

## A CASE OF ACUTE YELLOW ATROPHY OF THE LIVER.

By WILLIAM J. THOMPSON, M.D., F.R.C.P.I.,  
Physician to Jervis Street Hospital ;

AND

E. J. McWEENEY, M.A., M.D. R.U.I., F.R.C.P.I.,  
Professor of Pathology, Catholic University Medical School,  
Bacteriologist to the Local Government Board in Ireland.

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THAT acute yellow atrophy of the liver is a comparatively rare disease in this country is sufficiently demonstrated by the fact that no case has been reported at any of the Sections of the Royal Academy of Medicine in Ireland since its inception twenty-one years ago. Murchinson in his writings about this disease stated that during a period of nine years, and out of a total number of 27,000 patients admitted into the London hospitals, he had only seen one case, and Dr. Wickham Legg could only trace one case in the records of St. Bartholomew's Hospital for a similar period.

It is an interesting as well as an historical fact, according to Dr. William Hunter of Charing Cross Hospital, that it was Dublin physicians who in the beginning of the last century first in this country made observations about the disease—Cheyne and O'Brien in the year 1818, and Marsh four years later ; Morgani had previous to this described cases presenting features of the disease. The first accurate clinical account recorded was that by Bright in the year 1836. He described the changes which took place in the liver as a "diffuse inflammation affecting more the glandular portion than the connective tissue, leading to great diminution in the size of the organ, and accompanied by intense jaundice, severe

nervous symptoms, and often a hæmorrhagic tendency.' Two English observers—Busk in the year 1845, and Handfield Jones two years later—accurately described the characteristic microscopic appearance of the degenerated liver cells, and about the same time Continental investigators confirmed these observations and added more to what had already been made known.

The patient, E. B., was admitted into Jervis Street Hospital on the 24th August last. She was a well-nourished woman of medium height and build, with dark hair and complexion, aged twenty-seven years. She was born in Dublin, and had a good family and personal history. She had been married five years, had three children, the youngest of whom was four weeks old; the other two, aged respectively four and two years, were strong and healthy. She had not had any miscarriage, nor was there a history of a rash or sore throat, so that syphilis as a cause must be excluded. Her husband, a labourer by occupation, was not always steady or at regular work, which necessitated her doing washing, and in this way she contributed to the support of her family. She had not been in the habit of taking either porter or any other stimulant. She resided in a tenement house situated in one of the most congested parts of the city, where the surroundings could not be anything other than detrimental to a robust state of health. While carrying her last baby she enjoyed average health—as she did on the two former pregnancies. Parturition seemed to have been normal, and she was able to be up and about at the end of the week, and, as with the other two children, she nursed this child. During the third week after her confinement she felt sickness of stomach, loss of appetite, languor, and the jaundice first appeared at the end of three weeks after childbirth. A week later she first came under my care. At that time there was nothing in her condition to suspect anything other than an ordinary case of catarrhal jaundice. The liver dulness was not affected; no hardness or resistance of the organ could be detected, and the gall bladder was not distended; there was no pain on pressure. The tongue was thickly coated, enlarged, flabby and indented with teeth marks. The teeth were exceptionally good; the tonsils were normal, and no enlarged glands could be discovered. The

abdomen was soft and flaccid; no rigidity at or about the liver region; all the abdominal organs seemed normal, as well as the heart and lungs. There was marked constipation, and rather more sickness of stomach perhaps than one usually gets in a simple case. The skin and mucous membrane presented all the appearance of a well-marked case of jaundice, as also did the urine. The quantity of urine passed per day was below the average, 35 ounces. Urates were abundant, and there was a slight trace of albumen; no sugar, sp. gr. 1022, and the quantity of urea was less than normal. At this time leucin and tyrosin were not looked for. There was one other symptom which the patient complained of on admission—viz., a shooting, acute, spasmodic pain, lasting only a short time, apparently in the liver. She stated that the pain was deep-seated, and scarcely ever occurred in the exact place a second time, and her description of the pain was as it were the stab of a sharp instrument. External pressure had no effect in either relieving or increasing it, nor did the deep breath affect it. All through her illness this symptom was prominent, and towards the end became sometimes so severe that she had to cry out. Her temperature was normal, and pulse 62 per minute. For the first week in hospital this patient seemed to improve; took light nourishment well; the tongue became cleaner, and the jaundice was apparently not so intense. The bowels however required strong purgatives. There was no trouble with the breasts, as the milk ceased coming at once, although she had been suckling her child until her admission. During her second week in hospital the patient did not feel so well, the nausea and epigastric discomfort was very marked, the bowels became more stubborn, and the jaundice became more intense. From this until her death the patient got gradually worse, vomiting became almost incessant, the liver dulness diminished in area, and it was now found the amount of urea was diminished, and about half what it was on admission and leucin and tyrosin were present. The temperature now became irregular, varying from  $96.5^{\circ}$  to  $102^{\circ}$ , and the pulse became intermittent and fluctuated between  $80^{\circ}$  and  $120^{\circ}$ . There was no direct relationship between the temperature and pulse; when the temperature was high the pulse might be low, &c. At the end of the third week in hospital cerebral symptoms developed.

She gradually became drowsy and semi-conscious, was rest-

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less and had slight delirium ; the jaundice became very intense, and appeared as a greenish hue ; the tongue became dry and brown, and the amount of urine passed diminished to 13 ounces per day. The liver dulness practically disappeared in front and only posteriorly could it be slightly detected. She remained in this condition for three days, but gradually becoming more unconscious and weaker, and died about four and a half weeks after the jaundice first appeared. The vomiting was always gastric. At no time had she any hæmatemesis or any other hæmorrhagic manifestations. In the *post-mortem* room the body was not much wasted, considering the severity of her illness and the small quantity of nourishment she was able to make use of. On opening the abdomen there was found a small quantity of greenish-coloured fluid. The liver instead of presenting as usual was small, baggy-like and collapsed, and could only be seen by its being kept in position by the suspensory ligament. When removed it felt soft and spongy-like, with a wrinkled capsule, and weighed twenty-nine ounces. The stomach was small, and on opening it the mucous membrane was found to be pale and apparently healthy. The other abdominal organs seemed to the naked eye to be in a healthy condition. The spleen, the pancreas, and the kidneys seemed normal in size and on section. The uterus was normal, and there was a small ovarian cyst about the size of a marble. None of the abdominal glands were enlarged. The heart and lungs were healthy, and there was not any fluid in the pleural cavity or pericardium. I did not get examining the brain.

The clinical points of interest about this case are :—

- I. The rather protracted course—about  $4\frac{1}{2}$  weeks—although Hunter in his 29 collected cases gives 9, or 31 per cent., as running a course of from 3 to 8 weeks.
- II. The acute, short, spasmodic liver pain which was persistent throughout.
- III. The total absence of any hæmorrhagic symptoms.

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PROFESSOR E. J. McWEENEY then described the character of the specimen as follows : Liver weighed 29 ozs., small, very soft,

and pulpy; capsule wrinkled, pale red with yellowish mottlings. On section, mostly reddish with bright yellow patches, lobular marking obliterated. Dimensions of right lobe, 6 in. from side to side,  $5\frac{1}{2}$  in. from before backwards,  $1\frac{5}{8}$  in. thick. The left lobe was at most only half an inch thick. Gall-bladder normal. The microscopic examination was made by means of teased preparations, frozen and paraffin sections. It showed complete necrosis of the liver cells; patches of round-cell infiltration, which still preserved their nuclear staining; patchy pigmentation of the liver cells, with yellow granular matter which did not give an iron reaction (bile); moderate fatty degeneration; and the presence of minute, yellowish-brown globular concretions in groups and rows. These were very abundant in the tissue, and varied from  $12\mu$  to  $30\mu$  in diameter. Some of them seemed radially cleft, others were coated with a prickly layer of minute needle-shaped crystals, whilst others presented obscure concentric striation. There were also minute colourless acicular crystals lying singly and in sheaves. These objects were doubtless crystals of leucin and tyrosin. The globules were soluble in alkalies and weak  $\text{H}_2\text{SO}_4$ ; insoluble in ether, alcohol, and chloroform; partly soluble in water. A few bacilli were demonstrable on the sections, and of the numerous culture-tubes inoculated a few showed colonies. Study of the organisms, however, convinced him that they were merely ordinary putrefactive germs.

PROFESSOR O'SULLIVAN said he remembered one case of the kind, which belonged to Dr. Wallace Beatty. He had some sections of it which considerably resembled those shown, but his were more advanced. He was not sure that he would have taken Professor McWeeney's view of the condition of the liver cells, as he did not think the process was very far advanced. There was certainly not so much disintegration shown in the sections as in those of the case in his possession. The process seemed to be starting from the hepatic veins, the portal areas being less affected.

DR. H. C. EARL had seen portions of a liver from an undoubted case of this disease. A large portion of the right lobe was involved, and on section of this, more than half the affected portion was a brilliant red colour, and the rest an equally brilliant yellow. In the red area the liver cells had disappeared, and in the portal canals there was small round-cell infiltration. In the yellow areas were cells which looked absolutely normal. He was surprised at

the small amount of fat he found in the cells. He noticed in some parts of the yellow areas, where they bordered on the red, that the centres of the lobules were certainly more affected than the margins. The process seemed to be progressing from the centre of the lobule to the periphery.

PROFESSOR MCWEENEY, in replying, said he considered that the cells were completely necrotic, though their outlines were preserved. They did not give the ordinary nuclear stain. The protoplasm had stained diffusely with hæmatoxylin stain. The preparations showed no well-preserved liver cells. The yellow pigmentation was well marked in one of the sections. The bacteriology of these cases was very interesting, because it was very important to eliminate the theory of living bacteria. The generally accepted theory was that the disease was due to toxins. He had set going a number of aerobic and anaerobic cultures, but the material was not perfectly fresh. The anaerobic tubes remained sterile, and the aerobic showed some small white colonies which he considered unimportant. He cut some sections in paraffin, and found occasional bacilli, which he thought were ordinary putrefactive organisms. He thought the toxæmia theory still held the field.