was then so far advanced as to make surgery extremely difficult.

The hypertension found in the upper half of the stomach and lower oesophagus was probably due to (1) the increased inflow from the large spleen and its enlarged tortuous artery; (2) obstruction of the left gastric, splenic, and left gastroepiploic veins by fibrosis; and (3) high pressure in the normally low-pressure azygos system, since these veins were acting as collaterals for the venous return from the legs. No previous report of this complication could be found on reviewing the literature.

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Multiple Sclerosis in the African

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There is some doubt about whether multiple sclerosis occurs in indigenous Africans (Gelfand, 1957; Trowell, 1960; Haddock, 1965; Dean, 1967). During the period 1952-69 we have encountered only two cases, and these we report here.

CASE 1

The patient, a man aged 30, was admitted to the Kenyatta National Hospital on 9 July 1968 with a five-month history of difficulty in walking and weakness of the lower limbs. There were no constitutional symptoms. The onset of the disease was fairly rapid, occurring over a period of five days, starting with tremors in the left leg, unsteadiness of gait, and weakness in both legs, though he was still able to walk. His legs became progressively weaker and he noted burning sensations in his fingers. Ten days before admission he complained of being unable to control his urine. In June 1967 he had been in another hospital with similar complaints. He had stayed there for a month, making a slow but incomplete recovery.

Examination showed nystagmus, with a quick movement to the left, diplopia on looking to the right, and a doubtful left facial weakness. The optic discs were normal. Cerebellar ataxia and hypotonia were present in both arms, but more pronounced on the left. There were no sensory changes in the arms. The abdominal reflexes were absent. The legs were weak and spastic, with brisk reflexes and extensor plantar responses. There was some loss of vibration sense and slightly impaired position sense in the feet, with no loss of light touch or pain sensation. There was inco-ordination of movement in both legs of a cerebellar type and he was unable to stand. He was incontinent of urine.

Nystagmus, diplopia, and incontinence disappeared after a few weeks. All the other signs, however, remained static, though he was gradually able to walk a little with the aid of a stick. He was transferred to another hospital on 4 February 1969, and since then we have had no record of his progress.

Routine investigations were negative. The C.S.F. showed gross abnormalities compatible with multiple sclerosis. Other investigations showed: total phospholipid 99·4 μg/100 ml., lysolecithin 5·9%, sphingomyelin 27·6%, lecithin 54·2%, serine 3·5%, ethanolamine 12·7%, and phosphatidic acid 5·1%. Total protein 63·5 mg./100 ml., alpha-globulin 13·6 mg./100 ml. (21·4% of total); Lange curve 4433321000; and W.R. negative Acrylamide disc electrophoresis: prealbumin 4·6%, albumin 59·3%, beta-globulin 6·1%, IgB 10·6%, and IgA 2·1%.

CASE 2

This patient, a 25-year-old woman, from Machakos district, was seen in January 1966. She gave a two-year history of weakness, heaviness, and paraesthesiae of the left leg, failing vision, recurrent attacks of pain behind the eyes, and incontinence of urine. Her gait was both spastic and unsteady. There was pallor of the optic discs, more pronounced on the temporal side, but no nystagmus or other cranial nerve involvement. The tendon reflexes in the lower limbs were abnormally brisk, particularly the left knee jerk. The plantar responses were doubtfully extensor. The abdominal reflexes were absent. She had an intention tremor of both upper limbs. There was a loss of proprioception over the left leg. Sensation to pain and touch were intact. The C.S.F. showed: protein 25 mg./100 ml., Lange curve 5543211000, and a negative Kahn test. She was discharged after six weeks, when her walking had improved.

Eighteen months later she was readmitted ataxic. She gave a history that she had improved after her return home, but that since the birth of a child three months previously she had been unable to walk and was incontinent. Examination showed bilateral optic atrophy, no nystagmus, and a scanning speech. All deep reflexes were increased and she had left ankle clonus. The abdominal reflexes were present. The plantar responses were extensor. There was pronounced ataxia of the cerebellar type, affecting the legs more than the arms. Investigations were again negative, apart from those of the spinal fluid, which showed an abnormal Lange curve (555222000), a negative Kahn test, and a slightly raised protein (41 mg./100 ml.).

She was given a course of parenteral vitamin B injections. After four months in hospital the clinical picture had not appreciably altered, except that there was some improvement in the control of her bladder.

COMMENT

In these two cases the clinical features conform well with those of multiple sclerosis, though the definite diagnosis is made only at necropsy. Both patients were young, with evidence of involvement of the pyramidal tracts and ataxia of the cerebellar type with minimal sensory changes. They had disseminated lesions in the central nervous system, showing fluctuations in the clinical manifestations, and, lastly, the C.S.F. findings in each case were compatible with the diagnosis. We therefore feel that the diagnosis is justified in both these patients.

Dean (1967) produced evidence to show that the incidence of multiple sclerosis is higher among immigrants to South Africa from Europe than among the white population born in South Africa. He suggested that multiple sclerosis is a viral gastrointestinal infection of infancy and that lack of exposure to the virus in early life predisposes to the development of multiple sclerosis later on. Kuru (Mathews, *et al.*, 1968) has been shown to be due to a slow virus and presents as a progressive subacute cerebellar ataxia. A type of progressive cerebellar ataxia for which no aetiological cause has been established is seen not infrequently in Kenya. It could be that this may be the more usual response in the African to a slow virus if such be the cause of multiple sclerosis. Dietary and immunological factors have been considered as predisposing to the disease, but the applicability of these in Africa, where large sections of the population have little or no medical coverage, is hard to assess. The low incidence of multiple sclerosis in the indigenous African is by no means certain or accounted for.

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