Electrical storm induced by H1N1 A influenza infection

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Pandemic H1N1 influenza A virus is spreading worldwide. We report a case of electrical storm during H1N1 influenza A infection in a patient with congenital long-QT syndrome (LQTS) that was previously stable on β-blocker therapy. Possible causes for the association of A flu and LQTS recurrent ventricular arrhythmias are discussed.

Figure 1 Implantable cardioverter-defibrillator (ICD) log and electrogram. Implantable cardioverter-defibrillator holter (A) revealed that there was a remarkable increase in number of ventricular fibrillation (VF) appropriate detections on 3 November 2009 that resulted in 19 appropriate ICD therapies (13 shocks and 6 antitachycardia pacing). Ventricular fibrillation detection (B) showing that post-extrasystolic pause preceded arrhythmia onset. An example of ventricular fibrillation detection electrograms (C) and delivery of a successful 40 J defibrillation shock.
A new strain of H1N1 influenza A virus infecting human was identified in April 2009 and is spreading worldwide. As more people are being infected, new issues evolve. We report the case of a patient with congenital long-QT syndrome (LQTS) carrying an implantable cardioverter-defibrillator (ICD) that was stable on β-blocker therapy but developed an electrical storm in the course of H1N1 A flu.

A 22-year-old black woman, without structural heart disease, was hospitalized for recurrent syncope of unknown etiology, triggered by loud sounds and strong emotions. There was family history of sudden death in relatives, ECG showed a QTc interval of 482 ms, and symptomatic episodes of torsades de pointes were documented. A single-chamber ICD was implanted after recurrent symptomatic torsades occurred during propranolol uptitration to 40 mg t.i.d.

She remained asymptomatic for 1-month until she developed non-productive cough, fatigue, headache, and chills. After 2 days of flu-like symptoms, she felt rapid palpitations followed by 13 ICD shocks in a 5 h period. Implantable cardioverter-defibrillator interrogation (Figure 1) revealed that there were several appropriate detections in ventricular fibrillation zone that resulted in 19 ICD therapies. She reported full compliance to ambulatory therapy and did not take any QT-prolonging drugs, including antibiotics and antiviral agents. Termination of the electrical storm was successful by perfusing magnesium sulfate, increasing the resting pacing rate from 40 to 85 bpm, and increasing propranolol dosage. An ECG was obtained soon after initial therapy and showed QTc interval of 462 ms, which is within usual values for the patient. Blood chemistry at admission was unremarkable, including normal potassium, magnesium, and calcium serum concentrations. H1N1 influenza real-time PCR in throat swab was positive. There was no evidence of lower respiratory tract infection as assessed by examination, chest X-ray, and arterial blood gases.

Possible causes for the association of A flu and LQTS recurrent ventricular arrhythmias include fever, influenza myocarditis, or the effect of chance. Stress caused by shocks has also been described as a trigger for recurrent arrhythmias.

Fever is an established trigger of cardiac events in Brugada syndrome, but there are only anecdotal reports of fever-induced arrhythmias in LQT2 patients. Our patient’s clinical history suggests the diagnosis of LQT2 but the results of genetic testing are not yet available. Myocarditis has been observed in association with influenza in pandemics and interpandemic periods. However, arguing against myocarditis in this case is the lack of other accompanying symptoms, negative biochemical markers of myocardial necrosis and normal echocardiographic features.

Medical community should be made aware of the potential effect of H1N1 influenza infection in triggering ventricular arrhythmia in LQTS. This may have a great impact as ventricular arrhythmias are a cause of sudden death in LQTS and not all LQTS patients carry ICDs. Public health monitoring should explore this issue further.

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References