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LEPTOSPIROSIS IN THE ALDERSHOT COMMAND.

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Owing to a better appreciation of the clinical symptoms and the availability of a specific serum agglutination test, cases of Weil's disease are now being recognized on a larger scale than previously in many parts of the United Kingdom as well as in other countries.

Apart from the occupational incidence, e.g. occurrence of cases among meat and fish workers, sewer men, bargees, miners and others, this disease is occasionally contracted by persons bathing in canals, rivers and streams. It is possible that only a small proportion of the population is susceptible, otherwise it might have been expected that a certain number of the comrades and co-bathers of the cases here reported would also have been affected. Alternatively, it may be that immunity can be acquired by aborted or mild infections which escape detection, as has been suggested by certain French authors [1].

During the months of July and August, 1935, three cases of leptospiral jaundice occurred among the troops and were treated in the Cambridge Hospital. The patients gave a clear history of repeated bathing in the Basingstoke Canal within a few days of the onset of the disease. All the men were expert swimmers and were partial to swimming under water and using the crawl stroke, thus allowing an easy portal of entry by the nasopharynx. They had also bathed in certain swimming pools but as these were equipped with modern purification plant they are not likely to have been sources of infection.

Early in October a fourth case returned to Aldershot from leave and
Leptospirosis in the Aldershot Command

reported sick with jaundice, the diagnosis thus being made during convalescence. The history of this case suggests that the infection was acquired by bathing in the River Trent, in Lincolnshire, in an area where rats are harboured in considerable numbers in malt kilns in close proximity to the river bank.

History of Cases.

Case 1.—Trooper M., aged 23, had been on duty as a member of a "bathing picket" for a week during the hot period July 12 to 18, at Puckridge Flash, Basingstoke Canal, and had been in and out of the water almost all day. He stated that he had no abrasion of the skin.

He was admitted to hospital on July 24, 1935. The onset was sudden with severe pain in the head and neck, sore throat, slight vomiting and much weakness of the legs. His temperature after admission, 104° F., was the highest point reached during the eight-day course of his fever. By the fourth day the headache had abated and was replaced by extreme pain and tenderness of the calf muscles, accompanied by tingling sensations in the feet. Two days later double foot-drop became evident with absence of the ankle-jerks, but all other tendon and the superficial reflexes were normal. The sensory system was unaffected. After three days the power of the dorsi-flexors had largely returned and the calf pain was less severe. Copious epistaxis occurred on the fifth, seventh and eighth days, as much as a pint being lost on one occasion. There were no skin hæmorrhages or rash but jaundice appeared on July 29, and was moderately severe. Mild nephritis, shown by albumin, blood and epithelial casts, was present from an early stage. The Van den Bergh test gave a direct delayed and indirect positive reaction. A blood-count was not made until the end of the third week and
W. E. K. Coles

was then within normal limits. The patient progressed favourably apart from a slight return of fever between the seventeenth and thirty-third days, characterized by occasional recurrences of headache, mental instability and tingling in the legs, but there was no deepening of the jaundice.

He was discharged from hospital on September 17, 1935.

Laboratory confirmation of the diagnosis of this case was obtained as follows:

On August 6, 1935, a guinea-pig was inoculated intraperitoneally with centrifugalized urine deposit. This animal died on the eleventh day after inoculation and an autopsy showed the typical lesions of leptospiral jaundice, large numbers of leptospire being demonstrable in the liver. An attempt to produce the disease in a guinea-pig by intraperitoneal inoculation of 3 cubic centimetres of citrated blood withdrawn on August 1, 1935, had proved unsuccessful.

Blood-serum from the patient, taken on August 6, 1935 (fourteenth day of illness) was sent through the Royal Army Medical College to the Wellcome Bureau and the agglutination test, performed by Major H. C. Brown, was reported as follows:

<table>
<thead>
<tr>
<th>Dilution</th>
<th>1/10</th>
<th>1/30</th>
<th>1/100</th>
<th>1/300</th>
<th>1/1,000</th>
<th>1/3,000</th>
<th>1/10,000</th>
<th>1/30,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Result</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Trace</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
</tbody>
</table>

Leptospiræ were demonstrated by dark ground illumination in the urine of August 9, 1935.

Case 2.—Boy W., aged 16, was admitted to the Cambridge Hospital on July 26, 1935. He had bathed in the canal at Puckridge Flash on each Sunday of the three weeks prior to his illness. On the last occasion, five days before admission, he had a scratch on his right foot.

The onset of symptoms was sudden on July 26, and was characterized by severe headache, a feeling of tiredness, and weakness of the legs. He arrived at hospital at midday in a state of collapse and after admission he had a rigor. His temperature was 106°F, and pulse-rate 128. He vomited copiously and was incontinent. That evening his neck became stiff, the pupils were unequal and he had a divergent squint. Ophthalmoscopic examination revealed congestion of the optic discs. Kernig's sign was positive and the knee-jerks were found to be absent. His total white cell count was 18,000 per cubic millimetre. The polymorphonuclear proportion was 88 per cent. He thus simulated cerebrospinal meningitis very closely and a lumbar puncture was performed with removal of 25 cubic centimetres of clear fluid under slightly increased pressure. An intrathecal injection of 10 cubic centimetres of anti-meningococcal serum was made. The cerebrospinal fluid, when examined, was found to be normal in all respects and culture proved negative. The next morning his temperature was lower but he was semi-comatose. Nuchal rigidity was still present but Kernig's sign was doubtful. His urine was reported to show a thick cloud of albumin.
By July 29 he had improved considerably, but he now complained of double vision and pain behind his eyes, both of which symptoms persisted for three days. An increase in the swelling of the optic discs was now noted. Another lumbar puncture was carried out on this date and 45 cubic centimetres of clear fluid under definitely increased pressure were removed. Examination of this revealed a cell content of 510 per cubic millimetre, mainly polymorphonuclear cells. Amounts of globulin and sugar were normal and a culture was sterile. The fluid was not examined for leptospira. On July 30, the fifth day of disease, he became jaundiced. A haemorrhagic tendency shown by epistaxis and a small linear haemorrhage near the disc margin of one optic fundus did not become evident until the ninth day. The urine now contained granular and epithelial casts as well as albumin and bile, but no blood. The blood-urea was found to be raised to 96 milligrams per 100 cubic centimetres. The liver and spleen were never palpable and no rash was observed. Calf muscle pain was not a feature of this case but during the secondary rise of temperature, which occurred between the eighteenth and twenty-eighth days, the patient complained of pain in the arms and legs.

At the end of the month the jaundice and urinary changes had cleared up and there was a fair recovery of muscular power, and return of the knee-jerks. He became fit to leave hospital on September 16, 1935.

Guinea-pig inoculation by the intraperitoneal method using 2 cubic
centimetres of the patient's citrated blood taken on the eighth day proved negative as did a similar inoculation with urine deposit on the fifteenth day. Leptospira, however, were seen in the urine by dark ground illumination from the fifteenth day onwards.

Blood-serum taken from the patient on the sixteenth day for the agglutination test was reported on as follows:

<table>
<thead>
<tr>
<th>Dilution</th>
<th>Reaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/10</td>
<td>Trace</td>
</tr>
<tr>
<td>1/30</td>
<td>+</td>
</tr>
<tr>
<td>1/100</td>
<td>+</td>
</tr>
<tr>
<td>1/500</td>
<td>+ Trace</td>
</tr>
<tr>
<td>1/1,000</td>
<td>-</td>
</tr>
<tr>
<td>1/2,000</td>
<td>-</td>
</tr>
<tr>
<td>1/10,000</td>
<td>-</td>
</tr>
<tr>
<td>1/30,000</td>
<td>-</td>
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</table>

Major Brown commented on the "zone reaction" and wrote that it was similar to the results obtained in Paris from cases of leptospirosis with meningitic symptoms.

Case 3.—L/Cpl. W., aged 30, was stationed at Pirbright Camp where he bathed in the Basingstoke Canal every evening from the end of June until three days before he was taken ill on August 19, 1935. He was in training for Army swimming events and frequently swam beneath the water surface. He had no open cut or abrasion during this period. The usual depth of water was, he thinks, about five feet and much mud was often stirred up by parties of troops bathing. His first symptoms were headache and weakness of the legs. He was detained the following day and his evening temperature was found to be 102·4°F. He was admitted to hospital on the third day of disease, but did not appear very ill. He complained of pains in the back and in the legs, but it was not until the next day that
there developed the severe calf muscle pain which was the outstanding symptom of the case. The ankle-jerks were found to be absent, and this, apart from slight congestion of the vessels of the optic discs, was the only indication of neurological involvement. Albumin in the urine and granular and epithelial casts indicated a mild nephritis which cleared up entirely in a month. A blood-count on August 24 revealed white cells 16,600 with 84 per cent polymorphs. He vomited on a few occasions only. Jaundice did not appear until the sixth day and epistaxes, which were slight, occurred on the sixth, eighth and ninth days. The Van den Bergh test gave a positive direct delayed result. On the ninth day a rash appeared on the trunk; it consisted of small vesicles and pustules surmounting a red hæmorrhagic-looking base; it had disappeared by the end of a week. Scrutiny of the temperature chart reveals an actively febrile period of thirteen days following the onset of disease. All symptoms had cleared up by September 4, and thereafter convalescence was uneventful. The patient left hospital on October 5. Guinea-pig inoculation with the citrated blood of this patient as well as direct microscopic examination of the blood within the first few days proved negative. The centrifugalized urine on the eighth and ninth days appeared clear of leptospire, but on the twentieth day a few immobile leptospire were found. Blood-serum, taken on September 4 and tested at the Wellcome Bureau, was reported on as giving an adhesion test positive in a dilution of 1:3,000.

Case 4.—Pte. N., aged 25. The history in this case is that when on leave at his home in Gainsborough he was suddenly taken ill at the end of August. I am indebted to his private doctor for the early clinical notes.

Pte. N. appears to have bathed frequently throughout the month of August in the River Trent and to have made spectacular dives from a bridge stated to be thirty feet high, a fact confirmed by the doctor who was wont to view the proceedings from his surgery window and admire the athletic prowess of the man who was subsequently to become his patient. It is evident that the illness was sudden in onset and characterized by high fever, intense headache and very troublesome vomiting. The patient also complained of dimness of vision, giddiness, and much insomnia. Pains in the legs ensued and were excruciating, requiring frequent doses of morphia. Jaundice did not occur until about the sixth day and led to intense staining of the skin and mucous membranes. It was preceded the day before by a rash over the abdominal area characterized by light brown discrete spots having the size and shape of the flat surface of a split pea. The fever which was of about twelve days' duration is stated to have fallen by lysis. Before the jaundice became obvious this case quite naturally presented itself to his doctor successively as gastric influenza, toxæmia due to food poisoning, and finally atypical meningitis. As already stated the patient eventually became fit to travel to Aldershot on October 2 and was admitted to hospital as a case of jaundice with anæmia. He looked sallow and ill and the skin and conjunctivæ were still icteric. A well-marked secondary anæmia was
present, the haemoglobin reading being 50 per cent. The white cell count showed a slight leucocytosis. No albumin or bile could be detected in the urine, but granular and epithelial casts were present. The Van den Bergh test showed a delayed direct reaction. Apart from rises of temperature to 99°F. during the week after admission he remained afebrile and very soon his weight showed a progressive increase and his complement of red blood reached the normal. He was discharged cured on November 13. The urine was examined for leptospira with negative results. The blood-serum, taken on October 5, was sent to the Wellcome Bureau for the agglutination test with leptospira with the following result:—

<table>
<thead>
<tr>
<th>Concentration</th>
<th>Result</th>
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<tbody>
<tr>
<td>1/10</td>
<td>+</td>
</tr>
<tr>
<td>1/30</td>
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<tr>
<td>1/100</td>
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<tr>
<td>1/10,000</td>
<td>Trace</td>
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<tr>
<td>1/30,000</td>
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</table>

**DISCUSSION.**

No precise data relative to the incubation period of the disease is afforded by these four cases but it would appear to lie within the seven to thirteen days mentioned as the average period by Schüffner [2] and agreed to by later authors.

The key to the early diagnosis of leptospirosis lies first in bearing it in mind when examining patients with symptoms suggestive of influenza, especially if the degree of prostration is greater than is usual and the symptoms of headache and vomiting are more pronounced than would be expected. An immediate blood-count and full urine examination are of the greatest help as there is a definite polymorphonuclear leucocytosis and early evidence of nephritis. Congestion of the vessels of the optic discs is also a useful confirmatory sign. It was present in some degree in each of the three cases seen in the early stage. There was no marked conjunctival flushing. The blood-urea may be raised but there is no oedema or hypertension. The nervous system may be severely affected and a meningeal syndrome present, leading to a provisional diagnosis of cerebrospinal meningitis, but the fluid obtained by lumbar puncture is not suggestive of that condition. The characteristic muscle tenderness, in this series of cases, occurred about the third day and jaundice was not obvious, on the average, until the sixth day. The duration of primary fever averaged by the four cases was ten days, and a return of the fever in the third and fourth weeks was noticeable. A haemorrhagic tendency shown by epistaxes and skin rashes did not show itself typically until after the onset of jaundice. No enlargement of the liver, spleen or glands was clinically evident.

Much of the symptomatology of leptospiral and epidemic catarrhal jaundice is the same [3]. The main distinctions in a doubtful case are the relative or absolute lymphocytosis of the catarrhal jaundice and the negative serum agglutination reaction. This test as well as the adhesion test devised by Major Brown [4] appears to be specific and may be expected to be positive from as early as the sixth day of disease [4, 5].
Leptospirosis in the Aldershot Command

An interesting sequel of the disease is a thinning of the hair. Three out of the four patients complained of this. The loss is not necessarily confined to the hair of the scalp, as in one case hair could be plucked from the legs with the greatest of ease, and the bed linen was strewn with it.

Very little is known concerning the degree of infectivity of the human and so the patients were allowed to proceed on leave when convalescence was established. The average period elapsing from the onset of symptoms to discharge from hospital was fifty-eight days. When seen again a few weeks later they stated they had experienced only a moderate degree of lassitude and proneness to fatigue.

It has been reported that the total mortality among 452 cases in Holland, within a ten-year period, was 10.2 per cent [2], and that in 60 per cent of cases no jaundice was apparent [2]. Absence of jaundice has also been noted by Fletcher [6] in Malaya, Vervoort [7] in Sumatra and Taylor [8] in the Andaman Islands. For comparable results to be obtained in this country it is obvious that considerable familiarity with the signs and symptoms of the disease would be necessary and the serum agglutination reaction would assume a rôle of the utmost importance.

Occasional fatal cases in the British Isles have been reported in the last eighteen months and their post-mortem records reveal widespread areas of hemorrhage and degeneration throughout the internal organs.

Treatment on expectant lines proved sufficient for the cases under review as their condition, though grave at the outset, did not appear to threaten life. It should be remembered, however, that there is a real danger of hepatic and renal failure which would need to be contended with by glucose therapy, either by the oral route, or, in the case of much vomiting, by the veins. Dehydration might be an indication for an abundant fluid intake. In addition to these general measures special treatment by an anti-serum, obtained by horse inoculation, is now available from which the best results are obtained if it can be used within the first three or four days. In view of the lengthy persistence of residual agglutinins [9] and protective antibodies [10] in the blood of recovered cases it is possible that their serum could, in the same way, be successfully utilized. This method might be of particular value in Service stations where there has been a previous outbreak of leptospiral infection. As a sideline further useful research might well be directed to the estimation of the agglutinin content of the blood of habitual canal bathers.

I have to express thanks to Colonel T. C. C. Leslie, O.B.E., Commanding the Cambridge Hospital, for permission to forward these notes, to Lieutenant-Colonel C. J. Coppinger, O.B.E., for the laboratory work entailed, and to Major H. C. Brown of the Wellcome Bureau for so kindly carrying out the serological tests. Also to Dr. A. M. Pyle, civilian practitioner, who kindly sent me a description of the early clinical signs of Case 4.
BIBLIOGRAPHY.


