

Ventricular tachycardia storm management in a Covid-19 patient. The potential SARS-CoV-2 triggering rule.

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Abstract

We report the management of an electrical storm in a 68-years-old patient with previous history of coronary artery disease and severe left ventricular systolic dysfunction, affected by mild-symptomatic coronavirus disease 2019 (Covid-19) pneumonia. Covid-19 infection triggered a ventricular tachycardia storm requiring urgent catheter ablation procedure, in a patient who had never experienced sustained ventricular arrhythmias before. This case suggests the potential role of Covid-19 infection in precipitating life-threatening ventricular arrhythmias in patients with pre-existing cardiovascular diseases.

Introduction:

By December 2019, coronavirus disease 2019 (Covid-19) due to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, has rapidly spread all over the world, becoming in early March 2020 a global pandemic.

The predominant clinical course of SARS-CoV-2 infection is mostly characterized by respiratory symptoms, such as sore throat, cough, dyspnea, as well as fever and weakness, that can lead to pneumonia and severe acute respiratory distress syndrome. [1]

Additionally, Covid-19 infection has been associated with myocardial involvement and injury. [2-4]

Therefore, despite not being yet described, a potential increased risk of malignant ventricular arrhythmias exists.

We hereby present the case of an electrical storm in the setting of a SARS-CoV-2 infection.

Case Presentation:

A 68-year-old Caucasian woman presented to our emergency department at the end of February 2020, referring persistent fever unresponsive to oral antibiotic and antipyretic therapy for the past 6 days, associated with cough, shortness of breath and transient dizziness episodes.

Patient's medical history revealed a prior inferolateral ST elevation myocardial infarction (MI) in 2014. For the persistence of severe left ventricular dysfunction, she was implanted three months later with a single-chamber implantable cardioverter defibrillator (ICD) in primary prevention of sudden cardiac death.

Subsequent follow-up was uneventful, and she never experienced any device intervention.

At the time of hospital admission, most laboratory tests were within normal range, except for increased high-sensitivity C-reactive protein (C-PR) values (27.6 mg/L). Arterial blood gas analysis revealed oxygen saturation of 94% and oxygen partial pressure of 67 mm Hg. Two polymerase chain reaction assays of the

oropharyngeal swab sample were negative for the SARS-CoV-2 nucleic acid. Chest radiograph showed a pulmonary consolidation area in left inferior lung lobe, suggestive for pneumonia (Figure 1, A-B).

Non-invasive ventilation and intravenous empirical antibiotic therapy were attempted with clinical improvement within few days.

During the eighth day of hospital stay, the patient had multiple episodes of sustained ventricular tachycardia (VT)/ventricular fibrillation (VF) treated with ICD shocks (Figure 1, C). At device interrogation, frequent mild-symptomatic ventricular arrhythmia episodes were detected during the past 20 days, self-terminating or successfully interrupted with antitachycardia pacing (ATP). Electrical storm (12 episodes over 24 h) persisted despite intravenous infusion of beta blockers, amiodarone and deep sedation.

Repeated blood test showed C-RP reduction (16.8 mg/dL) while high-sensitivity troponin T level remained negative (< 15 ng/L). Chest radiograph control showed a progressive resolution of pneumonia findings.

Transthoracic echocardiogram confirmed the previous findings of dyskinesia of the inferior wall and aneurysm of the mid and apical parts, with reduced left ventricular ejection fraction of 37%.

Catheter ablation was performed the day after, with the patient afebrile in the previous 72 hours.

Left ventricle was first mapped using transseptal approach with a Pentaray catheter and electroanatomic CARTO3 mapping system (Biosense Webster, Inc., CA, USA). Voltage map showed a large post-MI scar zone in inferolateral LV wall with local abnormal ventricular activity signals (Figure 2).

Scar homogenization was performed with a 3.5 mm tip magnetic irrigated radiofrequency catheter (Navistar[®] RMT Thermocool, Biosense Webster) with a maximum power of 55 W in concert with a remote magnetic navigation system (NiobeTM, Stereotaxis, Inc., St. Louis, MO, USA).

At the end of ablation procedure, non-inducibility of clinical VT by programmed ventricular stimulation was confirmed.

During the following hospital stay, continuous ECG monitoring did not show further VT events and the patient remained afebrile, with resolution of pneumonia. Despite clinical improvement, nasopharyngeal swab was repeated before home discharge with a positive result for SARS-CoV-2 and this case was finally diagnosed with Covid-19 pneumonia.

Discussion:

The spectrum of clinical manifestations in Covid-19 is highly variable, potentially causing also asymptomatic or only mildly symptomatic presentation in a large number of cases, and laboratory detection of the viral nucleic acid prone to false-negative results. [5]

To date, this type of presentation with non-severe pneumonia, resolved without any specific antiviral therapy, and concurrent VT storm in a Covid-19 patient has never been reported.

It is known that patients with pre-existing cardiovascular disease have worse outcomes; some authors suggest that Covid-19 infection might be associated with a direct myocardial injury triggering life-threatening ventricular arrhythmias. [6]

Non-specific palpitations were reported as part of the presenting symptomology in 7.3% of patients admitted for Covid-19 disease [2], while cardiac arrhythmias were noted in 44.4% of severely ill hospitalized Covid-19 patients [3], although no specific data regarding the type of arrhythmia was reported.

For the first time we present the case of a patient with VT storm occurring during an atypical pneumonia later proved to be Covid-19.

Despite the important history of pre-existing cardiovascular disease, the patient had never experienced sustained ventricular arrhythmia documented by ICD interrogation, suggesting the role played by SARS-CoV-2 infection as potential arrhythmogenic trigger.

Mechanisms of cardiac arrhythmias in Covid-19 patients may be multifactorial and have not been completely understood yet. Cytokine storm triggered by an imbalanced type 1 and type 2 T helper cells [7], as well as hypoxia-induced intracellular calcium overload leading to early afterdepolarization, may represents a major trigger of reentrant VTs in these patients. [8, 9]

The present report confirms the complexity of the Covid-19 clinical profile and in case of ventricular storm might be helpful for the management of similar situations.

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Figure 1. Chest radiograph with left pulmonary consolidation area (arrow) in left inferior lung lobe. Anterior-posterior view (A) and lateral view (B). Electrogram with sustained ventricular tachycardia interrupted by DC-shock (C).

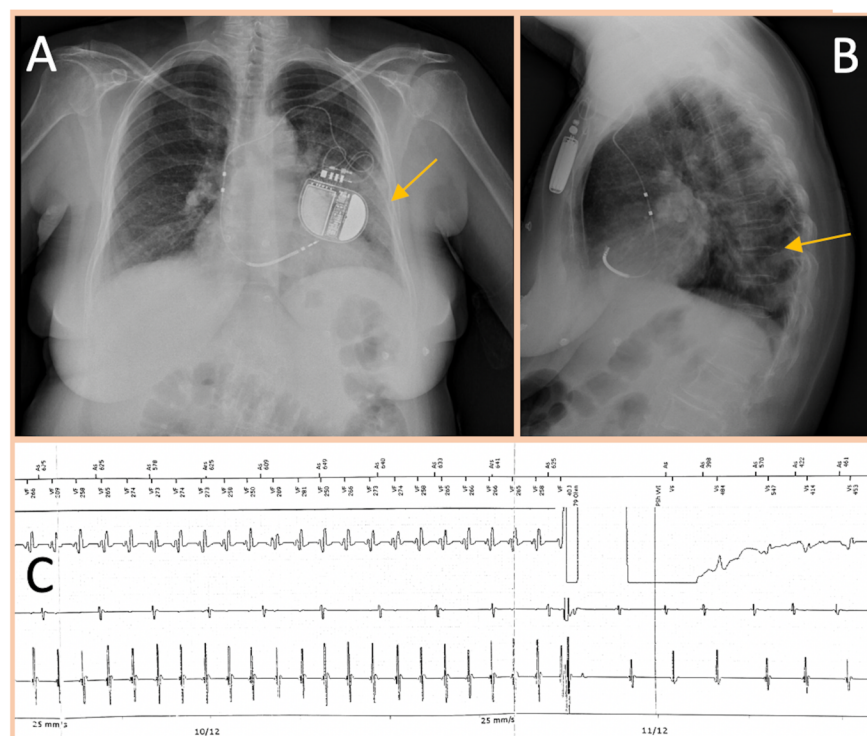


Figure 2. Left anterior oblique (LAO) fluoroscopic view during radiofrequency catheter ablation (A). Non-sustained ventricular tachycardia episode with clinical morphology during catheter ablation procedure (B). 3D-voltage map with low voltage scar (<1.5 mV) in posterolateral wall of the left ventricle (C). Red dots (VisiTag) represent radiofrequency pulses.

