ABSTRACT

The Zika virus (ZIKV), belonging to the Flaviviridae family, was first discovered in Africa in 1947. It is responsible for the nation-wide rise in arbovirus-related infections in Brazil. Its main transmission pathway relies on the bite or stinging of humans by virus-infected mosquitoes that belong to the Aedes genera, Aedes aegypti being the most common vector, given its wide distribution throughout tropical and subtropical regions. Transfusion-related transmission pathways have been thoroughly discussed recently due to the large number of asymptomatic patients and the virus’ strong epidemic potential; however, transmission is also possible through sexual and vertical pathways. Overall, clinical outcomes are positive. The most common symptoms experienced by patients are flu-like and similar to dengue (DENV) and chikungunya (CHIKV) virus infections. Primary diagnosis is based on clinical complaints that are typical in infections with this pathogen accompanied by specific molecular tests, more specifically, real-time reverse transcription polymerase chain reaction (RT-PCR). In a few cases, however, there are reports of severe consequences, including Guillain-Barré Syndrome (SGB), neurological damage and fetal malformations like microcephaly. There are no vaccines or ZIKV-specific antiviral therapies. Thus, the ZIKV remains a threat to hemotherapy infection prevention and leading disease control entities.

Keywords: Dengue, Hemotherapy, Zika Virus.

RESUMO

O vírus Zika é um Flavivírus descoberto na África em 1947, sendo responsável por causar uma arbovirose emergente no Brasil. Seu modo de transmissão é principalmente pela picada de mosquitos infectados do gênero Aedes, sendo que o Aedes aegypti é considerado o vetor mais comum, dada a sua ampla distribuição nos trópicos e subtropicais; no entanto, também é possível ocorrer a infecção através do ato sexual, materno-fetal, bem como transfusional. Este artigo trata de uma revisão de literatura a qual enfoca o histórico do Zika, sua estrutura viral e patogênese, epidemiologia, manifestações clínicas e diagnóstico, transfusões sanguíneas e os arbovírus. De particular importância, a via de transmissão transfusional tem sido discutida atualmente, devido ao grande número de casos assintomáticos e o potencial epidêmico do Zika em causar epidemias de grandes proporções. De maneira geral, a doença tem quadro clínico benigno, porém consequências clínicas graves têm sido relatadas, tais como a síndrome de Guillain-Barré (SGB), alterações neurológicas e malformações fetais, como microcefalia. Ainda não existem vacinas nem medicamentos antivirais específicos para o Zika, sendo o tratamento apenas de suporte; portanto, medidas de proteção tornam-se importantes para o controle vetorial do mosquito Aedes aegypti. Diante disso, o Zika continua a ser um desafio para a segurança transfusional e organizações líderes de prevenção e controle de doenças.

Palavras-chave: Dengue, Hemoterapia, Zika Vírus.
INTRODUCTION

Since its onset, only sporadic infections were reported in Africa and Asia, until the first Zika virus (ZIKV) epidemic occurred in the Pacific in 2007, on the Yap Islands (Federated States of Micronesia). In the period between 2013-2014, the virus was responsible for an epidemic in French Polynesia and subsequent spread to all of the Pacific Islands. In 2015, ZIKV emerged in the Americas, more intensively in Latin America and the Caribbean, as well as in Africa (MUSSO et al., 2017). The Brazilian Ministry of Health (MS) confirmed its autochthonous transmission in the northeast of the country in May 2015 (CUNHA et al., 2016).

The main transmission pathway of the virus to humans is through a mosquito sting by an infected Aedes. However, transmission can also occur through sexual, maternal-fetal and transfusion transmission pathways (SLAVOV et al., 2016, 2017). In addition, since ZIKV was detected in urine, saliva and in breast milk of humans, other potential transmission pathways must not be excluded (MUSSO et al., 2017).

Although signs of ZIKV infection are asymptomatic from 40% to 80% of infected individuals, patients may present nonspecific symptoms that resemble dengue virus (DENV) and chikungunya virus (CHIKV) infections. The most common signs and symptoms manifested by the patients are fever, bilateral non-purulent conjunctivitis, headache, myalgia, arthralgia with peri-articular edema of small joints and maculopapular exanthema (SLAVOV et al., 2016, 2017). In addition, cases of neurological manifestations and Guillain-Barré Syndrome (GBS) were considered epidemic in French Polynesia and Brazil. In early 2016, the World Health Organization (WHO) declared the recent ZIKV epidemics as an International Public Health Emergency (ESPII), because of its unexpected causal association with severe congenital brain abnormalities designated by medical entities as congenital Zika Syndrome, especially microcephaly during pregnancy (YUAN et al., 2017; BALMASEDA et al., 2017).

The primary diagnosis of ZIKV infection is based on the typical clinical symptoms of the patient, as well as a history of recent travels to regions that are endemic with ZIKV, or contact with anyone from those regions (CHEN, TANG, 2016). Differential diagnosis involves molecular tests such as the reverse transcription polymerase chain reaction (RT-PCR), a technique that can be applied in blood, serum, urine, seminal fluid or any other body fluid samples. Laboratorial test results are optimal if performed until one week after the onset of symptoms and the detection of the viral genome by RT-PCR is currently the most sensitive and specific method to confirm the diagnosis of infection by ZIKV (PINTO JUNIOR et al., 2015).

Treatments for the infection are symptomatic, since there are no vaccines or antiviral drugs that are specific to ZIKV (PINTO JUNIOR et al., 2015). Therefore, protective measures become important for controlling the spread of the disease through vectors, especially the Aedes aegypti mosquito.
(PINTO JUNIOR et al., 2015). In addition, the Centers for Disease Control and Prevention (CDC) recommend that pregnant women avoid unnecessary travels to areas of ZIKV transmission, as well as unprotected sexual contact with a partner who is at risk for HIV infection. Individuals who have traveled to endemic areas should postpone blood donation for at least 28 days (CHEN, TANG, 2016).

The objective of this work was to conduct a literature review on Zika virus infection, with emphasis on the transfusional approach of viral transmission.

ARBOVIRUS

Arboviruses (Arthropod-borne viruses) are viruses transmitted to humans and animals by infected hematophagous arthropods, especially mosquitoes and ticks, through bite, and are classified in this manner because of their replicative cycle, often and continuously replicating in insects (SANTOS, ROMANOS, WIGG, 2015). They are also classified as an RNA virus, very adaptable in nature, being the main reason as to why these viruses are the number one cause of zoonosis (PATY, 2013). This class includes more than 545 species, of which more than 150 are related to human incident zoonosis, diseases of great clinical importance due to the severity of the infections, which are of two types: infections involving the central nervous system (CNS) (myelitis, meningitis, encephalitis, behavioral changes, paralysis, paresis, seizures and coordination problems) and jaundice related hemorrhagic infections (hemorrhagic fevers with hepatic involvement) (VASCONCELOS, CALISHER, 2016).

The Flaviviridae family is composed of three genera: Flavivirus, Pestivirus and Hepacivirus (LOPES, NOZAWA, LINHARES, 2014). Among arboviruses that affect man, 13 are caused by the Flavivirus genus (SANTOS, ROMANOS, WIGG, 2015). The arthropod vectors, belonging to the Diptera order, Culicoidae superfamily and Culicidae family are of greater relevance in the tropics. Mosquitoes of the Culicidae family are grouped into three subfamilies: Toxorhynchitinae, Culicinae and Anophelininae, the latter two with a large number of insects with clinical importance. The subfamily Culicinae is the largest subfamily, and the most significant genera that occur in Brazil are: Aedes, Culex, Sabethes and Haemagogus and the subfamily Anophelininae, which includes the genera Anopheles that can also be found in Brazil (CONSOLI, DE OLIVEIRA, 1994).

The Aedes genus is the one that most often affects urban environments, with Aedes aegypti being the main vector in these types of environment, while Aedes albopictus is more widespread in rural areas (WILDER-SMITH et al., 2017). These two vectors are located in the tropics and subtropical regions and, preferentially, feed on human blood and are usually synanthropic. In addition, they are more active during the day and their reproduction occurs in domestic and communitary environments with egg incubation and hatching that can occur only in containers with water that sits still for long periods of time (WILDER-SMITH et al., 2017).
At least five arboviruses transmitted by mosquitoes to humans have epidemic potential in urban areas such as yellow fever virus (YFV), dengue virus (DENV), West Nile virus (WNV), ZIKV and Chikungunya virus (CHIKV) and have emerged in the hemispheres during recent centuries (GOULD et al., 2017), due to multiple factors such as urbanization, globalization and viral mutations that confer greater virulence to some viruses such as zika and chikungunya (WILDER-SMITH et al., 2017).

The DENV of the *Flaviviridae* family is the arbovirus of major clinical importance. It is typically found in tropical or subtropical climate and, according to recent estimates, it is responsible for approximately 390 million infections annually, of which 96 million are accompanied by clinical manifestations (BLITVICH, 2016). In addition, the ZIKV (*Flaviviridae* family, *Flavivirus* genus) has acquired clinical prominence through its rapid emergence and dissemination around the Brazilian territory, becoming ESPII due to the severity of clinical symptoms (VASCONCELOS, CALISHER, 2016, WILDER-SMITH et al., 2017).

**ZIKA VIRUS HISTORY**

The first ZIKV isolation occurred in April 1947, through the serum of a febrile Rhesus 766 sentinel monkey, during fieldwork by researchers at the Rockefeller Foundation in the zika forest, a forest reserve of approximately 25 acres, located on the shores of Lake Victory in the province of Entebbe in Uganda (DICK, KITCHEN, HADDOW, 1952, BURATTINI, 2017).

The characterization of this *Flavivirus* occurred by coincidence, based on research on the epidemiology of yellow fever (DICK, KITCHEN, HADDOW, 1952). The isolated strain was then named ZIKV766 because of the number of the sentinel monkey. A second isolate of ZIKV was made from a pool of *Aedes africanus* mosquitoes in January 1948, in the same forest, showing the participation of *A. africanus* as a potential vector for this virus (DICK, KITCHEN, HADDOW, 1952). Consequently, in the following 20 years, some isolates of ZIKV were obtained from *Aedes* spp. (*Aedes africanus*) and Malaysia (*Aedes aegypti*), leading to these probable epidemic vectors (MARCHETTE, GARCIA, RUDNICK, 1969, LANCIOTTI et al., 2008).

In 1969, ZIKV was isolated from *Aedes aegypti* mosquitoes collected in Malaysia outside the African continent. Almost a decade later, the first human infections were reported in 1977 on the island of Central Java, Indonesia (MARCHETTE, GARCIA, RUDNICK, 1969). Until the first epidemic on the Yap Islands in the Federated States of Micronesia in April 2007, only 14 cases of ZIKV in humans had been reported, however, none outside the African continent and Southwest Asia (DUFFY et al., 2009). Although the epidemic was brief (approximately 3 months), there is serological evidence suggesting that around 70% of islanders were infected with the virus. It demonstrated the high infectivity of ZIKV and its spreading potential as an emerging infectious disease (JIMENEZ et al., 2017).
VIRAL STRUCTURE AND PATHOGENESIS

ZIKV belongs to the Flavivirus genera and features the same genomic organization as all flaviviruses including DENV, WNV, YFV and Japanese encephalitis virus (JEV) (SHI, GAO, 2017). The ZIKV virions are approximately 60 nm in size and spherical in shape (SHARMA, LAL, 2017). ZIKV has a single-strand positive polarity RNA genome of 10,794 nucleotides with two non-coding regions (5’NCR and 3’NCR) flanking a single coding sequence (BURATTINI, 2017).

Two species of Aedes, A. aegypti and, to a lesser extent, A. albopictus, have been linked to almost all known ZIKV epidemics, although two other species, A. hensilli and A. polynesiensis, were considered to be the vectors in the Yap Islands and French Polynesia, respectively (PETERSEN et al., 2016). Approximately 40 to 80% of individuals infected with ZIKV are asymptomatic (SLAVOV et al., 2017). On average, ZIKV viremia persists for 10 days after infection, symptoms develop after about 6 days and may last for 1 to 2 weeks (FARIA et al., 2017). The incubation period from infection to clinical onset of ZIKV is thought to be 3 to 12 days after the bite of an infected female mosquito (JIMENEZ et al., 2017).

EPIDEMIOLOGY

It is still unknown how the urban migration of this virus occurs (SLAVOV et al., 2016). Probably during heavy rains, the wild mosquito population can progressively grow and spread the virus to nearby villages and from there to large urban centers, so the urban cycle can occur with human-to-human transmission (SLAVOV et al., 2016).

There are reports that the 2007 epidemic in the Yap Islands in the Federated States of Micronesia was the first major worldwide epidemic of ZIKV disease in humans, and only 14 cases had been previously reported (DUFFY et al., 2009).

There are several hypotheses being investigated to address the clinical severity of ZIKV disease in African and Asian continents, as well as the epidemics found in the Pacific Islands and the Americas that are now presenting severe cases, including neurological disease and significant fetal impairment (BURATTINI, 2017). The reasons suggested, but not yet confirmed, why manifestations are worse in severity in those regions include the genetic variation of viral strains, population immunity modulating the clinical presentation of the ZIKV infection because of wide exposure through decades, co-infection with other arboviruses and failures in the systems of identification and registration of cases. (BURATTINI, 2017).
Although the disease caused by ZIKV is usually self-limiting, there are reports of neurological changes and GBS following an infection by the virus. In adults, severe outcomes were first reported in French Polynesia during the 2013 epidemic. Subsequently an increase in GBS cases has also been reported in Brazil, Colombia, El Salvador, Suriname and Venezuela during the respective ZIKV epidemics (MLAKAR et al., 2016, SHAZ, BLOCH, 2017).

Other neurological complications potentially associated with ZIKV infection include encephalitis, meningoencephalitis, transverse and acute myelitis, auditory and ophthalmologic manifestations (MUSSO, GUBLER, 2016). Idiopathic thrombocytopenic purpura has also been reported, which is characterized by severe thrombocytopenia during or after the course of the infection (DA SILVA et al., 2017).

Laboratory approaches on the detection of ZIKV include molecular, serological and cultural methods. Most diagnostic tests are done by reference laboratories in the country or state (JIMENEZ et al., 2017). The initial diagnosis of the patient is made based on data such as clinical history, travel dates, destinations and activities. During the first week after the onset of symptoms, laboratory diagnosis can be performed using molecular methods such as real-time RT-PCR in blood, urine and/or saliva to detect the virus (JIMENEZ et al., 2017). Although serological tests are widely used - such as ELISA and immunofluorescence, they have a relatively high false positive rate in detecting specific IgG and IgM antibodies in the serum sample. Antibodies may be detectable in serum samples within 5 to 6 days of symptomatic disease, but existing tests have low specificity. Consequently, positive results should be confirmed with virus-specific neutralization tests (JIMENEZ et al., 2017).

The main routine diagnosis of ZIKV infection is the detection of viral nucleic acid through RT-PCR and the detection of IgM antibodies by enzyme-linked immunosorbent assay (IgM-ELISA) (PETERSEN et al., 2016). Therefore, testing of serum samples for RT-PCR obtained within the first week of clinical disease and MAC-ELISA testing of samples that are not tested for RT-PCR or that are considered negative for RT-PCR are considered the most accurate and precise methods of differential diagnosis (PETERSEN et al., 2016). Serology for ZIKV is generally tested through ELISA with a platelet neutralization test (PRNT) according to standard protocols. PRNT is the gold-standard for differentiation of anti-flavivirus antibodies since it is considered the most specific in primary flavivirus differentiation (MUSSO, GUBLER, 2016). However, PRNT is intensely laborious and expensive and is not widely performed (PETERSEN et al., 2016).

ARBOVIRUSES AND BLOOD TRANSFUSION

In all blood samples collected, high sensitivity laboratory tests are performed. Serological methods include: hepatitis B surface antigen (HBsAg), hepatitis B virus core antibody (anti-HBc), hepatitis C
(anti-HCV) antibody, human immunodeficiency virus antibody (anti-HIV), human T lymphotropic virus antibody I / II (anti-HTLV I / II), Venereal Disease Research Laboratory (VDRL) anti-
Trypanosoma cruzi (Chagas disease), while the viral nucleic acid test includes Nucleic Acid Test (NAT) for hepatitis B, C and HIV in order to investigate infectious agents that can be transmitted through blood transfusion pathways (UBIALI, 2015). Up to date, there are no licensed molecular or serological tests for ZIKV screening in blood donors, according to guidelines provided by the Brazilian Association of Hematology, Hemotherapy and Cell Therapy (ABHH) (UBIALI, 2015).

Recent studies have shown that ZIKV can be inactivated in all blood components using Amotosalen combined with ultraviolet A (UVA) in platelets and plasma treated with amustaline (S-303) and glutathione for red blood cells. Both systems use the same mechanism, they react with nucleic acids and modify them to avoid their replication, transcription and translation (LAUGHHUNN et al., 2017). Photochemical treatment using Psoralen (Amotosalen, S-59) in combination with UVA resulted in inactivation of a wide range of viruses, bacteria and protozoa. It is efficient for inactivation of pathogens in platelet and plasma concentrates, but it is not used in red blood cell concentrates, due to poor UVA absorption by the hemoglobin complexes in red blood cells (AUBRY et al., 2016).

The ideal technique for proper diagnosis of viremic samples is through molecular testing for viral RNA detection, but the immediate implementation of these tests for blood donor screening at this time seems unfeasible, not only because of the high cost, but also because ZIKV pathophysiology has not yet been fully elucidated (KASHIMA, SLAVOV, COVAS, 2015, ELLINGSON et al., 2017).

ARBOVIRUS TRANSFUSION RISK

Arboviruses infection prevalence is rising worldwide, representing new risks for patients undergoing blood transfusion therapies (PATY, 2013). The discovery of transfusion transmitted (TT) WNV in the US in 2002 marked a new paradigm, derived from viruses that cause asymptomatic short viremia in infected patients with high incidence of transmissibility, in contrast to some other viruses, such as hepatitis and HIV, where risk of transfusion is only present in patients who have a prolonged carrier state in a population with low and / or unstable incidence of infection (PETERSEN, BUSCH, 2010). Recent discoveries of TT infection by DENV and high prevalence of DENV in blood donations in some countries further justify this concern (PETERSEN, BUSCH, 2010).

The potential for CHIKV TT was demonstrated in the Caribbean during the epidemic in Martinique in 2014, where screening with NAT for CHIKV resulted in four positive plasma samples. Of these donors, two remained asymptomatic and the other two reported febrile syndrome 12 to 24 hours after blood donation (GALLIAN et al., 2014). All arboviruses present a potential risk in TT due to their ability to induce an asymptomatic viremic phase (PATY, 2013). Some examples of arboviruses that produce large epidemics of clinically significant human disease and which are proven or with
potential risks of TT can be seen in table 1 (PETERSEN, BUSCH, 2010).

### Table 1 - Arbovirus-related diseases epidemiological distribution.

<table>
<thead>
<tr>
<th>Organism</th>
<th>Vector</th>
<th>Vertebrate host</th>
<th>Geographical distribution</th>
<th>Syndrome</th>
<th>Transfusion transmission reported</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Togaviridae</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chikungunya</td>
<td>M</td>
<td>Humans, primates</td>
<td>Africa, Asia, Western Pacific</td>
<td>A</td>
<td>No</td>
</tr>
<tr>
<td>Rio Ross</td>
<td>M</td>
<td>Marsupials</td>
<td>Australia</td>
<td>A</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Flaviviridae</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dengue 1-4</td>
<td>M</td>
<td>Humans</td>
<td>Worldwide on the tropics</td>
<td>FH</td>
<td>Yes</td>
</tr>
<tr>
<td>Yellow fever</td>
<td>M</td>
<td>Humans, primates</td>
<td>Africa, South America</td>
<td>FH</td>
<td>Yes</td>
</tr>
<tr>
<td>Japanese encephalitis</td>
<td>M</td>
<td>Birds, swine</td>
<td>Asia</td>
<td>E</td>
<td>No</td>
</tr>
<tr>
<td>St. Louis encephalitis</td>
<td>M</td>
<td>Birds</td>
<td>Americas</td>
<td>E</td>
<td>Yes</td>
</tr>
<tr>
<td>WNL encephalitis</td>
<td>M</td>
<td>Birds</td>
<td>Asia, Africa, Americas, Europa</td>
<td>E</td>
<td>Yes</td>
</tr>
<tr>
<td>Tick-borne encephalitis (TBE)</td>
<td>T</td>
<td>Rodents</td>
<td>Europa, Asia</td>
<td>E</td>
<td>Yes</td>
</tr>
<tr>
<td>Zika</td>
<td>M</td>
<td>Humans, primates</td>
<td>Africa, Asia, Americas, Caribbean, Pacific</td>
<td>CZS, GBS</td>
<td>E, A</td>
</tr>
<tr>
<td><strong>Bunyaviridae</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rift Valley fever</td>
<td>M</td>
<td>Domesticated ungulates, rodents</td>
<td>Africa</td>
<td>FH, E</td>
<td>No</td>
</tr>
</tbody>
</table>

Note: M, mosquito; T, tick; E, encephalitis; HF, Hemorrhagic Fever; A, Arthralgia; CZS, Congenital Zika Syndrome; GBS, Guillain-Barré Syndrome.

### TRANSFUSION TRANSMITTED (TT) DENGUE AND ZIKA VIRUS

Screening for DENV has not been implemented because of the low rate of transfusion-transmitted dengue (TTD), although other interventions, including postponing short-term trips even to non-endemic tropical areas and pathogen inactivation technologies, have been largely carried out in endemic areas (MATOS et al., 2016). Screening for DENV in blood donation has been done experimentally in endemic areas, presenting frequencies greater than 1:500 donors positive for DENV RNA, but blood donation screening has not been adopted as a standard protocol (MATOS et al., 2016).

Brazil has one of the largest number of cases of DENV infection and, although there is currently no implementation of donor screening for this arbovirus, it is known that effective preventive measures ought to be adopted by medical entities (LEVI et al., 2015). For proper transfusion safety, the methods targeting DENV RNA (NAT) due to the short viremic phase preceding seroconversion should be performed, as detection of NS1 protein in the blood of suspected cases is a common phenomenon in endemic areas (MATOS et al., 2016). However, a marked decrease in the sensitivity of the tests in analyzed populations has been suggested in recent data, making it an unacceptable option for blood screening, a process that demands maximum sensitivity (MATOS et al., 2016). In contrast,
NAT for DENV was previously implemented in Puerto Rico, following the diagnosis of the hemorrhagic form of the disease occurring in a dialysis patient (LEVI et al., 2015).

The exact impact of ZIKV in blood supplies is uncertain. Current evidence suggests that TT by ZIKV is likely, but clinical penetrance has not been established. Although not unique to ZIKV, measures that are available to protect blood supply include donor selection, blood component quarantine, laboratory screening and / or the use of PRT (JIMENEZ et al., 2017. The application of these measures differs depending on whether they are located in an endemic area versus a non-endemic area (JIMENEZ et al., 2017). In table 2, it is possible to compare the cases of DENV and ZIKV.

### Table 2 - Cases of DENV and ZIKV.

<table>
<thead>
<tr>
<th>Organism</th>
<th>Tt sample</th>
<th>Diagnosis</th>
<th>Number of infected receptors</th>
<th>Geographical distribution</th>
<th>Year</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>dENV-1</td>
<td>Red blood cells and fresh plasma</td>
<td>RT-PCR</td>
<td>6</td>
<td>Hong Kong</td>
<td>2002</td>
<td>CHUANG et al., 2008</td>
</tr>
<tr>
<td>DENV-2</td>
<td>Red blood cells and fresh plasma</td>
<td>RT-PCR</td>
<td>2</td>
<td>Puerto Rico and Singapore</td>
<td>2007</td>
<td>TAMBYAH et al., 2008</td>
</tr>
<tr>
<td>DENV-4</td>
<td>Red blood cells, platelets and fresh plasma</td>
<td>TMA</td>
<td>6</td>
<td>Recife and Rio de Janeiro</td>
<td>2012</td>
<td>LEVI et al., 2015; SABINO et al., 2016</td>
</tr>
<tr>
<td>ZIKV</td>
<td>Red blood cells and platelets</td>
<td>RT-PCR and cell culture</td>
<td>4</td>
<td>Brazil</td>
<td>2016</td>
<td>JIMENEZ et al., 2017</td>
</tr>
</tbody>
</table>

Note: DENV 1, 2 and 4, Dengue virus 1, 2 and 4; ZIKV, Zika virus; RT-PCR, Real Time-Polimerase Chain Reaction; TMA, Transcription Mediated Amplification.

**CONCLUSIONS**

ZIKV infection is a serious, worldwide health issue. The possibility of viremic but asymptomatic patients being blood donors represents a serious concern for medical entities responsible for dialysis centers, since ZIKV and others arboviruses’ transmission is possible under those circumstances. Uncertainty still remains as to the particular/specific types of receptors susceptible to infection in a clinical exposure. However, recent discoveries suggest relative low-risk for health professionals.

Prevalence studies should be performed in both endemic and non-endemic regions in order to define infectivity through blood components and determine their ability to generate disease in the recipient, especially those at high risk, such as pregnant women and their children.

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