รายงานผู้ป่วย

Case Report

รายงานผู้ป่วย Guillain-Barré syndrome จากเชื้อไวรัสซิก้ารายแรกของประเทศไทย

Guillain-Barré syndrome in the Context of Zika Virus Infection : the First Report Case in Thailand

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Abstracts:

We reported a case of Guillain-Barré syndrome (GBS) who was infected by Zika virus and developed generalized weakness and respiratory failure. She was transferred and treatment in Buddhachinaraj Phitsanulok Hospital, a regional hospital of the lower northern part of Thailand and improved in 31 days and returned to have normal activity in three months. Laboratory show positive Zika virus in urine only, because of late collection. And we also review others reports from any countries in the context of epidemiology, distribution, clinical findingsand outcomes.

Key words: Guillain-Barré syndrome, Zika virus, Immune-mediated neuropathy

Introduction

Guillain-Barré syndrome (GBS) is an acute immune-mediated neuropathy that affects nerves controlling muscle strength and nerves transmitting pain, temperature and touch sensations. This can result in weakness and loss of sensation in the legs and/or arms. The worldwide incidence of GBS is estimated as 0.8-1.9 (median 1.1) cases per 100,000 people per year among all ages. The annual incidence of GBS increases with age (0.6per 100,000 per year in children and 2.7 per 100,000 per year in people aged 80 years and over) and the condition is slightly more frequent in males than in females.

Approximately two thirds of GBS cases are preceded by infections. Possible triggers are infections including bacteria (e.g. *campylobacter jejuni*) and viruses (e.g. dengue virus, chikungunya virus, cytomegalovirus, human immunodeficiency virus (HIV)). GBS may also be triggered by vaccine administration or surgery. GBS has a progressive, monophasic disease course, usually without relapse. Most patients with typical GBS present with rapidly progressive bilateral leg weakness with hypo/areflexia in the affected limbs.[1] In rare cases, patients can present with facial, oculomotor, bulbar (i.e. difficulty with swallowing and speaking) weakness, or primary sensory symptoms.

GBS is potentially life-threatening condition, with 20–30% of patients developing respiratory failure requiring ventilation and intensive care support. Up to 70% of patients have some degree of autonomic instability (i.e. arrhythmias and extremes in blood pressure), with 20% developing serious and potentially fatal autonomic dysfunction. There is a 5% mortality rate, despite optimal care.

During a Zika virus outbreak in French Polynesia[2,3] between October 2013 and April 2014, a 20-fold increase in GBS incidence was observed compared with the previous four years. A case-control study showed a strong association between Zika infection and GBS during the outbreak. Forty one patients (98%) with GBS had anti-Zika virus IgM or IgG. Thirty-seven patients (88%) experienced a transient illness lasting a median of 6 days (interquartile range 4-10) before the onset of neurological symptoms, suggesting recent Zika virus infection. Past dengue virus history did not differ significantly between patients with GBS and those in the control groups.[4] Based on a 66% attack rate of Zika virus infection in the general population, the risk of GBS was estimated to be 0.24 per 1,000 Zika virus infections.

In 2015 in the Brazilian state of Bahia, 42 GBS cases were reported[5], including 26 (62%) with a history of symptoms consistent with Zika virus infection. A total of 1708 cases of GBS was registered nationwide, representing a 19% increase of GBS cases from 2014 (total 1439 cases), although not all states reported an increase in incidence. As of June 16th 2016, in the context of Zika virus circulation, 13 countries and territories worldwide have reported an increased incidence of Guillain-Barré syndrome (GBS) and/or laboratory confirmation of Zika virus infection in people with GBS cases.[6] GBS has been reported in children as well as adults in the affected countries. Polio surveillance reports have indicated an increased incidence in cases of acute flaccid paralysis in children under 15 years in some countries with ongoing Zika virus transmission. These cases are currently under investigation.

Zika virus disease is caused by a virus transmitted by Aedes mosquitoes. People with Zika virus diseases visually have a mild fever, skin rash and conjunctivitis . No specific treatment or vaccine currently available and the best form of prevention is protection against mosquito bites. Zika virus is known to circulating Africa, Asia,America and the Pacific region.

Microcephaly is an uncommon condition where a body head circumference is less than expected based on the average for their age and sex and GBS in the typical form is an illness of the nerve that produces a lower, bilaterally and symmetrical sensorimotor develop ment deficit.

In 2015, forty two cases were reported, among which 26 (62%) had a history of symptoms consisted with Zika virus infection. GBS cases with laboratory confirm Zika virus infection were reported from Martinipro (two cases) and Perio to red (one case) and in French Polynesia 42 GBS cases during 2013-2014. Zika outbreak, 88% of the reported and illness competitively with Zika infection retrospective analysis (sero neutralization test) demonstrated that all 42 cases were positive for dengue and Zika virus infection .

Thailand, during 2015-2016, is one of eight countries and territories have reported an increased incidence of Zika infection and or laboratory confirmation (Figure 1) and the report is only one of GBS and Zika virus infection in Thailand.

Case Report

We reported a case of 52 years old female from Petchaboon who was refered with the symptoms of low grade fever and productive cough for three weeks prior to admission and two week of skin rash along body and limbs and one week of dysaesthesia of all linb and progressive weakness of both legs. She was admitted at provincial hospital and then tranferred to Buddhachinaraj Phitsanulok hospital because of more progressive weekness and respiratory failure. She was a healthy person with no any underlying condition and no any surgical operation before.

Physical examination at Buddhachinaraj Phitsanulok hospital showed mild high blood pressure 160/90 mm.Hg, heart rate 110 beats /min., respiratory rate 28 breaths /min., no fever, good consciousness and intubation already. General physical examination was in normal limit and neurological examination show:motor power of grade 2/5 of upper extremities and grade 0/5 of lower extremities with DTR all 0 grade, in sensory examination show picture of grove and stocking of all extremties in pinprick sensation, no clonus and barbinski reflex too .

Lumbar puncture was performed and showed no cell, no organism, high protein level (54 mg/dl.) with normal glucose level.

She was admitted to Intensive care unit (ICU) for respiratory support and also antimicrobials for aspiration pneumonia. Intravenous immunoglobulins started at standard dose for 5 days. In 31 days period of intensive care, the heart rate showed tachycardia at about 120 beats/min. in first two weeks and return to normal rate in the forth week, no others signs of autonomic disturbance in that period too. Mild fever from aspiration pneumonia were subsided in the third week and no evidence of significant pulmonany embolism and anyother complication.

She can spontaneous breath on the 28thday and on the 31thday. she was tranfered to regular ward for rehabilitation program in muscular power training and other supportive care.

The examination of plasma and urine show positive Zika virus by PCR technique in four serial specimen, positive only in urine that report same result from different two standard laboratory of the Ministry of Public Health and the Thai Red Cross Society.

She was transferred back to provincial hospital for continue rehabilitation program and after two sequencial follow up at one and three months. She returns to have normal activity and healthy again.

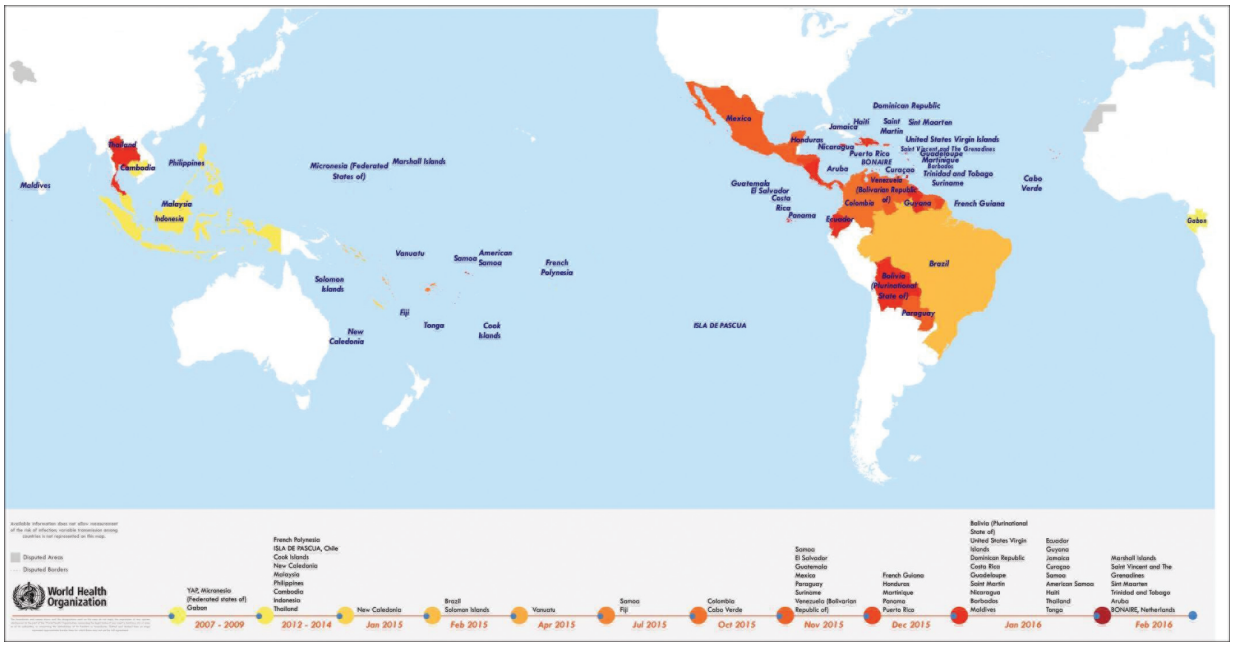

figure 1: Countries, territories and areas with local (autochthonous) Zika virus circulation, 2007-2016.[4]

Available information does not permit measurement of the risk of infection in any country; the variation in transmission intensity among countries is therefore not represented on this map. Zika virus is not necessarily present throughout the countries/territories shaded in this map.

http://www.who.int/emergencies/zika-virus/situation-report/19-february-2016/en/

Discussion

In the context of Zika virus circulation, eight countries or territories have reported increased GBS incidence and/or laboratory confirmation of a Zika virus infection among GBS cases.In 2015, 42 GBS cases were reported, among which 26 (62%) had a history of symptoms consistent with Zika virus infection in the state of Bahia in Brazil. A total of 1708 cases of GBS were registered nationwide, representing a 19% increase from the previous year (1439 cases of GBS in 2014), though not all states reported an increase in incidence.

Colombia reported an increase in the incidence of GBS as 201 GBS cases with history of suspected Zika virus infection were reported in the nine weeks to 14 February. Most of the cases are from Norte de Santander and Barranquilla-areas where many of the Zika virus cases have been registered. To date, none of these cases have been laboratory confirmed for Zika virus infection, or other possible causes.El Salvador recorded 118 GBS cases from 1 December 2015 to 8 January 2016, including five deaths, while the annual average number of GBS cases is 169. To date, none of those reported GBS cases have been laboratory confirmed for Zika virus infection or other causes.

GBS cases with laboratory confirmed Zika virus infections were reported from Martinique (two cases) and Puerto Rico (one case). In French Polynesia, 42 GBS cases were identified during the 2013-2014 Zika outbreak,88% of those reported an illness compatible with Zika infection.

Retrospective analysis (seroneutralisation test) demonstrated that all 42 cases were positive for dengue and Zika virus infection.

As with microcephaly, Zika virus is not proven to be a cause of increased GBS incidence in Brazil, Colombia, El Salvador, Suriname or Venezuela. However, a causal role for Zika virus is a strong possibility. Confounding factors include the contemporary circulation of dengue and chikungunya in the Americas, which are transmitted by the same species of mosquito. Further investigations are needed to identify the potential role of other factors (including infections) known to be associated, or potentially associated with GBS.

GBS is an acute, immune-mediate polyradiculoneuropathy typically occurring after minor viral bacterial infections. Motor function is usually affected, beginning distally and progressing proximally over up to 4-week period. Patients have generalised weakness, areflexia and a verying degree of sensory disturbances and involvement of cranial nerve. The risk of GBS increases with age and men are more commonly affected than women. The pathophysiology is incompletely understood, but is known to mostly occur 2-8 weeks after an infection. GBS is the leading cause of non-traumatic paralysis, with a global incidence of 1-4 per 100 000 persons-years. The range of infections reported to have preceded GBS include upper respiratory infections, notably influenza and pseudo-influenza, digestive tract infections, notably *Campylobacter jejuni,* as well cytomegalovirus and Epstein-Barr virus infections. The incidence of GBS case during the French Polynesian outbreak was estimated to be 0.24 per 1000 Zika virus infections, at the lower range of the 0.25 to 0.65 per 1000 observed following *C jejuni* infections. It is unlikely that GBS cases were missed during the study period, because routine procedures for systematic confirmation of diagnosis of GBS pre-existed the Zika virus epidemic and all cases were systematically referred to the Center for Health Professions Education (CHPE) for diagnosis confirmation. Although it is unknown whether attack rate of Zika epidemics will be as high in affected regions in Latin America than in the Pacific Islands (73% in Micronesia and 66% in French Polynesia), high numbers of cases of GBS might be expected in the coming months as the result of this association. The results of our study support that Zika virus should be added to the list of infections pathogens susceptible to case Guillain-Barré syndrome.

Because almost all of the patients with GBS were of Polynesian origin and because distribution of HLA alleles has been previously described as being involved in certain forms of GBS, a possible role of ethnicity in triggering GBS was hypothesized. However, the high incidence of GBS recently reported in Brazil, El Salvador, and Colombia during local Zika virus outbreaks suggests that, whenever involved, such host factors might not be specific to the ethnic groups living in French Polynesia.

In conclusion, this is the first study to document a large series of patient who developed a GBS following Zika virus infection, a virus that previously used to be considered as causing only mind disease. This patients with GBS reported symptomatic Zika virus infection that preceded the occurrence of neurological symptoms. All patients with GBS were of the acute motor axon neuropathy (AMAN) type, characterized by distal motor never involvement, the absence of typical patterns and levels of anti-glycolipid antibodies, and faster recovery than usually observed in typical GBS. Because Zika virus is spreading rapidly across the Americas, at risk countries need to be prepared to have adequate intensive care beds capacity to manage patients with GBS.

References

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