Acta Scientific Clinical Case Reports

Volume 3 Issue 9 September 2022

Case Report

Ventricular Fibrillation, Early Repolarization, and Hyperthyroidism: Which is the Linking? A Case Report

Aldo Coppolino* and Letizia Valeri

Department of Cardiology, S. Annunziata Hospital, Savigliano (CN), Italy

***Corresponding Author:** Aldo Coppolino, Department of Cardiology, S. Annunziata Hospital, Savigliano (CN), Italy.

Received: August 16, 2022

Published: August 25, 2022

Abstract

Thyrotoxic storm is the gravest form of hyperthyroidism and can generate cardiac complications such as arrhythmias. Ventricular fibrillation is the most severe of them. Hyperthyroidism can induce molecular e biological alterations in the cardiac cells, thus modifying the substrate and predisposing them to arrhythmias. We report a clinical case of ventricular fibrillation in a patient with a thyroid storm and an electrocardiogram pattern of early repolarization, such as an arrhythmogenic substrate.

Keywords: Ventricular Fibrillation; Electrocardiogram; Hyperthyroidism; Early Repolarization; Cardiac Arrest

Abbreviations

ECG: Electrocardiogram; ER: Early Repolarization Pattern; VF: Ventricular Fibrillation; T3: Triiodothyronine

Introduction

Thyrotoxic storm is the gravest form of hyperthyroidism and can generate cardiac complications such as arrhythmias. Ventricular fibrillation is the most severe of them. It has been elucidated that hyperthyroidism induces alterations in the potassium channels, thus modifying the substrate and predisposing to arrhythmias. We report a clinical case of ventricular fibrillation in a patient with a thyroid storm and an electrocardiogram pattern of early repolarization, such as an arrhythmogenic substrate.

Case Report

A 37-year-old male was referred to our hospital for seven recurrent cardiac arrest episodes by ventricular fibrillation. On admission, he seemed to be suffering from delirium. He had no medical or family history of electrocardiographic abnormality, cardiac disease or sudden cardiac death.

The blood pressure was 130/80 mmHg, heart rate 130 bpm, and body temperature 37.5° C.

The electrocardiogram (ECG) showed sinus rhythm and early repolarization pattern (ERP) characterized by J-point elevation > 2 mm in the inferior and V3–V6 leads. QTc interval was 450 msec.

No signs of ischemia were present (Figure 1).

Figure 1: This ECG shows the notching QRS in inferior and lateral leads, such as a typical Early Repolarization Pattern.

Potassium level and haemoglobin were 3.5 mEq/L and 14 mg/dL, respectively.

The renal and liver function, such as the toxicological tests, were normal.

Transthoracic echocardiography revealed normal left and right ventricular function and no significant valvular dysfunction.

The coronary angiography excluded coronary artery disease.

During the first hour of stay in our hospital, the patient experienced four other episodes of cardiac arrest by ventricular fibrillation triggered by ventricular ectopic beats and the R/T phenomenon (Figures 2a and 2b). We restored to sinus rhythm following defibrillation.

Figure 2a: The sequence of ventricular extrasystoles and successive ventricular fibrillation.

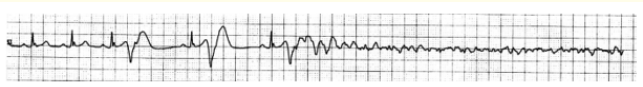

Figure 2b: Focus on the mechanism of ventricular fibrillation: phenotype of dispersion potential action by ventricular extrasystoles and its compensatory pause (trigger); R/T phenomenon induces ventricular fibrillation.

Subsequent results of the screening examination revealed hyperthyroidism: stimulating thyroid hormone (TSH) < 0. 01 IU/mL, triiodothyronine (FT3) 2.3 pg/mL, free thyroxine (FT4) 30.7 mg/dL.

Promptly we administered Metimazole 5 mg one time a day and Propranolol 40 mg three times a day.

In the following days, we observed an improvement in thyroid function. At the same time, the electrocardiogram recorded on the seventh day after admission showed the disappearance of the J wave in the inferior and V4-V6 leads (figure 3).

Figure 3: ECG recorded after normalization of thyroid function: the Early Repolarization Pattern disappeared entirely. The negative T wave in inferior and lateral leads could demonstrate the electric memory phenomenon.

The following cardiac resonance (MRI) was also normal.

Discussion

Early repolarization

Early repolarization (ER) was interpreted as a variant of the standard electrocardiogram (ECG). We frequently have observed ER in young, healthy subjects or athletes. Its characteristic is a notch or a slurring of the QRS-ST junction resulting in a so-called "J wave" with an elevation of the J point above the isoelectric line. The inferior leads or/and the lateral leads often reveal this pattern [1,2].

When Haissaguerre presented a study on the prevalence of ER patterns in patients resuscitated after a cardiac arrest related to idiopathic ventricular fibrillation (VF) [3], the ER pattern aroused enormous interest as an arrhythmogenic substrate. A relationship between ER pattern and malignant arrhythmias is supported by the experimental work of Antzelevitch., *et al*. which provided the cellular and ionic basis for the J point elevation and its potential arrhythmogenic role.

Transmural differences in the early phases of the action potential between the epicardium and endocardium cells are the molecular arrhythmogenic substrate. A prominent transient outward current (Ito)-mediated notch or spike and dome morphology of action potential in ventricular epicardium but not endocardium produce a transmural voltage gradient that registers as a J wave or J point elevation on the ECG [4-7].

Cardiac and extracardiac conditions are associated with the ER. We summarized these in table 1.

Causes of acquired Early Repolarization Pattern	
Cardiac Conditions	**Extracardiac**
Acute coronary syndrome Myocardial infarction (MI) Inflammatory diseases (pericarditis, myocarditis) Hypertrophic cardiomyopathy Left ventricular hypertrophy	Hypothermia Autonomic nervous system abnormalities Increased vagal tone Decreased sympathetic tone Cocaine use Antidepressants use Electrolyte disorders: hypercalcemia Neuropsychiatric disturbances Metabolic disorders

Table 1: It shows main causes of Early Repolarization Pattern

They increase the hazard of malignant cardiac events when ER appears too.

On the other hand, in the majority of people, ER occurs in the absence of cardiovascular diseases, such as an isolated ER pattern, and is so common that, in the absence of a possible aetiology, it is difficult to predict which subject is at increased risk of fatal arrhythmias [8-11].

Furthermore, ER has dynamic changes in the amplitude of the J wave in various situations [12]. Exercise, rapid atrial pacing and atropine or isoproterenol tend to decrease or even eliminate the ER pattern [13].

In contrast, bradycardia or beta-blockers increase the J wave pattern [13]. This variation may also be due to autonomic high vagal tone disorders. Augmentation of the ER pattern increases the risk of arrhythmias, and it often appears before a ventricular arrhythmic event [3,14-16].

Hyperthyroidism and cardiac interaction

Thyroid hormones significantly affect the heart and cardiovascular system, and hyperthyroidism and concomitant thyrotoxicosis increase the risk of cardiovascular symptoms and harmful effects.

Due to molecular and cellular effects on cardiomyocytes as upregulation of α-myosin heavy chain, intracellular calcium levels increase, prolongation of activation of sodium channels, upregulates β-adrenergic receptors, thyrotoxicosis brings hemodynamic effects such as preload augmentation, afterload and systemic vascular resistance decrement, myocardial contractility increase.

Therefore, patients present exercise intolerance, dyspnoea, angina-like chest pain, peripheral oedema, and thyrotoxic cardiomyopathy until congestive heart failure [17].

In addition, hyperthyroidism is characterized by a sympathovagal imbalance, with enhanced sympathetic and reduced vagal regulation of the heart rhythm. These autonomic dysfunctions commonly develop palpitation and sinus tachycardia due to the direct effect on the sinus node.

As well as, thyrotoxicosis influences the atrial cardiomyocytes' action potential duration and delayed rectifier potassium currents and can cause atrial fibrillation through the increase of the arrhythmogenic potential of pulmonary veins [18].

In an editorial comment about the article of Ueno., *et al*. Antzelevitch clarifies that the biologically relevant thyroid hormone triiodothyronine (T3) modulates contractile activity and repolarization in the heart. Precisely it modulates the transient outward current Ito [19].

Hyperthyroidism raises the expression of KCND3, the gene that encodes the α-subunit (Kv4.3) of the Ito channel, leading to the accentuation of Ito and the epicardial action potential notch [19,20].

Thus the transmural differences in the early phases of the action potential between the epicardium and endocardium cells appear, setting up the molecular arrhythmogenic substrate. It can become apparent in ECG through the J wave of ER.

Our case report and ECG findings

Few cases have been reported of ventricular fibrillation and thyroid storm [19,21,22]. Our clinical case adds to these and confirms that, in the absence of acute or chronic cardiac disease, thyrotoxicosis causes the preconditions for malignant arrhythmias.

In our opinion, the electrophysiological mechanism responsible for the arrhythmia is the dispersion of the action potential between the areas with a shorter action potential (epicardium) and sizes with longer ones (endocardium). The clinical phenotype of this

molecular mechanism is the brief appearance of the J wave on the ECG in the form of a typical "early repolarization pattern".

In summary and according to triangle Coumel theory, hyperthyroidism is the modulating factor that modifies the substrate, the modified substrate appears as an early repolarization pattern, and the triggers are ventricular extrasystolic beats.

The post extrasystolic pause enhances the dispersion of potential action. The consecutive ventricular extrasystole promotes re-excitation of an area already repolarized and triggers a chain phenomenon of recurrent ventricular fibrillation.

After normalization of thyroid function, the early repolarization pattern and episodes of ventricular fibrillation disappeared utterly.

Conclusion

This case report demonstrates that, in the absence of cardiac disease, the thyroid storm can generate ventricular fibrillation through the dispersion of the repolarizing gradient that gives rise to phase 2 reentry. The appearance of J wave elevation, defined as early repolarization, is the electrocardiographic marker of this substrate arrhythmic and results in an acquired form of ER.

Therefore, clinicians should pay attention to J-wave amplitude in the ECG of patients with thyrotoxicosis to stratify better the risk of malignant cardiac arrhythmias.

Conflict of Interest

No conflict of interest to declare.

Bibliography

1. Wasserburger R H and Alt W J. "The normal RS-T segment elevation variant". *The American Journal of Cardiology* 8 (1961): 184-192.
2. Macfarlane PW., *et al*. "The Early Repolarization Pattern: A Consensus Paper". *Journal of the American College of Cardiology* 66.4 (2015): 470-477.
3. Haissaguerre M., *et al*. "Sudden cardiac arrest associated with early repolarization". *The New England Journal of Medicine* 358 (2008): 2016-2023.
4. Gussak I and Antzelevitch C. "Early repolarization syndrome: clinical characteristics and possible cellular and ionic mechanisms". *Journal of Electrocardiology* 33 (2000): 299-309.
5. Yan GX and Antzelevitch C. "Cellular basis for the electrocardiographic J wave". *Circulation* 93 (1996): 372-379.
6. Gussak I and Antzelevitch C. "Early repolarization syndrome: clinical characteristics and possible cellular and ionic mechanisms". *Journal of Electrocardiology* 33 (2000): 299-309.
7. Antzelevitch C and Yan GX. "J wave syndromes". *Heart Rhythm* 7 (2010): 549-558.
8. Tikkanen JT., *et al*. "Long-term outcome associated with early repolarization on electrocardiography". *The New England Journal of Medicine* 361 (2009): 2529-2537.
9. Sinner M F., *et al*. "Association of early repolarization pattern on ecg with risk of cardiac and allcause mortality: a population-based prospective cohort study MONICA/KORA)". *PLoS Medicine* 7 (2010): e1000314.
10. Littmann L. "Early repolarization on electrocardiography". *The New England Journal of Medicine* 362 (2010): 1148
11. Ghosh S., *et al*. "Early repolarization associated with sudden cardiac death: Insights from noninvasive electrocardiographic imaging". *Heart Rhythm* 7 (2010): 534-537.
12. Nam G B., *et al*. "Augmentation of J waves and electrical storm in patients with early repolarization". *The New England Journal of Medicine* 358 (2008): 2016-2023.
13. Morace G., *et al*. "Effect of isoproterenol on the early repolarization syndrome". *American Heart Journal* 97 (1979): 343-347.
14. Kaneko, Y., *et al*. "Augmentation of the J wave following ventricular pause in "benign" early repolarization". *Internal Medicine* 49 (2010): 1671.
15. Cappato, R., *et al*. "J wave, QRS slurring and ST elevation in athletes with cardiac arrest in the absence of heart disease: marker risk or recent bystander?" *Circulation. Arrhythmia and Electrophysiology* 3 (2010): 305-311.
16. Wilhem M., *et al*. "Early repolarization, left ventricular diastolic function and left atrial size in professional soccer players". *The American Journal of Cardiology* 106 (2010): 569-574.
17. Sibel Ertek Arrigo F Cicero. "Hyperthyroidism and cardiovascular complications: a narrative review on the basis of pathophysiology". *Archives of Medical Science* 9.5 (2013): 944-952

18. Ahmad M., *et al*. "Hyperthyroidism and the Risk of Cardiac Arrhythmias: A Narrative Review". *Cureus* 14.4 (2022): e24378.

19. Ueno A., *et al*. "Ventricular fibrillation associated with early repolarization in a patient with thyroid storm". *The Journal of Interventional Cardiac Electrophysiology* 29.2 (2010): 93.

20. Shimoni Y and Severson D L. "Thyroid status and potassium currents in rat ventricular myocytes". *The American Journal of Physiology* 268 (1995): H576-H583.

21. Karashima S., *et al*. "Ventricular Fibrillation Associated with Dynamic Changes in J-Point Elevation in a Patient with Silent Thyroiditis". *Journal of the Endocrine Society* 2.2 (2018): 135-139.

22. Iiya M., *et al*. "Ventricular Fibrillation Induced by Coronary Vasospasm in a Patient with Early Repolarization and Hyperthyroidism". *Internal Medicine* 57.23 (2018): 3389-3392.