Guillain-Barré syndrome associated with H1N1 vaccination

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Guillain-Barré syndrome (GBS) has been described as adverse event following different types of vaccines, such as influenza, varicella, polio virus and hepatitis B¹. Since 2009 with the emergence of the pandemic H1N1 influenza virus infection in humans, new vaccines have been developed for influenza A (H1N1) as a preventive strategy to reduce transmission, protect groups at increased risk of infection, and decrease complications and death². In this context, we report two adults who developed GBS after H1N1 immunization.

CASES

Case 1

A previously healthy 33-year-old white man presented a 4-days history of progressive and ascending numbness and tingling in his feet and legs, followed by weakness of his lower limbs. There was no suggestive history of upper airway infection or diarrhea in the preceding weeks, but he had received H1N1 monovalent vaccine 15 days before symptoms onset. Neurological examination disclosed superficial and profound sensibility diminished in the lower limbs, crural paraparesis (muscle strength grade 4) and absent tendon reflexes. Laboratory work-up including complete blood count, TSH, tests for renal and hepatic function, vitamin B12 and rheumatic screening were all normal. Infectious diseases screening (HIV, syphilis, hepatitis, toxoplasmosis, cytomegalovirus, Epstein-bar and Campylobacter jejuni) were negative. CSF analysis showed protein-cytological dissociation: 2 leukocytes/mm³, protein 51mg/dL and normal glucose. Electrophysiological investigation was suggestive of acute demyelinating sensory-motor polyneuropathy, more severe in the lower limbs.

Case 2

A 23-year-old white man received the H1N1 monovalent vaccine and developed nonspecific symptoms as nausea, vomiting, cramps and muscle pain. On seventh day, lower limb weakness was observed with mild distal sensory complaints. Ascending weakness also affecting the upper limbs and respiratory insufficiency developed rapidly within 24 hours, requiring mechanical ventilation and he was transferred to the intensive care unit. Neurological examination showed flaccid tetraparesis with absent tendon reflexes in the four limbs. Laboratory studies were negative for infectious diseases and rheumatic screening. Complete blood count, creatinine, urea, glucose, TSH, B12 vitamin were all normal. A lumbar puncture was performed and CSF disclosed protein=69 mg/dL with 2 leukocytes/mm³ and normal glucose. Treatment with intravenous immunoglobulin (0.4 g/kg per day, during 5 days) resulted in weaning from ventilator and progressive motor improvement. Electroneuromyography revealed absence of F waves and diffuse signs of demyelination, suggesting an acute inflammatory demyelinating polyneuropathy.

DISCUSSION

GBS is usually preceded by an upper
airway respiratory or gastrointestinal infection. Although infrequent, it also may happen as a neurologic adverse event following immunization. The pathophysiology of vaccination-related GBS is not completely understood and probably an immune stimulation plays a role in its pathogenesis.\(^1,3\)

Recent studies showed no conclusive result proving a causal relationship between influenza A vaccine and GBS, nor a possible increased risk for GBS after H1N1 immunization.\(^4,5\) In this scenario, we cannot exclude that our patients had GBS as a coincidental finding, but the fact that no other secondary causes of GBS were found, and the symptoms of GBS developed very close to vaccination should be considered.

To summarize, our findings suggest a probably relationship between GBS and H1N1 monovalent vaccine. Further studies are necessary to unveil its real frequency and risk related to H1N1 immunization.

REFERENCES