Clinical reports

Guillain–Barré syndrome associated with rubella

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Summary: A 34 year old male patient with Guillain–Barré syndrome following rubella is described. Diagnosis of the infection was made by the detection of specific IgM and IgG antibody. This is a rare association and has not been previously reported in an adult without a characteristic rash.

Introduction

Rubella infection is generally a mild disease. It has an incubation period of 13–20 days after which a macular rash and lymphadenopathy may appear. Infection is subclinical in up to 25% of cases.¹ Neurological complications are rare and usually present as encephalitis or encephalomyelitis. We report a patient with severe Guillain–Barré syndrome (GBS) and serological evidence of recent rubella infection.

Case report

A previously fit 34 year old male motor mechanic was admitted to hospital with a 5-day history of increasing weakness in his lower limbs, unsteadiness of gait, paraesthesia of his hands and feet, back pain and dysphagia. Approximately 2 weeks before admission he had had a 'flu-like' illness with fever, myalgia and headaches. No rash was noted. There was no history of vaccination or exposure to toxins.

On admission he was afebrile and normotensive with no lymphadenopathy. The cranial nerves were normal. He had a symmetrical weakness (MRC grade 3/5) and generalized hypotonia in all limbs with absence of the supinator, biceps, knee and ankle tendon reflexes bilaterally. Triceps tendon reflexes and flexor plantar responses were present but reduced. All forms of sensation were impaired distally in the limbs. His condition deteriorated over the following 48 h with respiratory muscle involvement, loss of his gag reflex and increased limb weakness. The patient required assisted ventilation.

Initial investigations including urea, electrolytes, full blood count, ESR, arterial blood gases and chest X-ray were normal. The serum bilirubin was 43 µmol/l but this fell to normal levels within 3 days of admission. Other liver function tests were normal. The cerebrospinal fluid was clear, protein 0.73 g/l, glucose 4.2 mmol/l with no cells, and was sterile on culture. There were no oligoclonal bands.

Sera taken 5 days before and on the day following admission had rubella haemagglutination inhibition (HI) titres of 512 and were strongly positive for rubella IgM antibody by IgM-antibody enzyme-linked immunosorbent assays (ELISAs) (Sorin Ltd and Northumbria Biologicals). Rubella-specific IgM was also detected by sucrose density gradient ultracentrifugation (SDGU) followed by HI. Rubella-specific IgG antibody was detected at a level of > 500 IU (IMx, Abbott Laboratories Ltd). Serological tests for influenza viruses A and B, adenoviruses, legionella, *Mycoplasma pneumoniae*, Epstein–Barr virus, varicella-zoster virus, cytomegalovirus, herpes simplex, parvovirus B19 and hepatitis A and B were negative. Tests for syphilis were also unremarkable. Throat swabs and stool cultures did not yield any significant pathogens.

The patient was ventilated for a total of 9 weeks. During this period he developed severe flaccid quadriplegia with absence of all deep tendon reflexes and plantar responses. Signs of autonomic nervous system involvement included lability of blood pressure, episodes of tachycardia, bradycardia and asystole. Ophthalmoplegia was present for 5 days. Results from nerve conduction studies were consistent with a severe demyelinating neuropathy. The patient made a gradual recovery. Plasmapheresis was undertaken daily for the first 6 days and then twice more the following week but without any obvious benefit. A serum specimen

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taken after plasmapheresis had an HI titre of 128 and rubella IgG of 403 IU, and rubella IgM was no longer detectable. A further specimen was taken 3 weeks later and this showed a rise in the rubella IgG level to > 500 IU.

At present, 10 months after the onset of symptoms, he has some residual weakness although this continues to improve.

Discussion

GBS is preceded by a non-specific viral infection in 40–50% of cases. A specific pathogen, such as Epstein–Barr virus, hepatitis A, campylobacter or mycoplasma, is identified in a further 10%.² Previous reports of GBS following rubella have mainly relied on a clinical diagnosis³ or the demonstration of a significant rise in the HI titre.⁴ The introduction of ELISAs for the detection of rubella-specific IgM antibody enables the diagnosis to be made rapidly. However, positive IgM values in asymptomatic patients should be interpreted with caution as non-specific reactions can occur.⁵

References

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Only one other case has been reported where the demonstration of rubella-specific IgM antibody has confirmed the diagnosis of the preceding infection in a patient with GBS.⁶ However, the patient concerned had a characteristic rash and symptoms of rubella. Our patient had high levels of rubella-specific IgM and IgG. The IgM became undetectable about 5 weeks after the initial ‘flu-like’ illness and there was a demonstrable rise in the IgG level after plasmapheresis. These results are consistent with this having been a primary rubella infection. The onset of neurological symptoms within 2 weeks of this infection is suggestive of an association between these conditions. The possibility of recent rubella infection should be considered in patients with GBS where no other cause is found.

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