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Review

Recent trends in ZikV research: A step away from cure

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ABSTRACT

Zika virus (ZikV) is a member of *the Flaviviridae virus family*, genus *Flavivirus* has emerged as a potential threat to human health worldwide. Consequences of vertical infections includes microcephaly with brain and eye anomalies, and adult infections includes Guillain-Barre'syndrome (GBS), brain ischemia, myelitis and meningoencephalitis. To develop a better treatment, many efforts are being made, like drug-repurposing concept for FDA-approved drugs for antiviral activity are screened against ZikV infection and emerging as a promising alternative to expedite drug development and various vaccines like *DNA*, *ZPIV*, *LAIV*, *mRNA* and *AGS – v* vaccines have been designed and in under clinical trial phases. Moreover, few pharmacological agents like *Mycophenolicacid*, *Niclosamide*, *PHA*–690509, *Emricasan* and *Bortezomib* are most potent anti-ZikV candidates and highly effective single or combining treatment with these drugs. This article reviews the ZikV illness, transmission patterns, pathophysiology of disease, global efforts, challenges and the prospects for the development of vaccines and antiviral agents.

Contents

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1. Introduction

Zika virus has emerged as a potential threat to human health globally. It was first came into existence in the Uganda's Zika forest (1947) in rhesus monkeys through an observing group of sylvatic yellow fever. In the year 1952, the first case of ZikV infection was identified in humans at the same region of Uganda and the United Republic of Tanzania [1]. Outbreaks of ZikV infections have been confirmed in Africa, America, Asia-Pacific [2]. The first documented epidemicity of ZikV occurred in the Federated States of Micronesia (FSM) in 2007, where 49 cases were confirmed while others 59 were considered as most probable out of total 108 suspected cases. Before 2007, at least 14 cases of ZikV had been reported, although other cases were likely to have occurred but not reported, because the symptoms were similar to that of other arboviral infections like dengue, chikungunya and West Nile. Therefore, the precise and differential analysis of ZikV infection was difficult in that regions where dengue and chikungunya cases already have been reported. ZikV is related to other pathogenic vector-borne flaviviruses including dengue, West Nile and Japanese encephalitis viruses, but produces a comparatively mild disease in humans. Similar to other flaviviruses, ZikV is enveloped and icosahedral with a non segmented, positive sense RNA genome 10.7 kb in length with two flanking non-coding regions (50 & 30 NCR).

Viral and host proteases split this polyprotein into three structural proteins (capsids-C, premembrane/membrane protein −prM/M and the envelope-E) and seven non-structural (NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS5) [3] [4].

1.1. Clinical manifestations

The most common symptoms of ZikV infection are fever, rashes, joint pains, conjunctivitis (red eyes), pruritus, fatigue, muscle pain and headache (Fig. 1) but severity of the symptoms are varies in DENV, CHIKV and WNV (Table 1). The incubation period for infection with ZikV ranges from 2 to 12 days. ZikV infections are often asymptomatic (80% of cases) [5].

1.2. ZikV transmission in human

ZikV is transmitted to people through an *Aedes* mosquito with ZikV infection. The mosquito becomes infected with ZikV when it bites an infected person. Hence, a transmission cycle is being developed between mosquito to human, human to mosquito and

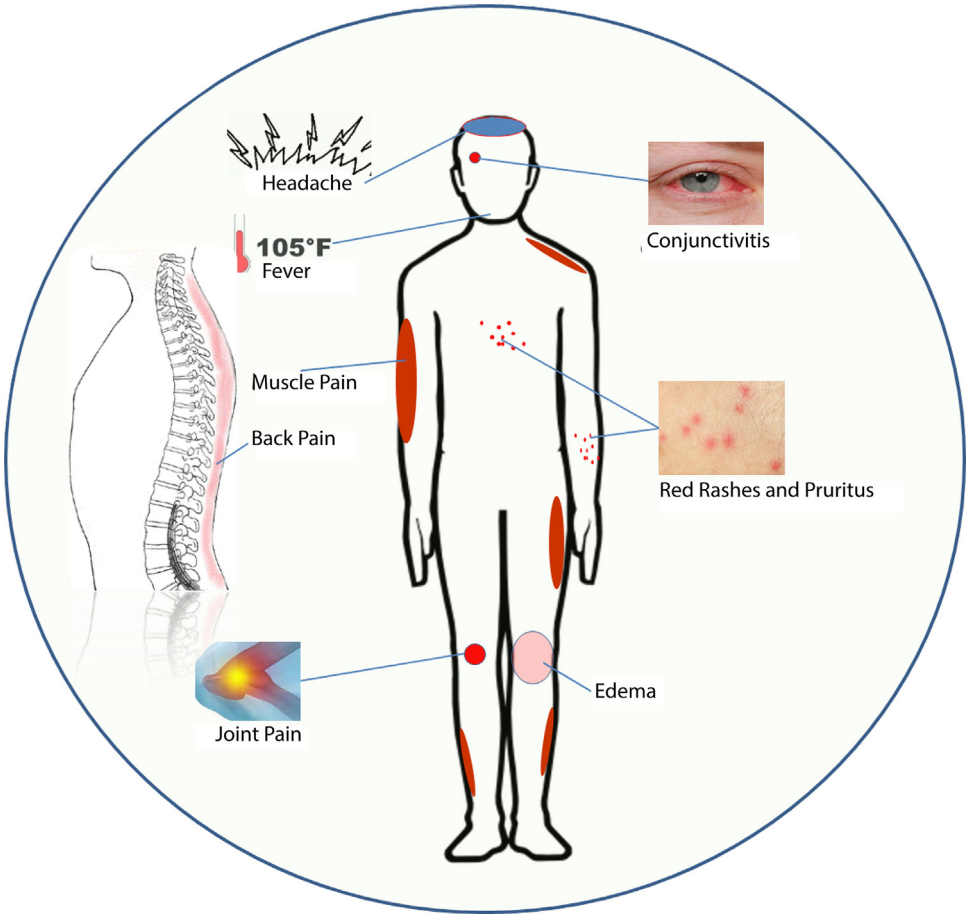

Fig. 1. Prevelent and adventious symptoms necessary at the onset of infection commonly caused by all prominent flaviruse(s) including ZikV, mapped to there respective positions. Appearence of these urges to make necessary tests for the undergoing infection with respect to the location of its existence.