AN UNUSUAL CASE OF MENINGOENCEPHALOMYELITIS IN NORTHERN IRELAND


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REPORT OF A SUSPECTED CASE OF LOUPING ILL IN NORTHERN IRELAND

Introduction

The louping ill virus, a variant strain of the Russian tick borne group of viruses, has been known to cause enzootic encephalomyelitis of sheep in the British Isles for over a century. The viral aetiology was established by Pool et al. (1930) in Scotland. Although it is not a frequent cause of human disease, there are reports of human infections among laboratory workers and persons working in close association with sheep in enzootic areas. In Scotland, England and Northern Ireland, most of the important publications on ovine encephalitis have come from workers in these areas (Pool et al., 1931; Greig et al., 1931; Brownlee and Wilson, 1932; Gordon et al., 1932; Hurst, 1950; Van Rooyen and Rhodes, 1948; Davison et al., 1948; Likar and Dane, 1958). The existence of infections by this virus is not widely appreciated and difficulties therefore arise in diagnosis. Von Zeipel et al. (1958) in Sweden, found neutralising antibodies against louping ill virus in 66 out of 159 patients with a clinical diagnosis of aseptic meningitis, meningoencephalitis and poliomyelitis. In this paper, we report a case of meningoencephalomyelitis which we believe may be due to louping ill virus and draw attention to the possible extent of infection by this virus in Northern Ireland.

Natural History

Louping ill virus is transmitted by a tick of the family Ixodidae (Ixodes ricinus). The tick acquires the virus agent in its larval or nymphal stage but does not become infective until the adult stage. Hurst (1950) stated that the virus does not reach the nervous system by the local peripheral nerves. He suggested that during the viraemic phase, the agent passes out of the blood on to the nasal mucosa and thence by way of the perineural lymphatics of the olfactory nerves to the subdural space. Although the exact route is not known, it is established that the virus disseminates widely and can occasionally be found in peripheral nerves and certain body tissues. In fact, the virus has been isolated from blood and C.S.F. during the acute phase of the illness.

Morphology

The diameter of the Russian spring-summer and louping ill strains

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is 15-25 m.u. by gradocol membrane filtration. These strains have been shown to pass through Berkefeld V and N or Seitz filters.

Case Report

The patient, J. H., 62 year old man, worked as a gardener in the suburban areas of Belfast for 9 years prior to his death. Although he had been in fairly good health before that date he had been attending hospital for the past 9 years with angina of effort, and on one occasion, in 1954, had been thought to have a myocardial infarct. There was nothing significant in the family history and he was married with three children, who are all well. He gave no history of being in close contact with sick birds or animals and he had not had any recent injection or vaccinations, nor had he dealt with any toxic substances.

He began to complain in March 1963 of a burning pain in both feet and calves. The pain was worse at night and within a few weeks he noticed pins and needles sensation and numbness. On admission to the Royal Victoria Hospital on 1/6/63 he was fully orientated. The cranial nerves were quite normal except for marked arteriosclerotic change in both fundi. The upper limbs showed no abnormality. In the lower limbs there was slight weakness of dorsiflexion of the feet. The knee and ankle jerks were absent and the plantar reflexes flexor in type. There was impaired pinprick and cotton wool sensation in a stocking area of both feet extending to two inches above the ankle. Joint position sense was impaired in the right toes and vibration sense below the right knee. All the pulses were present and equal, the blood pressure was 120/80, and the heart sounds normal.

Investigations at that stage revealed blood count in which haemoglobin was 96% and a leucocyte count of 11,900 with 80% polymorphs. X-ray chest and abdomen revealed no abnormality. Fractional test meal revealed the presence of free acid. Serum electrolytes and blood urea were normal. E.S.R. was 22 mm/1st hour. Lumbar puncture revealed clear fluid at 170 mms. C.S.F. pressure with a protein content of 200 mgs. % and 22 white cells per cu. mm., of which 20 were lymphocytes, globulin was increased, the W.R. was negative and there was no large change. Serum B12 and serum folic acids were normal and no Figlu was detected in the urine. There was no excess porphyrins in the urine and the serum protein electrophoresis showed no significant change.

The clinical diagnosis was peripheral neuritis of unknown aetiology. He was treated with large doses of vitamin, at first intravenously, and then by mouth. He felt slightly better and was discharged.

He was readmitted on the 20/8/63, two months later, with a history that in the previous three or four weeks his symptoms had become much worse and he now complained of severe weakness in the lower limbs, pain in the legs, and also numbness in the hands. At this stage there was a slight decrease in sensation in the upper limbs in a glove distribution. There was very considerable weakness in the legs with sensory loss which extended to just below the knees. Joint position sense and vibration sense were not greatly impaired in both lower limbs. Tendon reflexes in the legs remained absent. The anatomical diagnosis was again polyneuritis of unknown aetiology and he was discharged.

In October, 1963, about 6 months after the onset, he could no longer walk or stand, and was readmitted. On examination he had coarse nystagmus in all directions. There was no facial weakness and the rest of the cranial nerves were normal. In the upper limbs there was a very marked intention tremor with the left possibly more marked than the right and rapidly alternating movements were sluggishly performed. There was no definite weakness. In the lower limbs there was extreme weakness and wasting of all muscle groups. There was decreased resistance to passive stretch in the lower limbs. Sensation continued to be impaired in a stocking and glove distribution. Vibration sense was lost up to the clavicles and joint position sense was grossly impaired in both feet. All the tendon reflexes were absent. The plantar responses were now extensor in type.

He was absolutely clear in his thinking and detailed mental function tests revealed no abnormality. Investigations included: haemoglobin 90%, leucocyte count 8,500 with a normal differential and glucose tolerance test was normal. Serum alkaline and acid phosphatase, serum calcium and phosphorus, serum cholinesterase, blood urea and serum electrolytes were all normal. C.S.F. now showed a protein of 120 mgs. and 150 leucocytes per cu. mm., all of which were lymphocytes. W.R. in the blood and C.S.F. were negative.

Throughout the periods in hospital his temperature was not above 99°F. The clinical picture was now of spinocerebellar degeneration with peripheral neuropathy. Suddenly, on the 17/12/63 he complained of breathlessness with clinical evidence of left ventricular failure from which he died a few hours later.