Clinical Infectious Diseases

BRIEF REPORT

Transient Myocarditis Associated With Acute Zika Virus Infection

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Zika virus outbreak is spreading in the Americas. This emerging infection is associated with neurological complication. We report the first travel-acquired Zika acute infection complicated with myocarditis imported to mainland France. We recommend an electrocardiogram and troponin if any cardiac symptoms are present in a patient with acute Zika infection.

Keywords. Zika virus; myocarditis; imported viral disease; reemerging disease; sentinel surveillance.

A 45-year-old healthy outpatient presented with a fever that had started 5 days previously; he had diffuse joint pain, myalgia, headache, and diarrhea. He had been in the French West Indies (La Martinique) from 21 December 2015 to 1 January 2016. On his return he complained of a recent mild (assessed as 4/10 using a pain assessment tool) throbbing, squeezing mediothoracic pain without any spread to other parts of his body and no dyspnea.

Initial examination revealed a body temperature of 39°C, bilateral conjunctivitis, and hand edema but no skin rash. His blood pressure was normal, heart rate was 95 bpm; there was no sign of cardiac failure, and auscultation revealed no abnormal heart sounds.

Myocarditis was diagnosed, based on an ST-segment elevation in the anteroseptal region on electrocardiogram associated with an increase in the troponin I level (0.49 µg/L, normal <0.04 µg /L) and creatine phosphokinase (CPK) level (213 UI/L, normal <200 UI/L). The echocardiogram estimated the left ventricular ejection fraction at 55% and showed an inferior medial wall hypokinesia. The 24-hour electrocardiogram monitoring was normal.

Treatment with bisoprolol and ramipril was introduced. We observed a quick normalization of troponin and CPK levels and disappearance of chest pain and electrocardiographic changes. Fever and conjunctivitis were also resolved after 3 days of symptomatic treatment with paracetamol. Ten days later, cardiac magnetic resonance imaging showed a slight left ventricular dilatation (left ventricular telediastolic volume: 123 mL/m^2, normal range 41–81 mL/m^2; left ventricular telesystolic volume: 48 mL/m^2, normal range 11.9–20.74 mL/m^2; long axis: 62 mm, normal <56 mm; short axis: 58 mm, normal < 50 mm); there was no abnormality in myocardial tissue perfusion, no hypokinesia, and no edema.

The blood samples tested 5 days after the onset of symptoms came back positive for Zika virus (real-time polymerase reaction [RT-PCR]; RealStar Zika Virus RT-PCR Kit 1.0; Altona Diagnostics GmbH, Hamburg, Germany). Zika virus genome was detected at 4.0×10^3 RNA copies/mL (with reference to the European Viral Archive Zika Standard 1). IgM antibody capture-enzyme-linked immunosorbent assay (MAC-ELISA) and indirect ELISA performed by the French National Reference Center for arboviruses made it possible to detect anti-Zika immunoglobulin (Ig)M antibodies and anti-flaviviruses IgG antibodies. Other infections that could have been responsible for myopericarditis were biologically ruled out because RT-PCR (dengue, chikungunya, parvovirus B19, coxsackievirus, adenovirus, human immunodeficiency virus [HIV]) or serology (HIV, chikungunya) were negative or inconsistent with a recent infection (anti-dengue IgM antibodies negative and anti-dengue IgG antibodies positive).

DISCUSSION

Zika virus is transmitted by *Aedes* sp. mosquitoes. Since May 2015, Zika virus infection has spread from Brazil to other countries in South and Central America and in the Caribbean, including the French West Indies (La Martinique and Guadeloupe) [1]. Zika virus has been spreading rapidly on the American continent in recent months because of poor clinical manifestation in main cases and the intense proliferation of mosquitoes in tropical areas. Occasionally, Zika virus infection induces some neurological complications consistent with a selective neurotropism such as Guillain-Barré syndrome [2], acute myelitis [3], meningoencephalitis [4], and fetal irreversible cerebral complications such as microcephaly [5].

This case was unusual because the Zika virus–infected patient developed a symptomatic nonsevere and resolutive myocarditis within the first 2 weeks of disease onset. The most likely diagnosis of Zika virus–induced myocarditis relies on the following criteria: clinical, biological, and morphological evidence of myocarditis; and the evidence of Zika virus attested by blood tests; and no biological evidence for another simultaneous infection.

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As often occurs in rapidly resolutive virus-induced myocarditis, the evidence of Zika virus in myocardial tissue was not possible because an endomyocardial biopsy was considered too dangerous for the patient.

To our knowledge, this is the first report of a cardiac complication linked to Zika virus. Even so, a recent nonhuman primate model demonstrated Zika virus could target various organs, including the heart, early in the process; RNA Zika virus was detected in heart tissue in the 5 days post-infection [6]. The onset of acute myocarditis has been reported for the 2 main arboviral diseases, chikungunya and dengue [7], with the same 2 issues: the risk of a lack of diagnosis due to mild myocarditis, as the chest pain may be mistaken for other multiple pains commonly present in these diseases, and concerns about the long-term outcome with a possible risk for late onset of a dilated myocardiopathy [8], as described for other viral infections that therefore require a cardiac follow-up.

Zika virus disease should be discussed in patients with chest pain or confirmed acute myocarditis living in or returning from Zika-endemic areas. In addition, Zika-infected patients who present with any cardiac symptoms should have an electrocardiogram and receive dosages of enzymes (troponin I and CPK).

Note

Potential conflicts of interest. All authors: No reported conflicts. All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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