CASE REPORT

Brugada phenocopy caused by a compressive mediastinal tumor

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Recently, it has been shown that even experts in electrocardiography cannot differentiate the electrocardiographic pattern of genuine Brugada syndrome (BrS) from Brugada phenocopy (BrP). For this reason, this differentiation depends on the presence of established criteria both for BrS and BrP. In this manuscript, we present a patient with type 1 Brugada electrocardiographic pattern caused by expansive anterior mediastinal non-Hodgkin's lymphoma (NHL) with mechanical compression on the right ventricular outflow tract. The electrocardiographic pattern disappeared rapidly after antineoplastic measures.

KEYWORDS

Brugada phenocopy, Brugada syndrome, non-Hodgkin's lymphoma, sodium channel blocker

1 | CASE REPORT

An elderly Caucasian woman (79 years old) was admitted to the emergency room with complaint of precordial pain for 6 months with epigastric irradiation at rest, worsening in intensity and duration in the last 2 days associated with dyspnea and dry cough. 30 days before she had a syncopal episode with palpitations.

Negative family history for sudden death or syncope in first-degree relatives <45 years.

Physical examination: sinus rhythm, heart rate 96 bpm, diffuse hypophonesis, left basal crackles in the left lung and auscultation over the right lung produced a very muffled sound, painful distended abdomen palpation in the right hypochondrium, palpable liver at 3 cm below the right costal ridge. Palpable cervical ganglia on the right side.

Discrete troponin elevation (0.320) with normal CK-MB; normal electrolytes.

Initial 12-lead electrocardiogram (ECG) was performed (Figure 1), which led to thinking of right ventricle (RV) myocardial infarction. Coronary angiography revealed 70% proximal obstruction in the right coronary artery (RCA) (Figure 2) and ventriculography did not reveal

dyskinesia in any ventricle. Successful stent implantation was placed in the RCA (TIMI 3 and BLUSH 3). Even with the success of the procedure, serial ECGs did not show reversion of ST elevation (STE) in the right precordial leads.

Posteroanterior chest x-ray (Figure 3) showed a large pleural effusion on the right side that disappeared after treatment.

Computed tomography of the thorax showed an invasive expansive process (tumor) of the right pulmonary artery and the RV outflow tract (RVOT) (Figure 4).

Echocardiogram showed mild concentric left ventricle (LV) hypertrophy (septum and free wall = 12 mm), normal ventricular function (LV ejection fraction = 73%); chambers with normal dimensions; global contraction and preserved segment; moderate pericardial effusion without diastolic restriction; pulmonary artery systolic pressure = 65 mmHg.

Transcatheter needle aspiration biopsy revealed NHL. Chemotherapy treatment was started, associated with radiotherapy and immunotherapy with monoclonal antibodies and cytokines, which led to the reversal of type 1 Brugada ECG pattern (Figure 5). As an indispensable element for this diagnosis, we performed a provocative ajmaline test that was negative.

FIGURE 1 Initial ECG. STE in aVR and from V₁–V₃ decreasing progressively (this characteristic differentiates RV infarction from anteroapical LV infarction). Concomitantly, ST depression is observed in inferior and lateral walls. Conclusion: type 1 Brugada ECG pattern

FIGURE 2 Percutaneous coronary intervention. Occlusion of the proximal RCA (a), TIMI3 flow in the dominant RCA after insertion of drug-eluting stent (b)

FIGURE 3 Posteroanterior chest x-ray. Large pleural effusion before (a) and after treatment (b)

FIGURE 4 The computed tomography indicates the tumor extension (arrow)

2 | DISCUSSION

In the second consensus conference about BrS, the admitted criteria are: absence of apparent structural heart disease; absence of drugs effects, electrolyte disturbance and coronary artery disease; documented polymorphic ventricular tachycardia (PVT)/ventricular fibrillation (VF); family history of syncope and/or sudden cardiac death at <45 years in first-degree relatives; type 1 Brugada ECG pattern in proband and family members; induction of PVT/VF with programmed electrical stimulation; and syncope, cardiac arrest or nocturnal agonal respiration (Antzelevitch et al., 2005).

Several conditions have been suggested to mimic this ECG pattern without the same clinical implications. In our patient, the ECG pattern was most likely due to tumor compression of the RVOT, since the ECG normalized after tumor disappearance. It is difficult to decide if this “secondary” form of Brugada-like electrocardiographic changes can result in an increased risk for developing ventricular tachyarrhythmia in asymptomatic patients with BrS.

To our knowledge, a relation between RVOT compression by extracardiac mass and type 1 Brugada ECG pattern has been reported for the first time in 1999 by Tarín et al. (Tarín, Farre, Rubio, Tunon, & Castro-Dorticos, 1999), but in this case, they did not perform the sodium channel block test.

The present case has all the necessary criteria for BrP (Anselm, Gottschalk, & Baranchuk, 2014): type-1 Brugada ECG pattern, underlying identifiable condition, ECG normalization after resolution of the underlying condition, low clinical pretest probability of true BrS determined by a lack of symptoms, medical and family history, negative ajmaline provocative test.

3 | CONCLUSION

We presented a case of mediastinal non-Hodgkin's lymphoma that compressed the RVOT originating a type 1 Brugada ECG pattern. After the treatment and lymphoma reversal, this pattern disappeared. The negative provocative ajmaline test indicates low probability of Brugada syndrome.

FIGURE 5 ECG normalization after oncologic therapy. Low QRS voltage in the limb leads (<5 mm) and precordial leads (<10 mm), ST depression in inferior and lateral precordial leads

CONFLICT OF INTEREST

None.

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