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Letter to the Editor

Positive measles serology and new onset of type 1 diabetes presented with bilateral facial paralysis: a case report

Dear Editor,

A previously healthy 28-year-old woman had suffered from fever, nausea, vomiting, and generalized fatigue for one day before being transferred to this teaching hospital's emergency department in a confused state. Physical findings on admission were height of 150 cm and body weight of 88 kg, with a body mass index of 39 kg/m². She had a fever of 38°C. Laboratory data on admission are shown in Table 1. Her plasma glucose and HbA_{1c} levels were 421 mg/dL and 9.2 (normal range 4.3-5.7%), respectively. Serum test for glutamic acid decarboxylase antibody (AntiGAD) was 3 U/mL (positive if > 1 U/mL). The serum C-peptide level was 0.378 ng/dL (normal if > 1 ng/dL). A diagnosis of type 1 diabetes mellitus complicated by ketoacidosis was made based on the considerably decreased serum C-peptide level, antiGAD positivity, ketonuria, and metabolic acidosis (Table 1). She was treated with an intravenous infusion of saline and insulin, and eventually switched to intensive insulin therapy four times a day. On the second day of hospitalization she developed weakness on both sides of her face. On physical examination, there was bilateral facial nerve paralysis. An electroneuromyography demonstrated bilateral axonal neuropathy of the facial nerves and confirmed the diagnosis. A serological test for several viral antibodies was performed. The results revealed significant elevation of the measles IgM and IgG titers, but no abnormal results were shown in any of the other serological tests (Table 2). One week later, the patient's facial weakness had improved spontaneously with no residual weakness.

Measles virus infections generally occur in childhood, but infections in adolescence and adulthood can lead to complications. Pneumonia, hepatobiliary disease, encephalitis, acute renal failure, and type 1 diabetes (DM1) are among the various systemic disorders which have been associated with measles, with varying strengths of association.¹⁻⁴ Data on DM1 originates from the Swedish Childhood Diabetes Study, which showed a significantly higher rate of children who developed diabetes among those not vaccinated against measles. The authors hypothesized that measles vaccine could have a protective effect, or that measles infection could be a diabetogenic agent.⁴ But the association between measles and DM1 is still unclear.

Table 1 - Laboratory data on admission

Complete blood count	
WBC	11200/μL
Hb	13 g/dL
Plt	23.4 x 10 ⁴ /μL
Blood chemistry	
BUN	23 mg/dL
Cre	1.2 mg/dL
Alb	4.7 g/dL
Na	122 mEq/L
K	3.8 mEq/L
SGPT	17 IU/L
SGOT	15 IU/L
T-Chol	135 mg/dL
TG	324 mg/dL
Amylase	92 IU/L
Glu	421 mg/dL
HbA _{1c}	9.2 %
Urinalysis	
Glucose	3+
Protein	1+
Ketone	4+
Arterial blood gas analysis on 2 L/min oxygen by mask	
pH	7.143
pO ₂	98.0 mmHg
pCO ₂	23.7 mmHg
HCO ₃ ⁻	8.4 mmol/L

WBC, white blood cell; Hb, hemoglobin; Plt, platelet; BUN, blood urea nitrogen; Cre, creatinine; Alb, albumin; Na, sodium; K, potassium; SGPT, serum glutamic pyruvic transaminase; SGOT, serum glutamic oxaloacetic transaminase; T-Chol, total cholesterol; TG, triglyceride; Glu, glucose.

Table 2 - Results of serological test for viral antibodies (IU/mL)

Measles virus*	IgM	1.44 (0-1.2)
	IgG	1.86 (0-1.1)
Mumps virus	IgM	0.48
	IgG	0.09
Rubella virus	IgM	0.16
	IgG	> 400
Varicella zoster virus	IgM	0.67
	IgG	2.23
Cytomegalovirus	IgM	Negative
	IgG	420
EBV-anti VCA	IgM	0.93
	IgG	303
Herpes symplex virus	IgM	Negative
	IgG	52.662
<i>Borrelia burgdorferi</i>	IgM	0.42
	IgG	0.43
<i>Treponema pallidum</i>	Hemagg.	(-)

EBV, Ebstein Barr virus; VCA, viral capsid antigen; Hemagg, hemagglutination.
*Only measles antibodies were above the relevant reference range.

The differential diagnosis of the causes for bilateral facial paralysis covers a wide field, including genetic, infectious, traumatic, neoplastic, metabolic, neurological, vascular, iatrogenic, and idiopathic etiologies. Measles is not among the well-documented infectious etiologies, but three adult patients with acute renal failure and bilateral facial paralysis have been reported.⁵ In these patients, facial paralysis was the first neurologic sign, and then bulbar and respiratory weakness developed. Two of them died because of septicemia, and the only patient who survived had total deafness, blindness, and distal wasting. None of the patients had maculopapular rash.

The present patient could not give a reliable history and it is not possible to know whether she was vaccinated. However, she came from a rural area where compliance with the vaccination schedule was low. It is probable that she had an atypical presentation of measles, as expected in adults, because of fever and positive measles IgG and IgM antibodies. This case is interesting due to coexistence of bilateral facial paralysis, new onset of DM1, and positive measles serology. There is not a similar case in the literature. Although there are limitations with respect to the true causal relationship between measles and these two manifestations, this clinical picture should be kept in mind as a possible atypical presentation of measles infection in adults.

Conflict of interest

All authors declare to have no conflict of interest.

REFERENCES

1. Yasunaga H, Shi Y, Takeuchi M, et al. Measles-related hospitalizations and complications in Japan, 2007–2008. *Intern Med.* 2010;49:1965-70.
2. R Khatib, M Siddique, M Abbass. Measles associated hepatobiliary disease, an overview. *Infection.* 1993;21:112-4.
3. CY Lin, HC Hsu. Measles and acute glomerulonephritis. *Pediatrics.* 1983;71:398-401.
4. Blom L, Nystrom L, Dahlquist G. The Swedish Childhood Diabetes Study: vaccinations and infections as risk determinants for diabetes in childhood. *Diabetologia.* 1991;34:176-81.
5. Wairagkar NS, Gandhi BV, Katrak SM, et al. Acute renal failure with neurological involvement in adults associated with measles virus isolation. *Lancet.* 1999;18:992-95.

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