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Segmental lymph-node infarction after fine-needle aspiration

JD DAVIES, AJ WEBB

From the University Department of Pathology and Department of Surgery, Bristol Royal Infirmary, Bristol BS2 8HW

SUMMARY Wedge-shaped lymphoid depletion and sinus distention is described in the pole of an intramammary lymph node. The origin of the lesion appears to be traumatic venous thrombosis. Topographically the lesion differs from spontaneous venous infarction of lymph nodes, and resembles segmental infarction due to small arterial lesions. The unusually localised trauma of fine-needle aspiration appears to account for its distinctive microanatomical distribution.

Spontaneous infarction of non-tumorous human lymph nodes has only relatively recently been recognised.[1] Most such infarcts involve almost the entire node and appear to be due to venous occlusion.[2] By contrast arterial nodal infarction may be segmental in character, when it is usually associated with polyarteritic lesions.[3] Traumatic vascular occlusion leading to nodal infarction has not, to our knowledge, been documented previously. This case report illustrates traumatic venous nodal infarction with segmental distribution.

Case report

CLINICAL HISTORY

A 42-year-old woman presented with a three-week history of a painless discrete lump in the upper outer quadrant of the right breast. Fine needle aspiration biopsy of the lesion was performed. The puncture was haemorrhagic but some small lymphocytes, as seen in sluggish reactive nodal hyperplasia, were seen in smears of the aspirate. At operation two weeks later a lymph node was found lying in the axillary tail of the breast, in close proximity to a branch of the lateral thoracic artery. The node was bisected after resection and imprints made. The resultant smears contained few cells but were similar in appearance to those obtained in the fine-needle aspiration. Postoperative recovery was uneventful and no complications were found at an out-patient attendance two months afterwards. Seven years later, no subsequent mammary or lymphoreticular disease has come to light.

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PATHOLOGY

The lymph node measuring $1 \times 1 \times 0{\cdot}5$ cm was unremarkable on initial macroscopic examination, but subsequent retrospective inspection of the paraffin-embedded tissue showed loss of follicles towards one pole of each bisected slice. Microscopically a wedge-shaped area close to one pole was relatively depleted of lymphocytes (Fig. 1) and lacked follicles though these were prominent elsewhere in the cortex of the node. In the hilum of the node a medium-sized venous tributary draining the area was occluded by an organising thrombus (Fig. 2). There was fibrin extravasation close to the affected vein, haemosiderin-containing macrophages were present in the hilum, the affected wedge of the node and to a lesser extent in the perinodal connective tissue.

In the wedge-shaped segment of lymph node there was a decreased density of lymphocytes in the cortical, paracortical and medullary tissue. The basic connective tissue framework was preserved but the deeper sinuses were distended.

Discussion

Spontaneous infarction of small viscera has been described recently in human breast fibroadenomas,[4] lymph nodes[1] and salivary glands[5] and in the submandibular salivary glands of the dog.[6] In most lesions venous thrombosis occurs, and in some the venous thrombosis appears to be the primary event. Infarction in these organs is usually widespread and often involves most of the organ affected.

By contrast arterial occlusion appears to produce segmental or focal infarction in lymph nodes. This process has been described in polyarteritis

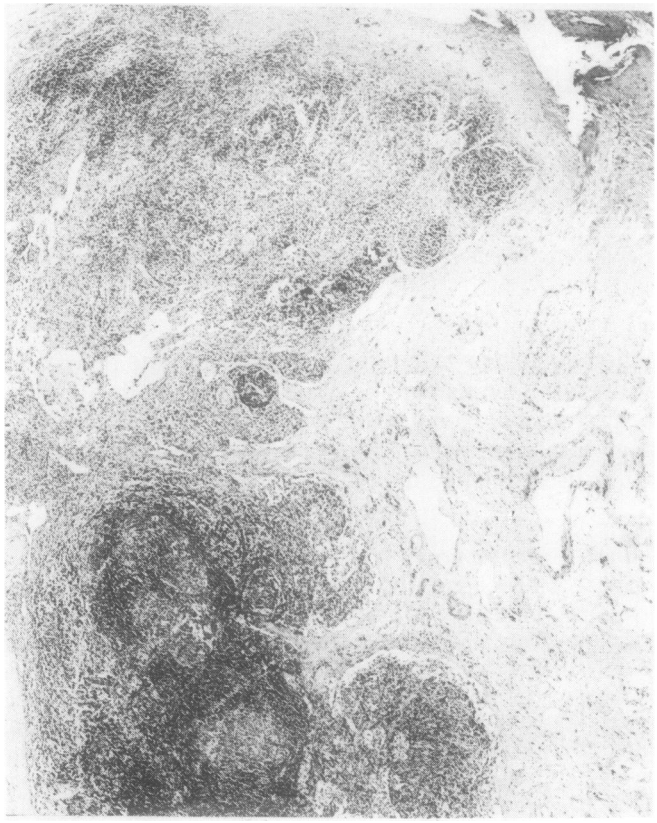

Fig. 1 *Upper pole of lymph node shows loss of follicles, decreased density of lymphocytes, and distention of deep sinuses. Traumatised hilar vein (top right) related to fibrinous extravasation (extreme top right). Haematoxylin and eosin ×30*

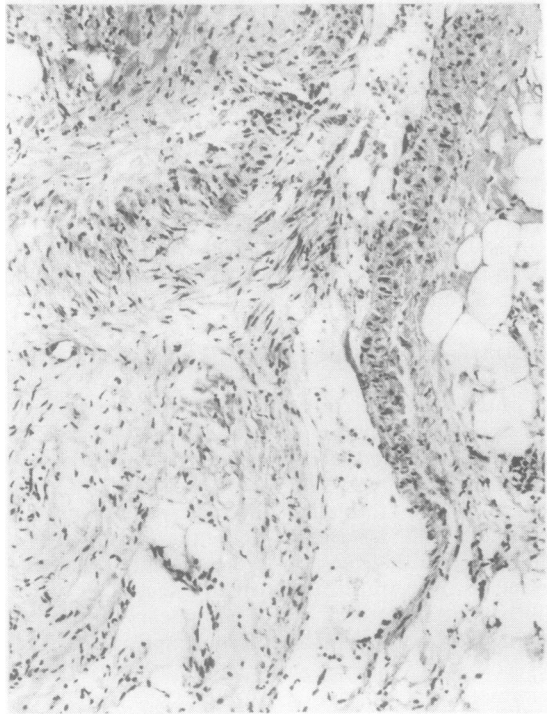

Fig. 2 *Deeper level of damaged hilar vein. Branch draining upper pole occluded by organising thrombus (upper centre) which projects into main channel running vertically. Martius Scarlet Blue ×100*

nodosum[3] and in embolic impaction of atheromatous debris.[7] Only rarely is spontaneous venous infarction focal (R Siebermann, personal communication, 1981). In our case the infarcted nodal tissue was restricted to a segmental area which was related to a relatively recently thrombosed vein.

The history suggests that the vascular damage was of traumatic origin, and there was no clinical indication of a widespread, or localised, thrombophlebitis. Further support for this traumatic interpretation is the presence of abundant haemosiderin accumulation in the nodal hilum, and fibrinous extravasation in the vicinity of the affected hilar vein. Such changes have not been a feature of cases of spontaneous venous infarction in either superficially[1 8] or deeply[9] situated lymph nodes. The presence of dilated lymphatic sinuses is a feature of nodal venous infarcts at this stage.[1] Possibly, the lymphoid depletion in our case represents a dynamic balance between the repopulation of nodal infarcts observed in experimental lesions[10] and the incomplete cellular depletion following occlusion of a single vascular component[11] observed in rodents. Blood-vessel occlusion of lymph nodes is associated in rodent nodes with eventual lymphoid repopulation of the infarcted tissue,[10] whereas afferent lymphatic vessel occlusion leads to a prolonged depletion of macrophages.[11]

Fine needle aspirational biopsy, using 18–22 gauge (0·5–0·8 mm diam) size,[12] is, in contrast to high-speed air-drill or Tru-cut procedures, a relatively atraumatic procedure. Only infrequent, and relatively minor lesions have been found in detailed examination of subsequently resected breast lesions,[13] (Davies JD, Webb AJ, unpublished observations), in the past nine years.

Our conclusions are that this exceptional segmental lymph nodal infarct associated with venous occlusion was produced by the essentially localised trauma which may follow fine-needle aspirational biopsy.

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Requests for reprints to: Dr JD Davies, University Department of Pathology, Level 9 Phase 1, Bristol Royal Infirmary, Bristol BS2 8HW, England.