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Acute Anteroseptal ST-Elevation Myocardial Infarction (STEMI) in the West Nile Virus Infection

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Purpose: To present a case of acute anteroseptal ST-elevation myocardial infarction in the West Nile virus (WNV) meningoencephalitis.

Methods & Materials: A 77-year-old patient was hospitalized in the late summer of 2017 on the second day of the illness manifested by a fever up to 38.6°C and diarrhea. The patient did not report recent travel but recalled mosquito bites. Past medical history included hypertension. At admission, routine laboratory tests, electrocardiogram (ECG) and chest x-ray were performed. In addition, cerebrospinal fluid (CSF), urine and blood samples were collected for a virological analysis.

Results: At admission, WBC count was 24.6 (reference range 3.4-9.7x10(9)/L) with neutrophilia (92%, range 44-72%) and very high levels of cardiac enzymes: creatinine phosphokinase 1856 (range 17-153 U/l), lactate dehydrogenase 433 (range 2-241 U/L), myoglobin 3116 (range 20-80 ug/L) and troponin I 17.640 (range 0.000-0.056 ug/L). ECG showed ST elevation. In the cardiac intensive care unit, an emergency coronary angiography was performed which confirmed the coronary artery stenosis. The patient's condition complicated on the 4th day of the illness by an altered level of consciousness with progression to coma, accompanied by neck stiffness and positive meningeal signs. Computed tomography of the brain was normal. Cerebrospinal fluid (CSF) showed pleocytosis with 26 cells/mm3, predominantly mononuclears (73%) and elevated protein level (1.151, range 0.170-0.370 g/L). Both CSF and urine were positive for WNV RNA by real-time and nested RT-PCR. Phylogenetic analysis showed WNV lineage 2. The patient was initially treated with acyclovir, ampicillin and cefepime parenterally with supportive therapy (antiedematous, antiaggregation and antihypertensive therapy). On the 8th day of the illness a respiratory insufficiency developed. The patient was intubated and mechanically ventilated, but developed hypotension and low oxygen saturation in spite of an adequate respiratory support. Despite the cardiopulmonary resuscitation, the patient died due to cardiopulmonary arrest.

Conclusion: Although cardiac involvement is not frequently reported in the course of a WNV infection, physicians should be aware of the possibility of a WNV-related myocardial infection.