Encephalitis and Polyradiculoneuritis Following Rubella Virus Infection - A Case Report -

Amongst neurological complications of rubella virus infection, polyradiculoneuritis as well as encephalitis is very rare. Only one case of postrubella polyradiculoneuritis combined with encephalitis has been reported to our knowledge. A 17-years-old male presented with suspected meningoencephalitis in a recent epidemic of rubella in a southern district of Korea. He developed symmetrical hyporeflexic weakness of all four extremities with urinary disturbance several days later. Rubella IgM antibody titer (enzyme linked immunosorbent assay) was 58 AU/mL in serum and 12 AU/mL in cerebrospinal fluid. Electrophysiologic studies showed peripheral polyradiculoneuropathy with multifocal conduction block. Considering the involvement of the central nerve as well as the peripheral nerve in an adult patient, this case is thought to be valuable in view of the pathophysiology of neurologic complication in rubella virus infection. *(JKMS 1997; 12: 168~70)*

Key Words : *Rubella, Polyradiculoneuritis, Encephalitis*

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INTRODUCTION

Encephalitis and polyradiculoneuritis are rare complications in rubella virus infection although a wide spectrum of postrubella neurological complications may follow. The incidence of encephalitis is estimated at 1 per 5,000 cases (1). Only nine cases of acute postrubella polyradiculoneuritis has been described (2~7). Concomitant polyradiculoneuritis with encephalitis following rubella in a patient is extremely rare. Only one case, to our knowledge, has been reported (8). Considering the involvement of the central nerve as well as the peripheral nerve in an adult patient, this case is thought to be valuable in view of the pathophysiology of neurologic complication in rubella virus infection.

CASE REPORT

We report an unvaccinated 17-year-old high school boy who was admitted to our hospital with suspected meningoencephalitis in a recent epidemic of rubella in a southern district of Korea. The patient had been healthy until 6 days previously when fever, sore throat and tender swollen lateral cervical lymph nodes developed, followed by the appearance of an erythematous maculopapular rash on the face, spreading to the trunk and extremities. The rash disappeared 3 days later. Two days before admission, severe headache and nausea developed and then increasingly worsened to confusion and aggressiveness. On admission day, he had a generalized tonic-clonic seizure. General physical examination disclosed mild fever and mild nuchal rigidity. Findings on neurological examination were unremarkable except for confused mentality. A lumbar puncture was done. Findings were : opening pressure ; 180 mmHg, white blood cells ; 25 /mm^3, protein ; 250 mg/dL, glucose ; 77 mg/dL. Microscopic and cytologic examinations of cerebrospinal fluid (CSF) showed nonspecific. Oligoclonal bands were not detected in the CSF. Myelin basic protein was 4 ng/mL (normal range ; below 4). Titers of enterovirus type 71, coxsachie virus A type 7 and type 9 (neutralization test) were 1:1 (normal ; below 1:1). Routine laboratory data, electrocardiogram, X-rays of the chest and the skull were unremarkable. Electroencephalography showed diffuse slowing. An enzyme linked immunosorbent assay (ELISA) revealed that rubella IgM antibody titer was 58 AU/mL in serum (normal range : below 15) and 12.6 AU/mL in CSF (normal range : below 1.2). During the following two days, his confused mentality cleared and there was no further seizure. On the third admission day, he developed symmetrical hyporeflexic flaccid weakness of all four extremities with urinary disturbance. There were no abnormalities in cranial nerves and sensory system. During the next three days, the weakness was more pronounced and unusual vasomotor autonomic dysfunc-

Table. Nerve conduction studies at 3rd day and 30th day

Nerve tested	Stim.point	Initial study	Follow up study	Normal range
Median motor				
Amplitude	W/E	3.9/1.8	2.6/2.4	4-18 mV
Conduction velocity		46.2	47.6	41-57 m/s
Distal latency		2.5	2.8	2.4-4.4 ms
F response		24.6	37.1	<31 ms
Ulnar motor				
Amplitude	W/E	9.2/4.1	7.1/4.4	6-16 mV
Conduction velocity		52.2	38.1	41-57 m/s
Distal latency		2.1	2.7	2.2-3.5 ms
F response		NR	NR	<32 ms
Peroneal motor				
Amplitude	A/K	0.12/0.04	1.6/1.4	2-12 mV
Conduction velocity		23.7	23.8	41-57 m/s
Distal latency		4.7	7.9	3.3-6.1 ms
F response		NR	NR	<55 ms
Tibial motor				
Amplitude	A/K	0.69/0.28	5.4/3.3	3-25 mV
Conduction velocity		40.0	31.6	41-53 m/s
Distal latency		4.2	5.4	2.7-6.1 ms
F response		NR	NR	<55 ms
Median sensory				
Amplitude	W/E	19/9	14/7	>20 uV
Conduction velocity		66	51	53-73 m/s
Distal latency		2.2	2.8	2.5-3.7 ms
Ulnar sensory				
Amplitude	W/E	17/10	11/8	>10 uV
Conduction velocity		55	61	53-73 m/s
Distal latency		2.5	2.6	1.8-3.5 ms
Sural				
Amplitude		13	9	6-47 uV
Distal latency		2.9	4.1	3.2-4.2 ms

W/E ; wrist/elbow, A/K ; ankle/knee, NR ; no response

tion (orthostatic hypertension and tachycardia) was noted (blood pressure : supine ; 180/120mmHg, sitting ; 230/140mmHg, pulse rate : supine ; 90/min, sitting ; 102/min). The electromyogram and nerve conduction studies, performed on three days after the onset of symtoms, showed axonal type of peripheral polyradiculoneuropathy with conduction block in the ulnar nerve (Table). Therapy with methylprednisolone (1 g/day, i.v., for 5 days) was administered. During the next one month, he had slow but progressive improvement. Follow-up electrophysiologic studies, performed on thirty days after the onset of symtoms, showed partial improvement. Rubella IgM antibody titer in CSF was 2.1 AU/mL. CSF protein, obtained on the 39th hospital day, was 115mg/dL. Three months after the onset of symtoms, flaccid quadriparesis and autonomic dysfunction became nearly normal.

DISCUSSION

The mortality associated with postrubella encephalitis is zero to 50% (1, 9). Our patient had a mild clinical course of the encephalitis. Postrubella encephalitis or polyradiculoneuritis has been known to be developed abruptly after the fading of the exanthem (10). Our patient, however, had a short course of encephalitis,

followed by a prolonged illness of the polyradiculoneuritis. The pathogenesis of postrubella neurologic complications is not yet completely known although two different mechanisms, the direct invasion of the virus and the host immune response, have been presented (11~14). Once a virus enters and spreads through the nervous system, the spectra of clinical syndrome are dependent on the nature of the organism as well as the host response. The adsorption of the virion of rubella has been known pantropic to nerve cells after penetration into the nervous system via the blood brain barrier and blood-CSF barrier (15). It was thought to be noticeable that the encephalitis preceded the neuritis in this patient. The elevation of the CSF protein, obtained on admission day, indicates the breakdown the blood brain barrier. We thought the abrupt development of the encephalitis likely resulted from direct invasion of the virus because a sufficient span of time was needed in order to organize the immune reaction. Meanwhile the polyradiculoneuritis, developed later, was likely resulted from the immune response since IgM antibody titer for rubella in CSF elevated intially and then decreased during the convalescent stage.

Orthostatic hypotension, as a manifestation of dysautonomia, is usually seen in acute inflammatory demyelinating polyradiculoneuropathy (Guillain-Barre syndrome). Hypertension and tachycardia are rarely seen but have been documented previously in Guillain-Barre syndrome and attributed to lesions of glossopharyngeal nerves which contain afferent fibers from the arterial baroreceptors (16~17) or increased plasma renin activity (18).

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