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Case Report

Brugada pattern unmasked during COVID-19 infection -:A case report

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ABSTRACT

The current pandemic caused by the coronavirus disease 2019 (COVID-19) continues affecting millions of people worldwide. Various cardiovascular manifestations have been associated with COVID-19 but only a few case reports of Brugada syndrome in acute respiratory syndrome by SARS-CoV-2 were published. The diagnosis, prognosis, and treatment remain a challenge and represent a concern in terms of management in this population. We describe a case of a 66-year-old patient with COVID-19 presenting a coved type-1 Brugada pattern in electrocardiogram. Drug challenge was performed for the diagnosis of Brugada syndrome and electrophysiological study for risk stratification.

<**Learning objective:** Brugada pattern can be unmasked during coronavirus disease 2019 (COVID-19) infection, regardless of fever. Supportive care and avoidance of drugs that may induce Brugada pattern and ventricular arrhythmias can prevent clinical complications in Brugada patients with COVID-19. In asymptomatic patients with suspected Brugada syndrome and a family history of sudden cardiac death, the correct diagnosis and risk stratification may be performed.>

Introduction

In 1992, Brugada et al. [1] published a case series of an arrhythmogenic disorder that later was named Brugada syndrome (BrS). This syndrome is characterized by ST-segment elevation in the right precordial leads and is associated with increased risk of ventricular fibrillation (VF) in the absence of a structural heart disease. Since the first publication, BrS has been extensively studied and efforts have been made to understand many questions and uncertainties about the diagnosis, risk assessment, and management [1,2].

The current pandemic caused by the coronavirus disease 2019 (COVID-19) continues affecting millions of people worldwide. Data suggest various cardiovascular manifestations associated with the infection, but only a few case reports of inherited arrhythmia syndromes associated with severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) were published [3].

We describe a case of Brugada pattern (BP) on electrocardiogram (ECG) unmasked during COVID-19 infection in a patient without previous diagnosis of BrS.

Case report

A 66-year-old male without known comorbidities presented to the hospital following a 4-day history of cough, sneezing, and anosmia. On physical examination, he was afebrile (96.98 °F or 36.1 °C), blood pressure was 131/88 mmHg, heart rate 81 beats/min, respiratory rate 18 breaths/min, and SaO2 95% on room air. Pulmonary auscultation revealed normal respiratory sounds.

Computed tomography (CT) imaging of the chest revealed bilateral lung involvement with typical-ground-glass opacities. The nasopharyngeal real-time reverse transcriptase-polymerase chain reaction test for SARS-CoV-2 was positive. Initial laboratory tests including blood count, electrolyte levels, metabolic panel, troponin, D-dimer, brain natriuretic peptide, and other inflammatory markers were normal.

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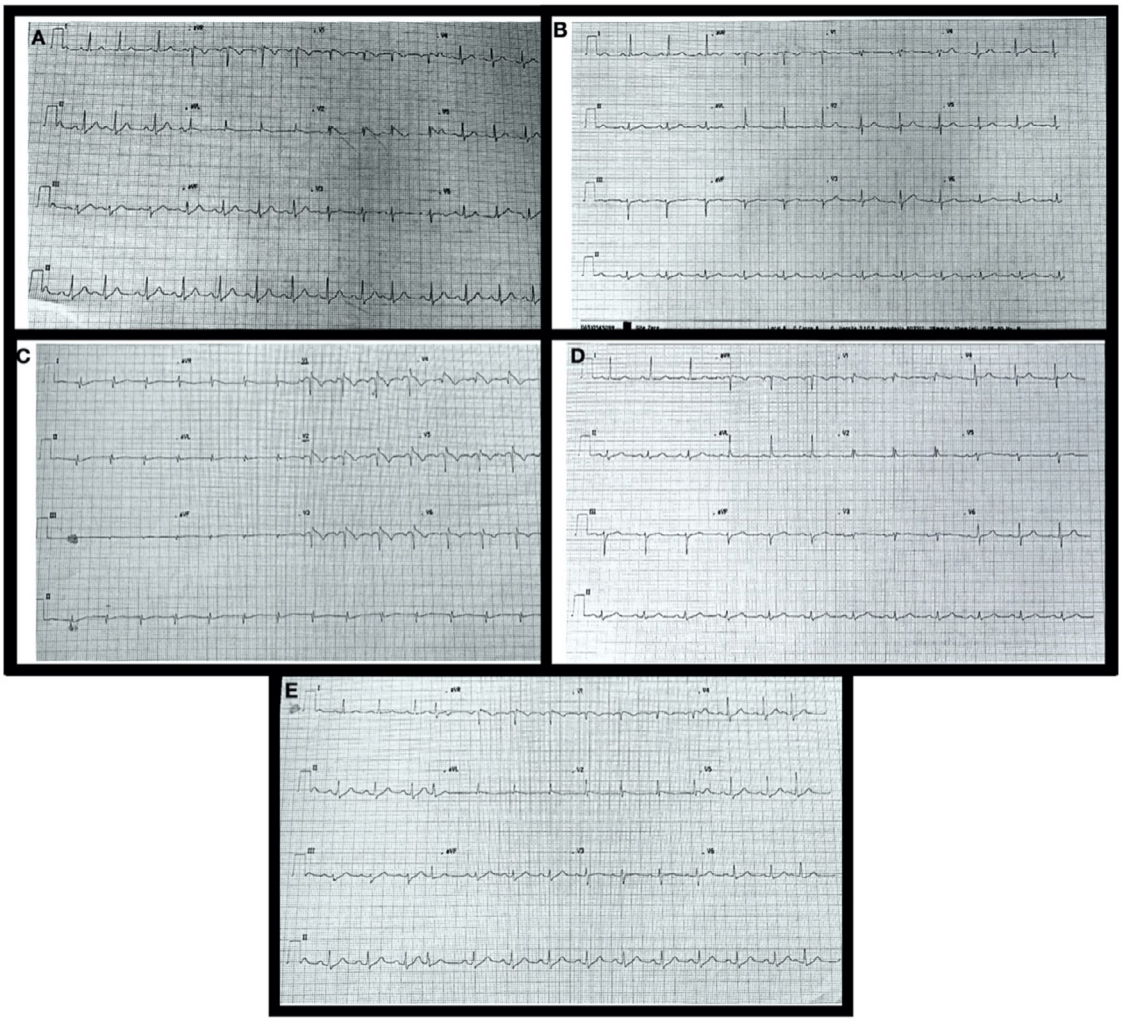

Fig. 1. (A) Type-1 BP on ECG. (B) ECG before admission (C) Type-1 BP on ECG with leads V1-V6 moved up to 2nd, 3rd, and 4th intercostal spaces. (D) BP resolved. Leads V1-V6 are positioned to 2nd, 3rd, and 4th intercostal spaces. (E) ECG at discharge without BP.
BP, Brugada pattern; ECG, electrocardiogram.

Twelve-lead ECG revealed normal sinus rhythm and coved ST-segment elevation of 5 mm, followed by negative T-wave in right precordial leads (Fig. 1A), consistent with type-1 BP. The patient denied previous history of syncope, agonal respiration, or palpitations. Family history was significant for early sudden cardiac death (SCD) - father at the age of 40 years. Previous ECG showed no BP (Fig. 1B).

The patient was started on ceftriaxone and was transferred to the intensive care unit for continuous monitoring. Medications that could induce BP and ventricular arrhythmias were avoided.

At the 2nd day of hospitalization, ECG still showed a type-1 BP on right precordial leads (Fig. 1C). Neither fever nor significant biochemical test abnormalities were reported. In order to exclude other etiologies for ST-segment elevation, the patient underwent an echocardiography which showed preserved left ventricular ejection fraction and no abnormalities. Coronary CT angiography revealed no significant coronary artery disease.

The patient remained in hospital for nine days. During the whole hospitalization, mild symptoms of COVID-19 infection persisted and no arrhythmic events occurred. Cardiac magnetic resonance imaging showed no abnormalities. BP disappeared eight days after admission (Fig. 1D) and the patient was discharged home with plan of outpatient follow-up.

Four months later, he was seen in consultation and ECG was similar to the day of discharge (Fig. 1E). The decision was made to proceed with drug challenge and electrophysiological study (EPS) for investigation of the probable diagnosis of BrS and risk stratification considering the family history of SCD and the BP during the infectious disease.

Ajmaline was intravenously administered according to the following protocol: 10 mg every 2 min up to a target dose of 1 mg/kg. Coved type-1 ECG pattern was documented in the right precordial leads (Fig. 2A). Programmed ventricular stimulation with three extrastimuli at the right ventricular apex resulted in inducible sustained VF (Fig. 2B). Implantable cardioverter-defibrillator (ICD) was therefore indicated.

Relatives were recruited for screening and BP was not identified in any family members. A next-generation sequencing test was performed and the result is pending.

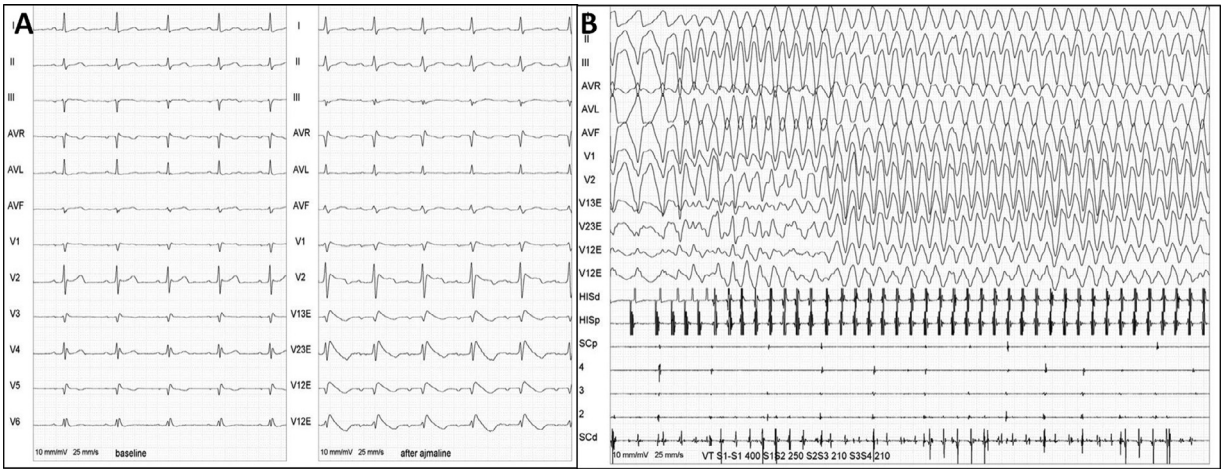

Fig. 2. (A) On the left, baseline electrocardiogram. On the right, Type-1 Brugada pattern during ajmaline infusion. (B) Electrophysiological study-induced ventricular fibrillation.

Discussion

The diagnosis of BrS is based on the presence of either spontaneous or induced type-1 ECG pattern on at least one right precordial lead in standard or superior position. Other causes of ST-segment elevation must be excluded [2]. Risk assessment and treatment are major challenges in the management in BrS patients [4] .

Current guidelines recommend that patients with BrS and syncope, aborted cardiac arrest, or sustained ventricular tachycardia/VF should undergo ICD implantation [5]. However, BrS subjects with no symptoms and no risk factor have no benefit from the device. Despite being asymptomatic, patients with BP may be at risk for VF therefore it is important to establish the diagnosis and potential risks of those individuals suspected of BrS [2,4,5].

Fever has been reported as a trigger for ventricular arrhythmias in BrS patients and may unmask the BP [4,6]. In a retrospective study, BrS subjects who developed fever-induced type 1 ECG were associated with intermediate risk of life-threatening arrhythmic events. The authors suggested that BP during high temperatures involves a more complex physiopathologic mechanism compared with drug-induced type 1 ECG [6].

Cardiovascular involvements such as heart failure, arrhythmias, and myocarditis have been reported in COVID-19 patients. Data suggest that the damage is due to either direct viral infection effect to the myocardium or indirect toxicity caused by systemic infection [7]. The inflammatory response activation presented by patients with COVID-19 infection may induce fever which in turn could explain the BP on ECG. The biochemical capacity of cardiac sodium channels may decrease during fever, leading to a reduced sodium flow responsible for ventricular arrhythmia [4,7].

There have been few reports of BP in acute COVID-19 infection [8–10]. In most of them, BP was induced by fever [8]. In the publication of Lugenbiel et al, BP increased with inflammation progress, regardless of the temperature [9]. In rare reports BP was not related to fever [10].

Data suggest and recommend supportive care to avoid fatal events in inherited arrhythmia syndromes [4]. Although it is not possible to conclude a cause-effect, in the present case medications that could induce ventricular arrhythmias were avoided besides intensive care and no arrhythmic events occurred during hospitalization.

ICD remains the most effective therapy for SCD prevention in BrS. In our case, although the patient was asymptomatic, BP was unmasked in a context of acute infection, and family history was remarkable for SCD. For this reason, ajmaline test was performed followed by EPS which resulted in inducible VF. Therefore, ICD was indicated [5].

Different from the most previously reported cases, BP occurred in the setting of COVID-19 with normal inflammatory markers and no fever, suggesting direct effect of SARS-CoV-2 in cardiac ion channels. To our knowledge, this is the first case report of BP in the setting of COVID-19 in which the diagnoses of BrS, risk stratification, and treatment were established according to current guidelines [5]. Also, genetic testing was performed to better help familiar screening.

Disclosures

None.

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Declaration of Competing Interest

The authors declare that there is no conflict of interest.

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