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Case Report

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COVID-19 Revealing Brugada Syndrome

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Introduction

SARS-cov-2, responsible of COVID-19, is a novel coronavirus which first appeared in Wuhan City (China) in December 2019. The epidemic propagated wildly fast in China first, then to the rest of the world. It is still spreading geographically, contaminating over 5 million people worldwide and is responsible of more than 350000 deaths as of today.
A few infected individuals were detected by showing cardiac symptoms such as palpitations or chest pain, rather than respiratory symptoms.

The association of Brugada syndrome and COVID-19 is rarely described. We report a case of a patient who presented this association, emphasizing the difficulties of the pharmacological treatment.

Keywords: 2019-nCoV, intensive care, brugada syndrome.

Case report

42-year-old female, no particular medical history (cardiovascular nor familiar sudden death), consulting at the emergency room for a five days dyspnea, associated with left sub sternal chest pain. The patient had a feverish sensation but had no temperature measurement was taken.

She was admitted in a COVID19 dedicated medical structure where a PCR test was realized and found positive. Following a respiratory aggravation, the patient was transferred to the intensive care unit.

During her admission, the patient presented fever (39.5°C), tachycardia (101 bmp) and tachypnea (22 cycles/min). SpO2 levels 82% at breathing room air.

Electronic panel test was taken revealing a mild hypocalcemia (8.3 mmol/L), normal Sodium (136 mmol/L) and Potassium (4.0 mmol/L) levels. CRP was 182.7 mg/L and BNP 20 pg/mL. other laboratory tests were normal, including troponins and hemocultures.

Bedside chest X-ray showed bilateral multifocal interstitial opacities and a normal cardiac silhouette. Chest CT scan showed ground glass opacities extended at 50% of the parenchyma in favor of viral pneumopathy.

EKG revealed ST-segment elevation at right precordial leads V1 and V2 (figure 1) and left bundle branch hemi block. Echocardiography showed a mild anteroseptal hypokinesis, with preserved ejection fraction of 55%.

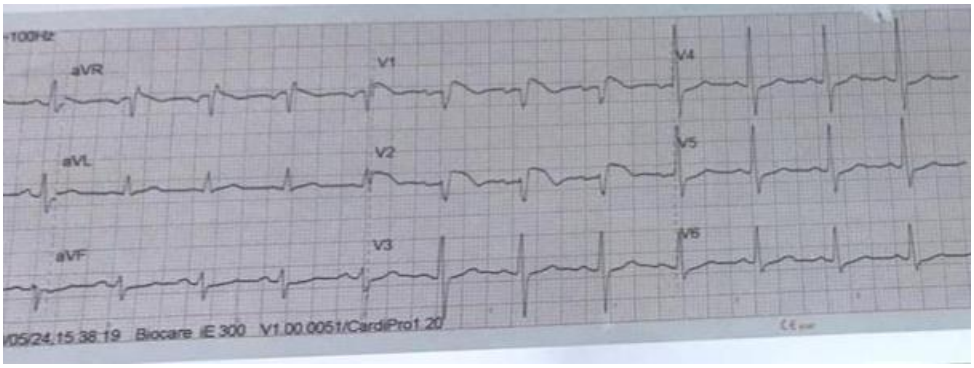

Figure 1: EKG showing a type-1 Brugada pattern.

Therapeutic care was based on antipyretics (15mg/kg) the first days, bi-antibiotherapy (ceftriaxon 2g/day + Moxifloxacin 400mg x2/day), anti-coagulation (0.4UIx3), and oxygenotherapy (high concentration mask; 9L/min). We could not administer the association Hydroxychloroquin + Azythromycin in the presence of preexistent repolarization troubles.

The patient had a favorable outcome with minimum oxygen aid to maintain a good O2 arterial saturation. Temperature dropped. Daily troponin tests were negative and CRP decreased to 100.4mg/L. ST-segment elevation diminished in the anteroseptal territory, which is in favor of Brugada syndrome. (Figure 2). The patient is to benefit of an automatic defibrillator after showing negative PCR tests.

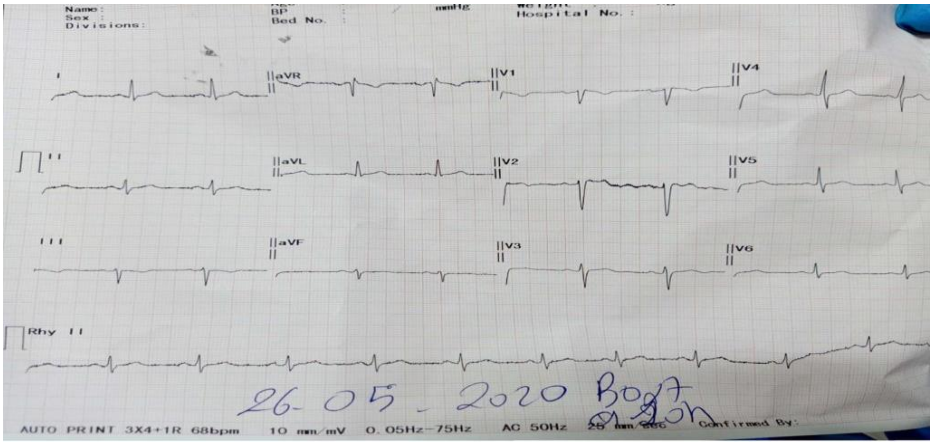

Figure 2: Decrease of ST-segment elevation in right precordial leads.

Comment

Brugada syndrome is a familial arrhythmia disorder characterized with a pattern of type 1 Brugada in right precordial leads (coved ST-segment elevation and inverted T-wave in V1 -V2 leads) and high risk of ventricular fibrillation and sudden death. [2].

A connection between fever and type 1 Brugada is well-known and widely reported. [3]. As an elevated body temperature is responsible of inactivating sodium channels, which is more significant in patients with mutated sodium channels. The decrease of Sodium flow can lead to dangerous transmural heterogeneity leading to ventricular arrhythmias that can be fatal. [4].

It could be interesting to find out whether the virus itself interacts with myocadiac ionic channels and provoke electric changes that are typical in Brugada syndrome.

It is important to be prudent while dealing with COVID19 therapy molecules during this pandemic, as they can lead to undesirable cardiovascular effects. [5].

Conclusion

Patients presenting with Brugada syndrome and COVID19 infections must be carefully monitored in an intensive care unit until no fever is detected, regardless of their respiratory status. This justifies a strict use of antipyretics. Considering the use of implantable defibrillator is highly encouraged.

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