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Case Report

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CHA2DS2-Vasc Scoring System as A Guidance for Managing Atrial Fibrillation in A COVID-19 Patient

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Abstract

Cardiovascular disease (CVD) is a frequent comorbidity in coronavirus disease 2019 (COVID-19) making atrial fibrillation a common clinical manifestation in hospitalized coronavirus patients. Medications used to treat atrial fibrillation include antiarrhythmic drugs and anticoagulants based on the balance between hemorrhagic risk using HAS-BLED score and thrombotic risk using the CHA2DS2-VASc Score. So through the column of this article, we describe a 75 years old man with COVID-19 infection presenting with atrial arrhythmias, which resolved with rate and rhythm control strategies, and supportive care.

Keywords: *CHA2DS2-VASc, Atrial fibrillation, COVID-19, Intensive care unit.*

Introduction

Coronavirus disease-2019 (COVID-19) is an infectious disease caused by severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) [1-2] The disease was first identified in2019 in Wuhan, China, and has since spread globally, therefore, the World Health Organization (WHO) declared the novel coronavirus outbreak a pandemic [3-4].

COVID-19 may have deleterious effects on the cardiovascular (CV) system, and patients with preexisting CV disease. Several recent Chinese studies have since demonstrated the sequelae of CV events, from simple arrhythmias to sudden cardiac death. [5-6]

Case Report

we report a history that goes back to 27/07/20 of a 78 years old man, starting by the appearance of a flu-like syndrome made of unencrypted fever and chills followed by the appearance of a dry cough and dyspnea of progressive aggravation, which motivated a consultation, the thoracic CT showed an aspect suggestive of viral infection, supplemented by a positive COVID PCR. patient was admitted and referred to the dedicated COVID-9 ICU.

Examination on admission shows a conscious patient, his blood pressure was at 130/80 mmHg and his heart rate was at HR 88 pulses per minute. His respiratory rate at 33c /min and arterial oxygen saturation at 80% at room air without fever (body temperature at 36°.9C), However, we noticed bilateral humming groans on pleuropulmonary auscultation, without any other particular cardio-vascular signs.

The CHA2DS2-VASc Score for Atrial Fibrillation was at 2. The chest-CT revealed air bronchogram associated with ranges of diffuse ground glass opacities at the level of the two pulmonary fields, the viral origin of which is very likely with severe parenchyma involvement (50%-75%).

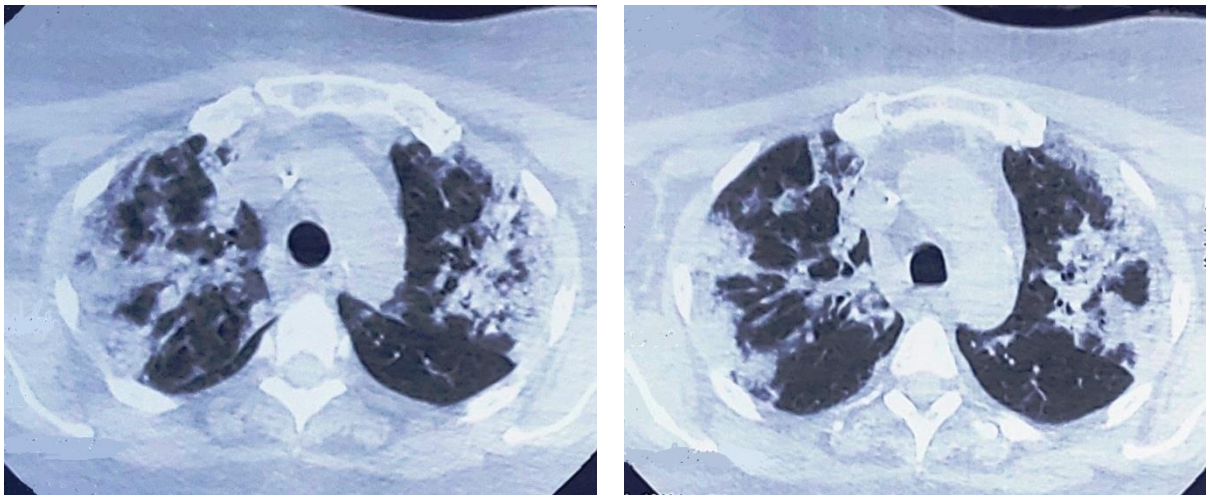

Figure 1: Chest-CT scan revealing air bronchogram associated with ranges of diffuse ground glass opacities.

The ECG shows an:

atrial fibrillation at 109hpm, no P waves, "garbage baseline" PR cannot be measured irregular ventricular response normal QRS QT corrected at 380 msec

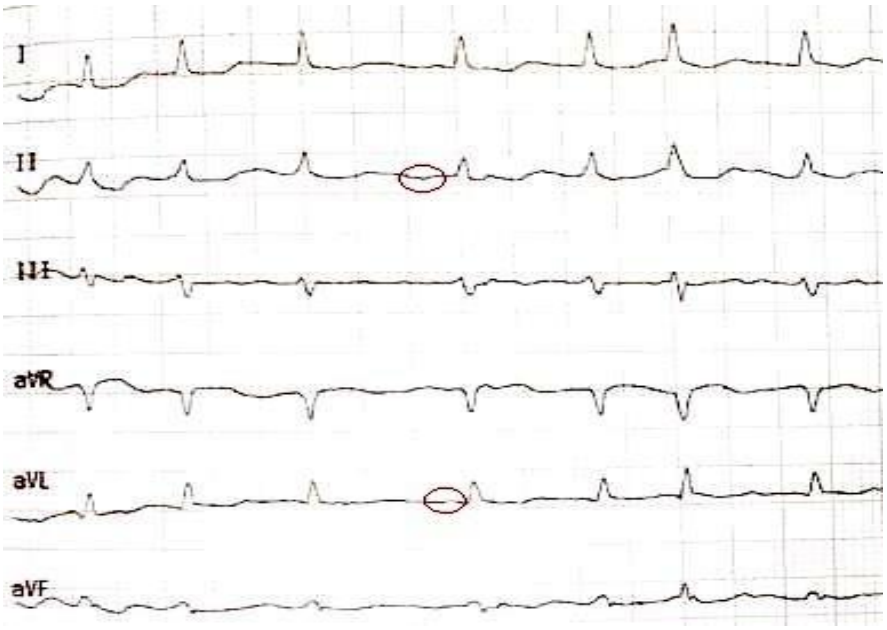

Figure 2: EKG revealing small deflection of variable amplitude (f waves).

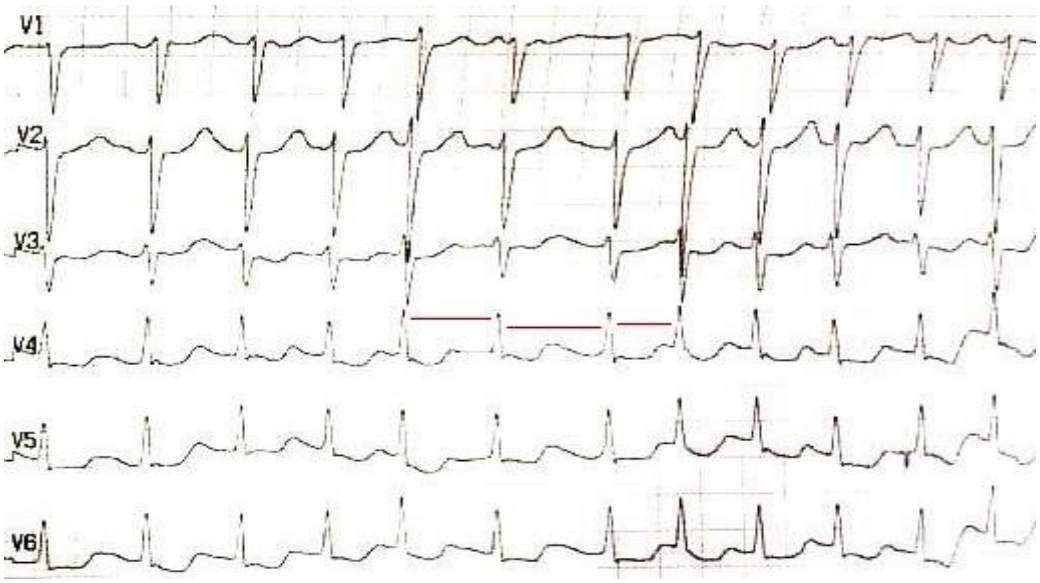

Figure 3: EKG revealing an irregularly irregular ventricular rate demonstrating narrow QRS complexes

The echocardiography showed;

For the left ventricle: normal in size with normal wall thickness and systolic function, an ejection fraction at 50%, without neither dyskinesia nor thrombosis.

For the right ventricle: no valvular leakage or stenosis, a right ventricle systolic function associated with a concentric left ventricular hypertrophy both aorta and the inferior vena cava are normal. The patient's biological assessment revealed a white blood cell number of 11,150 cells / mm 3 (Neutrophils 10380, lymphocytes 330), hemoglobin of 13.6 g / dl, thrombocytes at 160,000 cells / mm 3. Prothrombin time and partial thromboplastin time were normal (TP at 70% and TCA at 26s for a witness of 23s).

Natremia:145 mmol / l, kalemia: 3.5mmol / l, correct liver and renal function (urea: 0.72 g / l and creatinine: 15.9 mg / l, ASAT : 41 IU / l and ALT: 20 IU / l), fasting blood sugar at 1.23 g / l, C-reactive protein at 180 mg / l, Troponin at 31.4 ug / l, ferritin at 2000 ng\ml therapeutic management included oxygen therapy, non-invasive ventilation, medical treatment associated, as the national protocol suggested Hydroxychloroquine 200 mg 3 times a day, Azythromycin 500mg the first day then 250mg per day, methylprednisone at 80mg a day for 7 days and curative anticoagulation treatment including enoxaparin 100 UI\kg (1mg\kg) twice a day On the 6th day, , the symptomatology worsened by the appearance of sever dyspnea, the patient was intubated and passed away within 24 hours of COVID-19 complications.

Discussion

It has been recently reported that CV compromise is a common complication of patients who are hospitalized with COVID-19 infection and is associated with a higher risk of mortality.[7] Cardiac arrhythmias are also frequent clinical manifestations. Data provided by the New York State Department of Health reported that AF is among the top ten COVID-19 comorbidities, specifically occupying the seventh position.[8] In effect, some forms of arrhythmia were present in 60% of 85 fatal cases, which suggests that cardiac arrhythmia is associated with an increased risk of mortality in COVID-19 patients [9].

First, the symptomatic presentation of AF spans from silent AF to a variety of mild to moderate symptoms as it was the case for our patient (palpitations, dizziness, chest discomfort, etc.), and to hemodynamic instability. The former is often discovered through opportunistic screening or a routine ECG in general practice, which has been proven highly effective to catch AF [10].

The mechanisms by which subjects with AF may be at increased risk are not known, but probably rely on both the cell entry mechanisms of the virus and the inflammatory host response. As for patients with other cardiac diseases, such as heart failure, it has been shown that prevalent AF is associated with higher levels of angiotensin-converting enzyme 2 (ACE2), the peptide through which the virus binds human cells [11-12].

ACE2 up-regulation may potentially increase the susceptibility to COVID-19 [11] Interestingly, ACE2 levels also correlate with left atrial structural and functional remodeling, which are substrates of increased susceptibility to AF [12]. Studies also showed that metabolic derangements and catecholaminergic stress are the main factors of Arrhythmias in a COVID-19 patient [13-14], also Inflammatory cytokines, including C-reactive protein which was in the case of our patient at 180 mg / l, interleukin-6, and tumor necrosis factor-α, may have an important role in AF pathogenesis.[15], Despite these emerging studies, the arrhythmogenic effect of COVID-19 might still be underreported and was noted in up to 17% of patients; the literature still lacks more precise differentiation [16]. Importantly, the presence of AF together with inadequate rate control might have a negative impact on patients' prognosis.

With a large prevalence of AF in the general, and especially the elderly, population [17] and the known connection of arrhythmic burden and viral disease,[18] AF may influence

mortality Guidelines for the management of AF in various conditions and populations have recommended that assessment of thrombotic risk in patients with NVAF should be based on the CHA2DS2-VASc scoring system.[19] According to the guidelines, NVAF patients with CHA2DS2-VASc score of 0 to 1, defined as low thrombotic risk, were not necessary to take antithrombotic therapy. however, guidance for managing AF in COVID-19 patients is currently not available. our patient had a *CHA2DS2-VASc Score of* 2 and therefore antithrombotic therapy was indicated.

Conclusion

Although it is probable that well-controlled AF does not increase the risk in COVID-19 patients, people living with AF might be more vulnerable to the effects of SARS-CoV-2 infection. Also, assessment of thrombotic risk using the CHA2DS2-VASc scoring system can serve as a guidance for managing AF in COVID-19 patients.

Competing interests

The authors declare no competing interests.

Authors' contribution

All the authors contributed equally in drafting of the manuscript. All the authors read and agreed to the final manuscript

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