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Reprinted from the Transactions of the Pathological Society of London for 1879.]

Note on the morbid anatomy of hypertrophic cirrhosis of the liver.

By Robert Saundby, M.D. Edin., M.R.C.P. Lond.

For a long time the French pathologists have been aware that there are cases of hepatic cirrhosis which do not conform to the dictum of Bichat, "Cet état ne se complique jamais du volume extraordinaire du foie." In 1859 MM. Charcot and Luys presented a memoir to the Société de Biologie, in which they maintained that the new growth in hypertrophic cirrhosis penetrates the lobules, is therefore intralobular, while in ordinary cirrhosis it surrounds the lobules and is perilobular. In 1871 M. Ollivier contributed a series of papers to the Union Médicale, in which he sought to establish a separate clinical identity for these cases. In 1874 M. Hayem published a paper in the Archives de Physiologie, which contained an account of two cases of hypertrophic cirrhosis with microscopical descriptions, showing that the growth invaded the lobules, and, according to this observer, followed the course of the capillary blood-vessels. A few months later M. Cornil drew attention, in the pages of the same journal, to the numerous biliary canaliculi present in the dilated portal canals, fissures, and spaces of cirrhotic livers, especially of the hypertrophic form; an appearance which M. Cornil had described in 1871 in a case of acute yellow atrophy, which had run a somewhat protracted course. In 1875, M. Hanot wrote his inaugural thesis upon a form of hypertrophic cirrhosis accompanied by jaundice, for which he sought to establish a definite clinical and pathological position. During life the disease may be recognised, according to M. Hanot, by the history of early and persistent jaundice, the even enlargement of the liver, and the absence of ascites; anatomically, the liver is enlarged, its surface is

smooth, often covered by traces of perihepatitis ; the organ is tough on section, its cut surface is yellow or olive green in colour. Microscopical examination reveals, in addition to the new growth, which he describes as intralobular, a great development of biliary canaliculi in the extralobular connective tissue. M. Hanot regards this form of cirrhosis as originating in an inflammation of the bile ducts leading to obstruction to the outflow of bile, dilatation of the capillary ducts, and extension of the catarrhal inflammation to these latter, which become at first filled with cells, and finally lined with epithelium.

The doctrine of a form of cirrhosis originating from changes in the bile ducts was taught by Rokitanski, but opposed by Frerichs. In 1866 Wyss published an account of the structural changes in jaundice, and described some degree of new connective-tissue growth which, he says, never invades the lobules. In 1872 Heinrich Meyer, experimenting on animals, found an increase of the connective tissue both around the vessels and in the lobules. Dr. Beale published a case of cirrhosis resulting from obstruction of the common duct. In 1873 Dr. Wickham Legg described the results of ligature of the common bile duct in cats,[1] the operation being followed by enlargement of the liver and a formation of new connective tissue around and within the lobules he ascribed these changes not to the obstruction to the flow of bile, but to the extension of inflammation along the duct from the seat of the ligature.

In 1876 MM. Charcot and Gombault[2] published an account of the changes in the liver after ligaturing the bile duct in guinea-pigs, the principal features of which were enlargement of the liver, development of new connective tissue around and within the lobules, multiplication of the biliary canaliculi, and atrophy of the liver cells. They observed that the newly formed canaliculi could be traced in certain places in direct continuity with the rows of hepatic cells. In a subsequent paper[3] they adopt M. Hanot's description of the clinical characters of hypertrophic cirrhosis with jaundice, as those of biliary cirrhosis, which is characterised anatomically by the new growth surrounding *each* lobule (monolobular), invading the lobules (intralobular), and the development of a numerous network of

[1] 'St. Bartholomew's Hospital Reports,' 1873, p. 161.

[2] "Note sur les Alterations du Foie Consecutive à la Ligature du Canal Cholédoque," 'Arch. de Phys.,' Mai—Juin, 1876.

[3] "Contribution à l'Étude Anatomique des differentes formes de la Cirrhose du Foie," 'Arch. de Phys.,' Sept.—Oct., 1876.

biliary canaliculi. As to the last appearance, M. Charcot[1] says:—"C'est là le phénomène fondamental dans la série des lesions de la cirrhose hypertrophique avec ictère, et suivant toute apparence, le premier en date, celui d'où dérivent tous les autres." On the other hand, the ordinary atrophic form is described as "portal cirrhosis;" the new growth surrounding many lobules (multilobular) not invading them (perilobular), while any development of even biliary canaliculi is exceptional and accidental, being ascribed to a local compression of a part of the ducts by the new growth.

From the above quotation it is plain that M. Charcot regards the appearance of an abundant network of biliary canaliculi as characteristic of biliary cirrhosis, and also as the evidence of a primary affection of the excretory apparatus of the bile. By previous observers, Cornil, Waldeyer, Klebs, and others, their presence had been noted in atrophic as well as hypertrophic cirrhosis; but M. Charcot thinks biliary cirrhosis in some cases becomes atrophic, and he admits their accidental presence under other conditions. When my attention was first drawn to this subject, I went through my preparations and cases, and in an article in the 'British and Foreign Medico-Chirurgical Review' for July, 1877, I said, "We believe that the evidence is incontrovertible that chronic obstructive jaundice, or the resulting inflammatory changes, causes a form of cirrhosis of the liver, and we admit the truth, at least, provisionally, of M. Hanot's three clinical features—(*a*) jaundice, (*b*) enlargement of the organ, and (*c*) absence of ascites—as characterising this special form. Moreover, from a careful review of a large number of specimens of cirrhosis, we admit that the growth differs from ordinary cirrhosis by extending within the lobules more frequently and to a greater extent than in the latter. But we are not inclined to admit that this new formation of canaliculi is essential or pathognomonic, but is related to certain secondary changes which may or may not be present." This opinion was founded upon old preparations, and especially on a section of syphilitic cirrhosis, in which the canaliculi were very abundant, but the evidence I have now to offer is still more conclusive, as the cases were carefully observed with reference to the points to be decided.

The first case was that of a patient who presented during life an exact conformity with the clinical description of M. Hanot (persistent jaundice, enlargement of the liver, absence of ascites). At the autopsy the liver was found to be much enlarged, smooth on the

[1] 'Leçons sur les Maladies du Foie,' p. 212.

surface, tough on section, yellow coloured; there was no alteration of the external bile apparatus. The portal canals, fissures, and spaces showed a new growth of connective tissue, with much nuclear proliferation, and some appearance of newly-formed biliary canaliculi in the fissures and spaces where the lobules were most encroached upon. The new growth invaded the lobules. No network of biliary canaliculi could be seen in the portal canals, and the drawing gives the best illustration of the condition which the organ afforded.

Here, therefore, was a case which conformed in all respects to the description of biliary cirrhosis, but in which "le phénomène fondamental" was almost wanting.

The second case presented during life the clinical characters of ordinary cirrhosis, except that the liver was enlarged. There was no jaundice; ascites was present, with chronic peritonitis and perihepatitis. The liver was enlarged, weighing seventy-six ounces, and presented the common appearance of the association of fatty infiltration with cirrhosis. The drawing (Pl. XVII, fig. 2) shows numerous canaliculi in the new growth around the lobules, the hepatic cells being very fatty. This was a case of hypertrophic cirrhosis, if you please, as the liver was enlarged, but it conforms in no other particular to M. Hanot's type; its surface was coarsely granular, while the bands of connective tissue were multilobular. The ascites may be ascribed to the chronic peritonitis present, but even then the case differs essentially from all we have been told as characteristic of biliary cirrhosis, from which not the least important distinction is the absence of jaundice.

The third case was that of a young woman, with general enlargement of the glands; her liver was enlarged, she had no jaundice, and no ascites. The cause of death was acute tuberculosis. The liver was pale on section, and studded with little yellow granulations; under the microscope these presented an appearance very similar to that already described in the first case, but the newly-formed canaliculi were even better marked.

I am inclined to regard this as an early stage of the cirrhotic liver of phthisis, of which the following is an example in a more fully developed condition.

This, my last case, was one of phthisis in a boy aged 15. There was neither jaundice nor ascites. The liver was enlarged, weighing 45 ounces (the enlargement being relative to the size of the body),

and distinctly cirrhosed. The appearances seen in sections of this liver (Pl. XVII, fig. 1) are quite as good examples of newly developed biliary canaliculi as have been figured by any of the authors quoted. In another section the direct continuity of the newly-formed ducts with the rows of hepatic cells may be traced.

This last case seems to be convincing proof that the network of newly-formed biliary canaliculi cannot be regarded as characteristic, even in its most pronounced condition, of the cirrhosis of biliary origin, described by MM. Hanot and Charcot as hypertrophic cirrhosis with jaundice; while the first case seems to indicate that very little alteration of the ducts may be present in a liver which conforms in all other respects to that type. In M. Hayem's cases, previously alluded to, no alteration of the canaliculi is referred to in the text or indicated in the plates, so that in his cases, also, which appear to me, although M. Hanot disputes this, to have been cases of biliary cirrhosis,[1] the "fundamental phenomenon" was absent.

The above communication was written early in 1878, but I did not have an opportunity of laying it before the Society till February, 1879. I am now enabled to quote in support of my views a paper by Dr. Ludwig Brieger,[2] in which he describes the multiplication of biliary canaliculi as present in cases of undoubted alcoholic cirrhosis, in the cirrhotic livers of tubercular patients, and in some forms of atrophic nutmeg liver.

February 18*th*, 1879.

[1] M. Hayem's first case dated his illness from "jaundice and dysentery," fifteen years before, and on admission "there was a yellow straw-coloured subicteric colour of the skin," though there was no bile in the urine. The second case had been "attacked by jaundice for some months" before admission, and the urine contained a little bile-pigment, which, however, passed off.

[2] "Beiträge zur Lehre von der Fibrösen Hepatitis," 'Virchow's Archiv,' Band lxxv, Heft. 1.

DESCRIPTION OF PLATE XVII.

Plate XVII illustrates Dr. Robert Saundby's Note on the Morbid Anatomy of Hypertrophic Cirrhosis of the Liver. From drawings by himself.

FIG. 1. Multiplication of biliary canaliculi in a portal canal and around the lobules, from a case of phthisical cirrhosis of the liver. Hartnack, oc. 3, obj. 4.

FIG. 2. Similar appearances, highly magnified, from a cirrhosed and fatty liver without jaundice. Hartnack, oc. 3, obj. 8.

Fig. 1.

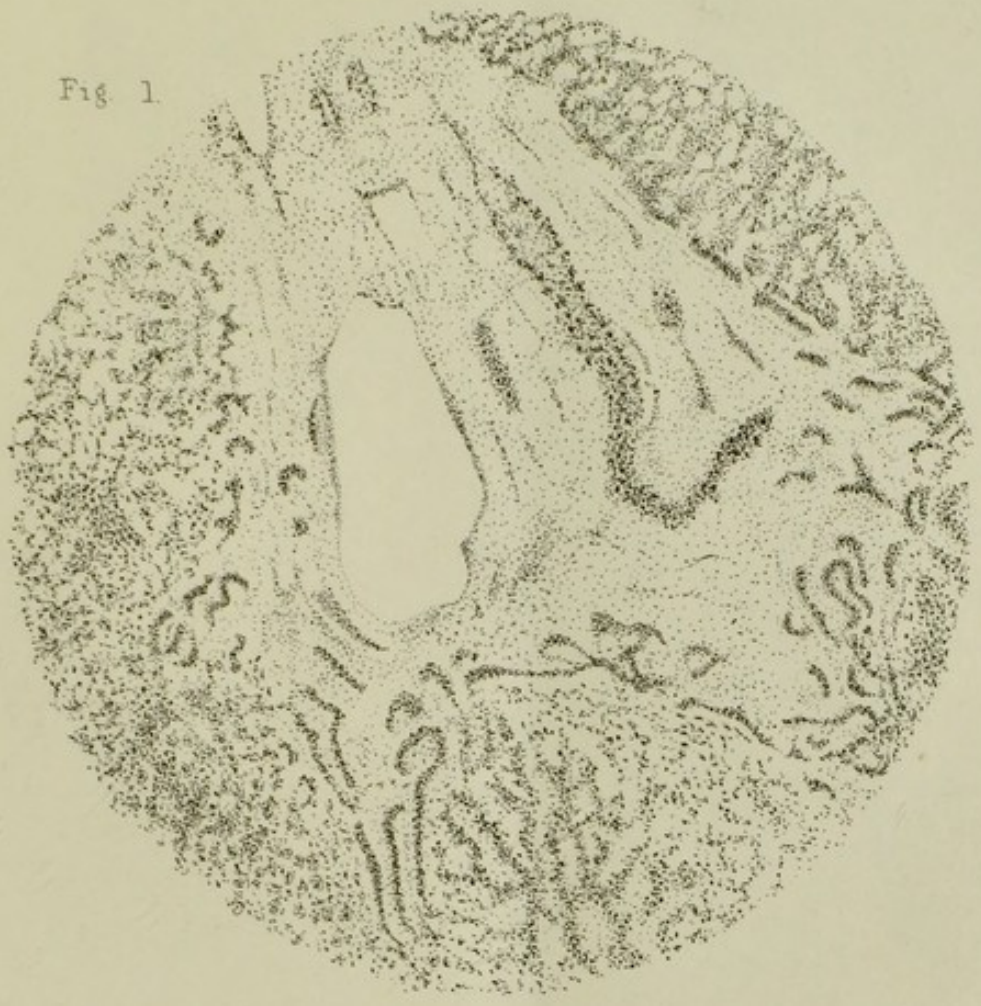

Fig. 2.