

THE PATHOLOGY OF THE FATTY, AMYLOID, AND PIGMENTARY INFILTRATIONS OF THE LIVER.¹

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WHEN I last had the opportunity of addressing you, I pointed out at some length the normal structure of the liver, and you will doubtless remember, in tracing the lobular distribution of the various vessels, we noticed the branches of the hepatic artery and portal or interlobular veins were either accompanied by a bile-duct of equal size, or, whilst separated from it by a more or less distinct interval, their mutual connexion was shown by threads or bands of their common fibrous sheath stretched between them. The intra-lobular or hepatic vein is always isolated and separated from the other vessels of the liver by a portion of hepatic parenchyma, so that in the microscopical examination of any given section of the liver there can be no difficulty in at once distinguishing between an afferent and an efferent lobular vein. At the periphery of the lobules we noticed a divergence of the various vessels; the portal vein, passing between two or more lobules, was seen to distribute its various terminal branches just within the margin of the periphery. The hepatic artery penetrated half-way into the lobule, and there broke up into its capillary network, whilst in the centre surrounding the hepatic vein we found its commencing venous network; so that, roughly speaking, we may divide the lobular distribution of the vessels into three concentric zones—that of the portal vein, the hepatic artery, and the hepatic vein. I say we may make this classification roughly, for it must ever be borne in mind that these zones are not sharply marked off, and in a strictly physiological sense do not exist, but pathologically the division is useful. Fatty degeneration, amyloid degeneration, and cyanotic atrophy, are found affecting respectively the zone of the portal vein, hepatic artery, and hepatic vein.

In the consideration of the pathology of the fatty liver it is important we should bear in mind the exact meaning of the words infiltration and metamorphosis. In metamorphosis the appearance of fat in a structure is but the forerunner of the complete transformation and final destruction of its normal histological characters, whilst fatty infiltration is but the deposition of fat in a part without of necessity the destruction of its physiological characters; in the one case the fat is the result of protoplasmic decomposition, in the other it is but a superfluous constituent brought to the cell from without, and, if removed, leaves the cell in its normal state. The microscopical characters of the two affections are also different. In metamorphosis the cell—I use the term in its general sense—is seen to contain scattered throughout its protoplasm and nucleus a number of minute oil-globules, having no tendency to run together; the cell is, in reality, converted into a compound granular corpuscle, the periplast of which, after a time, ruptures, the oil-globules being set free and the cell destroyed, whilst in infiltration the cell rarely contains more than two oil-globules, which have a great tendency to join and form a single large globule which pushes the nucleus to one side, in many cases so obscuring it that its presence cannot be detected, but it invariably reappears if the oil is reabsorbed.

I do not propose to enter upon the consideration of the general pathological question of the deposition of fat in the body, but will simply content myself with the remark that this takes place—1st, whenever the food contains an excess of fat, or of aliment capable of conversion into fat; or 2ndly, owing to the process of oxidation being imperfectly carried on both generally and locally. It is in this second sense that we are most interested, for any disease interfering with the oxygen-carriers of the body necessarily causes a deficient oxidation of the fat. And here we have the simple explanation of the frequent occurrence of fatty liver in cases of anæmia and pulmonary disease; for in the latter we have imperfect oxygenation of the blood, and consequent imper-

fect oxidation of the fat, whilst in the former there is a diminution in the blood of the red corpuscles.

The fatty liver with which the pathologist is so familiar is caused by a fatty infiltration, not a fatty metamorphosis. That the fatty matter enters the liver cells from the blood is strikingly shown by the fact that the infiltration invariably begins in the area of distribution of the portal vein. In the section I have placed under the microscope you will notice each lobule is distinctly mapped out; by the use of the staining agent, perosmic acid, this is most beautifully shown. As the infiltration advances the portal zone becomes wholly impregnated with oil, whilst the middle or hepatic zone shows it in an earlier stage. The vitality of the cells is, however, never materially impaired. The surface of the fatty liver is smooth and glossy, whilst its edges are thickened and rounded; it pits on pressure, the pits remaining, and is of a flabby or doughy consistence. It is enlarged particularly in the latter stage, but has a lower specific gravity than usual. On section in the early stage of the disease the organ is found to be congested, with the borders of the lobules of a canary colour, distinctly isolating them. The centre of the lobule is congested, but in the later stages the lobules are found to be of a universal buff colour, there being no differentiation present, the organ is likewise very anæmic and feels greasy, whilst a number of oil-globules may easily be scraped from the surface with a knife.

The waxy or amyloid degeneration of tissues is caused by their infiltration with a peculiar translucent homogeneous substance, having the appearance of ground glass. There are various theories as to the nature of this substance. Virchow looks upon it as vegetable cellulose in consequence of the blue reaction of both with iodine and sulphuric acid. Meckel, because of its reddish-brown reaction with sulphuric acid, considers it a form of cholesteroline. Dr. Dickinson thinks it is simply dealkalised fibrine deposited in the various tissues, such constituents as potash and phosphoric acid being wanted. It seems to be of an albuminoid nature, most probably poured out from the blood in a fluid condition, and subsequently coagulating. As fibrinogen exhibits constantly the well-known tendency to become solid, this, more than any other blood-albuminoid, seems to claim attention. But whatever may be the exact character of the infiltration, this much is certain, that the normal composition of the blood is altered, for although we search the blood of persons affected with amyloid disease, we search in vain for a material having the same chemical reactions as the effused substance. The simultaneous appearance of the degeneration in various organs after some such wasting disease as a prolonged suppuration, or a pulmonary phthisis, and the invariable commencement of the disease in the smaller nutrient vessels, point, I think, clearly to this conclusion.

The liver, perhaps, is the most frequent seat of the disease; the organ becomes much larger and heavier than normal, varying in weight from six to eight pounds; its specific gravity, as we should expect, is also increased, the surface is smooth and polished, and the capsule appears stretched, whilst in consistence it resembles india-rubber, it is hard but elastic, pitting on pressure, but the pits readily disappear. On section the organ looks waxy, glossy, and of a dull smoked pink colour, very much like smoked salmon, or urine which contains blood. On the surface irregular stellate-shaped yellow marks are seen, which is the liver tissue in a state of infiltration; the organ is very anæmic on the addition of a watery solution of iodine, consisting of equal parts of solution of iodine and water; the waxy parts are stained of a mahogany-brown tint.

The last infiltration of the liver which I propose to consider is the pigmentary, or that condition which is commonly spoken of as the nutmeg liver, or that of cyanotic or red atrophy. In this the liver is the seat of long-continued mechanical hyperæmia, the result of lesion of the valves of the heart, or pulmonary disease, or, in fact, any disorder giving rise to passive congestion of the systemic veins, in consequence of which dilatation and thickening of the hepatic veins ensue. In the earlier stages of the disease the organ is increased in size, owing to the large quantity of blood it contains, but it shortly undergoes a gradual diminution in bulk in consequence of cellular atrophy. On section the liver is found to be much congested, large quantities of blood oozing out from the dilated vessels; the sectional surface presents a well-marked mottled appearance; the centres of the lobules at first have a bluish tinge, but after a time dark red points appear, whilst the periphery is pale.

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