On the pathology of hepatic abscess / by F. N. Macnamara.

(14)

ON THE PATHOLOGY OF HEPATIC ABSCESS.

From the Indian Annals of Medical

By F. N. Macnamara, M.D. Asst. Surg. B.M.S.

The term *suppurative inflammation* which is applied to the changes preceding abscess of the liver, expresses the view that is almost universally held regarding the immediate cause of the abscess, and the nature of those changes.

In a recently published work,* Dr. Wilks writing of inflammation of the liver, says, "in cases of suppuration the adjacent tissue may be found in the various inflammatory stages; and before the pus is formed and during the pyrexia a hepatitis of course exists, but in this stage it is rarely seen; and as an idiopathic affection, perhaps never in this country. In tropical climates, however, a hepatitis is a common affection leading to various results as abscess or gangrene." Again, "in such a case you may see the various processes preceding the abscess; before the tissue breaks down you perceive the lobules in their natural position and shape, but of a yellow colour from the infiltration of pus, and preceding this the tissue of a deep red, and the microscope is necessary to discover the inflammatory cells in it. You find in the liver as in the lungs that the pyæmic inflammation is first recognised by a congestion, and the points of suppuration are thus seen surrounded by this deep red halo."

* Lectures on Pathological Anatomy by Dr. Samuel Wilks Asst. Physician to Guy's Hospital, and Lecturer on Pathology. 1861.

This is an accurate description of the appearances which present themselves in one of the earlier stages of liver abscess, but as will be presently shown the yellow *quasi* pus infiltrated mass, surrounded by the deep red halo is not the first of the appearances, nor is it preceded by a point of congestion in the liver abscess which usually accompanies dysentery.

Before describing the appearances which anticipate the abscess, I would allude briefly to two or three points in the general history of that affection. And, first, I think there can be no doubt as to the fact of the very common association of liver abscess with dysentery—the nature however of their connection is uncertain, and it will be only incidentally alluded to it in this paper. I suspect Dr. Budd's doctrine may be in many cases true, so far as it holds the abscess to be due to the poisonous effects of morbid matter taken up by the portal vein, and precipitated upon the liver. And may it not be that the liver abscess (or the state of the liver which precedes it), so impairs the functional ability of the organ that the large intestine is compelled to take on some kind of compensatory action, and is thereby either predisposed to the attack of the dysenteric poison (if such there be) or, as an immediate result of unwonted overaction, becomes the subject of dysenteric changes?

Another point to be noticed in the history of liver abscess, is its rarity in many localities where dysentery abounds. Thus very few cases of liver abscess presented themselves in connection with the dysentery of the Crimean war. Dr. Marston, in a paper published in the Medical Times for September 1856, states that out of the great number of soldiers suffering from dysentery who came under his charge in the Malta Hospital, only two were the subject of liver abscess. Dr. Baly's testimony is to the same effect as regards the dysentery of the Millbank Penitentiary, and there is much published evidence of the like kind, for other parts of Europe. Very different is the case in this country; all Indian Writers on the subject, bear testimony to the constant association of liver abscess, and dysentery. In the Madras Presidency, out of 51 cases of death from dysentery, 26 cases of hepatic abscess are reported. Dr. Morehead reports in Bombay, 12 cases of hepatic abscess out of thirty deaths from dysentery. Dr. Parkes reports seven cases out of 25 deaths. These reports would give us 42.45 as the per centage of liver abscess in deaths from dysentery, or taking Dr. Morehead's, and Dr. Parkes'

cases only, 34 per cent, and this per centage agrees with the results of my own observations. In 48 autopsies which Dr. Marston made of patients dying from dysentery, he found only two liver abscesses—little more than four per cent. What is the explanation of this vast difference? Why is liver abscess so much more commonly associated with dysentery in the East Indies than in Europe? Can the two, as has been suggested, be without mutual relation, but running their course together dependant upon one and the same cause? If so the cause of dysentery must differ here and in Europe, or the cause being the same, there must be some state of the liver in those who become the subjects of dysentery in India which predisposes to hepatic abscess.

Abscess of the liver has been noticed by the French surgeons as often occurring in Algeria in connection with dysentery, and they have observed that the association is most frequent in old men, and men who have been long in the country.

Dr. Parkes has,* as his 5th variety of hepatitis—"Hepatitis "suddenly arising and denoted by the usual nosological "symptoms." This, he observes, "appears to be a disease "chiefly of new comers; and is probably a consequence of "great overaction of the liver. According to my limited "experience, although sometimes seen, it is by no means the "usual form, nor has it much tendency to terminate in suppura-"tion." Such is my own experience, and indeed the symptoms which usually attend the formation of a liver abscess when associated with dysentery, are in the beginning very obscure—perhaps amounting at most to a little indefinite uneasiness about the liver region with ill defined tenderness there, and a more depressed condition of the system, or excited state of pulse than can be accounted for by the purely dysenteric symptoms.

Having premised so much regarding the history of liver abscess, I may describe the condition of the liver, general and microscopical, which almost constantly presents itself in Europeans dying of dysentery with liver abscess.

The liver is usually, but not always enlarged, and seldom to any great extent. To the finger its surface is firm, and the cut surface to the eye has a normal consistency, and density, but the substance is friable, and readily gives way before the handle of the scalpel.

---

* Remarks on the Dysentery and Hepatitis of India, by Dr. Parkes. 1846.

The colour of the liver is very constant, and characteristic, an uniform pale drab colour, this pervades the whole substance and without any of that mottling, or concentral alternation of yellow and brown substance which is commonly seen in the lobules of the fatty livers of phthisical patients. The shape of the liver is generally preserved, unless when it is affected by the bulging of an abscess.*

Such a liver may become very much congested, as indeed it is often found in patients who have died in the earlier stage of acute dysentery. But though the congestion hides the drab colour, it may be discovered when the bloody serum which exudes is scraped away. Pale superficial patches of fatty degeneration may be seen in these as in otherwise healthy livers, they are not however of interest in the present enquiry unless as showing that the liver may become, like other organs, the subject of very local changes.

On cutting into the diseased liver we discover the changes which immediately precede an abscess, or accompany its earliest stages. Scattered through the substance may be seen one or more patches in which a degeneration has clearly occurred. These vary in size from a pin's head to a large pea, but seldom pass beyond this, till they have entered into the next stage. They are of a paler colour than the surrounding parenchyma, and have a softer consistence—as regards both colour and consistence they merge gradually into it. These patches, present the first discernable change which precedes the abscess. Some or all of them may have passed into the next stage, and if so, we find them larger, may be the size of a walnut, but, if we do not hit the central portion we see but a soft, straw coloured, spot fading off into the neighbouring substance, no congestion, no halo of redness. In the centre of the area of degeneration we shall find the tissue liquescent, or completely fluid, presenting to the eye the appearance of true pus, and here we have an abscess, yet no inflammation, and the puriform fluid may not present a single pus corpuscle to the most searching microscopical examination. There may be a central cavity, the fluid contents having disappeared, but whether the cavity contains fluid or no, its parietes are soft, ragged, and spongiform. In the third stage, and this is more common in larger than with smaller patches, there is a surrounding halo of congestion. I have

* The enlarged, drab coloured liver is frequently found in the same class of subjects dying from other diseases.

noticed that where two patches approximate, the redness may extend between them, while there is none on the further part of their circumference.; this points to the meaning of the congestion, and proves clearly that it does not precede the local degeneration.

Such is the commencement of the abscess, and too often it spreads, more and more tissue breaking down, and the neighbouring cavities uniting to form the immense abscesses we not unoften see at the post mortem examinations of Europeans of the poorer class, who die in Calcutta. Things may, however, take a turn for the better, and if nature be equal to the effort, and be well supported, the cavity or degenerated patch becomes encysted by lymph thrown out by the vessels of the halo, the fluid may be absorbed, and the dead tissue dry up into a semi-solid mass.

In the liver of an European who died in the Medical College Hospital of dysentery, I found in the right lobe near its upper edge a congested spot about the size of a walnut. In the centre of this were two white masses, each the size of a large pea, separated from one another by a little fibrous tissue. They had the consistence and appearance of cream cheese, and each was enclosed in a fine membranous cyst. There was a little moisture between the outer surface of each cyst and the parenchyma. On microscopic examination, the contents of the cysts proved to consist chiefly of fat, mixed with granular matter, and the debris of liver cells. These cysts with their contents presented the happiest termination of liver abscess, but they appeared to have become the focus of a new congestion under the disturbing influences of the attack of dysentery from which the patient died.

Before describing the microscopical appearances, I would draw attention to the great frequency of fatty degenerations in the tissues of that class of Europeans, *viz.* soldiers and sailors, upon whom our observations are chiefly made. This fact was, first pointed out by my brother Mr. N. C. Macnamara who while serving with the 1st Fusiliers at Dinapore had abundant facilities for observing on the point. In a paper published in the 5th vol. of the "Indian Annals" he stated, that in almost every post mortem he had made on soldiers of that Regiment, he found fatty degeneration of the liver, kidneys, vessels, and heart, sometimes of one, sometimes of all these organs. He rightly traced the cause of the change to the climate acting on men who were overfed with animal food, and alcoholic drinks, and who passed their days in a crowded,

darkened barrack, often far the greater part of it, not given to eating or drinking, in sleep.

As regards the liver we must carefully distinguish between fatty infiltration, and fatty degeneration, the one may no doubt run on into the other, but under favoring circumstances the fatty infiltration may pass away, leaving the secreting structure quite intact.

The granular degeneration which I am about to describe, frequently, I suspect, runs a parallel course with the fatty infiltration, dependent upon the same causes as far as the mode of life of the sufferer is concerned. To the granular degeneration may succeed a true fatty degeneration, for the cells, loaded with an abnormally concentrated plasma, and surrounded by it, will, under the influence of some temporary congestion, become incapable of nourishing themselves as they should, and hence the fatty change in their contents. I had already recognized this change when Frerich's book* on the liver came into my hands, and my belief in the order of the sequence was confirmed by finding that he takes the same view of it. He says (Vol. 1. page 292). In allusion to the ordinary fatty liver of tuberculous patients, "In the latter cases the fat is deposited " in the interior of the cells directly from the blood; in the " former cases (that is in the cases where exudation processes " constitute the causes of fatty degenerations) the tissue first " becomes infiltrated with abnormally concentrated blas- " tema in consequence of which the endosmotic properties of " the cells are altered, and their nutrition is impaired. Under " such circumstances the cells are seen in the first place to " become filled with a granular albuminous precipitate, ob- " scuring the nucleus which only becomes visible on the " addition of acetic acid; it is not till afterwards that oil " globules make their appearance in large numbers, but even " then they are seldom remarkable for their size. This fatty " degeneration of the liver which we believe to differ from " fatty infiltration, is usually limited in its extent to the " circumference of inflammatory deposits, recent exudations, " cicatrices: &c. It is found extending over the whole organ, " apparently as a terminal stage of infiltration of colloid " matter, in the so called lardaceous, or waxy liver."

I have, however, found this state of granular degeneration extending throughout the liver, under other circumstances than those alluded to in the latter part of the foregoing quotation.

* Translated by Dr. Murchison, published by the new Sydenham Society.

The most prominent microscopic change I have been able to discover in the dysenteric liver is a granular degeneration of the cells, attended in the more advanced stage with a deposition of intercellular granular matter. This granular change may be equally present in livers in which abscesses have formed, and in those in which there are no indications of them.

The cells undergoing this degeneration often look scaly, and their edges are generally ragged and disintegrated. Some cells look in fact like a mere aggregation of fine granular matter held together by the cohesion of the particles, and not at all sustained by any cell wall. In other cells, not so advanced in degeneration, the cell wall and the nucleus may be detected, but the latter looking as if choked by the quantity of granular substance deposited about it. In many cells the most careful examination has failed to show me any nucleus.

I have on three or four occasions when examining these disintegrating cells observed that they appeared set, I might almost say scattered, in a granular matrix, which has seemed in more than one instance to have a semifibrillated structure. Such a precipitate of albuminous molecular matter both inter and intracellular may arise from repeated attacks of slight congestion of the liver, or from a long continued engorgement of the organ.

The amount of fat varies much; in some instances, I could scarcely detect a single fat globule, and this too in cells which had undergone extreme degeneration, at other times much fat was mixed up with the granular matter, but I think fat is most commonly present in cells in which the granular change is commencing. Not unfrequently we see abscesses of considerable size looking as if they had resulted from a simple liquefaction of the tissue, and it is then almost impossible to say where the wall of the cavity is, by such imperceptible gradation does the softening increase from some distance around into the central fluid portion. In the liquescent part and neighbourhood, fat is always present in large quantity. In the white patches there is also an increased quantity of fat, and there the degeneration of the cells may be seen in an extreme state. In the central fluid portion the chief constituent is fat, and granular matter, liver cells in all stages of disintegration, and pieces of tubules. Pus globules may be altogether absent, or present in more or less abundance. It has appeared to me that the fluid approaches to typical pus in proportion to the degree of the surrounding congestion. In the instance of the

only inflammatory abscess I have seen in it's earliest stage, the very small quantity of fluid that was present, had all the characters of true pus.

Excepting in one instance I have not examined any liver with this form of associated dysenteric abscess in which I did not discover a granular change. The exception to which I allude is a remarkable one, and presents several points of interest in connection with this subject.

The patient a middle aged European, had been sometime in India. He was a man of very careful, abstemious habits and his occupation obliged him to be much in the open air. He contracted dysentery through exposure at the end of the rains, and died shortly after of empyema due to the bursting of an hepatic abscess into the right pleural cavity. The liver had a slightly congested appearance, there was one large abscess in the right lobe, in the left lobe there were numerous patches of a pale lemon colour, circular in form, varying in size from a pin's head to that of a large walnut, a sharp line of demarcation separated the white softening from the tissue around; about the largest patches only was there a slight halo of congestion. I especially noticed in this liver the presence of congestion between neighbouring patches, and *only between* the patches. In several of the largest patches there was complete central liquefaction, the central cavity containing a fluid which to the eye was undistinguishable from the most laudable pus. The fluid had a slightly acid reaction. This is the only case in which I have seen a line of demarcation between the dead and the surrounding tissue, in all others, unless where a cyst had formed, the white softening merged into the neighboured tissue. In this liver too there was a most marked difference between the cells of the degenerate area, and those of the living tissue, even though taken from the closest neighbourhood on either side of the line of demarcation. The cells of the general mass were fairly healthy the nucleus and cell-wall clear and well defined, while within the area of white softening, the cells were disintegrating, many of them looking like ragged edged masses of granules, retaining only somewhat of the size and shape of liver cells. Fat globules were abundant in and amongst these cells. The fluid had the usual constitution. Not a single pus cell could be detected in several slides of it which were put under the microscope.

In this case we have an instance of local death occurring at many points quite unpreceded by local congestion, or inflammation, and this in fairly healthy tissue. The determining

cause of the secondary abscesses was probably the arrest of morbid matter taken up from the primary abscess. It is of course impossible to say what was the mode of origin, or the cause of the primary abscess, and I would here carefully guard against the supposition that I contend all hepatic abscesses originate in the way I have been describing. The following case proves the contrary. An European was admitted with acute dysentery into Hospital under my care on the 23rd of May. He was a seaman, and had been some years in India, the last two in Calcutta; he confessed to being a very hard drinker, and ascribed the present attack, which commenced on the 16th, to hard drinking. The dysentery was very acute, there was well marked tenderness over the liver, and its edge could be felt projecting below the ribs. The man got rapidly worse and died on the 1st June.

Post Mortem Examination of Liver.—Liver much enlarged, edges rounded, of firm consistence, colour paler than natural, but this could only be discovered after wiping away the bloody serum which exuded freely. In one place towards the edge of the right lobe, was a highly congested patch the size of an egg, and in the very centre of this a small collection of pus not larger than a pin's head. The cells throughout the liver were much infiltrated with fat, but were otherwise healthy looking, the nucleus clear and bright, and the cell wall distinct. The cells in the congested area retained their distinctness, but towards the centre there were many altered in shape and varying in size, some were very small. The central fluid was chiefly pus, but mixed up with the pus cells, were liver cells, free fat globules, and a few blood corpuscles, and fibro-cells. I thought I could trace all the gradations of appearance between the liver cells and fibro-cells on the one hand, and the liver cells and pus cells on the other, the gradations marking the degree of failure on the part of the textural germ to grow into a healthy liver cell.

I have already alluded to the causes which I think produce this granular degeneration of the liver. Shortly, I believe them to be exposure to the sun, the heat of the climate, malarious influences; these, operating on a class of men who eat largely, and as a rule indulge very freely in alcoholic liquors, would suffice to bring about a state of chronic engorgement of the liver substance, aggravated by occasional attacks, of temporary congestion, and as the result of this granular degeneration.

The liver having fallen into this degenerate state, breaks

down at points under some pressure such as an acute congestion might afford, or owing to the arrest of some morbid matter; influences which in a healthy structure might bring about inflammation, or produce but transient effects. The unhealthy tissue is overtaxed, its deficient vitality gives way, and death and subsequent liquefaction ensue. The local death is (in almost every case) as Professor Paget expresses himself "the extreme of degeneration."

In this death of portions of the hepatic substance we have an example of the consequence, to which the same eminent Pathologist is alluding in speaking of the liability of old and intemperate persons, to local mortifications after slight injuries. "Already degenerate they perish through the addition of "what in healthier persons would have led to only some dege-"neration, or to the inflammatory process in the injured "part."*

Since then the form of liver abscess which most commonly accompanies dysentery commences not with inflammation, but in the degeneration and death of the tissue, may we not find in the fact, some help towards an explanation of the common association of the two diseases? I myself believe that the explanation so ably propounded by Dr. Budd is in some cases a just one—that the portal blood becomes contaminated with morbid matter, and that this arrested in the degenerated liver substance commences the abscess, yet not always by setting up inflammation, but by destroying the nutrition, of the part and thus causing its death. This supposition removes the objection which has been raised against Dr. Budd's theory on the score of absence of inflammation in the portal vessels.

Though it is not my wish in this paper to enter at length into the so often vexed subject,the connection of hepatic abscess, and dysentery, I cannot refrain from alluding to one point in the clinical history of the latter which bears upon it.

During the earlier stages of acute dysentery there is always much congestion of the liver, and a great outpour of bile. As long as inflammatory products are present in the stools, so long are they loaded with bile, the gelatinous, and blood stained mucus, sometimes swimming in a pot-full of deep amber coloured fluid. The bile disappears with the inflammatory products, and I have been accustomed to look upon the change in the color of the stools as a most trustworthy sign of the arrest of the

* Lectures on Surgical Pathology, Vol. I.

dysenteric action; there is I believe no better sign of this than the expulsion of white, or almost white fecal matter. Now though the defenders of the Ipecacuanha, and perhaps of the mercurial treatment of dysentery, look upon this outpour of bile as due to the so called emulgent action of their remedies, and have greatly prided themselves on their power, and manifest effects, is it not really the result of a reflex action exerted upon the liver by the diseased intestinal mucous surface, and is not this reflex action the cause of the congestion of the liver which accompanies the first stages of dysentery? And may it not be that one effect of this congestion upon the degenerated structure of the liver is so to interfere with its nutrition as to bring about a local death at one or more points? May not this in many cases be the explanation of the association of dysentery with hepatic abscess?