Guillain-Barré syndrome after rubella

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Summary

A case of polyneuropathy (Guillain-Barré syndrome) developing only 6 days after rubella infection in a 26-year-old woman is described. Serological confirmation of the viral infection by detecting the specific IgM antibody has not been previously reported in any of the rubella cases associated with the syndrome.

Case report

A 26-year-old Ugandan Asian was admitted to hospital with increasing weakness of her upper and lower limbs. Two weeks previously she had had a rash, a temperature of 38°C and bilateral occipital lymph gland enlargement. The rash, which started on her face and spread over the whole body, was macular and lasted for 48 hr. She felt unwell, had a slight cough and then developed arthralgia, mainly in the hands. Six days later she had paraesthesia of the hands and the feet which gradually increased in severity together with weakness of the limbs. She was unable to feel her feet on the ground and the calf muscles became painful and tender, but there was no sphincter involvement.

On admission she had a temperature of 37.5°C which settled spontaneously. There were a few enlarged occipital lymph nodes and slight enlarged tonsils. Her cranial nerves were normal, but there was symmetrical diffuse weakness of her arms and legs, most marked distally in the upper limbs, with absence of the supinator and ankle jerks on both sides. The triceps jerks were reduced but the biceps jerks were present and equal. Plantar responses were flexor. All forms of sensation were diminished in glove distribution in the hands and over the whole of both lower limbs, most marked distally. There was also some impairment of superficial sensation over the lower abdomen.

During the two days which followed admission her condition deteriorated and as a result she was started on ACTH, 60 units intramuscularly daily. Improvement in her neurological state was apparent within a couple of days and progressed steadily over the next 10 days. The dose of ACTH was gradually

reduced and was discontinued after 2 weeks. She was well enough to be discharged 3 weeks after admission and had virtually recovered 8 weeks after the onset of the neurological symptoms.

Rubella haemagglutination inhibition titre was 1:5120 and the rubella specific IgM antibody activity was 24%, as demonstrated by gel filtration with Sephadex G-200. The rubella complement fixation titre was 1:40 and antibody titres to adenovirus, influenza viruses A and B, parainfluenza viruses 1 and 3, measles virus and *Mycoplasma pneumoniae* were unremarkable. The cerebrospinal fluid was clear, pressure 110 mm, protein 0.75 g/l, glucose 3.33 mmol/l and white blood cells $3 \times 10^6/l$ ($2 \times 10^6/l$ mononuclear). None of the cerebro-spinal fluid specimen was available for virological examination.

Discussion

Polyneuropathy is a rare complication of rubella. In the last 10 years since the rubella haemagglutination inhibition test came into routine use in most laboratories, only two cases of acute polyneuropathy associated with this viral infection have been described (Tomlinson, 1975). Before the introduction of the test there were a few other reports (Miller, 1956; Miller, Stanton and Gibbons, 1956; Pampiglione, Young and Ramsay, 1963; Leneman, 1966) in which the diagnosis of rubella was made on clinical grounds only. In the present case, the detection of specific IgM antibody, which appears after primary rubella and persists for about 4 weeks (Vesikari and Vaheri, 1968), confirmed the diagnosis. The onset of the neurological disorder within a week after the rubella strongly suggests an association between the two conditions.

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