Fatal Case of Acute Hæmorrhagic Hepatitis following Toxæmia of Pregnancy.

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MRS. F. The patient, in this case a ii-gravida, aged 34, was admitted under Mr. T. G. Stevens on August 25, at 10.30 in the evening. She had then been having pains for about two hours. The os was the size of half-a-crown, and she was in a very excitable and restless condition, with a feeble pulse of about 100, and temperature a little over normal.

She had a history of gastro-intestinal disturbance for the previous fortnight, with some vomiting and almost continuous pain in the upper part of the abdomen, together with slight diarrhœa, anorexia, and swelling of the legs. The symptoms, however, which seems to have struck her husband most forcibly during this period were the intense restlessness and mental distress, which she also manifested on admission.

She thought she had been passing less urine than usual, and complained of great thirst together with headache in the frontal region and epigastric pain.

There was considerable œdema of the lower extremities. and her complexion was very sallow, though not icteric.

The imminent onset of eclampsia was anticipated, in view of the fact that the urine (catheter specimen) gave a very heavy cloud of albumen on boiling. The patient was therefore freely purged and given a hot pack. As the pains were getting stronger her restlessness became so great that hyoscine gr. 1/100 and morphine gr. ¼ were administered subcutaneously, and at the end of the second stage, which occurred about five hours after admission, a few whiffs of chloroform were given.

On the following morning one of us (R.H.S.) was called to see the patient about 9.30, because she was jaundiced and had acute

epigastric pain. The temperature and pulse were both about 100, she was obviously suffering great pain, and she had great hyperæsthesia in the umbilical region and all down the right side; there was also severe referred pain in the *left* shoulder and hæmatemesis, these last two features being unlike a case of gallstones. The patient was markedly jaundiced and very restless. The abdomen was rigid and gave a tympanitic note to percussion. No tumour could be felt, but the liver dulness seemed reduced. A catheter drew off about two ounces of port-wine-coloured urine, which was the only specimen obtained after delivery. The vomiting, which was incessant, was of the "coffee-ground" type, with streaks of bright blood. It did not smell of phosphorus and was not luminous in the dark. The urine contained many blood-cells and much blood-pigment. It was loaded with albumen and gave a faint bile reaction. The most interesting thing about the urine was that it contained scattered leucin-cones, though no tyrosin-tufts were discovered. An urea estimation was unfortunately omitted, but it would not have been of great value as it would have been impossible to collect the urine for 24 hours owing to the fact that the patient had incontinence.

All these symptoms and signs, including the reduced area of liver dulness, were sufficient to justify a diagnosis of acute yellow atrophy, but here it is only fair to say that post mortem the liver was found in no way reduced in size, but merely compressed by the marked degree of meteorism present. Mr. T. G. Stevens, of St. Mary's, and Sir Wilmot Herringham, of St. Bartholomew's, both saw the patient, and concurred in the diagnosis.

The patient's condition became rapidly worse, although the temperature came down. The vomiting continued, though hæmatemesis ceased. There was a continuous trickle of hæmorrhage from the uterus, although it felt firm and well retracted. This metrorrhagia was uncontrollable by ergotin or hot douches. Morphia was administered on three or four occasions for the pain and restlessness. The patient complained of great hunger and thirst, but immediately vomited fluids taken by the mouth. Saline solution was given by the rectum and retained.

On the night of the 27th-28th the patient became delirious and passed fæces under her. These apparently contained altered blood. On the 28th she was, if possible, more restless, continually trying to rise and sinking back exhausted after each attempt. The temperature was rather subnormal and the jaundice had deepened.

In the evening she passed from delirium into coma, and died about forty hours after delivery.

A post mortem examination of this case was performed within

six hours of death. The body was somewhat emaciated and deeply jaundiced. There was marked abdominal distension.

The brain was somewhat œdematous, but was otherwise perfectly normal. The pericardium contained about half an ounce of straw-coloured fluid. The heart was of normal size; its valves showed no signs of disease, but the musculature was softened throughout.

The lungs were markedly congested at the bases, but were crepitant throughout. The pleural cavity was normal.

The stomach and intestines were distended throughout, the former being half full of a dark "coffee-ground" fluid.

Liver from a case of very *acute hepatic toxæmia of pregnancy*. Patient died in thirty-six hours, deeply jaundiced, after continuous vomiting of "coffee-ground" material and a very violent delirium. The extravasation of blood throughout the section suggesting an acute toxæmia rapidly destroying the vessel walls and leading to widespread extravasation, *before* cellular necrotic changes had time to take place.

The spleen was soft and slightly enlarged, but the kidneys appeared comparatively normal. Microscopically, they showed a moderate amount of cloudy swelling, but no fat. The ureters and

bladder were normal, the latter containing an ounce or so of very dark-coloured urine.

The liver weighed 56 ounces and was somewhat light in colour. Scattered over its surface hæmorrhagic areas could be seen beneath the capsule which was not thickened. These hæmorrhagic areas were found on section to be scattered broadcast throughout the organ. Microscopic section showed the liver substance broken up by multiple areas of hæmorrhage, the liver cells being entirely destroyed in places, while in other places they were not markedly changed (see plate).

The pathological findings suggest the adoption of the title acute hæmorrhagic hepatitis, for the size and colour of the liver do not correspond with the findings in cases of so-called acute yellow atrophy. To us the case suggests a very acute toxæmia, the action of which was primarily upon the walls of the liver capillaries causing rupture and a consequent flooding of the adjacent liver substance with blood.