LEPTOSPIROSIS IN CAPE TOWN

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This case is presented because reports of leptospirosis diagnosed in life are very rare in South Africa.[1-3]

CASE REPORT

A 53-year-old Coloured male was admitted to Somerset Hospital on 29 September 1965. His occupation until 1950 (not since) was as a labourer in the docks, cleaning the boilers in the tug-boats. In 1950, he developed bilateral pulmonary tuberculosis and was in a tuberculosis hospital for 1 year, and received antituberculous therapy for a period of 1 year and 9 months. Six months after leaving hospital he developed a sudden right hemiparesis. Until this present illness, he was walking about at home and in the immediate vicinity. He is of poor social background, with a history of excess alcohol consumption.

Present Illness

This commenced suddenly on 20 September 1965. He became anorexic and a companion noticed that he had a yellow discoloration of his eyes for the past week. He did not observe any dark urine or change in the colour of his stools; however, he is a very poor witness. No therapy was administered either before or during his present illness. There was no history of headaches.

Examination on Admission

He was drowsy, deeply jaundiced, apyrexial, and had generalized purpura.

There were no other abnormalities apart from pallor, clubbing of the fingers, long-standing right spastic hemiparesis, and diminished movement of the right side of the chest with crepitations. There was no hepatomegaly.

Special Investigations

The urine contained bilirubin but at no stage was proteinuria present. Hb. 9 G/100 ml.; WBC 20,000/cu.mm.; neutrophils 76%, lymphocytes 16%; ESR 90 mm. in the first hour (Westergren). Blood smear—red blood cells were hypochromic, white blood cells were normal. Platelet count 144,000/cu.mm. Prothrombin index 95%.

Serum bilirubin: total 24·0 mg./100 ml., conjugated bilirubin 17·8 mg./100 ml. Serum albumin 3·4 G/100 ml., serum globulin 4·1 G/100 ml. Alkaline phosphatase 7 units. Thymol turbidity 24·0 units. Zinc turbidity 12 units. Serum amylase 112 units. SGOT 79 units. Blood urea 106 mg./100 ml.

Serum electrolytes: serum sodium 125 mEq./l., serum potassium 3 mEq./l., serum chloride 98 mEq./l., serum bicarbonate 30·3 mEq./l.

X-ray of the chest showed diffuse calcific foci throughout the lung fields with fibrosis, especially at the apices and a large bullae at the right apex.

There were no acid-fast bacilli present in the sputum and the blood Wassermann reaction was negative.

In view of the association of jaundice, purpura, and polymorphonuclear leucocytosis, leptospirosis was suspected on admission. On 30 September 1965, leptospiral agglutination titre was 1/10,000 for *Leptospira icterohaemorrhagica*. Urine microscopy and guinea-pig inoculation were negative.

Management

Initially, he was managed with the regime for liver failure, namely oral Neomycin and no proteins in the diet. The following day, he was mentally much better and the purpura was fading. Blood urea fell to 23 mg./100 ml. on 4 October and remained normal thereafter. Neomycin was discontinued and he was given a normal diet.

The blood agglutination titre for leptospirosis was repeated on 19 October and had risen to a titre of 1:200,000. Urine microscopy and guinea-pig inoculation were negative.

On 26 October the CSF agglutination titre was 1:300.

The patient continued to improve clinically and the serum bilirubin decreased, with slight fluctuations (Table I).

TABLE I

Date	*Total bilirubin (mg./100 ml.)*	*Conjugated bilirubin (mg./100 ml.)*
30/ 9/65	24	17·8
4/10/65	8·6	7·4
9/10/65	5·9	4·4
19/10/65	1·5	1·2
26/10/65	1·4	0·9
1/11/65	3·5	3·1
8/11/65	0·5	
11/11/65	2·0	1·9
19/11/65	0·5	

An intercurrent pneumococcal bronchitis which developed on 7 October 1965, responded to cloxacillin.

The anaemia was investigated. Serum iron 31·5 μg./100 ml.; total iron-binding capacity 314·5 μg./100 ml.; percentage saturation 10%. Serum folate 3·3 mμg. by *L. casei* assay; serum B_{12} 2,024 μμg./ml. by Englena assay; occult blood negative.

Barium meal and barium enema were normal. The xylose excretion test was low, 2 G in a 5-hour specimen of urine. The cause of the anaemia was probably nutritional, either owing to deficient intake and/or malabsorption. It was corrected with intramuscular iron and oral folic acid. The patient was discharged well on 19 November 1965.

The public health authorities carried out investigations in the vicinity of his home, but no rats were found.

DISCUSSION

This is one of the few cases of leptospirosis diagnosed during life, in South Africa. It is of note that the patient had no headache, and no conjunctivitis. The fluctuations in the bilirubin have been noted in other cases in the literature.[4] The exact significance of this is not clear. The patient made a fairly rapid, spontaneous recovery without any specific therapy.

CONCLUSION

A case of *Leptospirosis icterohaemorrhagica* in Cape Town is described. Although the organism was not isolated, the titre of 1/10,000 rising to 1/200,000 is diagnostic. The patient recovered without any systemic antibiotic therapy.

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