

NOTE ON A CASE OF CIRRHOSIS OF LIVER.

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[Read in the Section of Pathology, February 17, 1905.]

Mrs. C., aged thirty, was admitted to the Adelaide Hospital on the 24th August, 1904, under my care. Sixteen months previously she had been confined, and for some time before the birth of her baby her legs and hands were swollen. These swellings quite cleared up after her confinement, and she remained in good health up to June of last year, when she noticed she was getting thin and was very easily tired. No further symptoms developed until early in August, when she observed her abdomen was becoming larger. She consulted Dr. Brew, of Enniskerry, who, recognising that ascites was the cause of her abdominal enlargement, advised her to come into hospital. I may say briefly that beyond the discomfort caused by the ascites she had no symptoms. She felt well; her appetite was good and bowels regular. She had no vomiting and no history of any hæmatemesis. Her urine was sp. gr. 1030, and contained no albumen, sugar or bile. It was fairly high-coloured. Her heart was healthy. There were some distended venules on the cheek, but nothing else to give any clue as to the cause of the ascites. On account of the ascites it was impossible to make any detailed examination of the abdomen, and so I asked Mr. Gordon to remove the fluid by a laparotomy. No ovarian disease or tubercular peritonitis was discovered, but the liver was found to be cirrhotic. The removal of the fluid caused a considerable change in her symptoms. She became rather seriously ill, lost all appetite, her pulse was exceedingly small and compressible; she became jaundiced, and bile appeared in the urine. After remaining in this condition for a few days she gradually recovered. The same symptoms, only rather more intense, developed after her abdomen was a second time tapped, but this time only with a trocar and canula, and though I thought on one day she was not going to survive, she again

rallied. On the fourth occasion that the fluid was removed Mr. Gordon again performed a laparotomy, and grafted the omentum on the peritoneum, but she never really rallied from the operation, and died on the 25th November. A limited *post-mortem* was made and the liver removed. It is, as you can see, a typical example of atrophic cirrhosis.

The great interest in this case is the presence of a marked multilobular cirrhosis of the liver in a young woman, who was a life-long teetotaller. No history of syphilis was obtained, nor do I think there was the slightest reason to suspect any such taint.

On looking up the article on cirrhosis of the liver in Clifford Allbutt's System of Medicine, I find it stated "that the excessive use of alcohol is by far the most common cause of cirrhosis of the liver in all countries; by its side all the other causes together are insignificant"; and again, "in conclusion, it may be safely stated that in nearly all the patients who present definite signs of cirrhosis the disease has arisen as a consequence of alcoholic excess, syphilis, or chronic malarial infection."

DR. H. C. EARL said that the connection between alcoholic excess and cirrhosis of the liver had been assumed, and partly proved, but he thought that too much importance was placed on the alcoholic excess. In quite half the cases he had seen there was certainly none. It was hard to say whether there was syphilis or not, but at any rate it was impossible to get a history of it. He had seen cases of typical cirrhosis in very young people—for example, in a child of seven and in a child of eleven. It was quite obvious that there was something else besides alcoholic excess, because one found patients in hospital who drank all they could get, and had no cirrhosis, and it was by no means a common affection among them.

DR. MATSON asked if there was any tubercular history, or whether any tubercular foci existed in any part of the body? Also, was there

any enlargement of the spleen, and how soon after the draining did the patient die? In view of acute yellow atrophy could the pregnancy throw any light on the causation? As regards alcohol producing cirrhosis, he wondered if the kind of alcohol made any difference, as in England it was usually whisky or brandy that was drunk, and in Ireland stout.

PROFESSOR O'SULLIVAN said that his own experience coincided with that of the President. One made *post-mortem* examinations on numbers of drunkards in this city, and fatty infiltration of the liver was usually found, while cirrhosis of the liver was the exception; and he was inclined to agree with Dr. Matson that it might be due to the kind of spirit consumed. At the same time in experimental cirrhosis the disposition of the animal appeared to exercise a certain amount of influence on the result—that is, some animals of the same species would get cirrhosis and others would not, as if there was a tendency in some individuals to the formation of increased connective tissue. One ought not to forget the interesting work that was being done in Montreal by Professor Adami on the effect of organisms of low virulence acting for a long time on the organs. He remembered that there was an announcement made that cirrhosis of the liver was due to an extremely minute coccus, but this was subsequently corrected, as it was found that it was a form taken by the colon bacillus. Adami and his pupils believed that many of the chronic interstitial changes in the body are due to micro-organisms of low virulence, no matter what the specific nature is.

COLONEL MCNEECE said that during the last 15 years he had made many *post-mortem* examinations, but had never seen a case of cirrhosis of the liver. The cases which did occur at Netley were in men who had served 15 years in India. Formerly each man was served out with a dram of rum in the morning (and many men besides bought more drink), but that had been done away with for the last 12 years or so, and now they saw nothing of the hob-nailed liver in the service, so that he thought it was due to a sort of crude alcohol manufactured in India from beet-root. He had read the accounts of the investigations in Canada, and it might have something to do with the case before them—of cirrhosis occurring in a strictly temperate person.

DR. KIRKPATRICK asked if Dr. Peacocke could give them any explanation of the occurrence of ascites in these cases of cirrhosis

of the liver? The teaching they had had was that the fibrous tissue pressed on the portal veins and caused a very chronic form of ascites. Now, it seemed that many cases ran almost their whole course without any ascites appearing; also that in those cases where it did appear it was a terminal symptom. A few years ago some cases were collected together to show the results of Talma's operation, and these seemed to show that it gave bad results in cases of cirrhosis of the liver, but in cases due to perihepatitis it had given brilliant results, so that he thought the old explanation—that the ascites was due to mechanical obstruction—could no longer be supported, and he believed that it was a terminal symptom. Whether the cirrhosis started primarily as a result of change in the liver cells or as a result of an irritant reaching the connective tissue in the portal systems through the blood-vessels, it did not seem to him that that could afford a full explanation of the symptoms. A patient would go on for a considerable time without any symptoms, then, more or less suddenly, he began to lose flesh and present symptoms of loss of vitality; ascites supervened, and death resulted in a short time. If the symptoms were due to a crowding out of the liver cells by connective tissue, one would expect the process to be more gradual; and if this crowding out of the liver cells was to be a full explanation, then surely the ascites ought to be an early symptom and last for a long time; but in many cases we found that it occurred as a terminal symptom. Could Dr. Peacocke give any explanation of the mode of onset?

DR. PEACOCKE, replying, said that anyone who had experience of *post-mortem* examinations knew that cirrhosis of the liver occurred in cases without any alcoholic history, and *vice versa*. In this case he had got no tubercular history. The *post-mortem* was limited, but they saw no tubercular foci. The spleen was, perhaps, slightly enlarged. The ascites was first noticed early in August. The patient came into hospital on the 24th of August, and died on the 25th of November, and it was a day or two after the 4th tapping that she died.