Indian Pacing and Electrophysiology Journal 21 (2021) 257–259

Contents lists available at ScienceDirect

Indian Pacing and Electrophysiology Journal

journal homepage: www.elsevier.com/locate/IPEJ

Challenging ST elevation during night shift

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ARTICLE INFO

Article history:
Received 13 January 2021
Received in revised form
22 March 2021
Accepted 26 March 2021
Available online 3 April 2021

Keywords:
St elevation
STEMI
Brugada syndrome
Electrolyte imbalance

1. Introduction

A woman in her 50s, during her home quarantine period for an asymptomatic SARS-CoV2 infection, alerted the emergency department after a syncope. Drowsiness, altered mental status, nausea, and vomiting were reported in the previous days. No chest pain was reported.

She was recently diagnosed with diabetes mellitus; however, she did not perform any further medical investigation due to COVID-19 positivity. She was on treatment for essential arterial hypertension with an angiotensin II receptor blocker. The paramedics found the patient awake and confused. A 12 lead ECG (Fig. 1) was performed and sent to the cardiology department of our hospital.

Which are the possible diagnoses ?

Would you call the Cath lab for this patient without any further investigation?

2. Case report

2.1. Interpretation

The ECG showed regular sinus rhythm at 97 bpm, with evident P wave, prolonged PR interval (220 ms), wide QRS (200 ms) with a right bundle-branch block like morphology and an extreme QRS axis deviation (from −120° to −90°). There was a marked coved shape ST segment elevation from V1 to V3 with negative T waves and a slight ST segment depression in lead I and AvL with a positive T wave. The Qtc was prolonged.

2.2. Clinical course

An arterial blood gas analysis (ABG) was performed as soon as the patient was admitted to the Emergency Department. The ABG showed a picture of severe metabolic acidosis with compensatory respiratory alkalosis (pH 6.98, K 8.4 mEq/L, HCO3- 2.6 mg/dl, lactates 7 mg/dl, blood glucose 403 mg/dl, pO2 81 mmHg, pCO2 21 mmHg). Further blood exams revealed a picture of acute renal failure (creatinine 3.9 mg/dl). The patient was hemodynamically stable and no ventilatory support was required.

Rapid i.v. HCO3- infusion and i.v. insulin infusion were started aiming to correct hyperkalemia and pH.

After 50 min both the patient mental status and the ABG were improving (pH 7.3, K 7.2 mEq/L, blood glucose 312 mg/dl and lactates 5.1 mg/dl).

2.3. A 12 lead ECG was repeated

The ECG (Fig. 2) showed regular sinus rhythm with a normalized atrio-ventricular conduction (PR interval 180 ms), and a narrow QRS (80 ms) with persistence of incomplete right bundle-branch block, ST segment resolution and positive tent shaped T wave in precordial leads.

3. Discussion

The ECG findings showed in Fig. 1 in a patient with syncope, could be consistent with acute anterior myocardial infarction, type 1 Brugada pattern, class Ic antiarrhythmic drug intoxication, electrolytes abnormalities or myocardial injury in the context of SARS-CoV2 infection.

Anterior STEMI was ruled out by an urgent echocardiogram performed in the Emergency Department, showing a left ventricular systolic function at the lower range l (left ventricular ejection fraction 50%), without any regional wall motion abnormality and no significant elevation of cardiac Troponine. The patient was not on

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Peer review under responsibility of Indian Heart Rhythm Society.

https://doi.org/10.1016/j.ipej.2021.03.003

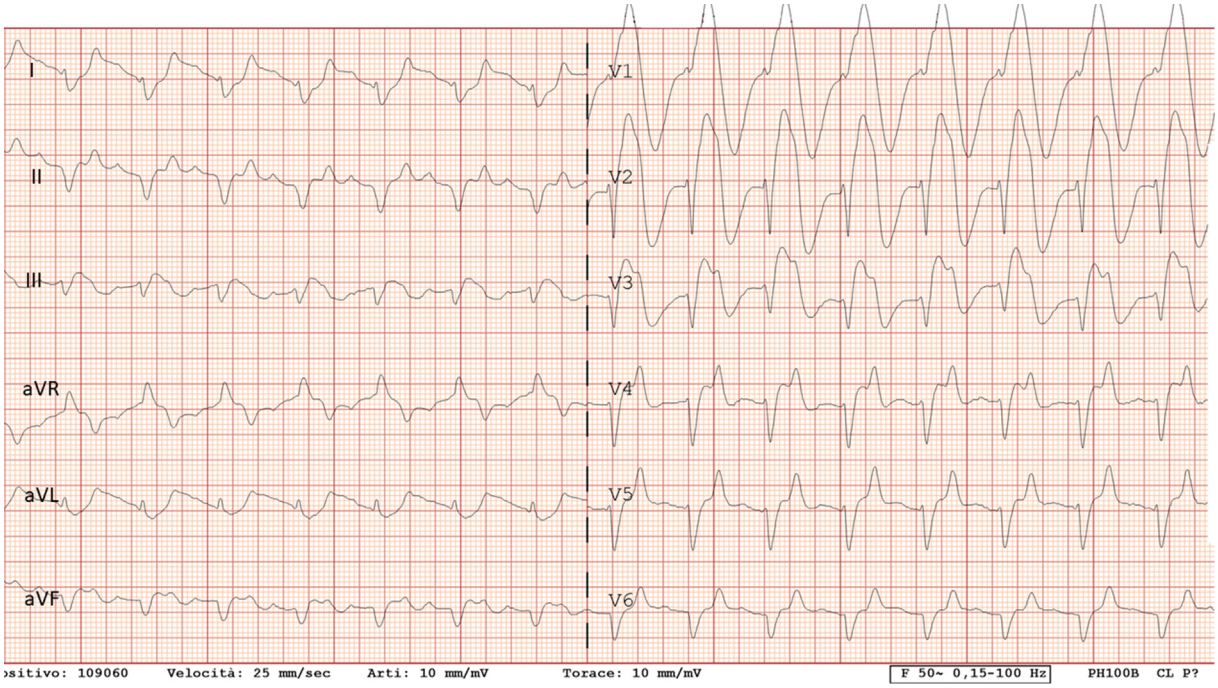

Fig. 1. The first ECG received during night shift.

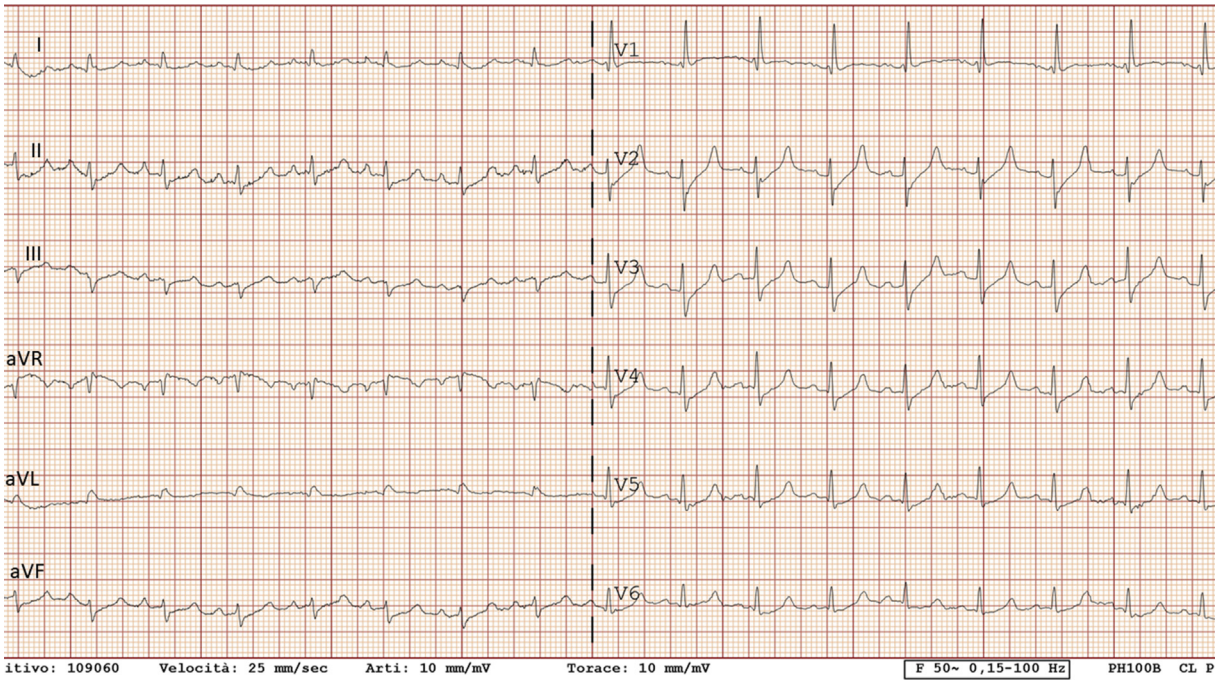

Fig. 2. ECG recorder after 50 min of treatment.

treatment with any antiarrhythmic drug, ruling out the hypothesis of class Ic antiarrhythmic drug intoxication. It is well known that both hyperkalemia and pH changes can lead to progressive electrocardiographic abnormalities, specifically slowing of intraventricular conduction (revealed by QRS widening), hyperacute T waves and more rarely ST segment elevation. ECG changes due to acidosis have often been considered not intrinsically related to pH changes, but rather to hyperkalemia, usually present in the same patient [1,2] . In the present case there was a rapid pH correction compared with a slower change in the potassium concentration after initiation of therapies, suggesting a greater influence of pH on ECG morphology in the context of acute hyperkalemia.

The Brugada syndrome could not be ruled out in the acute setting. We observed the normalization of Brugada pattern following the correction of pH and potassium concentration; however, since hyperkalemia has been reported to unmask type 1 Brugada pattern [3], we performed an ajmaline test after the patient's recovery, without induction of diagnostic Brugada pattern.

ST segment elevation observed in patients with Brugada Syndrome is a consequence of the accentuation of the action potential notch in phase II in epicardial cell. This is the result of imbalance of inward (sodium and calcium) and outward (potassium) currents caused by a mutation in the genes encoding cardiac sodium channels that leads to reduced sodium current. Cardiac sodium channels are inactivated by extracellular hyperkalemia as well, thereby reproducing a comparable ionic currents imbalance and overlapping ECG alterations [4].

Another differential diagnosis could be represented by a COVID-19 induced ST elevation. The most common ECG changes during SARS-CoV2 infection are reduction in heart rate and increase in both QRS duration and QTc interval. However, ST segment elevation associated with myocardial injury) has been described [5]. Due to the clinical picture (absence of chest pain, no cardiac involvement at instrumental and laboratory exams) and considering the rapid resolution of ST segment elevation with the initial therapy, this hypothesis was ruled out.

In conclusion, we report a case of a Brugada phenocopy induced by hyperkalemia and acidosis .

Declaration of competing interest

All authors have no disclosure.

References

[1] Reid JA, Enson Y, HarveY RM, et al. The effect of variations in blood pH upon the electrocardiogram in man. £ Circulation 1965;31(3):369–73.
[2] Weiss James N, Zhilin Qu, et al. Electrophysiology of hypokalemia and hyperkalemia. Circulation: Arrhythmia and Electrophysiology 2017;10(3):e004667.
[3] Postema Pieter G, Alexander PJ, DeVries Hans, et al. Familial Brugada syndrome uncovered by hyperkalaemic diabetic ketoacidosis. Europace 2011;13(10):1509–10.
[4] Antzelevitch C. The Brugada syndrome: diagnostic criteria and cellular mechanisms. Eur Heart J 2001;22(5):356–63.
[5] Yatskar L, Harari R, Shah B. ST-segment elevation in patients with covid-19: a case series. N Engl J Med 2020;382:2478–80.