Medical Memorandum

Polyarteritis Nodosa Presenting as Acute Diabetes Mellitus

In view of the manner in which the following case presented, it is felt that it may be of value as yet another form in which polyarteritis nodosa may be encountered.

CASE REPORT

The patient, a South Indian pipe-fitter aged 30, was admitted to hospital on December 17, 1951, having had fever and headache for three days. Three months previously he had been an in-patient for a disease that resembled enteric fever, and his serum at that time gave a Widal reaction of 1 in 250 to *Bact. typhosum* H. He had a continuous high temperature for 20 days, but with relative bradycardia and a palpable spleen. He was discharged after five weeks. The only medicine received was aspirin in small amounts.

On examination his temperature was 102° F. (38.9° C.), pulse 110, respirations 28; no physical signs were found. Total white cells were 10,000 (74% polymorphs, 16% lymphocytes, 8% monocytes, 1% eosinophils, 1% basophils). No malaria parasites were seen. The Widal reaction was negative. His temperature fell to normal at the end of 72 hours, and he was symptomless; he had been given aspirin, 5 gr. (0.32 g.) thrice daily, and Dover's powder, 15 gr. (1 g.) at night, for three days.

Early on the sixth morning he complained of severe pain in the left abdomen, lateral to the umbilicus; there was deep tenderness in this area and the abdomen was soft. He was given $\frac{1}{4}$ gr. (16 mg.) of morphine and 2 ml. of nikethamide intramuscularly. Later in the morning the tip of the spleen could be felt for the first time. At 3 p.m. he had a "fit," observed by the nurse, which consisted of twitching of the face, both eyes, hands, and legs. After this he was very quiet for 10 minutes, but then became noisy and started throwing himself about the bed for 30 minutes; this was followed by a similar fit to the first. When seen by the doctor the patient was in coma, but the corneal reflex was present; there were no localizing signs, but both pupils were widely dilated. No reflex changes were found, but there were gentle squirming movements of both arms. His blood pressure was 120/80.

A catheter specimen of urine showed a heavy cloud of albumin, sugar +, and acetone +. Many pus cells, a few red blood cells, and granular casts were present in the deposit. The blood sugar was 265 mg. per 100 ml. Cerebrospinal fluid examination showed a yellowish fluid, under normal pressure; proteins 0.04 g.%; no globulin; chlorides 656 mg. per 100 ml.; a few red blood cells; and 9 lymphocytes per c.mm. The flow increased on jugular pressure.

The following measures were taken on the assumption that this was a diabetic coma: normal saline intravenously, gastric lavage with normal sodium bicarbonate solution, 50 units of insulin subcutaneously, and 2 gr. (0.13 g.) of phenobarbitone sodium intramuscularly.

He showed great restlessness, but this was controlled by 4 ml. of paraldehyde intramuscularly and 1/100 gr. of hyoscine. The blood sugar on the following morning (seventh day) was 50 mg. per 100 ml., and shortly after he vomited dark-brown material suggesting altered blood. He was still in coma. His blood pressure was 130/90. Urine was collected from an indwelling catheter, and penicillin, 1,000,000 units four-hourly, was ordered. Urine output was only fair after substitution of 5% glucose in half normal saline; the acidosis was controlled with small amounts of insulin, the carbon-dioxide-combining power rising from 29 to 60 vols.% over the next 48 hours, sugar and acetone disappearing from

the urine. In addition the patient was given 8 g. of potassium chloride as a rectal drip of 2% potassium chloride over the next 24 hours. The chest remained free of adventitious sounds, though the temperature rose to 100° F. (37.8° C.).

On the ninth day the blood pressure rose to 170/115, with a pulse of 116. Intravenous therapy was stopped and 2,000 ml. of 5% glucose in normal saline was given per rectum. There was no relief from the deep coma; the urine continued to show albumin +, W.B.C. ++, R.B.C. +, but no casts, and it was acid. Ophthalmoscopic examination revealed early papilloedema but no disease of vessels and no exudates or haemorrhage. Plasma cholesterol was 70 mg. per 100 ml., and the Widal reaction was again negative. On this day it was noted that arterial pulsation in both femoral arteries ceased just below the groins and there were bilateral colour changes in the legs below the knees. The temperature of both limbs was reduced.

On the tenth day the blood pressure rose to 200/120 and the condition of both legs was the same. There was no change in his general condition; he continued to secrete urine on a total rectal intake of 1,400 ml. He died quietly on the fifteenth day. The temperature rose terminally to 102° F. (38.9° C.).

Post-mortem Examination.—An emaciated male with bilateral dry gangrene of legs below the knees. The following points were of interest: (1) 2 oz. (28 ml.) of straw-coloured fluid in the pericardium; a small discoloured area in the posterior myocardium of the left ventricle suggesting a healed infarction. (2) Pleura and lungs appeared normal. (3) Liver showed an area of yellowish discoloration in the left lobe. (4) Greater omentum showed a small area of greenish discoloration in its free edge. (5) Pancreas apparently normal. (6) Spleen showed a massive infarct. (7) Both kidneys showed massive infarcts progressed to necrosis. Adrenals apparently normal. (8) The brain showed no evidence of meningitis, abscess, haemorrhage, thrombosis, or emboli. The pituitary fossa and pituitary gland were apparently normal.

Histological examination showed an acute arteritis in both the larger and the smaller vessels, well seen in the femoral arteries and the arteries of the spleen, liver, brain, and kidney. There was a panarteritis with degenerative changes in the intima and media of both arterioles and larger vessels, with considerable perivascular inflammatory cell reaction and penetration of all layers. In the afferent arterioles of the kidney, fibrinoid degeneration was seen with arteriolonecrosis. The aneurysmal formation in the larger vessels was of interest in that it is seldom seen in the modern form of polyarteritis (Robb-Smith, 1952, personal communication).

COMMENT

In this case the only known possible antigens were the *Bact. typhosum* and aspirin, but the former rests only on the evidence of a positive Widal reaction, which may have been an anamnestic reaction in the absence of positive blood, stool, or urine cultures.

During the patient's last illness the diagnosis of polyarteritis nodosa was not considered until the appearance of bilateral arterial block in the legs. Then in retrospect the onset of acute abdominal pain followed by convulsions and cerebral vascular accident suggested the diagnosis. The absence of an eosinophilia was not thought to be of significance.

It is believed that the acute diabetes was due to an associated acute haemorrhagic pancreatitis.

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