PIGMENTED VILLO-NODULAR TENOSYNOVITIS

A Case Report

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SUMMARY: An unusually situated pigmented villo-nodular tenosynovitis in a young male patient is described. Of particular interest was the large size and diffuse slimy seaweed-like character of this growth. The ankle joint is an uncommon location for this type of lesion. Mostly they have been reported in the knee joint where they cause pain and swelling (Jaffe, Lichtenstein and Sutro 1941).

Case report

A 33 year old male patient presented with a non-tender soft swelling behind the medial malleolus of left ankle. He stated that the swelling had been present for 6 months, gradually increasing in size but unaccompanied by other symptoms. There was no history of preceding trauma. X-ray examination of the ankle joint was normal.

On surgical exploration a yellowish nodular growth was found arising by a short stalk from the long flexor tendon sheath behind the medial malleolus and matted villous projections originating from the surface of it were intimately wrapped round the tendon. It was not infiltrating the adjoining bone and there was only a small effusion. The lesion was locally excised without sacrificing other structures. No loss of ankle joint function or clinical recurrence have been noted since then.

Macroscopically

The lesion was a villo-nodular mass measuring 7 x 4 x 1.5 cm, the surface of which was yellowish brown in colour. There were nodules of variable size and consistency amongst this slimy seaweed-like growth; the cut surface presented a vaguely lobulated appearance.

On microscopic examination the lining synovium showed hyperplastic villous projections (Fig. 1). The supporting stroma was composed of loose collagenous fibres containing thin-walled blood vessels and polyhedral cells.

The cytoplasm of these cells was foamy in places and heavily laden with dark brown pigment of haemosiderin (Fig. 2). This pigment was not only present within these stromal and lining synovial cells but was also lying free. The nodules revealed a similar histological pattern. They were lined by pigmented synovial cells and the stroma was composed of tightly packed roundish cells containing scattered iron pigment in their cytoplasm (Figs. 3 and 4). Lipid containing cells and multinucleated giant cells were also observed and occasional mitotic figures were found (Figs. 4 and 5). Some of the nodules were heavily collagenised and relatively acellular.

Comment

The findings described are those of a heavily pigmented villo-nodular tenosynovitis arising from a tendon sheath in the location of ankle joint. Although the knee joint is the usual site for this lesion, the process rarely involves the ankle, hip, shoulder or even the elbow joint. The giant cell tumour of tendon sheath is a closely related condition.
Fig. 1. Lining synovium showing hyperplastic villous projections.

Fig. 2. Supporting stroma cells are heavily laden with dark pigment. Lipid-containing histiocytes are also present in clusters.

Fig. 3. Pigmented synovial cells and tightly packed stromal cells containing scattered iron pigment in their cytoplasm.

Fig. 4. Scattered pigment is present in the cytoplasm of stromal cells.
Pigmented Villo-Nodular Tenosynovitis

but it is a localised nodular tumour growing towards the skin whereas this frond-like papillary lesion grows into the joint space (Ackerman 1968). Encapsulation and the well demarcated and lobulated configuration, the absence of a typical granulomatous reaction in these lesions together with the high recurrence rate are factors more suggestive of a neoplastic formation than an inflammatory reaction (Stewart and Wright 1950).

The nodular and cystic elements in villo-nodular synovitis could be confused clinically with bursa and ganglia of joints and tendon sheaths but differ histologically. Bursal cysts usually result from repeated trauma or excessive pressure.

The synovium is thickened and fibrotic, synovial membrane may be destroyed, and the lining consists of granulation tissue and inspissated fibrinous material.

The tumour has also to be distinguished from synovial sarcoma and can be done so by the presence of xanthoma cells, the scarcity of spindle cells and the absence of a characteristic biphasic epithelial or pseudoglandular differentiation. Mitotic figures are quite common in either lesion. Diffuse pigmented synovitis has been produced experimentally in the knee joints of dogs by repeated intra-articular injections of autogenous blood but this cannot account for all the cytological features of a fully developed lesion. Jaffe (Jaffe, Lichenstein and Sutro 1941) studied the effects of repeated haemorrhages in joints of a haemophiliac. The synovial membrane of such joints became villous, hyper-vascular and heavily pigmented with haemosiderin, even the articular surfaces of bone ends in these joints were effected. But the synovial membrane did not reveal other typical features of pigmented villo-nodular synovitis such as lipid filled cells, multinucleated giant cells and sheets of compact stromal cells which are close to histiocytes in their cellular properties. In mildly affected haemophiliac joints the haemosiderin has been found to be confined to the lining synovial cells in heavily discoloured areas (Collins 1951).

Fig. 5. Section of nodule showing lipid containing cells, multinucleated giant cells and occasional mitotic figures.
The numerous names offered for this condition reflect the uncertainty about the histogenesis. It has been suggested that the lesion could be a true tumour in oncological sense as it arises from the linings of joints, tendon sheaths and ligamentous tissues. Because of the benign nature and the presence of giant cells it has been called in the past "benign giant cell synovioma" (Stewart and Wright 1950). The idea receives support from the fact that the recurrence is common after incomplete primary excision, that it erodes adjacent bones and that it grows to large proportions. All evidence supports the opinion of Jaffe that the condition is an inflammatory reaction. The inciting agents are unknown. Trauma, haemorrhage or infection may aggravate the condition but it is doubtful if they are the primary aetiological factors.

REFERENCES


