PLACE 2026
PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

CONGRESSO
ROMA, 25 - 27 Giugno 2026

Cardiologia Clinica – Cominicazioni Orali

A novel cause of ventricular fibrillation in hyperthyroidism:

QT shortening on an early repolarization pattern

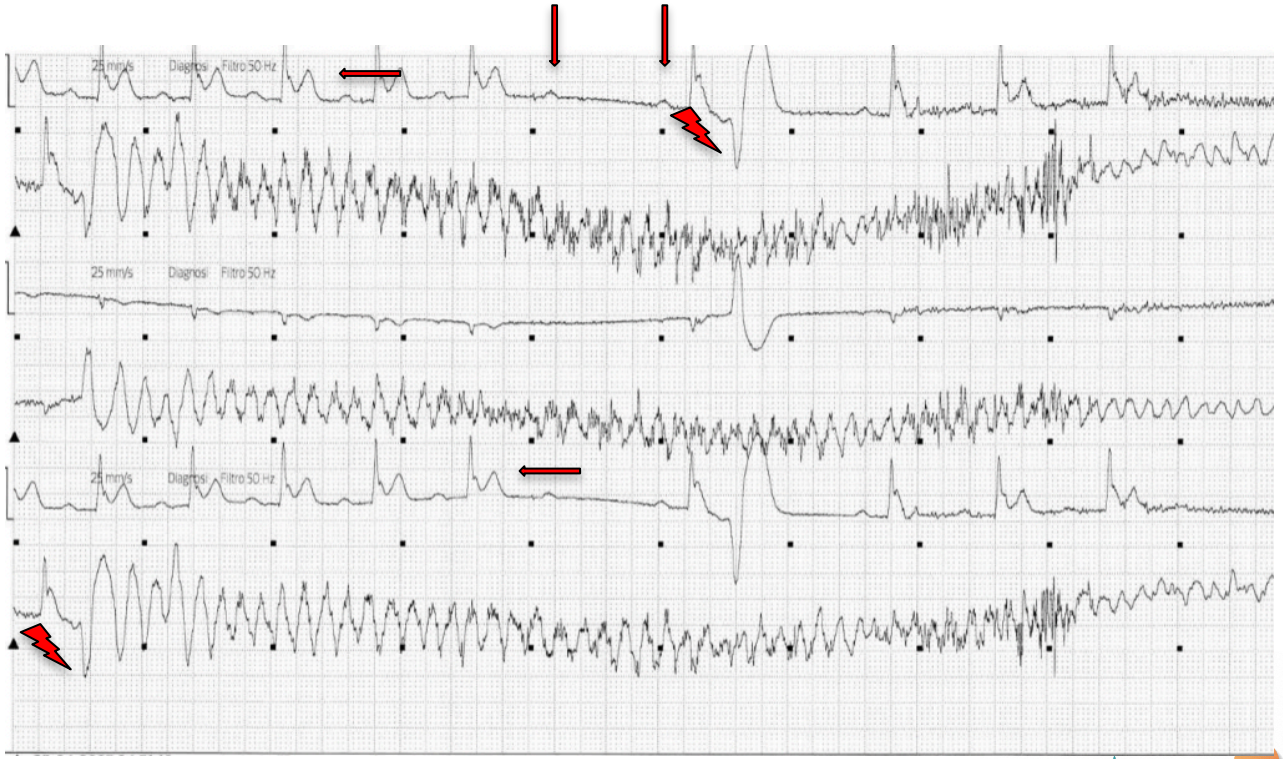
Claudia Cataldi

Case Presentation

A 41-years-old African man, without prior cardiological history, had two episodes of LOC accompanied by seizures during sleep.

During the first hours of ICU monitoring, a torsade de pointe, degenerating in VF, was interrupted by DC-shock

Isolated PVCs, a 1st-degree atrio-ventricular block (AVB) and sporadic episodes of 2nd-degree type-1 AVB were noticed.



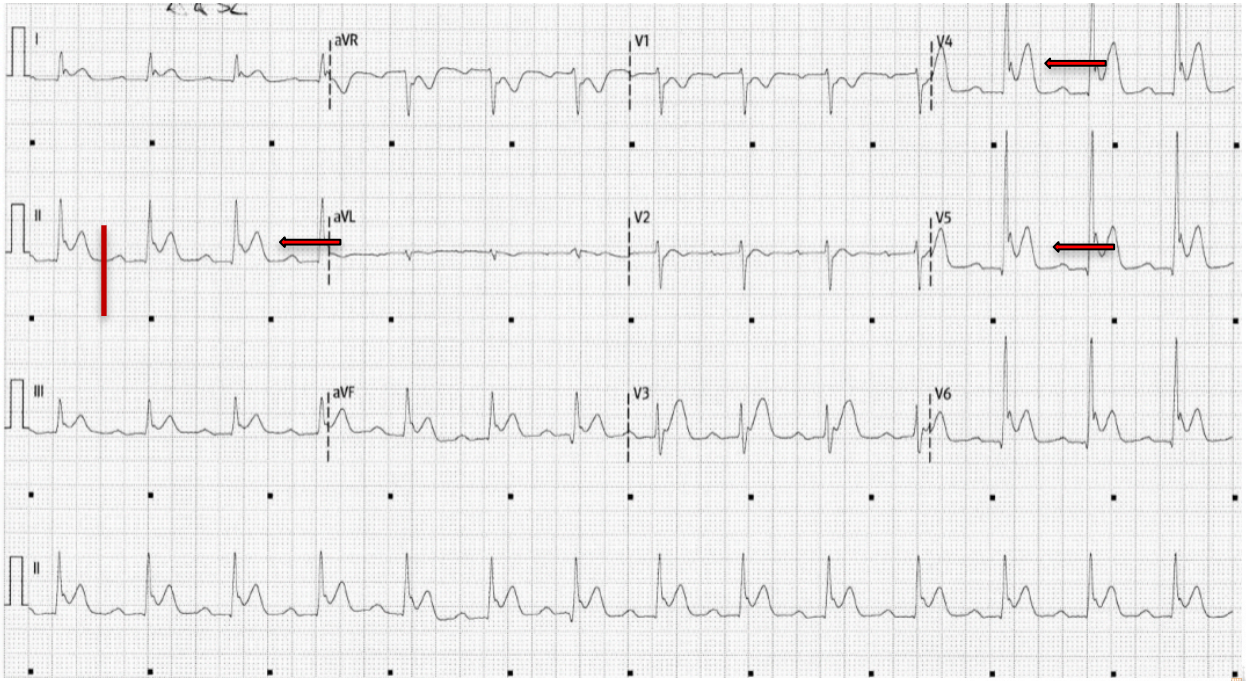
Case Presentation

HR = 83 bpm PR = 272 ms QT = 318 ms QTc = 358 ms

A manifest early repolarization pattern was observed at admission, with an associated relatively short QTc (358 ms).

Coronary angiography and cardiac MRI resulted negative.

Blood tests revealed overt hyperthyroidism, so anti-thyroid therapy and metoprolol were started.

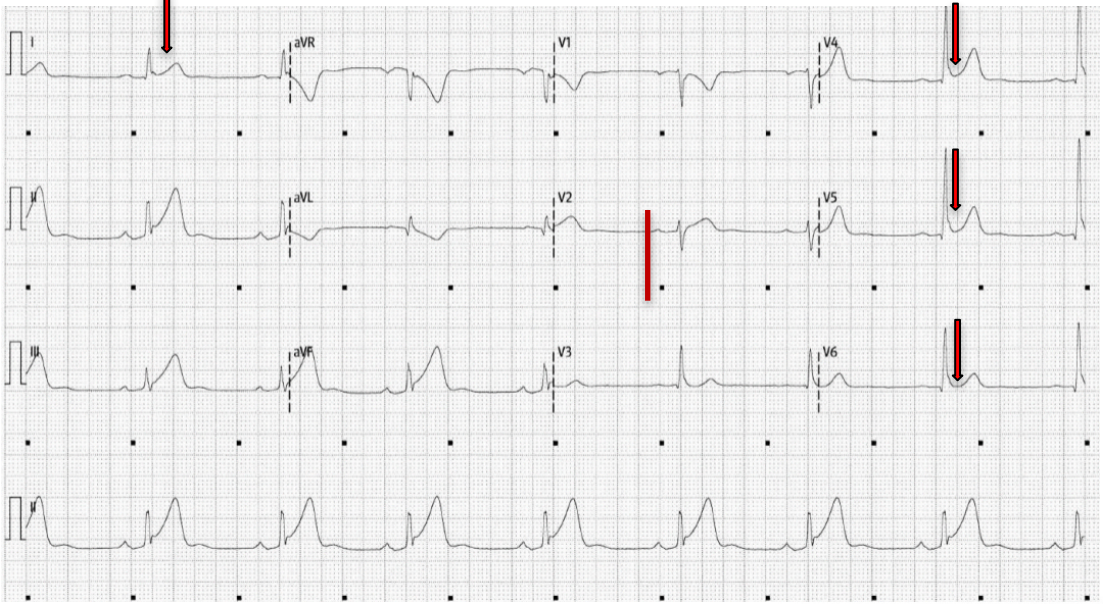


Case Presentation

Thyroid function improvement and normalization ran parallel to the ECG evolution: progressively, the J wave reduced its amplitude, the QTc interval reached normal values, while the ERP was still present.

An influence of hyperthyroidism in triggering life-threatening arrhythmias was considered. Anyway, given the persistence of the ERP and of 2nd AVB episodes, despite the normalization of thyroid function, suggested high probability of arrhythmic recurrence, and a potential genetic substrate. That's why a trans-venous ICD was considered, to guarantee overdrive pacing.

HR = 47 bpm PR = 220 ms QT = 430 ms QTc = 407 ms

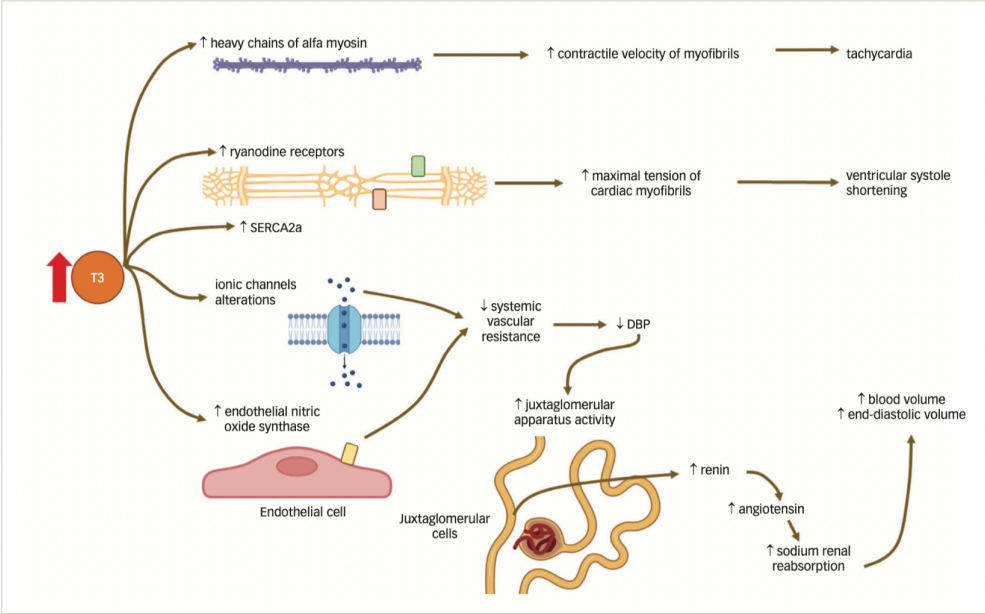


>>> Genetic test is under way.

Discussion

Thyroid hormones are potential modifiers of ventricular repolarization, throughout genomic and nongenomic mechanisms.

Positively Regulated Genes	Negatively Regulated Genes
α-myosin heavy chain	β-myosin heavy chain
Sarcoplasmic reticulum Ca^{2+} ATPase	Phospholamban
Na^{+}/K^{+} ATPase	Adenylyl cyclase catalytic sub-units
β1-adrenergic receptor	Na^{+}/Ca^{2+} exchanged
Voltage-gated potassium channels	Thyroid hormone receptor α1



DBP = diastolic blood pressure; SERCA2a = sarco/endoplasmic reticulum Ca^{2+} ATPase 2; T3 = triiodothyronine.

Quiroz-Aldave JE, touchREV Endocrinol. 2023

Discussion

How hyperthyroidism modifies ventricular repolarization?

Both QTci prolongation and worsening of an early repolarization pattern (ERP) have been described in hyperthyroidism, as a basis for VF but nowadays no QTci shortening was reported as a cause for VF.

Case Report

QT Prolongation due to Graves' Disease

Zain Kulairi, Nisha Deol, Renee Tolly, Rohan Manocha, and Maliha Naseer

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CASE REPORT

Ventricular fibrillation associated with early repolarization in a patient with thyroid storm

Akira Ueno · Takeshi Yamamoto · Naoki Sato · Keiji Tanaka

Discussion

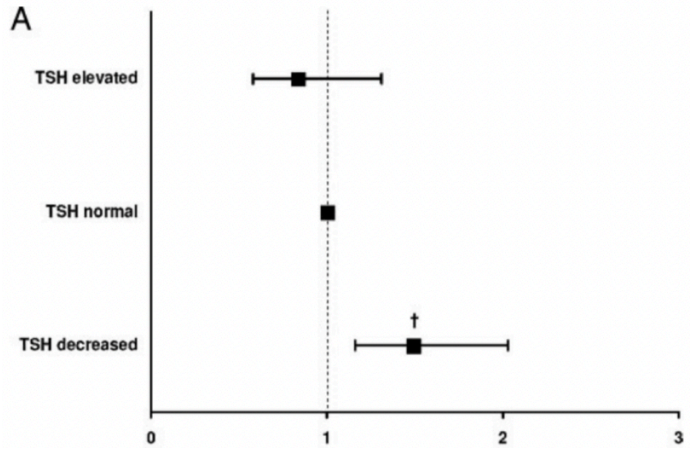
QTci prolongation was observed in small sample reports.
A significative shorter QTci in correlation with low TSH levels was found in a consistent population study (excluding overt hyperthyroid status).

The exacerbation of the epicardium-endocardium transmural voltage gradient has been correlated to ERP related VF during thyrotoxicosis. Circulating FT3 can contribute to the modulation of the transient outward current (Ito).

The association of a ERP with a short QTc interval and VF has never been described before, so the overlap between genetic and hormonal arrhythmic mechanism was suspected.

BRIEF REPORT

The Relation of Thyroid Function and Ventricular Repolarization: Decreased Serum Thyrotropin Levels Are Associated with Short Rate-Adjusted QT Intervals

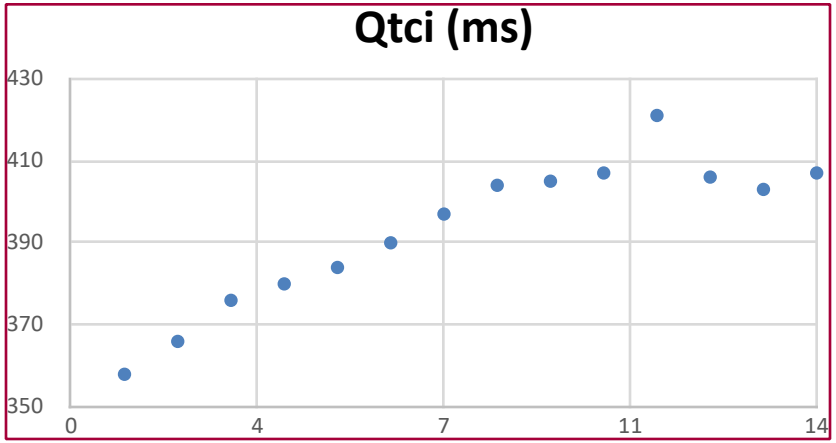


Dorr et al. J Clin Endocrinol Metab. 2006

Discussion

β-Blockers and parasympathetic activation can exacerbate the J-point elevation. That could make treatment of thyroid storm more challenging in patients with ERP.

In our case, the ECG evolution guided the therapeutic approach: β-Blockers were withdrawn during the follow-up, when the patient reached euthyroid status, without arrhythmic recurrence.



Conclusions

- This case reminds us that thyrotoxicosis can rarely cause life-threatening arrhythmias, even in the absence of underlying structural heart disease;
- The behavior of the repolarization process during thyroid storm is varying;
- This may depend on the combination of genomic and non-genomic effects of thyroid hormones, on the level of circulating TSH, T4 and T3, on the membrane density of β-adrenergic receptors, on the catecholaminergic and androgenic sensitivity.
- This is the first description of a ventricular fibrillation in association with a relatively short QTc interval, ERS and hyperthyroidism;

- We’re waiting for genetic test that could lead to the identification of a novel type of ERS with a particular hormone – sensitivity. .

GRAZIE PER L'ATTENZIONE

Like an electrical current through my veins