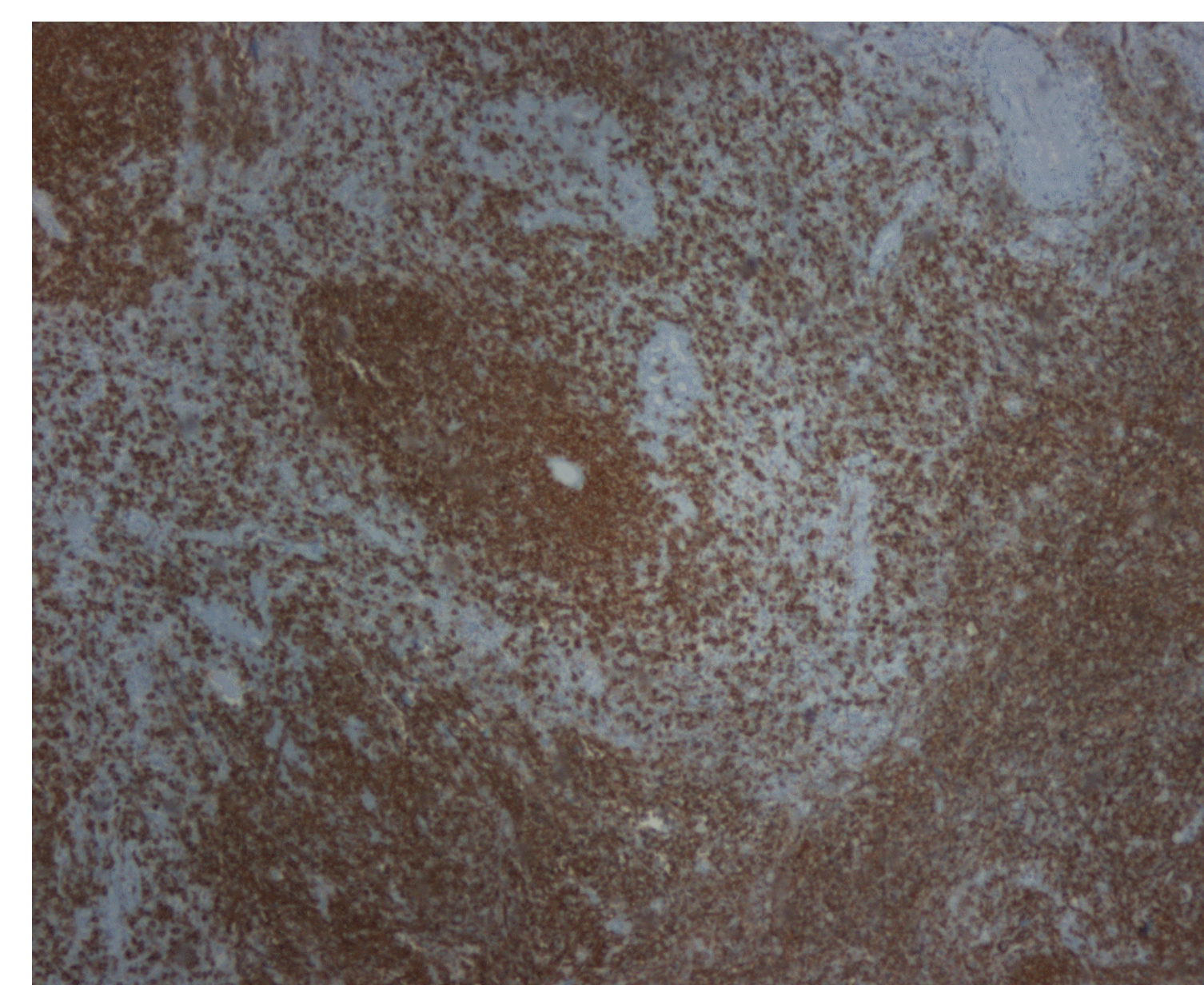


Figure 3.

Immunohistochemical stains showed that the follicles were positive for CD20 (shown). They were also positive for CD10, and pax5. They were negative for bcl-2. Interfollicular areas were positive for CD3, CD5, and CD43. CD23 showed follicular dendritic cells in the follicles, IHC 10x.

DISCUSSION

Differentiation of LESA from marginal B-cell lymphoma HIV (+) patients is often challenging and requires correlation of HIV-associated morphological findings, immunohistochemical stains, and immunophenotyping by flow cytometry. LESA in HIV(+) patients has also been known as HIV-associated salivary gland disease and salivary diffuse infiltrative lymphocytosis syndrome. Since a number of inflammatory, infectious and neoplastic conditions may involve the parotid gland in HIV-infected patients, care should be taken not to confuse these morphologic changes with an extranodal marginal B-cell lymphoma.

Molecular genetic results need to be interpreted with care, since the detection of B-cell clones in cases of LESA does not always correlate with the morphological evidence of lymphoma. Morphology indeed plays a critical role in the diagnosis and differentiation of these entities. In our case, the diffuse lesion with epithelial islands, the somewhat conserved follicles with CD20 positivity, and the immunohistochemical staining pattern pointed towards a benign entity.

Even though LESA is a benign condition, it is associated with a high risk (44x fold increase) for development of extranodal marginal zone B-cell lymphoma in patients with LESA. Close follow-up of patients diagnosed with LESA is recommended, emphasizing the importance of correctly separating this entity from lymphoma.

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