CASE REPORTS

Encephalitis and polyneuritis complicating varicella zoster infection

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Summary

A 70-year-old patient is described who developed symptoms and signs of a Guillain-Barré-type neuropathy and encephalopathy beginning 6 weeks after a cutaneous varicella zoster eruption. The patient made a complete symptomatic recovery over a period of 4 months. It is suggested that the delayed onset neuropathy and encephalopathy in this patient may have been due to an allergic inflammatory response to an antigen common to peripheral and central myelin.

Introduction

A patient is reported who developed a varicella zoster infection which was followed by an encephalitic illness and peripheral neuropathy of the Guillain-Barré type. Although both encephalopathy and neuropathy are recognized complications of varicella zoster, the authors are not aware that the occurrence of these complications together has been previously reported.

Case report

A 70-year-old previously healthy male teetotaler presented with a 2-week history of progressive lower limb weakness. Six weeks earlier he had developed a typical zoster eruption over the right lower abdomen which had resolved spontaneously over 2 weeks. Clinical examination revealed a global weakness of the lower limbs to MRC grade 4 with areflexia of all 4 limbs and flexor plantar responses; sensory examination was normal. Eight days later he was admitted to hospital with increasing confusion. He was apyrexial but was temporo-spatially disorientated and experiencing visual hallucinations. On examination, the cranial nerves were normal but there was a severe weakness of the upper and lower limbs. In the arms power was globally reduced to MRC grade 3 and in the legs to MRC grade 3 proximally and MRC grade 2 distally. All deep tendon reflexes and the plantar responses were absent. Sensibility to pain and light touch was reduced in the hands and feet and vibration sense was lost below the xiphisternum and below both wrists; physical examination was otherwise normal. Labor-

atory findings included Hb 13.7 g/dl; WBC $8.2 \times 10^9/l$; ESR of 55 mm/hr. Other investigations including vitamin B₁₂ and folate concentrations, urea and electrolytes, random blood sugar, serum immunoglobulins, thyroid and liver function tests, Wasserman reaction and a chest X-ray were all normal. The cerebrospinal fluid (CSF) was under normal pressure with no pleocytosis. The CSF protein was 0.1 g/l and the glucose 5.0 mmol/l. An electro-encephalogram (EEG) showed frequent generalized bursts of high voltage delta activity consistent with an encephalitis. Nerve conduction velocity (NCV) in the posterior tibial nerves was reduced to 32 metres/second (m/sec) and 24 m/sec on the right and left respectively with prolonged terminal latencies of 20.4 milliseconds (msec) and 14.5 msec. Nerve conduction velocity in the left lateral popliteal nerve was 32 m/sec with a terminal latency of 15.9 msec. No response was obtained from the right lateral popliteal nerve. In the upper limbs NCV was reduced to 27 m/sec and 25 m/sec in the forearm segments of the left ulnar and median nerves respectively. The patient remained confused and disorientated for 10 days but by 2 weeks after his admission, intellectual function had greatly improved and a repeat EEG showed occasional bilateral bursts of theta activity. Power in the limbs began to improve 4 weeks after the onset of his illness, by 8 weeks he was walking with the aid of sticks, and by 4 months power had returned to normal although the Achilles tendon reflexes were still absent.

Discussion

Varicella zoster (VZ) infections usually involve the posterior root ganglia or the sensory ganglia of the cranial nerves, and clinical evidence of involvement elsewhere in the nervous system is rare. Recognized neurological complications include encephalomyelitis (Dayan, Ogul and Graveson, 1972; Gardner-Thorpe, Foster and Barwick, 1976), polyneuropathy (McCormick *et al.*, 1969), cranial nerve palsies and anterior nerve root syndromes (Rose, Brett and Burston, 1964).

The time course of the neurological illness in this

patient was similar to that described in other reports of encephalitis or neuropathy following VZ and it therefore seems likely that the neurological changes in this patient were attributable to his antecedent VZ infection. The possible pathogenesis of these complications is of interest. Although direct involvement of the CNS with VZ virus has been described (Dayan *et al.*, 1974) other pathological studies have reported foci of demyelination and collections of macrophages and other inflammatory cells, suggesting an 'auto-immune' response (Gardner-Thorpe *et al.*, 1976). It has also been suggested that the pathogenesis of VZ polyneuropathy involves an inflammatory allergic demyelination (McCormick *et al.*, 1969). If the encephalopathy and Guillain-Barré syndrome which occur after VZ are due to allergic demyelination, the fact that these complications occurred together in this patient might suggest that antigens common to peripheral and central myelin were involved in their pathogenesis as has been postulated in a recent experimental study (Saida *et al.*, 1979).

Full recovery from the encephalopathy and polyneuropathy occurred after several months confirm-

ing the previously reported favourable prognosis in these complications (Rose *et al.*, 1964).

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