

THE PATHOLOGY OF TUBERCLE.

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The subject of the pathology of tubercle is with the modern improvements in the methods of its examination becoming every day more interesting, by renewed consideration of its manner of growth and deposit. I believe such knowledge will yet be attained as will aid us in *preventing* the initiatory deposit of that growth, which, when deposited, unfortunately resists too powerfully our efforts to procure its removal. I revert to the subject of tubercle, strong in the belief of the frequent association of *tubercular phthisis and simple catarrhal pneumonia*.

I will avoid unnecessary theorizing and intend my paper to be more an answer to two questions, viz. :—

What is tubercle? and how is it produced?

Firstly—As to what tubercle is. According to the old doctrine of Lænnec, tubercle was regarded as “the product of a peculiar constitutional disease”; that it never could be developed out of a simple inflammation, such as in the lungs from an acute or chronic pneumonia, or take its rise from a bronchial hemorrhage; that it was a *specific growth ab initio*.

Virchow, whose merits in this field of pathological research are very great, goes, I think, too far in asserting as he does, that even the doctrine of miliary tuberculosis is founded upon error, and that *all* the alleged miliary tubercles of the lungs are *bronchitic, peri-bronchitic and pneumonic deposits*. This remark I think not altogether true, since it is by no means of rare occurrence for the same transparent gray granules, which in acute miliary tuberculosis are scattered in great number through the lungs and most organs, and whose tubercular nature can hardly be questioned—to be met with also in phthisical lungs, but I think there is sometimes a danger of mistaking for tubercles, dry concretions of pus present in partially occluded bronchial tubes. We frequently see by the side of miliary tubercle, yellow cheesy deposits occurring in the form of miliary granules, and which, I think, we must consider as original tubercles, having undergone the caseous or semi-fatty change.

We have no proof that these cheesy miliary deposits are the products of vesicular pneumonia. We have no criterion by which to distinguish a cheesy tubercle from a cheesy miliary granule of inflammatory origin—and when examined microscopically, these deposits show themselves to possess some of those elements which enter into the formation of a true grey miliary tubercle. On examining microscopically a section of a grey miliary tubercle, we see its structure to be as follows :—A large quantity of fibrine partially organized, forming a meshwork or framework for the support of other elements, which framework stains dark, blue or black in hæmatoxylin solutions.

In and between the fibres are found clear spaces which presumably have been filled during life with *mucus or serum*, and supported by the framework are found *cells* of three distinct kinds :—1st. The small, shrivelled, contracted and dark cell, which Niemeyer has regarded as the tubercular cell *par excellence*, and which are undoubtedly shrivelled and contracted leucocytes. They have no demonstrable nucleus, and are found abundantly through the deposit and in the tissue surrounding that deposit. They are identical in their microscopical characters with cells frequently found at the peripheral part of scirrhous, described as indifferent cells, and which vary from the tubercular cells only in their number and in their less compact arrangement.

2nd. Cells larger than the preceding ones, with their protoplasm differentiable into nuclear and perinuclear portions; these are epithelial cells shed from the alveolar wall during the process of inflammation.

3rd. I may mention giant cells occasionally found in miliary tubercles about to undergo fatty change; these are simply large masses of protoplasm with an attempt at the formation of a nucleus. For my own part I have never succeeded in satisfactorily demonstrating giant cells in connection with tubercle. I have found them in connection with necrosed bone, and also in amyloid degeneration of the kidney, but never in tubercle.

This deposit so composed, fills the whole of the alveolus of the lung. When there deposited it is adherent to the alveolar walls and spreads by infiltration of the adjoining tissue with those small cells first described.

In comparing the microscopical character of a