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CASE REPORT

Guillain-Barré Syndrome Following Suspected Zika Virus Infection: A Rare but Important Neurological Manifestation

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ABSTRACT

Zika virus infection is typically a mild, self-limited febrile rash illness, but growing evidence suggested potential neurological complications during outbreaks. We report a case of acute flaccid paralysis consistent with Guillain-Barré syndrome (GBS) occurring shortly after a clinically compatible illness suggestive of Zika virus infection in a returning traveler. The patient developed rapidly progressive, symmetrical limb weakness with areflexia, albuminocytologic dissociation in cerebrospinal fluid, and electrophysiological findings supportive of acute inflammatory demyelinating polyneuropathy. He improved after intravenous immunoglobulin and supportive care. This case highlights the importance of recognizing post-infectious neuropathy following suspected Zika virus disease, especially in settings where laboratory confirmation may be limited and serology may be complicated by flavivirus cross-reactivity.

Keywords: Guillain-Barré syndrome, Zika virus infection, acute flaccid paralysis, post-infectious neuropathy

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INTRODUCTION

Zika virus (ZIKV), a mosquito-borne flavivirus, was historically considered a sporadic cause of mild human illness until large outbreaks occurred outside Africa and Asia. The first major outbreak in the Pacific was described on Yap Island in 2007, characterized by fever, rash, conjunctivitis, and arthralgia, establishing ZIKV as an emerging global pathogen capable of rapid geographic expansion [1]. Subsequent reports emphasized ZIKV transmission potential in regions endemic for Aedes mosquitoes and noted that clinical presentation can overlap with other arboviral infections, complicating syndromic diagnosis [2]. A major outbreak in French Polynesia during 2013-2014 further underscored the capacity of ZIKV to cause widespread community transmission [3]. In 2015, autochthonous transmission in Brazil was confirmed, marking introduction into the Americas and prompting heightened international surveillance [4].

Guillain-Barré syndrome (GBS) is an acute, immune-mediated polyradiculoneuropathy and one of the most common causes of acute flaccid paralysis worldwide. It typically presents with rapidly progressive, symmetric limb weakness, areflexia, and variable sensory or autonomic involvement, often following an infectious trigger [5,6]. The immunopathogenesis is believed to involve molecular mimicry and antibody-mediated or complement-mediated injury to peripheral nerves, with clinical phenotypes influenced by preceding infections and host factors [6,7]. Standardized case definitions (e.g., Brighton Collaboration) improve diagnostic consistency for clinical & epidemiologic evaluation [8].

Early disease-modifying therapy with intravenous immunoglobulin (IVIg) or plasma exchange can shorten recovery time, while meticulous supportive care remains critical due to risks of respiratory failure and dysautonomia [9,10].

Given the expanding range of ZIKV and early alerts linking ZIKV circulation with neurological syndromes, including GBS, awareness of this association is essential for timely diagnosis and management [4]. This report describes GBS occurring after a clinically compatible illness suggestive of ZIKV infection, highlighting diagnostic considerations and implications for clinicians during outbreak settings.

Case Presentation

A 34-year-old previously healthy man presented with progressive weakness of both lower limbs for 3 days, followed by difficulty rising from a chair and climbing stairs. One week earlier, he had returned from a coastal region with known Aedes exposure. Two to three days after return, he developed a low-grade fever, generalized pruritic maculopapular rash, mild non-purulent conjunctival redness, and arthralgia involving wrists and ankles. Symptoms resolved spontaneously within 4 days without antibiotics.

On examination, he was afebrile and hemodynamically stable. Neurological examination revealed symmetrical flaccid weakness (Medical Research Council grade 3/5 proximally and 4/5 distally in lower limbs; 4/5 in upper limbs), generalized areflexia, and mild distal paresthesia without a sensory level. Cranial nerves were intact. No bowel or bladder dysfunction was noted. Over the next 24 hours, weakness progressed, and he developed mild tachycardia and intermittent orthostatic dizziness.

Baseline laboratory tests (complete blood count, electrolytes, renal and liver function) were within normal limits. Cerebrospinal fluid analysis performed on day 6 from onset of weakness showed elevated protein with a normal cell count (albuminocytologic dissociation). Nerve conduction studies demonstrated prolonged distal motor latencies, slowed conduction velocities, and temporal dispersion consistent with a demyelinating polyradiculoneuropathy. Serologic testing for dengue was negative for acute infection; ZIKV-specific testing was not available locally, and recent flavivirus exposure could not be definitively confirmed.

A diagnosis of GBS (Brighton level supportive) following a suspected ZIKV-like illness was made. The patient received IVIg at standard dosing over 5 days, with close monitoring of respiratory function and autonomic parameters. He did not require mechanical ventilation. Over the subsequent two weeks, strength stabilized and gradually improved. At 6-week follow-up, he was ambulatory with minimal assistance and had near-complete recovery of upper limb strength.

Discussion

This case illustrates a temporal sequence consistent with post-infectious GBS following a clinically compatible ZIKV-like illness. ZIKV infection commonly manifests as a mild febrile rash syndrome with conjunctivitis and arthralgia, features present in this patient, and similar to those described in early outbreak reports [1-4]. In 2015, public health communications highlighted concerns regarding increases in neurological syndromes, including GBS, in areas with ZIKV circulation, supporting clinical vigilance even when laboratory confirmation is not feasible [4].

The diagnosis of GBS in this patient was supported by classic clinical findings (rapidly progressive symmetrical weakness and areflexia), cerebrospinal fluid albuminocytologic dissociation, and electrophysiological evidence of demyelinating neuropathy. These features align with established descriptions of GBS and its major subtypes [5-7]. Standardized criteria such as the Brighton Collaboration case definition strengthen diagnostic certainty and comparability in surveillance contexts where emerging infections are suspected triggers [8].

A key challenge in linking an individual GBS case to ZIKV is diagnostic limitation. During the acute phase, nucleic acid detection is time-sensitive, and serology can be complicated by cross-reactivity with other flaviviruses, particularly dengue, which may co-circulate

in the same regions [2]. In many real-world settings—especially early in an outbreak—specific testing may be unavailable, making "suspected" rather than confirmed ZIKV infection a pragmatic classification based on clinical syndrome and exposure history [4]. Therefore, clinicians should interpret such associations cautiously while ensuring appropriate management of GBS.

Therapeutically, IVIg is an evidence-supported disease-modifying treatment for GBS and is generally comparable in efficacy to plasma exchange, with practical advantages in many settings [9,10]. The patient's stabilization and gradual recovery are consistent with expected outcomes when treatment and supportive care are instituted early, alongside careful monitoring for respiratory compromise and autonomic instability [6,7,9].

Conclusion

GBS should be considered in patients presenting with acute flaccid paralysis following a compatible arboviral illness during periods of suspected ZIKV transmission. Even when ZIKV confirmation is not possible, prompt recognition of GBS, use of standardized diagnostic frameworks, early initiation of IVIg or plasma exchange, and vigilant supportive care can improve outcomes.

Patient Consent: Written informed consent was obtained from the patient for publication of this case report and accompanying clinical details. Identifying information has been omitted.

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