The Process of Ulceration in Articular Cartilages

As Presented to the University of Edinburgh and the Anatomical Museum

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The study of ulceration in articular cartilages offers profound insights into the intricate cellular mechanisms involved in tissue degeneration and regeneration. John Goodsir's detailed observations on this topic shed light on the non-vascular nature of cartilage and the role of cellular processes in its ulceration. By employing microscopic techniques, Goodsir meticulously examines the changes occurring at the cellular level during the ulceration of articular cartilages, particularly under pathological conditions such as scrofulous disease and inflammatory states. This compilation will explore Goodsir's findings, emphasising the cellular transformations, the interaction between cartilage and false membranes, and the underlying principles of cartilage absorption and ulceration.

Non-Vascular Nature of Cartilage

Goodsir begins by addressing the longstanding debate about the vascularity of cartilages. He clarifies that while cartilage is not entirely devoid of blood vessels, it is significantly less vascular compared to other tissues. In adult human articular cartilages, blood vessels are rarely detected, indicating that the nourishment and pathological changes in cartilage cannot primarily be attributed to vascular activity. This distinction sets the stage for understanding the unique process of ulceration in cartilage, where traditional blood vessels and lymphatics are not the active agents.

Microscopic Observations of Ulcerated Cartilage

Using microscopic techniques, Goodsir examined thin sections of ulcerated articular cartilage, particularly those covered by gelatinous or false membranes. He described distinct changes at the cellular level.

On the edge of the ulcerated cartilage, the corpuscles undergo significant changes. They enlarge, becoming rounded or ovoid, and their interiors fill with multiple nucleated cells. At the ulcerated edge, these enlarged corpuscles open into the diseased membrane, with some ovoid masses partially released from their cavities and others integrated into the false membrane. This indicates an active interaction between the cartilage and the overlying pathological membrane. When the false membrane is removed, the cartilage appears rough

and honey-combed, with nipple-like projections of the membrane penetrating into its surface. These projections and the openings of the enlarged corpuscles on the ulcerated surface suggest a dynamic cellular process at play.

Role of False Membrane and Vascular Structures

The false membrane covering the ulcerated cartilage is vascular, containing loops of capillary vessels that penetrate its nipple-like projections. However, the cartilage itself does not exhibit any vascular changes during ulceration. A layer of nucleated particles exists between the capillaries and the ulcerated cartilage surface, indicating that the cellular layer of the false membrane plays a crucial role in the absorption and transformation processes.

Cellular Mechanisms in Ulceration

Goodsir proposed that the cellular activities, rather than vascular actions, drive the ulceration process. He suggested that new cells formed in the false membrane absorb the hyaline matter of the cartilage, transforming it into a soft cellular texture rather than directly removing it. This cellular transformation underpins the absorption process, challenging the traditional view that veins and lymphatics are the primary agents of absorption.

Ulceration in Scrofulous and Inflammatory Conditions

In severe pathological conditions, such as scrofulous disease or extensive joint inflammation, the ulceration extends from the cartilage's attached surface. Vascular cellular projections from the bone invade the cartilage, meeting similar projections from the false membrane. This dual invasion can lead to the cartilage being riddled, broken into scales, undermined, or entirely absorbed into the joint cavity fluid. Goodsir emphasised that the observed changes support the theory that cellular formations, not vascular structures, are the immediate agents of cartilage absorption. The cells of the false membrane transform the cartilage into cellular tissue, a process indicative of transformation rather than simple removal.

The following is Goodsir’s work viz.

“The Process Of Ulceration In Articular Cartilages

The question as to the vascularity of cartilages cannot now excite much interest, when we know that all the textures are in themselves destitute of blood-vessels, which are accessory parts, carriers of nourishment, not active agents in its deposition. We do not consider

cartilage as a texture into which no blood-vessels pass, but only as less vascular than some of the others. In a large mass of cartilage, as in those of the bulky mammals, or in the thick cartilages of the foetal skeleton, canals containing blood-vessels are found here and there; but in the thin articular cartilages of the adult human subject few or no vessels can be detected.

It is evident, therefore, that in the process of ulceration in cartilage, it cannot be the usual blood-vessels of the part which are the active agents.[1] Still less likely is it that lymphatics, the existence of which has never been asserted in this texture, are the absorbing instruments.

If a thin section, at right angles, be made through the articular cartilage of a joint, at any part where it is covered by gelatinous membrane in scrofulous disease, or by false membrane in simple inflammatory condition of the joint, and if this section be examined, it will be found to present the following appearances.

On one edge of the section is the cartilage unaltered, with its corpuscles natural in position and size. On the opposite edge, is the gelatinous, or false membrane, both consisting essentially of nucleated particles, intermixed, especially in the latter, with fibres and blood-vessels; and, in the former, with tubercular granular matter. In the immediate vicinity, and on both sides of the irregular edge of the section of cartilage, where it is connected to the membrane, certain remarkable appearances are seen. These consist, on the side of the cartilage, of a change in the shape and size of the cartilage-corpuscles. Instead of being of their usual form, they are larger, rounded, or oviform; and, instead of two or three nucleated cells in their interior, contain a mass of them. At the very edge of the ulcerated cartilage, the cellular contents of the enlarged cartilage-corpuscles communicate with the diseased membrane by openings more or less extended. Some of the ovoidal masses in the enlarged corpuscles may be seen half-released from their cavities by the removal of the cartilage; and others of them may be observed in the substance of the false membrane, close to the cartilage, where they have been left by the entire removal of the cartilage which originally surrounded them.

If a portion of the false membrane be gradually torn off the cartilage, the latter will appear rough and honey-combed. Into each depression on its surface a nipple-like projection of the false membrane penetrates. The cavities of the enlarged corpuscles of the cartilage open on the ulcerated surface by orifices of a size proportional to the extent of absorption of the walls of the corpuscle, and of the free surface of the cartilage.

The texture of the cartilage does not exhibit, during the progress of the ulceration, any trace of vascularity. The false membrane is vascular, and loops of capillary vessels dip into the substance of the nipple-like projections which fill the depressions on the ulcerated surface of the cartilage;[2] but, with the exception of the enlargement of the corpuscules, and the peculiar development of their contents, no change has occurred in it. A layer of nucleated particles always exists between the loops of capillaries and the ulcerated surface.

The cartilage, where it is not covered by the false membrane, is unchanged in structure. The membrane generally adheres with some firmness to the ulcerating surface; in other instances it is loosely applied to it; but in all, the latter is accurately moulded to the former.

In scrofulous disease of the cancellated texture of the heads of bones, or in cases where the joint only is affected, but to the extent of total destruction of the cartilage over part or the whole of its extent, the latter is, during the progress of the ulceration, attacked from its attached surface. Nipple-shaped processes of vascular cellular texture pass from the bone into the attached surface of the cartilage, the latter undergoing the change already described. The processes from the two surfaces may thus meet half-way in the substance of the cartilage, or they may pass from the attached, and project through a sound portion of the surface of the cartilage, like little vascular nipples or granulations. The cartilage may thus be riddled, or it may be broken up into scales of varying size and thickness, or it may be undermined for a greater or less extent, or be thrown into the fluid of the cavity of the joint in small detached portions, or it may entirely disappear.

On the principles already laid down, if absorbents exist, as we have reason to believe they do in the false membrane, neither they nor the veins are to be considered as the active or immediate agents in the absorption of the cartilage. They certainly are not so in the absorption of the walls of the corpuscules, and this, as well as the analogy of similar processes, gives weight to the opinion to which I have come, that they are not the immediate instruments in the absorption of the free surface. The cells of new formation appear to be the immediate agents in this action. They absorb into their substance the hyaline matter of the cartilage, the latter probably not being removed at once from the spot, but merely converted into soft cellular texture; the process being one of transformation rather than removal."[3]

Conclusion

John Goodsir's meticulous examination of the ulceration process in articular cartilages provides valuable insights into the cellular mechanisms underlying tissue degeneration. His use of microscopy reveals significant changes in cartilage-corpuscles and their interaction with diseased membranes, highlighting the role of cellular activity in cartilage absorption. Goodsir's work challenged traditional views on vascular involvement in tissue absorption, proposing instead that new cellular formations are the primary agents of this process. By advancing our understanding of these cellular processes, Goodsir's findings offered a more nuanced perspective on tissue degeneration and regeneration, potentially informing future medical approaches to joint diseases and conditions.

[1] See Mr. Aston Key's Paper in the *London Med. Chir. Trans*, vol. xviii. Part I., "On the Ulcerative Process in Joints."
[2] The vascular loops described and figured by Mr. Liston are not vessels in the cartilage, but the vessels described in the text. Liston. *Lond. Med. Chir. Trans*.
[3] Turner, William (ed.) and Lonsdale, Henry (contrib.). *The Anatomical Memoirs Of John Goodsir F.R.S. Late Professor Of Anatomy In The University Of Edinburgh, Volume II* (Edinburgh: Adam and Charles Black, 1868): 408-411.