Cardiovascular Involvement in the Zika Virus Infection

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Abstract

The evidences highlight that 13.5% of the echocardiographic studies performed in children with congenital Zika virus infection presented amendments of congenital heart disease (CHD). Several recent studies have shown that the pathogenesis of neurological complications of the Zika virus, as Guillain-Barre Syndrome, in relation to other clinical manifestations there are still no conclusive findings. Scientific studies have observed the possibility of cardiac involvement be underdiagnosed due to the mercy of the infection per ZIKV, in their majority.

The Zika virus (ZIKV) was identified in the Zika forest of Uganda for the first time in 1947 by means of insulation in the serum of Macaque rhesus; however its first outbreak was documented in 2007, expanding since then its broadcast area (Chan et al., 2016). The WHO (2017), in his last situational report, published at the end of the first quarter of 2017, highlights the transmission of ZIKV in 84 countries, and keeps a global risk assessment, remaining with a high level of vigilance, even with the reduction of notification of cases in some regions. This fact is confirmed by the most recent data from the Ministry of Health of Brazil (2017) through epidemiological bulletin. In 2016, there were 215319 probable cases of fever by the virus and eight laboratory confirmed deaths, already in 2017, 9351 probable cases were identified, and absence of deaths with laboratory confirmation.

Several recent studies have shown that the pathogenesis of neurological complications of ZIKV (Chan et al., 2016), as Guillain-Barre Syndrome (Cao-Lormeau et al., 2016), but in relation to other clinical manifestations there is still no conclusive findings (Chan et al., 2016).
As the infection by the Zika virus has similarities with the prenatal infection of congenital rubella virus other organs can be affected (Di Cavalcanti et al., 2017). There is a large tropism of ZIKV to the various tissues in the human being, probably because of the wide distribution of receptors (AXL, TYRO3 e DC-SIGN) to the Zika virus in different organs (Chan et al., 2016).

One of the affected organs, in potential, by this infection is the heart. However, there are few studies showing the cardiac alterations associated to ZIKV. To evaluate possible echocardiographic findings in infants with suspected congenital Zika syndrome, Di Cavalcanti et al. (2017) conducted a retrospective study. Furthermore, it has been observed myocarditis in mice inoculated with ZIKV and the presence of viral RNA in the cardiac muscle of mice infected by ZIKV (Chan et al., 2016).

Aletti et al. (2017) documented the first case of cardiac complication associated with the Zika virus infection in humans, characterizing as transitory myocarditis associated to arthralgia, myalgia, headache and fever, this framework compatible with arboviruses. The patient presented chest pain of moderate intensity, having confirmed the diagnosis of cardiac involvement by the increase of creatinine phosphokinase (CPK), troponin and repolarization changes in ECG, associated with serological confirmation of infection by ZIKV.

In the same way, Carta et al. (2017), recent evidence, detected the presence of arrhythmias in patients with cardiac symptoms from endemic area. The main manifestations at the electrocardiogram (ECG) were: atrial fibrillation, ventricular arrhythmias, non-sustained atrial tachycardia. To the study with Doppler echocardiography, the majority presented heart failure with reduced ejection fraction.

The findings of Zonneveld et al. (2016), in his series of cases, are compatible with the hypothesis of the ZIKV be causing acute myocarditis in patients. There were observed in critical patients evaluated with unfavorable outcome, due to death, only elevation of CPK and of its CK-MB fraction, without electrocardiographic changes suggesting acute myocardial infarction.

Di Cavalcanti et al. (2017) observed that 13.5% of the echocardiographic studies performed in children with infection by congenital ZIKV presented amendments of congenital heart disease (CHD); however, only one individual presented some clinical signs compatible with the cardiac (dyspnea). The abnormalities found were: atrial septum defects (35%), defects in the apical vsd hemodynamically insignificant (57%) and only one case with large membranous ventricular septal defect. These findings show a three times higher incidence of CHD in patients with congenital ZIKV in relation to the general population with less than 1 year of age.

Despite these findings, Sarno et al. (2016), in the case study of fetal death in mothers with positive epidemiology for infection by ZIKV and fetal ultrasound evidencing commitment of cephalic perimeter and fetal hydrops, found ZIKV RNA only in samples of the central nervous system and amniotic liquid, excluding the presence of virus in cardiac tissue. However, due to the sample size, it should be stressed that findings cannot be taken.

Due to the lack of reports and to the possibility of death due to cardiac involvement, Zonneveld et al. (2016) describes this presentation as “atypical”. Krittanaewong et al (2017) raises the possibility of cardiac involvement being underdiagnosed due to the mercy of the infection per ZIKV, in their majority.

In this way, the arboviruses caused by the Zika virus must be discussed in patients with cardiovascular complaints, in the context of endemic areas and with positive epidemiology for disease. In addition, the attending physician in the case of heart disease induced by ZIKV must request at least a screening electrocardiogram and troponin I and CPK (Aletti et al, 2017).
References