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SYPHILIS AND LARDACEOUS DISEASE.

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When discussing the part played by syphilis in the etiology of aneurisms and atheroma of the aorta,[1] I was led to consider a doubtful point in the pathology of tertiary syphilis itself and its relation to lardaceous disease. I cannot help thinking that the relation between syphilis and lardaceous disease is one which throws some light on the pathological process in both affections.

Probably most medical men believe that tertiary manifestations of syphilis are due to the local presence of microbes, although these microbes of syphilis remain still to be discovered.[2] From this point of view gummatous formation in syphilis must be supposed to bear much the same relation to the microbe of syphilis that "caseous pneumonia" and the synovial swelling in tuberculous joint affections do to the bacillus tuberculosis. When the discovery of the real microbe of syphilis takes place, as it must some time, the microbe will almost certainly be found present in tertiary, as well as in primary and secondary manifestations of syphilis, though in the tertiary period it will probably be found almost completely localized to the lesions which it produces.[3] On the other hand, in the supposed sequelæ of syphilis, such as tabes dorsalis, the microbe will probably not be found present, and a clear demarcation will thus be established between these supposed "post-tertiary" or "quaternary" manifestations of syphilis and the true tertiary lesions.

It might be argued that because syphilitic gummata are apparently not infectious they cannot contain the microbe of syphilis. The fact, however, that the secretions from tertiary syphilitic ulcers have not been shown to be infectious is no proof that the local presence of a microbe is not the cause of tertiary lesions. If it had not been that a

[1] See this Journal,

[2] The supposed discovery of syphilis bacilli by S. Lustgarten (Medizinische Jahrbücher, Vienna, 1885, pp. 89 et seq.) was at first thought to solve the question, but further experiments have failed to confirm Lustgarten's views as to the specific nature of his bacilli. It has been suggested that some of the bacilli found by him in the primary sores may have been "smegma-bacilli," whilst the few found present in internal gummata may really have been tubercle bacilli, a secondary infection having taken place or a tuberculous caseous nodule having been mistaken for a gumma. The whole evidence, as well as that concerning the claims of other microbes, which have been described as possibly specific of syphilis, was carefully discussed by Prof. P. Baumgarten in his Lehrbuch der pathologischen Mykologie, 1890, vol. ii. pp. 681–686.

[3] See also the additional note at the end of the paper.

special method of staining had rendered it comparatively easy to detect the presence of the tubercle bacillus, and if there had not been animals easily infected by inoculation with the tubercle bacillus, the presence of this bacillus would never have been proved to be the cause of lupus.

It would seem as if the undiscovered microbe of syphilis, after it has run its usual course and worn itself out in the secondary stage, often remains in a latent or quiescent state scattered about the tissues of the body,[1] and that although unable to give rise to general symptoms in its host, it nevertheless retains the power of causing local harm by gummatous formation, especially when the tissues of the body have become weakened either by general causes, such as alcoholism, or by local causes, such as a traumatism limited to some special part of the body.

Many considerations are in favor of the view that gummata are caused by the local action of a microbe. I shall here confine myself to putting forward an argument in favor of this view which has not yet, as far as I know, been brought forward. It must be taken only for what it is worth, but, as fresh evidence, may be of provisional use, until the question be finally settled by the discovery of the real microbe.

Lardaceous disease is acknowledged to occur in syphilitic patients at times without any suppuration, but besides this fact there exists another curious connection between syphilis and lardaceous disease, namely, that a localized lardaceous formation in the neighborhood of visceral gummata may sometimes be recognized;[2] the latter occurrence, although hardly noted by authors, is very striking when observed. It is my intention here to use these facts as fresh evidence in favor of the view that a microbe is present in tertiary syphilitic lesions. I will endeavor to make myself clear:—

By the recent experiments of Dr. N. P. Krawkow (*Cent. für Allg. Path. und path. Anat.*, May 20, 1895) it has certainly been made clear that lardaceous disease is usually the result of absorption into the blood of products of the metabolism of parasitic microbes. Krawkow failed

2 There can be no doubt that methyl violet is a far more reliable stain than iodine for the lardaceous material; so Cohnheim and Cornil found it, and this is the opinion of N. P. Krawkow, who has recently worked on the etiology of the lardaceous formation (Cent. für Allg. Path. und path. Anat., vol. vi. p. 338). Lesser degrees of lardaceous disease would often escape observation but for careful microscopical examination, and doubtless for want of microscopical examination they often do remain unobserved, especially in the kidneys. I would point out that a routine series of microscopical examinations for early stages of lardaceous disease should be made in the kidneys from cases of chronic parenchymatous nephritis and "mixed" nephritis. This pathological anatomical study is required just as much as are further experiments on the production in animals of lardaceous disease before the laws determining the formation of this pathological material in the tissues of human beings can be finally determined. It is possible that there may be some kinds of chronic parenchymatous nephritis always associated with a slight lardaceous change in the kidneys, and that in such cases the nephritis and the lardaceous change may be both due to the excretion by the kidneys of toxins produced by the action of microbes in other parts of the body, for instance, by pyogenic microbes in the lungs of advanced cases of chronic pulmonary tuberculosis.

1 See also the additional note at the end of this paper.

to produce lardaceous disease in animals when the suppuration was induced by turpentine instead of by pyogenic microbes,[1] whereas he actually succeeded in inducing lardaceous formation in the tissues of a rabbit by repeated injections of the mere filtrate of a culture of the bacillus pyocyaneus, that is, by the metabolic products of a microbe without the microbes themselves.[2]

In human beings lardaceous disease hardly occurs without definite suppuration except in cases of syphilis. Krawkow seems to have shown that when it arises from suppuration it is due to the microbes of suppuration. There seems to be little doubt that it occurs sometimes in cases of syphilis when there has been no suppuration at all or only very slight suppuration; it seems, therefore, not unreasonable to suppose that in such cases of syphilis the lardaceous formation is due likewise to a microbe, although not a pyogenic microbe, namely, to the as yet undiscovered microbe of syphilis.

The occasional localization of the lardaceous change in the neighborhood of syphilitic gummata may be taken as further evidence both that gummata are due to the local presence of the microbe of syphilis (whatever the microbe may ultimately turn out to be), and that this microbe has the power of causing lardaceous disease without the help of pyogenic microbes. This further evidence depends on the fact, as pointed out below, that if these theories be accepted, the localization of lardaceous formation in the neighborhood of syphilitic gummata can easily be explained, whereas it cannot be so easily explained by any other theories.

Granting that a gumma is a formation caused by the local presence of a specific microbe of syphilis (just as a tubercle is caused by the local presence of the tubercle bacillus), then we can understand that the tissue in the neighborhood of a gumma is more likely to be affected by the products of this microbe than are tissues further removed from the gumma. The metabolic products of the microbes inhabiting and causing the gumma must to some extent leak out and infiltrate the neighboring tissues, before being carried off by the lymphatics. If, as seems probable, one of the properties of these microbic products is to cause lardaceous formation in certain tissues (especially the walls of the minute bloodvessels) on which they can act, then it is not surprising that the lardaceous change should occasionally be found especially marked in the immediate neighborhood of gummata, or that it should

[1] He points out, however, that an abscess induced by the injection of turpentine may easily become secondarily infected with pyogenic microbes, and thus lardaceous disease, readily brought about by the action of these microbes, might be incorrectly attributed to aseptic suppuration caused by turpentine.

[2] Krawkow suggests that the lardaceous material may be a product of this microbic metabolism, and follows those who think that it is deposited in the affected tissues by a process of degeneration.

take place there before occurring in more distant parts. In other words, it seems likely that the microbic products, manufactured within the gumma, should affect the surrounding parts before being carried off into the general circulation; after which these microbic products gradually produce the characteristic changes of lardaceous disease in the spleen and other organs peculiarly liable to this change before they are finally excreted by the kidneys.

In this short paper I have endeavored to show how lardaceous formation in the tissues, when occurring in association with active tertiary syphilitic manifestations, and especially the lardaceous formation occurring in the immediate neighborhood of gummata, may be taken as additional evidence of the already probable view that active tertiary syphilitic manifestations are caused by the presence of a specific microbe at the site of the lesion. The conclusions which appear most probable may, I believe, be summed up thus:

That there is a specific microbe of syphilis, the local presence of which is the cause of the tertiary lesions of syphilis; that the metabolic products of this microbe have, like those of some other microbes, such as the staphylococcus pyogenes aureus and the bacillus pyocyaneus, the peculiar property of producing lardaceous formation in the tissues of the hosts.

Additional note on the view that tertiary manifestations of syphilis are caused by the local presence of a specific microbe.

By those who held that syphilis when it has reached the tertiary stage cannot be transmitted by inheritance from parents to their children, it might formerly have been alleged that the exceptions to this rule are apparent only, and that really in such apparent exceptions remnants of secondary syphilis must have existed in one of the parents. This explanation is, however, now untenable, for no one would call syphilis after it has existed fifteen years secondary syphilis, and cases are known where parents fifteen years or more after the commencement of syphilis have transmitted it to their offspring. Professor Fournier proves this by instances, and considers that of cases where syphilis has been transmitted to the fœtus, in about one case out of ten the parental syphilis dates from six years back or more.[1]

If, on the other hand, syphilis be allowed to be due to a microbe which, after the secondary stage, is supposed often to remain in the body in a latent or almost latent condition (though able to become

[1] Out of 562 cases where the pernicious effects of hereditary syphilis showed themselves, in sixty cases the parental syphilis was acquired six years previously or more. See Professor Alfred Fournier, L'Hérédité Syphilitique, Paris, 1891, p. 122.

active again as the producer of the local manifestations in tertiary syphilis), then the whole matter may be more easily explained.

According to this view, after the secondary period of syphilis is over, the surviving microbes of syphilis are supposed to be lying quiescent in different tissues of the body, but may occasionally be washed out by the blood-stream into the general circulation. If we accept this explanation it does not seem astonishing that parents while suffering from active tertiary syphilis produce children untainted by syphilis, just as parents whilst suffering from active tuberculous disease produce children perfectly free of congenital tuberculosis. Neither is it surprising that children of parents suffering from active or even latent tertiary syphilis may at times be born with congenital syphilis, in the same way that children of mothers suffering from active tuberculosis may be occasionally born with congenital tuberculosis, as has been conclusively proved by the observations of Birch-Hirschfeld and Schmorl (*Beiträge zur path. Anat.*, 1891, vol. ix. p. 428); P. Londe (*Rev. de la Tuberculose*, 1893); and Schmorl and Kockel (*Beiträge zur path. Anat.*, 1894, vol. xvi. p. 313).

In fact, just as syphilitic gummata and gummatous infiltration may be compared to tuberculous caseous nodules and tuberculous disease of synovial membranes, so, according to this theory, the process by which the fœtus may become infected from a mother in the tertiary period of syphilis may be compared to the process which Schmorl, Birch-Hirschfeld, etc., believe to be the one by which tuberculosis spreads from the mother to the fœtus. Professor Birch-Hirschfeld[1] suggested that in cases of congenital tuberculosis the tubercle bacilli "grew through" from the maternal into the fœtal portion of the placenta, and thus infected the fœtal circulation; this view has, moreover, gained support from the observations of R. Kockel and M. Lungwitz on "Placental Tuberculosis in Cows" (*Beiträge zur path. Anat.*, 1894, vol. xvi. pp. 294 *et seq.*).

We can easily understand, then, that if there be any microbes of syphilis in the general circulation of a woman at some period of her pregnancy, these microbes may reach and be lodged in the maternal portion of the placenta; there they may develop and multiply locally and thus "grow through" into the fœtal portion of the placenta, where some may enter the fœtal circulation and give rise to congenital syphilis.

This theory, moreover, explains why a quiescent syphilis in the mother is probably more likely to be transmitted to the offspring than a quiescent syphilis in the father, for in the case of the mother there is the

[1] This was from analogy with what was observed to take place in the case of pregnant animals inoculated with anthrax. See Birch-Hirschfeld, "Ueber die Pforten der placentaren Infection des Fœtus," in Beiträge zur path. Anat., 1891, vol. ix. pp. 383 *et seq.* Birch-Hirschfeld suggested that the process of infection of the fœtus in congenital syphilis and congenital tuberculosis might be similar to that observed by him in the case of anthrax.

long period of pregnancy, during which the ovum may become infected in the way described. This theory will also elucidate cases, where a healthy child is born of syphilitic parents (not necessarily undergoing anti-syphilitic treatment at the time), though subsequent children suffer from congenital syphilis.[1] In such a case one must suppose that during the earlier pregnancy the syphilis microbes were lying quiescent in the mother's body, or at least that any which chanced to get into the general circulation failed to infect the placenta; but that during subsequent pregnancies the fœtus became infected either by some of the (previously quiescent) microbes getting into the general circulation and lodging in the placenta, or else by some tertiary syphilitic process localized in the neighborhood, reaching the fœtal circulation by spreading through the placenta.

By analogy mercury may be supposed to owe its influence on syphilis to some specific action on the microbe. No one will question the striking use of mercury seen at times in cases of tertiary syphilis as well as in its earlier stages. Those who think that primary and secondary syphilis are due to the presence of a microbe, on which mercury exercises an antagonistic action, must therefore admit that the beneficial effect of mercury in tertiary syphilis may be used as a slight argument in favor of the view that the tertiary as well as the primary and secondary manifestations of syphilis are due to the presence of a specific microbe. It would, however, be unprofitable here to discuss at greater length well-known evidence in support of a view which already appears probable; so probable, in fact, that the only additional evidence any longer really needed is the evidence which would convert the theory into a fact, namely, the discovery of the microbe itself.

[1] See Fournier, op. cit. pp. 263–267.

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