Tietze's disease

H. U. CAMERON AND V. L. FORNASIER

From the Department of Pathology, Princess Margaret Hospital, Toronto, Ontario, Canada

SYNOPSIS The material from a case of Tietze's disease has been examined histologically. A review of the literature on the pathology of this lesion correlated with the findings from this case indicates that there is a pathological lesion in the costal cartilage in this disease. This consists of an increased vascularity and degenerative changes with patchy loss of ground substance leading to a fibrillar appearance. Cleft formation may occur with mucoid debris which may undergo calcification. Hypertrophic changes may be present at the periphery.

The aetiology still remains obscure.

The aetiology of Tietze's disease or costochondritis remains unknown. As the diagnosis is seldom in doubt and the condition is benign and self limiting, material for pathological examination is rare. This report describes the findings from one case of Tietze's disease and reviews the literature on the histopathology of this obscure lesion.

Materials and Methods

A 22-year-old girl presented with a painful swelling at the second right costochondral junction. This had been present for two months and appeared to be enlarging. Although the diagnosis of Tietze's disease was made, there was some question in the mind of the clinician, and, as the patient refused to be reassured, the lump was explored. On splitting the perichondrium a swollen costochondral junction was found. The mass was shaved down and the shavings were sent for pathological examination.

The shavings were fixed in 10% formalin, embedded in paraffin, and sections cut. They were stained with haematoxylin and eosin, Mallory's trichrome, Van Gieson, Toluidine Blue, Alcian Green, PAS, reticulin, and PTAH.

Observations

The hyaline cartilage showed a generalized pallor, and the centrally lying cells did not show the normal perilacunar halo. There was an increase in vascularity within the cartilage (fig 1). At the periphery there were columns of cartilage, indicating proliferative activity. The surrounding perichondrium appeared normal.

Clefts were present and the increased staining reaction with Alcian Green in the region of the

Fig 1 Alcian Green. Oil immersion. $\times 1000$. A blood vessel is present in a central area of cartilage degeneration.

Fig 2

Fig 2 *Alcian Green. $\times 100$. This shows a cleft in the cartilage. The walls of the cleft stain more deeply than the surrounding cartilage. Some mucoid material is present within the cleft.*

Fig 3

Fig 3 *Alcian Green. $\times 400$. The patchy areas of 'unmasked' collagen fibres are readily seen.*

clefts indicated increased production of mucopolysaccharides (fig 2). This is identical to the reaction one would expect to find around the cysts in a cystic meniscus. Occasional clefts contained a hyaline mucoid material.

In patchy areas matrix had been lost and the collagen fibres in the cartilage became 'unmasked' (fig 3). Cartilage is a fibrous composite. Normally, it is extremely difficult histologically to distinguish the collagen fibres or fibrous phase from the mucopolysaccharide ground substance or filler phase. When the ground substance is leached out, as occurs when cartilage is subjected to proteolytic digestion, eg, with papain, the collagen fibres can clearly be seen on histological examination, especially when viewed with polarized light. The source of the enzymes, if any, in this condition is not obvious as the surrounding chondrocytes, which are the most obvious source as they contain proteolytic enzymes

in their lysosomes, appear normal.

Discussion

Those cases with pathological reports were then reviewed.

As most authors were reporting on one case only and the changes were quite mild, little emphasis has been laid upon them. However, when the review of the literature was correlated with the findings from our case it would appear that Tietze's disease does have a recognizable pathological picture.

There is an increase in vascularity (Gill, Jones, and Pollak, 1942; Geddes, 1945; Leger and Moinnereau, 1950). Degenerative changes occur with the formation of clefts, which may undergo calcification (Tietze, 1921; Harttung, 1923; Leger and Moinnereau, 1950), and patchy loss of ground substance leading to a fibrillar appearance (Tietze,

1921; Harttung, 1923; Gill *et al*, 1942). Some hypertrophic changes may be present at the periphery (Gill *et al*, 1942; Geddes, 1945; Leger and Moynereau, 1950).

It must be pointed out that most tissues are limited in the way they can respond to a stimulus. In cartilage in the present condition, the histological appearance, as in a cystic meniscus, does not shed much light on the aetiology.

References

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