CLINICAL NOTES

A CASE OF WEIL'S DISEASE.


On September 20th, 1936, a man, aged 43 years, living in one of the new houses on the Mount Merrion Estate, entered his back yard and saw a rat there. His neighbour had been chasing it with a dog and it had run into his yard. He went over towards it but it disappeared under a bush. He picked up a piece of wire netting and tried to dislodge it, when it ran up the netting and bit him on the left thumb. He went in, washed the wound and bandaged it up. It became sore later so he poulticed it. A few days later the thumb was much inflamed but with continued poulticing it healed completely.

On October 4th, a fortnight after he had been bitten, he developed a feverish attack with the symptoms of what is usually termed "flu". His temperature rose to 104° F., he had herpes round his mouth, severe pains in his back and legs, some headache and also nausea. The attack did not clear up as was expected: on Oct. 7th he started hiccoughing, and this persisted for the next three days with only short intermissions. By this time he looked very sick and appeared to be slightly jaundiced, his eyes were a little bloodshot, but his liver was not enlarged and the wound in his thumb had not broken down. Every ten seconds or so he was racked with a hiccup and was in very great distress.

We discussed the possibility of rat-bite fever, but did not think that this was the cause of his condition. We felt, anyway, that N.A.B. was too risky to give as he might be developing some liver trouble, and we thought that the cause of his hiccough was either incipient jaundice or renal failure. We suggested chloretone in 5-grain capsules and, curiously enough, the hiccough ceased and did not recur. Examination of a sample of urine showed neither bile salts nor bile pigments, but it was loaded with albumin. There were no red cells and very few casts.

Three days later his temperature had not settled down; he was just as ill, but no jaundice had appeared. His blood urea was estimated and found to be 60 mg. per cent. A Widal reaction was done but was negative for B. typhosus, B. paratyphosus B. and brucella abortus.

He then gradually improved, his temperature dropped to normal and he was able to get up on the 21st, a fortnight after the attack started, although he was still very weak.

He was up for only two days after the attack when his temperature again rose to 102° F. He started vomiting and dry retching, and he became just as ill as he had been previously. His urine contained some albumin, his blood urea was 56 mg. per cent., and his blood count was: red cells 4,130,000, white cells 5,000, hemoglobin 80 per cent., colour index 0.97.

A week later, on Oct. 30th, as his condition had not improved, we asked Dr. E. T. Freeman to see him, and he suggested a diagnosis of spirochetal jaundice. A catheter specimen of his urine was injected into a guinea pig with a negative result.

An agglutination test can also be done in this disease similar to a Widal reaction and Dr. Brown, of the Wellcome Bureau of Scientific Research, very kindly examined the serum and reported that it agglutinated a formalised culture of Leptospira icterohemorrhagica, human strain, in all dilutions from 1/10 to 1/30,000. This, of course, established the diagnosis of Weil's disease.

The patient steadily improved, the relapse lasting a fortnight in all, and at the end of November he was up and out. His blood urea was normal, 25 mg. per cent., his urine was free from albumin, and except for general weakness he had quite recovered, although he had lost just two stones weight.

The disease of spirochetosis icterohæmorrhagica, or better, Weil's disease, since neither icterus nor hæmorrhage is an essential part of the condition, has been recognised since 1836. Stokes and Ryle in 1916 published an account of several cases in the troops in Flanders and similar outbreaks were reported amongst the French,
German and Italian troops. Since then isolated cases and localised outbreaks have been reported throughout Europe and particularly in Holland, one factor being common to all, contact with rats or rat-infested areas. Thus the disease more commonly occurs amongst workers in sewers, abattoirs, refuse dumps and coal mines, particularly in wet pits, amongst the fish workers in Aberdeen, and in those who bathe or suffer accidental or suicidal immersion in canals.

Schüffner in 1934 reported that 452 cases had occurred in Holland in the previous ten years. In the same year Fairley reported a fatal case in a sewer worker in London. He was struck by the frequency of jaundice in these workers and investigated the history of previous attacks of jaundice in eight other workers and succeeded in proving in these cases by agglutination reactions that the condition from which they had suffered previously was indeed Weil's disease. In one case the blood was taken 12 years after the attack of jaundice. Davidson this year reported 45 cases amongst the fish workers in Aberdeen. In Japan the disease is endemic and has a high mortality of about 30 per cent. Up to 1935 only 12 cases in all had been reported in the United States of America.

Examination of rats from different parts of the United Kingdom has shown that from 5 to 35 per cent. are carriers of the leptospira and in the Netherlands it has been found that 45 per cent. of adult rats harbour the organism although only 3 per cent. of young ones are affected. In the light of these figures the reports of infection are surprisingly few.

Infection occurs through broken skin or through the mucosa of the eye, nose, mouth or upper respiratory tract. Experimental swallowing of infected water usually fails to produce the disease, presumably owing to the antiseptic action of the acid in the stomach and of the bile. However, it is of interest that the disease occurs in a greater proportion of those who fall into infected waters than of those who bathe in them.

The incubation period is ten days, and the course of the disease may be divided into three stages each lasting a week.

In the first stage organisms are found in the blood stream but not in the urine. The onset is sudden with fever, headache, pains in the back and legs, nausea, vomiting and often abdominal pain, and so is usually mistaken for influenza. Pink eye, conjunctivitis, is of considerable importance in early diagnosis, if it is present, as jaundice only appears about the 4th to 7th days. A leucocytosis of 10,000 to 20,000 is usual.

In the second stage anti-bodies are found in the blood stream and the organisms have disappeared but are excreted in the urine instead. The temperature drops by lysis but the jaundice, if present, deepens. If no jaundice has developed and this fails to appear in 40 per cent. the case is considered to be a mild one and should by this time be almost free from symptoms. Hæmorrhages, either as a purpuric rash or as large ecchymoses, may appear and, if extensive, indicate a profound toxæmia. Slight bleeding from the nose, lungs or alimentary tract occurs in more than half of the
cases. If death occurs, it is during this stage and is from renal failure. The mortality rate is about 5 per cent. in Europe.

In the third or convalescent stage the symptoms have largely disappeared, but in severe cases jaundice may persist for three weeks and convalescence may be very slow. During this week a second rise of temperature occurs in about half the cases without any increase of jaundice or other symptoms. Thus it is not a true relapse.

The final proof of the accuracy of the diagnosis must depend in every case on bacteriological or serological tests. During the first week leptospira may be found in the blood on guinea pig inoculation but disappear fairly rapidly. In the second week they are excreted in the urine but rarely give a positive result on guinea pig inoculation. Davidson obtained this in only four out of twenty-three proved cases of the disease. Thus the diagnosis depends on agglutination tests which become positive in the second week and remain so for years. With living leptospira agglutination occurs in the lower dilutions, about 1/10 to 1/1000, and lysis occurs in the higher, 1/300 to 1/30,000. This makes the reading of results difficult as it masks the agglutination. To overcome this formalised cultures of leptospira are used and give agglutinations in as great dilutions as 1/30,000.

Treatment is symptomatic and there is an anti-serum prepared which is useful, especially if given early in the disease.

One other case has been reported in this country. The patient, who had a deep cut in his foot, developed the disease after bathing in a pool at Baldonnel in a rat-infested area. Comdt. O'Connor obtained thirteen rats from this area and was able to demonstrate leptospira in three-quarters of these. He suggested that "since the rat is the established vector of this disease, spirochetal jaundice has probably occurred in Ireland in the past and may occur again." This case proves the accuracy of his forecast and shows that infected rats probably are to be found in many different parts of Ireland.

References.