It is of interest that in the present case Nelson's treponemal immobilization test was negative, as this test is claimed to differentiate between true and biological false-positive Wassermann reactions. Nelson *et al.* (1950) state that the treponemal immobilizing antibody does not occur in the serum of normal persons or of those suffering from non-treponemal diseases. They have shown that of 57 lepers seropositive by standard tests (76% of those tested), 47 were negative with the treponemal immobilizing test. The incidence of positive reactions with the latter test was approximately the expected incidence of syphilis in the social and socio-economic groups from which the lepers were drawn.

It is feasible that our patient contracted leprosy as a result of prolonged contact with coloured troops.

Summary

A case of lepromatous leprosy exhibiting a chronic biologically false-positive Wassermann reaction is presented. The importance of considering the possibility of a biological false-positive Wassermann reaction in the absence of convincing clinical evidence of syphilis is stressed. Steps must be taken to determine the cause of the false-positive reaction. Histologically the lesions somewhat resembled sarcoid.

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Medical Memorandum

Two Cases of Polyarteritis Nodosa with Uraemia

Polyarteritis nodosa is now being diagnosed during life with increasing frequency (Klein, 1949). The following cases manifested general symptoms, particularly kidney involvement with uraemia. The first patient died in a uraemic state, whilst the second apparently recovered.

CASE 1

A house-painter aged 62, the father of three healthy children, was admitted to the surgical department on March 29, with complaints of severe pain and superficial ulcerations in the lower extremities of two months' duration. The family history was irrelevant. He had suffered from bronchial asthma for the past 17 years, and had been using ephedrine to relieve his attacks. Physical examination revealed limited chest excursions with musical rales in both lungs; the heart sounds were muffled, but no murmurs were heard. The blood pressure was 120/70 mm. Hg, and the pulse rate 74. The lower extremities were warm, with small petechiae and small areas of superficial necrosis over the toes and the medial and lateral areas of both legs. The right dorsalis pedis artery was not palpable. The E.S.R. and full blood count were normal, the blood urea was 42 mg. per 100 ml., W.R. and Kahn were negative; a chest radiograph showed increased lung and hilar markings, and the aorta enlarged and elongated.

A tentative diagnosis of thromboangiitis obliterans was made, and periarterial sympathectomy was performed on both femoral arteries (Leriche). At operation the arteries were found to be constricted. The patient made an un-

eventful recovery, the ulceration of both legs improving and after leaving hospital healing completely and never reappearing. He continued to have occasional asthmatic attacks. The pain in the legs recurred three months after operation, but was less severe, and appeared on exposure to cold; he continued thus for about a year and a half, until readmission. Four weeks before this he began to suffer from very severe frontal and occipital headache, accompanied by dizziness, but without vomiting. The headache was severer at night than during the day; analgesics gave relief for a few hours only. He noticed that his legs had become swollen, and two weeks later his face also became swollen, especially around the eyes. On several occasions his urine was red. There was no lumbar pain and no frequency or dysuria. His urine contained albumin, many red cells, and a few white cells and casts. The blood urea was 56 mg. per 100 ml. He looked pale, with slight oedema of the face, especially around the orbits, and of the legs. Physical examination was normal throughout, but there was tenderness on fist percussion over the loins. His blood pressure was 200/120. *Fundi*: Disk boundaries blurred, blood vessels enlarged, arterial reflexes increased, many haemorrhages and cotton-wool exudates.

A few days after admission the patient developed a severe generalized epileptiform seizure, with loss of consciousness and white foam at the mouth. His blood pressure was 250/150. He was given aminophylline intravenously, and the attack subsided. On the sixth day after admission his temperature rose to 102° F. (38.9° C.). He complained of slight pain in the pharynx, but examination revealed no apparent reason for the fever. The white cells numbered 10,800, a few days later increasing to 19,200 per c.mm. The differential count showed a shift to the left, the eosinophils remaining between 2 and 3%. The urine showed no change. Sulphadiazine and penicillin were given with no effect on the temperature. The headaches increased. The blood urea rose to 77-98 mg. per 100 ml. Plasma euglobulin was increased. Cold fraction strongly positive—irreversible at room temperature. On the fourteenth day after admission he developed a slight cough. On physical examination there was slight dullness at the base of the right lung, with many crepitations. An x-ray film of the lungs showed density of the right base. Eye-ground findings showed improvement. The haemorrhages and exudates were absorbed. On the twentieth day the patient complained of abdominal pain; the abdomen was soft and there was no localized tenderness. The white cell count was 20,000 per c.mm., and the urine showed no change.

The protean nature of the complaints—headache, fever, cough, abdominal pain, pain in the extremities, the fluctuating nature of the eye-ground changes, as well as the anaemia, leucocytosis, the increased E.S.R., the blood-protein changes, and the renal insufficiency—suggested polyarteritis nodosa.

During the following week the patient's condition deteriorated. The urinary findings became more pronounced, especially the haematuria. The blood urea increased in a few days from 98 to 262 mg. per 100 ml., and he died on the twenty-sixth day after admission, in a uraemic state.

Typical changes of polyarteritis nodosa in the kidneys, myocardium, liver, spleen, pancreas, and mediastinal fat tissue were seen at necropsy.

CASE 2

A married woman aged 25, with two healthy children, emigrated to Israel from Yemen in 1948. Three years before admission, when she arrived from Yemen at an immigrant transit camp in Aden, she had suffered for a few months from a febrile disease of unknown nature, and apparently recovered. Eighteen months later, a few weeks after arriving in Israel, she started to run a mild fever, fluctuating during the day, accompanied by severe headaches, pain in the neck, shoulders, and abdomen. She vomited three or four times daily. The attending physician suspected pregnancy, since menstruation stopped with the onset of the illness. This condition lasted for a fortnight, after which she felt better, and during the following month

she was able to work in the fields. Every now and then she felt feverish and weak, and suffered from headache and pain in the shoulders and upper abdomen. Albuminuria was found and she was sent to a hospital in town, where she stayed seven months, during which time she had recurrent periods of chilliness, severe headache, abdominal pain, dizziness, nausea, and vomiting. At that time no abnormal findings were detected on physical examination except for an enlarged and tender liver.

During her seven months' stay in the hospital the urine albumin fluctuated between 2 and 4.5%, and the blood urea between 18 and 180 mg. per 100 ml. The sedimentation rate fluctuated, but was always increased. Blood cultures were repeatedly negative, as well as blood examinations for typhoid and brucellosis. Sputum examination for tubercle bacilli was negative. Radiographs of the lungs, spinal column, gastro-intestinal tract, kidneys, and urinary tract did not show any abnormalities. Sternal puncture did not show a deviation from normal. The patient was given penicillin, streptomycin, and aureomycin with no effect on the course of the fever. The general condition continued to deteriorate, the anaemia became severe, and the differential count at one time showed 7% eosinophils. Finally, she was given three blood transfusions, which apparently had a good effect: her temperature became normal and she left the hospital slightly improved and without fever. Erythrocytes numbered 3,000,000 per c.mm.; urine showed albumin ++; blood urea, 40 mg. per 100 ml.

She returned home and felt well for two days, after which she had a new relapse of fever, severe headache, shoulder pain, chills, ringing in the ears, abdominal pain, and vomiting. In addition for the first time she had blurred vision, and also swelling of different parts of the body with itching which lasted for a few hours. A few days later she was readmitted. She was very weak and the state of nutrition was very poor; her skin was pale grey. Physical examination was normal throughout, with blood pressure 130/80, no oedema, and the urine containing albumin 4%; and many red and white cells but no casts. *Blood*: R.B.C. 3,500,000 per c.mm.; Hb 10.5 g. %; W.B.C., 9,700 per c.mm., differential count normal except for 7% eosinophils; reticulocytes 0.9%; thrombocytes, 150,000; bleeding-time, 4 minutes; clotting-time, 3 minutes; sugar, 72 mg. per 100 ml.; urea 52 mg. per 100 ml.; cholesterol, 256 mg. per 100 ml.; Weltmann, 10; total proteins, 7.4 g. (albumin 4 g., globulin 3.4 g.); euglobulin, increased; cold fraction positive, reversible. *Wassermann*, negative. *Kidney function*: standard urea clearance, 68%. *Sputum*: negative for tubercle bacilli. *Stools*: negative for ova (including schistosoma) and amoebae. *Rectoscopy*: Atrophied mucosa, no ulcers or granulation tissue seen. *Fundi*: Arterial constriction, increased reflexes of the arteries, haemorrhages in all layers of the retina, deep spotted and superficial flame-shaped, some of them being in a stage of reabsorption; multiple cotton-wool exudates. X-ray film of lungs and kidneys showed nothing abnormal. X-ray film of heart: slight left ventricular hypertrophy. E.C.G. normal.

The patient stayed for six weeks in the ward afebrile, while the urine albumin fluctuated between 1.5 and 4%, and the blood urea between 40 and 100 mg. per 100 ml. The eosinophils in the blood increased to 18%; blood cultures were negative several times. Repeated examinations of the fundi showed reabsorption of the haemorrhages and exudates.

A biopsy of the right pectoral muscle was made which, however, was negative for polyarteritis nodosa. Upon further inquiry the pathologist, who had received a biopsy specimen taken before the patient's admission to our hospital, informed us that the specimen showed the typical histological picture of acute periarteritis nodosa—fibrinoid degeneration of the intima and inflammatory changes in and around the arterial wall (see illustration). Decisive proof of the diagnosis was thus obtained.

The patient was in hospital for six weeks, her general condition gradually improving. She gained in weight; the

albuminuria dropped to 0.5%. Blood urea dropped to 39 mg. per 100 ml.; the fundi showed great improvement; and she was discharged in an improved condition. Three and twelve months later she was found to be in good health. The blood count and blood pressure were normal, and the urine contained only traces of albumin. She gained in weight, looked well nourished, had no pain, and was able to perform her household duties and help her husband in the fields.

We were dealing with a disease running for over 10 months with febrile and afebrile periods, severe headache, abdominal and joint pains, normocytic anaemia, slight leukocytosis, and eosinophilia, hyperglobulinaemia with severe and constant involvement of the kidneys, marked rise of blood urea, massive albuminuria, haematuria, severe and

Polyarteritis nodosa. Section through artery of subcutaneous tissue.

rapidly changing eye-ground fundi findings with no hypertension. The fluctuating nature of the kidney manifestations, the febrile episodes, the abdominal pain, as well as the marked leukocytosis with eosinophilia, indicated the presence of a systemic disease leading to severe involvement of the kidney. The absence of skin eruptions and of pathological findings in the heart, and the presence of leukocytosis and eosinophilia, made the diagnosis of Libman-Sachs disease improbable. Subacute bacterial endocarditis was ruled out because of the absence of cardiac involvement, splenomegaly, clubbing of the fingers, and petechiae. The diagnosis of polyarteritis nodosa fitted the clinical picture best.

COMMENT

Prognosis of polyarteritis nodosa has had a better outlook during the past few years. Since the clinical manifestations of the disease are now better known, cases are diagnosed that previously would not have been recognized until necropsy. Our second case, the diagnosis of which was proved by biopsy, manifested clinically severe kidney involvement up to renal insufficiency. This patient made a remarkable recovery, and is now, at least 30 months after the onset of the disease, apparently in good health. The clinical course of this case indicates that prognosis, even when major organs, such as the kidney, are severely involved, need not be fatal.

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