THE HISTOLOGICAL APPEARANCES OF CHONDRODERMATITIS CHRONICA HELICIS

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Winkler (1915) described eight cases of a small, painful benign growth, occurring on or near the upper part of the free border of the helix of the auricle and suggested the name “chondrodermatitis nodularis chronica helicis” for the condition. Shortly afterwards Foerster (1917) described four cases of a similar condition under the name of “painful nodular growth of the ear.” Further examples of the condition have since been reported by several authors.

The condition usually occurs in adult males, commonly over the age of 50 years. The patients complain of a small painful lump on the upper part of the free border of one ear, usually of several months’ duration. The pain is aggravated if the patient lies on the affected side, and is compared with the “darting” or “knife-like” pain of a corn. There is rarely any history of bleeding or discharge from the lesion. On examination there is a small (usually 4-6 mm. diameter), firm, well-defined pinkish nodule, on or near the free border of the upper part of the helix, and usually immovable on the underlying cartilage. The centre of the lesion is covered with a crust-like scale, removal of which reveals a cup-shaped depression with a firm base. Removal of the scale may be accompanied by temporary relief of the pain.

There is less agreement as to the aetio-pathology of the condition, and the following study was undertaken in an effort to clarify the position. Before it was completed Shuman and Helwig (1954) published in America a series of cases from the files of the American Armed Forces Institute of Pathology which have added considerably to the understanding of the condition.

Winkler considers the primary aetiological agent to be a degenerative change in the auricular cartilage with subsequent infection and chronic inflammatory changes. The degenerated cartilage acts as a foreign body and maintains the inflammatory stimulus and a reactive hyperplasia occurs in the overlying epithelium. Foerster supports this view and considers that the anatomical peculiarities and variation in form which occur in the scapha-helix may help to explain the location of the lesion. Ebenius (1941) considers that there are probably several aetiological factors including trauma, circulatory disturbances, and abnormalities of the auricular skin. Carol and van Haren (1941), from their study of serial sections from eight cases of chondrodermatitis, believe the condition to be due to a primary epithelial change, resulting in the formation of a clavus, pressure from which causes epithelial ulceration and necrosis of the underlying corium and in some instances of the cartilage also. Shuman and Helwig consider that several factors, including developmental abnormalities, poor blood supply, and trauma, are concerned in the aetiology of the condition.

Material

The present study is based on the examination of serial sections from 12 cases of chondrodermatitis chronica helicis excised at the Liverpool Radium Institute during a five-year period. The patients were all men, the youngest of whom was 40 years of age, and the duration of the condition varied between one month and six years.

Histological Appearances

All of the specimens show characteristic histological features in the epithelium, connective tissue, and perichondrium. The duration of the lesion does not appear to alter these characteristic appearances.

At the edge of the lesion there is a relatively abrupt change from the normal epithelium to epithelium showing acanthosis, with irregular broadening and deepening of the rete pegs and hyperkeratosis associated also with keratotic plugging of the follicles (Fig. 1). This epithelial hyperplasia may be so marked as to suggest an early malignant change, particularly if seen in tangential sections. In the central 1–2 mm. the epithelium is invaginated in a crater-like manner by a keratinous plug composed chiefly of hyper-
KERATOTIC AND PARAKERATOTIC EPITHELIAL SQUAMES (Fig. 2). The epithelium forming the walls and base of the crater, although stretched, is hyperplastic and hyperkeratotic, with areas of parakeratosis. Hyperkeratotic and parakeratotic squames are shed from the base and walls of the crater into the central mass with which they become continuous (Fig. 3). An occasional leucocyte is present in the epithelial wall of the depression. The deeper part of the keratin plug and the underlying epithelium are widely infiltrated with polymorphonuclear leucocytes. This inflammatory process, associated with the stretching of the epithelium, results in ulceration over a variable extent of the epithelium at the base of the crater (Fig. 4). The floor of this ulcer is formed by a band of eosinophilic fibrinous material in which leucocytes and parakeratotic epithelial squames are entangled. The fibrinous material extends laterally to form a thin membrane deep to the basal layer of the neighbouring epithelium and also into any available spaces in the adjacent corium.

Prolongations of the inflammatory process into the adjacent corium are seen in tangential sections as small isolated epithelial islets with an inflammatory reaction on their deep surface (Figs. 1 and 5).

The underlying corium shows a proliferation of fibrovascular tissue which is characterized by an absence of elastic tissue (Fig. 6) and is infiltrated with an occasional lymphocyte and neutrophil polymorphonuclear leucocyte. This reaction is most intense around the fibrinous material in the floor of the ulcer at which site the appearances are very similar to a granuloma pyogenicum (Fig. 7). The fibrovascular reaction fades out towards the periphery of the lesion where there is patchy perivascular and perifollicular lymphocytic infiltration.

A perichondritis beneath the area of ulceration was present in all of the cases, the vascular granulation tissue spreading down to become continuous with the perichondrium (Fig. 4).

The relationship of the underlying cartilage to the overlying pathological change is variable, and appears to bear a relationship in some cases to the severity of the secondary infection. In the present series of cases the cartilage was involved in three; in two, the secondary infection was of greater severity than usual, leading to an increase in the amount of the fibrinoid material and in the extent of the fibrovascular reaction, both of which spread down to involve the cartilage. In one of these cases there was an acute abscess in the corium. In the third case showing involvement of the cartilage the floor of the cup-shaped depression was formed by necrotic cartilage, the fibrinoid material being limited to a narrow zone beneath the basement membrane of the epithelium at the edges of the ulcer. Possibly associated with the failure of the fibrinoid reaction the vascular component of the fibrovascular reaction was very poorly developed in this case.

Six of the cases showed no evidence of degeneration of the cartilage at the site of the painful nodule; in the remaining three cases the specimens did not include sufficient cartilage on which to base a reliable opinion.

**Discussion**

The histological features described above are essentially similar to those described by Ebenius, Carol, and van Haren, and Shuman and Helwig. From the aetio-pathological standpoint they support the view held by Carol and van Haren that chondrodermatitis helicis is primarily an epithelial change with a localized area of acanthosis and hyperkeratosis, the centre of the lesion being occupied by a horny plug of hyper- and parakeratotic epithelial squames which invaginates the epithelium in a cup-like manner. Infection and stretching of the epithelium at the base of the depression lead to ulceration and secondary infection in the corium; the floor of the ulcer is formed of fibrinous material in which epithelial squames and polymorphs are entangled. Secondary changes occur in the corium, the most characteristic of which is a fibrovascular proliferation, chiefly around the fibrinoid material and extending down to the perichondrium. The underlying cartilage may or may not be involved in the inflammatory process, but the fact that in the present series definite degeneration of the cartilage, occurring in association with the area of chondrodermatitis, was present in only three cases does not lend support to the view held by Winkler and others that the primary cause of the condition is degeneration of the cartilage.

Mucoid degeneration of the auricular cartilage was noted in four of the cases, but it did not appear to bear any specific relationship to the area of chondrodermatitis, and there was no evidence of it giving rise to any secondary changes in either the adjacent connective tissues or the overlying epithelium.

A study of three cases of chondronecrosis occurring after radiation therapy for carcinoma of the pinna did not show any evidence in favour of the theory that necrotic cartilage gives rise to any secondary change in the overlying epithelium which in these cases showed typical post-radiation atrophy. Ulceration over the area of chondronecrosis was present, but in contrast to chondro-
**Fig. 1.** Tangential section from the edge of the lesion showing acanthosis, hyperkeratosis and parakeratosis associated with a fibrovascular reaction in the corium which is spreading down to become continuous with the perichondrium (× 40).

**Fig. 2.** Tangential section taken from nearer the centre of the lesion showing the "crater-like" invagination of the epithelium by the keratin plug and a subepithelial focus of infection. (Post-radiation specimen, × 40.)

**Fig. 3.** Detailed structure of the keratin plug, with parakeratotic squames at the surface (left side of photograph) and degenerated and infected squames at the base (× 100).

**Fig. 4.** Mid-line section, showing ulceration of the epithelium at the base of the crater; the ulcer floor is formed by hyaline fibrinous material and is covered by the keratin plug (× 40).

**Fig. 5.** Peripheral extension of acute infection and fibrinoid production beneath the basement membrane of an epithelial downgrowth. (Same slide as Fig. 1, × 100.)

**Fig. 6.** Characteristic fibrovascular reaction with absence of elastic tissue. In this section the fibrovascular tissue is invading the elastic auricular cartilage. (Moore's elastic tissue stain, × 40.)

**Fig. 7.** Fibrovascular tissue adjacent to the area of ulceration (× 400).
dermatitis helicis the ulcer was at the surface and not at the base of a cup-shaped invagination, and the horny plug and fibrovascular tissue were absent, the granulation tissue forming the ulcer floor being less vascular and containing elastic fibrils.

It may be argued that the radiation had prevented the normal tissue response to the presence of degenerative cartilage. That this is not so is suggested by the fact that the typical epithelial changes of chondrodermatitis helicis were still present in two patients in this series who had been treated by radiotherapy before surgical excision.

The presence of secondary infection may alter the characteristic histological appearances of chondrodermatitis chronica helicis by causing more widespread tissue involvement, including abscess formation in the corium and destruction of the cartilage; this may give rise to an erroneous conception of the pathology of the condition.

There is still no convincing evidence as to what initiates the pathological change in chondrodermatitis chronica helicis, nor its peculiar location and predominance in males. The histological similarity to clavus suggests that pressure, possibly during sleep, may have some bearing on the condition.

**Summary**

The histological features of 12 cases of chondrodermatitis chronica helicis are described. The condition is primarily an epithelial change with hyperkeratosis, acanthosis, and parakeratosis. A central keratin plug is formed which invaginates the epithelium in a "crater-like" manner. The epithelium below the keratin plug becomes ulcerated, associated with the production of fibrinoid material and a fibrovascular proliferation in the corium. Perichondritis is always present. The cartilage may or may not be involved.

**References**


