Two Cases of Guillain-Barré Syndrome and Encephalitis after Measles

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During recent years complications from measles infection have attracted great interest. Apart from encephalitis, which has been estimated to occur in 1 per 1,000 cases of measles (Miller *et al.*, 1956), subacute sclerosing panencephalitis and multiple sclerosis have been thought to be associated with the disease (Pette and Kuwert, 1965; Dayan, 1969).

Polyradiculoneuritis (Guillain-Barré syndrome) has been associated with measles in a few cases (Leneman, 1966). This report describes the cases of two children who fell ill with measles during the same epidemic and who both developed signs of polyradiculoneuritis and encephalitis.

Case 1

A 9-year-old girl who had not had prophylaxis against measles fell ill with cough and fever on 23 February 1970. Two days later a morbilliform rash appeared, and five days after that she complained of pain in her arms and legs and was unable to walk. On admission to the clinic on 3 March she was afebrile. Achilles tendon reflexes could not be elicited. The following day there was slight nuchal rigidity, facial muscle weakness, slight ataxia, and very weak patellar reflexes. The C.S.F. was clear and contained 11 mononuclear cells/mm³ and 48 mg of protein/100 ml.

The neurological symptoms gradually increased and on 13 March there was fatigue, anxiety, flaccid paralysis of the legs, weakness of the facial and arm muscles, and urinary incontinence. C.S.F. protein had risen to 760 mg/100 ml and electrophoresis indicated massive damage to the blood-C.S.F. barrier. E.E.G. was slightly abnormal over the left hemisphere.

Prednisolone was given, initially 40 mg/28 kg/day, later in lower doses, totalling 1.4 g during eight weeks. On 9 April she was alert, her arms were stronger, but the legs were still paretic. C.S.F. protein was 400 mg/100 ml. On 5 May she moved her legs a little and controlled her bladder. E.E.G. abnormality was less severe and conduction velocity of the right ulnar nerve was 10 m/sec (lower normal value 45 m/sec). After an intensive training programme she was able to walk unaided again in December. In July 1971 bilateral foot extensor weakness persisted. Conduction velocity was 42 m/sec in the ulnar nerve and 35 m/sec in the peroneal nerve.

Virological Studies.—No virus was isolated from the C.S.F. on admittance. Measles complement fixation test titre of serum was 1/32 12 days after the onset of the rash and 1/8 three months later. Haemagglutination inhibition test titres were 1/40 and 1/20, and neutralizing antibody titres 1/256 and 1/512 respectively.

Case 2

A 5½-year-old boy, not immunized against measles, fell ill on 9 March 1970 with cough, vomiting, and high temperature. A morbilliform rash appeared two days later followed by diarrhoea and, on 16 March, drowsiness, irritability, and weakness of the legs. On admission two days later he was afebrile, with slight nuchal rigidity and signs of dehydration and ketonuria, but the blood picture and serum electrolytes were normal. The C.S.F. was clear, with no white cells. Protein concentration was 30 mg/100 ml.

He had penicillin for bilateral maxillary sinusitis and, during two weeks, supplementary intravenous fluids. Serum electrolytes remained normal. Signs of cerebral depression and irritation persisted, spontaneous movements of arms and legs ceased, and stretch reflexes disappeared. On 24 March he had attacks of nystagmus. Blood pressure was 140/100 mm Hg. Early next morning he had generalized convulsions with right-sided dominance persisting in spite of anti-convulsive treatment for 1½ hours. Respiration was assisted and there was no cyanosis. The C.S.F. protein was 260 mg/100 ml and electrophoresis indicated massive damage to the blood-C.S.F. barrier. The E.E.G. was severely non-specifically abnormal, with a maximum on the left side. Alertness returned.

On 3 April spontaneous movements of the extremities were seen, and 12 days later he sat in a wheelchair. C.S.F. protein had fallen to 199 mg/100 ml. On 6 May he laughed, talked, moved his hands precisely, but could not stand unaided. Peroneal nerve conduction velocity was 10 m/sec and the E.E.G. was less abnormal. On 6 July the C.S.F. protein was normal (25 mg/100 ml). Peroneal nerve conduction velocity was 30 m/sec but had returned to normal (52 m/sec) on 28 July. He was then in full physical activity and no behavioural abnormalities had been noted by his mother.

Virological Studies.—No virus was isolated from the C.S.F. six and 16 days after the onset of the rash. Four weeks after the onset of the rash complement fixation titre of the serum was 1/16, haemagglutination inhibition titre 1/40, and neutralizing antibody titre 1/256. No significant alterations of the titres occurred during several months, but after 15 months serum gave a negative complement fixation reaction.

Comment

Several features of polyradiculoneuritis suggest that it is an autoallergic disease. Thus if the Guillain-Barré syndrome is the human counterpart of experimental allergic neuritis, measles encephalitis may be an equivalent of experimental allergic encephalomyelitis. In analogy with experimental allergic encephalomyelitis (Åström and Waksman, 1962), and in concordance with the present findings, measles encephalitis might be associated with sensitization to antigens of the central nervous system, possibly also accompanied by lesions throughout the nervous system. The latent period between exanthema and the first recording of neurological symptoms was five days in the present cases. Since viremia and invasion of the central nervous system occurs well before the onset of rash (Pampiglione, 1964) the latent period recorded would be long enough to allow for the development of immunity against components of nerve tissue.

The laboratory diagnosis of measles infection was supported by several serological data. Although in accordance with previous experience (Enders, 1962) measles virus could not be isolated from the C.S.F., these data together with the clinical picture showed beyond doubt that the exanthematous disease associated with the neurological complications was measles.

The occurrence of two cases of polyradiculoneuritis after measles during the same epidemic may be mere coincidence. On the other hand it should be noted that some measles epidemics seem to be more prone than others to produce neurological complications (Doutlik, 1966), and measles virus strains of varying neurotropism have been produced (Burnstein *et al.*, 1964).

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