LETTERS TO THE EDITOR

True lipoma of Glisson’s capsule

Descriptions of about 20 examples of fatty lesions on the surface of the liver designated as pseudolipoma of the hepatic, or Glisson’s, capsule have been described. They are not thought to be of neoplastic origin, and no true lipomas of Glisson’s capsule have been described. True lipomas, however, are said to occur deeper within the hepatic parenchyma. We describe a lesion, discovered at necropsy, which we believe to be a true lipoma of Glisson’s capsule.

Case report

A 75 year old diabetic woman presented with a stroke, and subsequently died with bronchopneumonia. No previous abdominal surgery or transcutaneous hepatic biopsy had been carried out. At necropsy there was a recent haemorrhage in the right cerebral hemisphere, with bilateral basal bronchopneumonia. On the anterior border of the left lobe of the liver, incorporated within Glisson’s capsule, there was a yellow mass measuring $3.3 \times 2.7 \times 2.0$ cm (fig 1). Histologically, this had a distinct multinodular pattern consisting throughout of mature fat cells, showing only slight variation in size and shape; it was divided by thin trabeculae of fibrous tissue, and had a clear vascular network. The adjacent Glisson’s capsule was infiltrated by small clusters of mature fat cells (fig 2).

Hepatic pseudolipomas are fatty masses mainly found on the diaphragmatic aspect of Glisson’s capsule; they are more common in men than in women, usually asymptomatic, and so are only seen at necropsy. It has been suggested that they are caused by traumatic inclusion of fat cells within the hepatic capsule during surgical operations, or by transcutaneous biopsy of the liver. Another hypothesis is that they are epiploic appendices which have undergone torsion with subsequent detachment from the colon, later becoming attached to the surface of the liver. Histologically, they comprise fat and fibrous tissue, with calcification, necrosis, or inflammation. In our specimen there were none of these degenerative features, but there were fat cells in Glisson’s capsule adjacent to the lesion. We therefore believe that this is a true lipoma of Glisson’s capsule, perhaps arising in fat cells formed by metaplasia from fibroblasts within the capsule of the liver.

FIGURE 1 Part of the left lobe of the liver, with the lipoma of Glisson’s capsule on the anterior border.

FIGURE 2 Histological appearances of the lipoma, with a clear vascular network (upper insert). There are fat cells in the adjacent hepatic capsule (lower) (haematoxylin and eosin).

Benign lymphoepithelial lesion of salivary gland in a patient with AIDS

Involvement of the parotid and occasionally other salivary glands, with or without a Sjögren-like syndrome, in human immunodeficiency virus (HIV) seropositive patients has been well documented, particularly in North America. We report a case of benign lymphoepithelial lesion (BLL) of the salivary gland in a homosexual patient with persistent generalised lymphadenopathy syndrome (PGLS), as to our knowledge this condition, which can cause diagnostic problems, has not yet been described in the British literature.

A 35 year old homosexual man presented with a seven month history of a swelling in the right side of the neck. He felt otherwise well and there was no evidence of xerostomia or xerophthalmia. On examination there was a diffuse swelling, about $2.5 \times 4.6$ cm, was composed of uniform grey tissue, and included a cyst $2.0$ cm in diameter containing clear gelatinous material. Histologically it showed salivary tissue containing a relatively well circumscribed lymphoid infiltrate incorporating several irregular epithymoepithelial islands (figure). A few cystically dilated large ducts and several small ducts were present and their lining epithelium was invaded by lymphocytes. The lymphoid infiltrate was polymorphic and showed a mixture of small and large lymphocytes, plasma cells, and many multinucleated histiocytic giant cells. A small number of haemosiderin laden macrophages were present. There were also several reactive follicles containing tingible body macrophages with poorly defined mantle zones. Immunocytochemically the plasma cells were polyclonal and no light chain restriction was shown in the lymphoid cells. The subsequent lymph node biopsy specimen from the groin exhibited typical features of PGLS, but there was no evidence of lymphoma.

This case illustrates most of the features of the salivary lesions which have been reported in HIV positive patients or in those at risk for AIDS. Cysts or dilated ducts lined by cuboidal or squamous epithelium are characteristic; often the epithelium is infiltrated by lymphocytes. The cysts are usually surrounded by lymphoid tissue showing large follicles and features similar to those seen in PGLS. Epimyoepithelial islands are seen in a proportion of cases. In this particular case we preferred the term benign lymphoepithelial lesion, as used by Smith et al, because the most striking feature was the formation of numerous epimyoepithelial islands without any obvious clinical features of Sjögren’s syndrome.

The aetiology of the condition has been the subject of debate. A few patients have presented with features of Sjögren’s syndrome and the sicca complex, and an autoimmune basis for the condition had been proposed by Ulrich and Jaffe. Autoantibody tests, where performed, have generally been negative. Another theory is that the lesion represents PGLS affecting intraparotid lymph nodes surrounding salivary tissue, with dilatation of ducts entrapped in the hyperplastic lymphoid
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