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When Syncope Looks Like Epilepsy: A Case Report of, AV Dissociation Causing Vasovagal like Collapse in Hypothyroidism

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Abstract

To describe the difference between syncope and epileptic seizures is tricky, especially when patients have convulsive movements. Doctors sometimes get it wrong, which means people end up taking seizure medications they don't need while the real issue goes untreated. Every so often, an underlying problem like hypothyroidism triggers trouble with the autonomic nervous system and leads to syncope.

Case Presentation: We saw a patient would have epilepsy, but it turned out she was having vasovagal-type syncope related to her untreated hypothyroidism. What helped us figure it out? We used video-EEG, ECG, tilt table tests, and checked her thyroid. This made the real diagnosis clear and shaped her treatment plan.

Discussion: To discuss why this happens! Reflex syncope comes down to sudden changes in the autonomic nervous system that drop blood pressure and slowdown the heart rate. Sometimes, these episodes look a lot like seizures because of the muscle jerks or stiffening. New research (from 2020 to 2025) shows thyroid problems especially hypothyroidism throw off the body's autonomic balance, making vasovagal syncope more likely to happen. To investigate syncope apart from epilepsy, we need to dig into the patient's history, run EEG and ECG at the same time, and do autonomic function tests. We've included diagnostic flowcharts and helpful figures.

Conclusion: When someone has unusual seizures or doesn't respond to seizure meds, doctors need to think about syncope mimics especially if there's a thyroid issue. Thyroid dysfunction can push people toward reflex syncope, so a step-by-step approach (history, tilt table, EEG/ECG together) is key.

Keywords: ***Syncope; Vasovagal syncope; Epileptic seizure mimics; Hypothyroidism; Autonomic nervous system dysfunction; Convulsive syncope; Video-EEG monitoring; Tilt table test; Electrocardiography; Misdiagnosis of epilepsy***

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1. Introduction

People come in with sudden blackouts all the time, but figuring out why isn't always easy. Most of them think "epilepsy" right away, but vasovagal syncope where the nervous system overreacts can look just like a seizure, especially if there's jerking or muscle stiffening. Lately, studies are showing that hypothyroidism messes with the body's nerve signals, making vasovagal episodes more likely to happen.

2. Case Presentation

A 32-year-old, right-handed woman patient was sent to the hospital for a severe headache at L occipital area, gripping in nature but did not seem to radiate anywhere else. Denies photophobia, neck stiffness/fever. Systems review says no chest pain/SOB palpitation, no bowel motion changes, and no dysuria. With no family history of HTN and T2DM, hyperlipidemia. The patient initially admitted under the care of neuro team and subsequently transfer of care of cardiology, EEG was normal CT brain was done shown normal study. There is no established infarct or acute intracranial hemorrhage seen. No mass effect or hydrocephalus is demonstrated. She was referred to cardiology team as her ECG showed intermittent AV dissociation with LBBB pattern (FIG. 1a, b). Cardiac enzymes were normal. Electrolytes, including calcium/magnesium/phosphate were normal. Her TFTs show low TSH with normal T3 and T4 (she is on thyroxin replacement for hypothyroidism). Her echocardiogram report shows IVS/AVS are intact. She was moved to cardiac care unit for further monitoring (FIG. 2). While inpatient, she has one episode of giddiness with the blurring of vision, no palpitation/sweating/nausea/LOC. The patient was put on holter which showed intermittent AV dissociation, min hour 43 Max HR 135, No sig pause (1.99sec), subsequently patient was put on continuous monitoring. Telemetry which again showed intermittent AV dissociation. Before patient was discharged Neuro team reviewed once more as a differential to patient's presentation is an underlying epileptic disorder. Neuro-team unconvinced that it is a seizure disorder. According to her previous history, she has been on thyroxin 100 µg (daily) for 8 years. From last 3 years, she was having syncope during sleep. This syncope/seizure last for 1 - 2 minutes occurred at a frequency of three to five in a year.

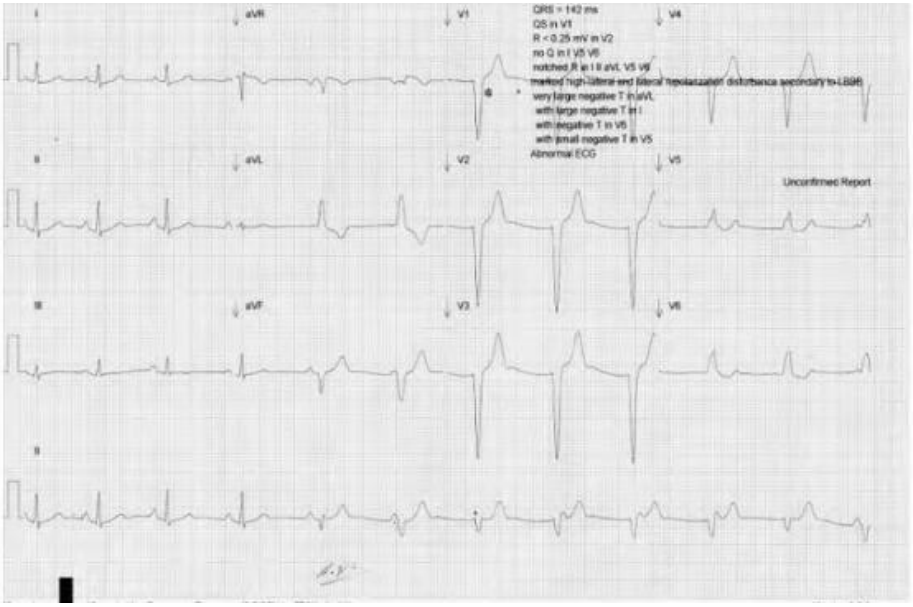

FIG. 1a. **ECG showing runs of ventricular complexes with left axis deviation (Heart rate: 66 bpm).**

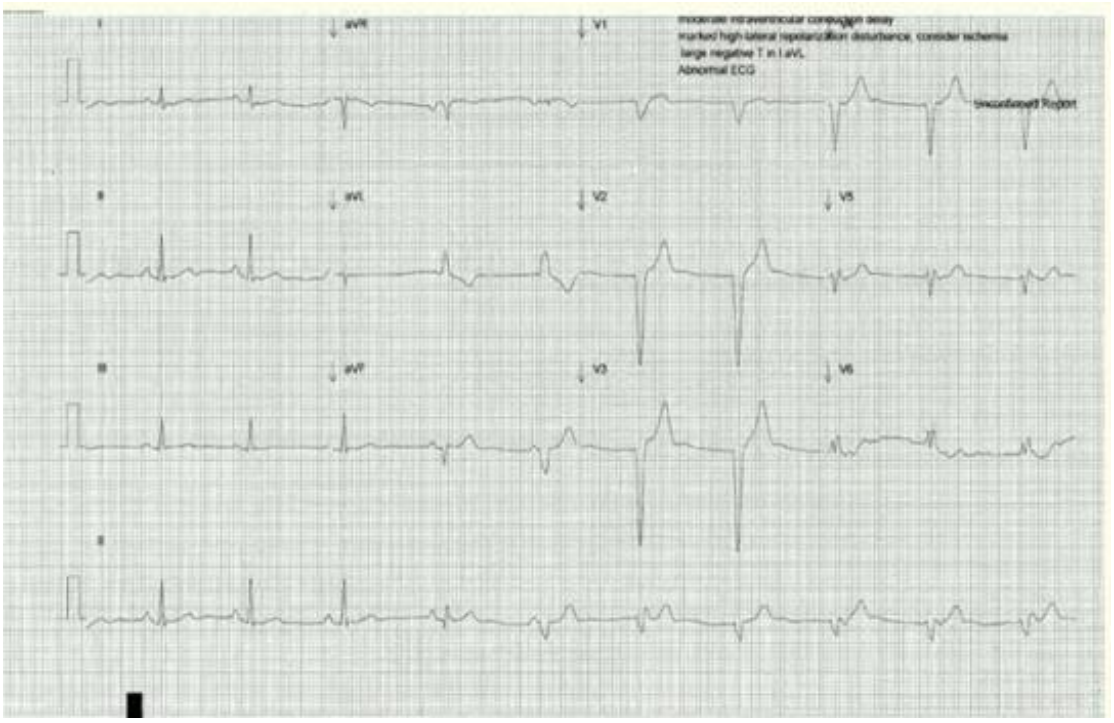

FIG. 1b. **ECG with different types of premature ventricular complexes, also with left axis deviation (Heart rate: 61 bpm).**

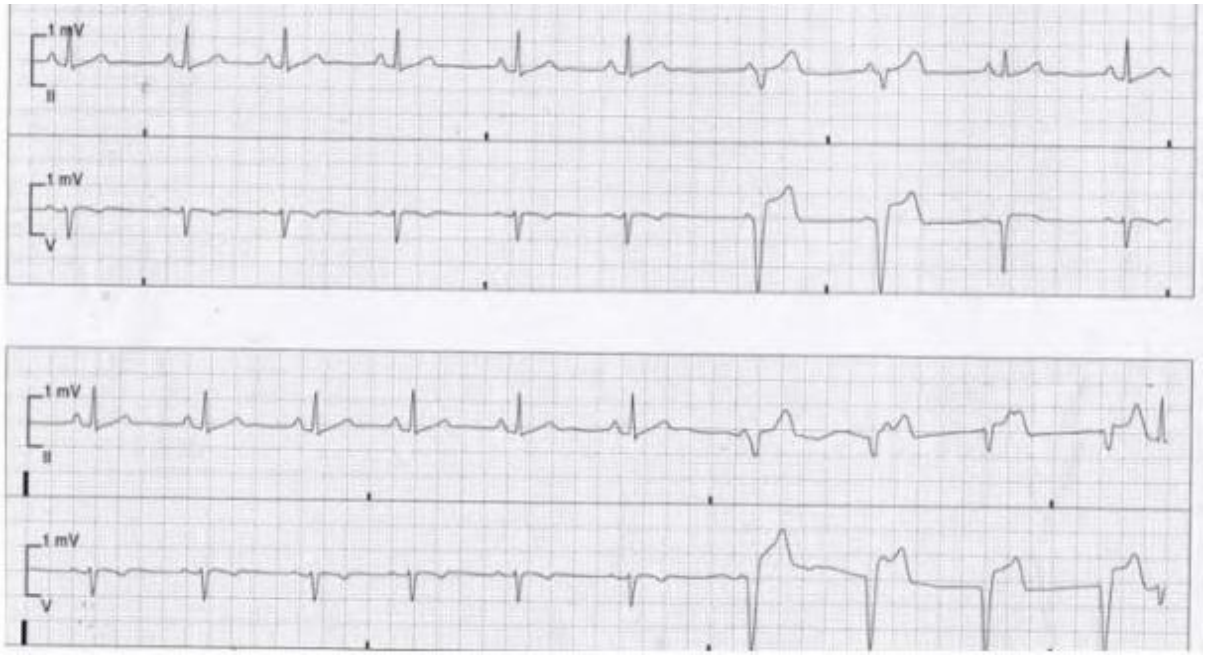

FIG. 2. **ARR, CPT, STIII 0.3mm, STV 0.3mm, NBP D…, RESP 39br/m HR 57 BPM, PVC/ min 0 BPM STaVL -0.1MM NBP S…., NBP M.**

Holter monitor tracing showing intermittent AV dissociation in a hypothyroid patient.

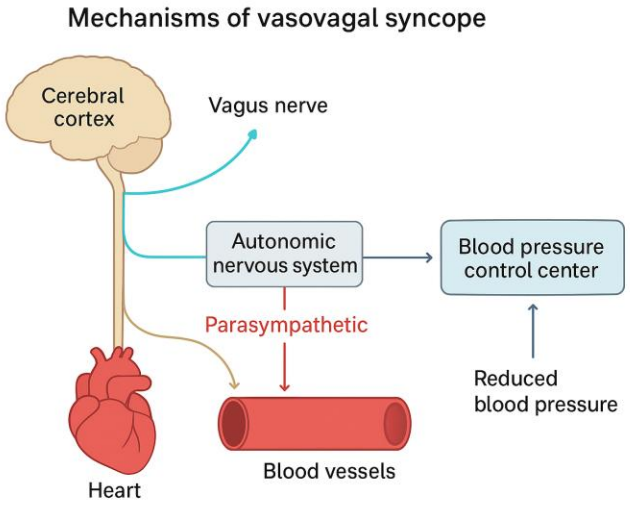

FIG. 3. **Diagram showing how vasovagal syncope happens nervous system imbalance triggers bradycardia and low blood pressure, starving the brain of blood for a moment.**

TABLE 1. **Effect of Thyroid Hormone on Cardiac Gene Expression. Adopted from [13].**

Positively regulated	**Negatively regulated**
αMyosin heavy chain Sarcoplasmic reticulum Ca2+-ATPase	βMyosin heavy chain
Sarcoplasmic reticulum Ca2+-ATPase	Phospholamban
Na+/K+ATPase β1-Adrenergic receptor	Adenylyl cyclase catalytic subunits
β1-Adrenergic receptor Negatively Regulated	Thyroid hormone receptor _1
Atrial natriuretic hormone	Na+/Ca2+ exchanger
Voltage-gated potassium channels	
(Kv1.5, Kv4.2, Kv4.3)	

TABLE 2. **Cardiovascular Risks Associated with Hypothyroidism. Adopted from [13].**

Impaired cardiac contractility and diastolic function
Increased systemic vascular resistance
Decreased endothelial-derived relaxation factor
Increased serum cholesterol
Increased C-reactive protein
Increased homocysteine

3. Discussion

We report a case of atrioventricular block associated with cardiogenic syncope in a patient with an eight-year history of hypothyroidism. Despite being on thyroxine replacement therapy, the patient experienced recurrent syncopal episodes, with Holter monitoring and continuous telemetry revealing intermittent AV dissociation. This case highlights the complexity of

managing conduction disturbances in patients with long-standing thyroid deficiency, as the neurology team ruled out seizure activity, pointing toward a primary cardiac etiology [3-5].

The relationship between thyroid status and cardiac rhythm is rooted in the central role thyroid hormone plays in cardiac gene expression. Both structural and functional genes in cardiomyocytes are mediated by thyroid hormone through thyroid hormone receptor dependent transcriptional regulation, which influences contractile proteins, calcium-handling proteins, and various ion channels [1,2,6,7]. Specifically, thyroid hormone regulates the action potential in cardiac myocytes by modulating electrophysiological properties and the expression of ion channels [2,6]. The sinoatrial node, serving as the physiological pacemaker, relies on pacemaker-related genes such as hyperpolarization-activated cyclic nucleotide-gated channels 2 and 4, which are transcriptionally regulated by thyroid hormone [8]. Consequently, alterations in thyroid status can significantly disrupt pacemaker activity and heart rate regulation [7,8].

Hypothyroidism induces a state of decreased cardiac contractility, reduced cardiac output, and increased systemic vascular resistance [1,9,10]. These changes, alongside impaired diastolic relaxation and accelerated atherosclerosis, stem from altered cardiac gene expression and diminished electrophysiological stability [2,6,9]. While hypothyroidism is recognized as a reversible cause of conduction abnormalities, including complete AV block and ventricular arrhythmias, the precise mechanism remains incompletely understood [3,4,11]. It likely involves a combination of myocardial edema, altered autonomic tone, interstitial fibrosis, and ion channel remodeling [6,7].

In clinical practice, cardiac syncope or AV block typically occurs in elderly patients or those with severe, untreated hypothyroidism [3,4,12]. The observed incidence of AV dissociation in these states appears fundamentally linked to metabolic derangements that facilitate nodal inflammation or infiltration [12]. Furthermore, age-related AV node calcification may act as a predisposing substrate that, when coupled with the biochemical milieu of hypothyroidism, increases the likelihood of high-grade conduction blocks [10,12]. Additionally, thyroid hormone deficiency may modulate autonomic tone, further inhibiting nodal depolarization and contributing to symptomatic bradyarrhythmias [7].

Evidence suggests that correcting the underlying thyroid hormone deficiency typically resolves these conduction abnormalities, implying that metabolic restoration can restore normal atrioventricular synchrony [3,4]. However, clinical vigilance is required, as refractory cases such as the one described here may indicate concurrent electrolyte imbalances or underlying subclinical conduction system disease necessitating further diagnostic investigation [11]. In severe cases of myxedema, it is crucial to distinguish between transient metabolic conduction delay and irreversible cardiac injury, as the latter may necessitate permanent pacemaker implantation if sinus rhythm fails to recover following the achievement of a euthyroid state [9]. In this case, treatment is directed toward identifying and correcting the underlying cause of the syncopal events to prevent further morbidity or mortality [3,4].

4. Conclusion

This case drives home the importance of spotting vasovagal syncope in patients who seem to have epilepsy, especially if they have thyroid problems. Getting the diagnosis right means people avoid unnecessary seizure meds and instead get the right treatment for their actual problem, autonomic instability.

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