Ventricular tachycardia unveiling severe undiagnosed hypothyroidism

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Hypothyroidism is typically associated with bradyarrhythmias, but can rarely precipitate life-threatening ventricular arrhythmias. We present a case of severe hypothyroidism manifesting as polymorphic ventricular tachycardia (VT). A previously healthy woman in her early 50s presented with an acute onset of breathlessness and on examination had hypotension and tachycardia. ECG revealed polymorphic VT, promptly terminated by defibrillation using 200J biphasic shock. Investigations uncovered severe primary hypothyroidism (thyroid-stimulating hormone: 142 mIU/l) and left ventricular (LV) dysfunction with ejection fraction (EF) of 35%. Coronary angiogram was normal. Treatment with levothyroxine and standard heart failure therapy was initiated. In conclusion, at 3- and 6-month follow-ups, the patient remained asymptomatic and had no episodes of tachyarrhythmias without antiarrhythmic drugs, and her LV function normalized (EF: 55%). This case highlights the importance of considering hypothyroidism in patients presenting with unexplained ventricular arrhythmias. ***Cardiovasc Endocrinol Metab*** **14: 1–4**

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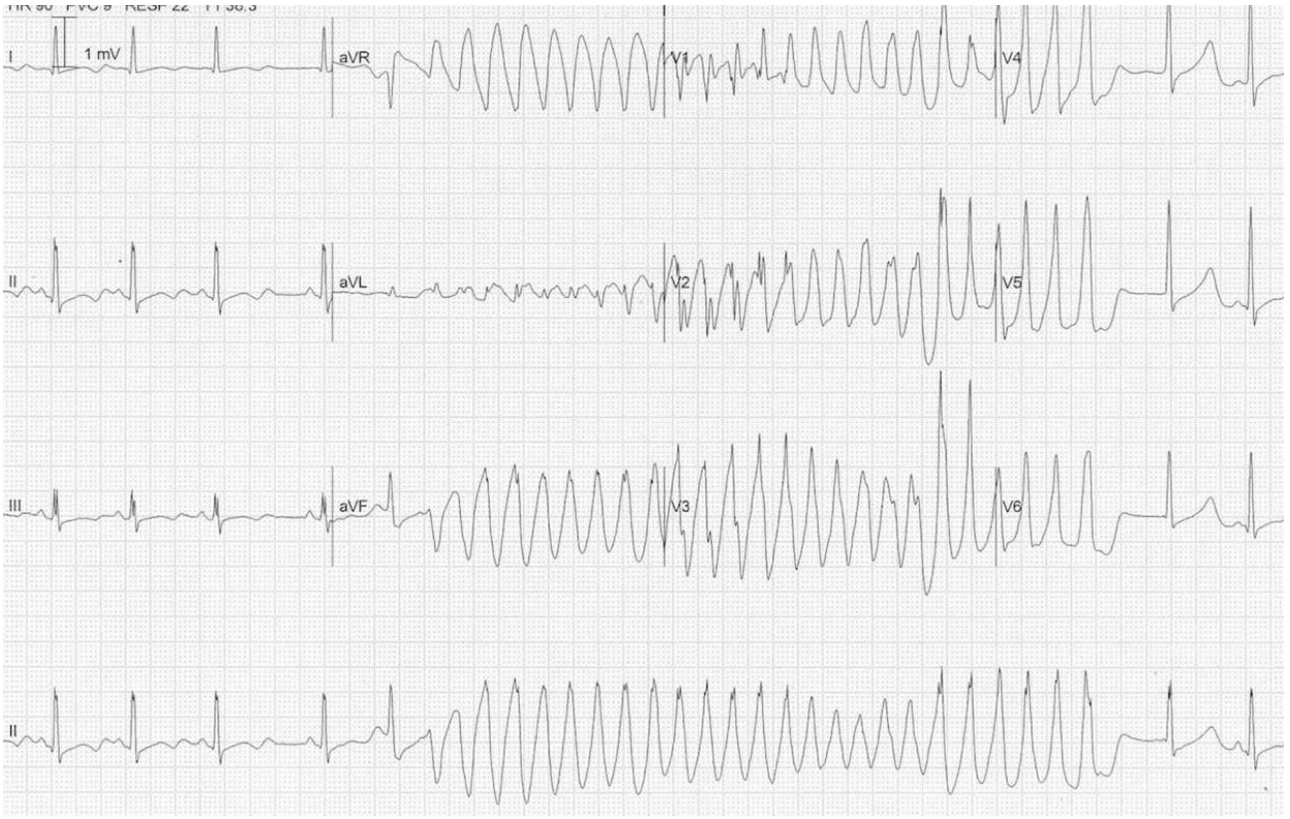
Background

Thyroid hormones play a crucial role in regulating various aspects of normal cardiac function, including myocardial contractility, heart rate, and ventricular repolarization, through both genomic and nongenomic actions [1]. Overt hypothyroidism is diagnosed when thyroid-stimulating hormone (TSH) levels are above 10 mIU/l with low triiodothyronine (T3) and thyroxine (T4) levels. The burden of thyroid disorders in India is on the rise, with the current community prevalence of hypothyroidism being 10.9% [2]. While hypothyroidism is commonly associated with bradyarrhythmias, conduction abnormalities, and impaired myocardial performance, the manifestation of ventricular arrhythmias in this condition is relatively rare [3,4]. The prevalence of any ventricular arrhythmias in euthyroid individuals is 1.31%, in hypothyroid individuals is 6.58%, and ventricular tachycardia (VT) in hypothyroid individuals in particular is 2.6% [5]. Various potential mechanisms, including dysfunctional potassium, calcium channel activity, prolonged QT interval, diminished heart rate variability, and disrupted sympathetic-parasympathetic balance, synergistically contribute to increased vulnerability to ventricular arrhythmias [1,3,4,6]. The association between hypothyroidism and ventricular tachyarrhythmias, particularly sustained VT, remains an underrecognized and underreported phenomenon. This case report highlights the rare and potentially life-threatening complication of sustained VT precipitated by severe, undiagnosed hypothyroidism in an otherwise healthy middle-aged woman. It emphasizes the importance of timely detection and treatment of thyroid dysfunction to prevent such adverse cardiovascular events and improve patient outcomes.

Case presentation

A woman in her early 50s with no prior comorbidities presented to the emergency department with acute onset of breathlessness. On examination, the patient was in altered sensorium with cold extremities, SBP of 60 mmHg, heart rate of 190 beats per minute, and respiratory rate of 32 cycles per minute. Her medical and drug history was unremarkable, with no prior cardiac or thyroid-related issues. The ECG demonstrated rapid, irregular wide QRS complexes with varying morphology and amplitude, consistent with polymorphic VT (Fig. 1). Given the patient's hemodynamic instability, immediate defibrillation with 200 J of biphasic shock was performed, successfully converting the rhythm to

Fig. 1



At admission, ECG showing polymorphic ventricular tachycardia (VT).

sinus. Subsequently, intravenous magnesium sulfate 2 g bolus over 15 min was administered to stabilize the myocardium and prevent arrhythmia recurrence [7,8]. The patient was then transferred to the ICU for close monitoring.

The laboratory investigations revealed a normal complete blood count, liver function tests, renal function tests, serum electrolytes, magnesium, and calcium levels. The lipid profile, however, was deranged, with a total cholesterol of 510 mg/dl [reference range (RR): 150–220], triglycerides of 615 mg/dl (RR: 25–160), low-density lipoprotein of 285 mg/dl (RR: 100–150), and high-density lipoprotein of 102 mg/dl (RR: 30–70). Significantly, the thyroid profile revealed markedly elevated levels of TSH at 142 mIU/l (RR: 0.5–4.5), low levels of T3 at 33 ng/dl (RR: 70–190), low levels of thyroxine (T4) level at 0.73 mcg/dl (RR: 5–11), and thyroid peroxidase antibodies measured at 1000 IU/ml (RR: <30). These findings indicated severe primary hypothyroidism of autoimmune etiology, which had, hitherto gone unrecognized. The chest X-ray was normal, and the 2D echocardiogram showed left ventricular (LV) global hypokinesia with an ejection fraction (EF) of 35%. The treatment for heart failure with reduced EF was initiated and Levothyroxine was started at a low dose of 0.8 mcg/kg bodyweight. The patient stabilized, and no further VT episodes were documented over the next 24 h. Coronary angiogram revealed normal epicardial arteries.

Outcome and follow-up

No further ventricular ectopy was documented across 48-hour monitoring while inpatient and continuous ECG telemetry confirmed a maintained normal sinus rhythm. The patient was discharged in a stable condition on levothyroxine. At 6-month follow-up, T3 was 102 ng/dl, T4 was 7.5 mcg/dl, and TSH was 2.5 mIU/l and the patient was asymptomatic with no recurrence of arrhythmias and the cardiac contractility improved and 2D echocardiogram showed normal LV systolic function with an EF of 55%.

Discussion

Hypothyroidism has been strongly associated with various cardiovascular abnormalities including cardiac arrhythmias [1,2,4,5]. Only a few case reports have shown the occurrence of VT and rare forms of polymorphic VT-Torsades de pointes (TdP) in the background of severe hypothyroidism [9–12]. Our case highlights the link between overt hypothyroidism and the risk of developing VT, in the absence of an underlying structural heart

disease. The pathophysiological mechanisms underlying this association between hypothyroidism and cardiac arrhythmias have been extensively elucidated with thyroid hormones T3 and T4 having wide-ranging regulatory effects on cardiovascular hemodynamics as well as cardiac ion channels [1,3].

Thyroid hormone T3 directly regulates the expression and functioning of vital cardiomyocyte ion channels such as sodium, calcium, and potassium channels [1]. Hypothyroidism-induced calcium and potassium channel dysfunction can abnormally prolong ventricular repolarization, as manifested by QT interval prolongation on ECG [4]. This creates an arrhythmogenic substrate for early afterdepolarizations that can trigger polymorphic VT and TdP [9–12]. Additional attributed mechanisms are imbalanced autonomic regulation, diminished heart rate variability, and increasing susceptibility to ventricular irritability. Dyslipidemia, a common feature of hypothyroidism, further exacerbates the arrhythmogenic risk by destabilizing cardiac cell membranes, altering ion channel activity, and promoting oxidative stress, as described in previous studies [1,9]. The risk for adverse cardiovascular events and arrhythmias rises incrementally with greater severity and duration of thyroid deficiency. Hypothyroidism, which leads to a state of cardiovascular depression with decreased myocardial contractility, when untreated can lead to reduced EF and heart failure as seen in our case [1,5,6,13].

Our patient had polymorphic VT that reverted to sinus rhythm with defibrillation. The 2D echocardiogram showed LV systolic dysfunction with a reduced EF of 35% along with global hypokinesia. Coronary angiography, however, revealed normal epicardial arteries. Upon extensive workup, VT precipitated by overt hypothyroidism was noted. With the initiation of levothyroxine replacement, the patient remained free of recurrent ventricular arrhythmias throughout the hospitalization with ECG telemetry. At 3- and 6-month follow-up, she continues to be asymptomatic and a repeat 2D echocardiogram revealed normal LV function with EF of 55%. This outcome supports the reversibility of both electrical instability and myocardial contractile depression in our case of severe hypothyroidism. It highlights the significant chronotropic and ionotropic influences exerted by optimal thyroid hormone levels for maintaining cardiac function [1,6].

Akin to the case reported by Goyal *et al.* [10], where severe hypothyroidism manifested as polymorphic VT, the underlying pathophysiological derangements likely involved electrolyte imbalances, dysfunctional cardiac ion channels, and prolonged ventricular repolarization, all exacerbated by the profound thyroid hormone deficiency. Consistent with the findings of Kandan and Saha [11], who documented TdP in severe hypothyroidism, our case highlights how the electrophysiological instability induced by hypothyroidism, characterized by impaired repolarization reserve and early afterdepolarizations, can predispose individuals to potentially lethal ventricular arrhythmias.

Interestingly, despite severe hypothyroidism, our patient did not exhibit pericardial effusion or myxedema, which are commonly associated with chronic thyroid hormone deficiency. Pericardial effusion in hypothyroidism typically results from increased capillary permeability and impaired lymphatic drainage, leading to fluid accumulation in the pericardial sac [14]. Several factors, however, might have prevented its development in this patient, including early detection of thyroid dysfunction, shorter disease duration, and residual thyroid function. Dutta *et al.* emphasize that thyroid symptomatology varies significantly across individuals, with certain patients avoiding complications due to favorable physiology or early intervention [15]. Similarly, myxedema, a life-threatening complication characterized by widespread tissue fluid accumulation and neurological involvement, typically occurs in longstanding untreated hypothyroidism, often triggered by infections or other stressors [16]. The absence of such precipitating factors, combined with the timely initiation of levothyroxine, likely explains why our patient did not progress to myxedema. This variability in clinical presentation underscores the importance of individualized assessment and management in hypothyroid patients.

The TSH cutoffs and exact thyroid hormone levels that increase arrhythmic risk and warrant preemptive treatment remain unclear. Screening ECGs for corrected QT interval (QTc) prolongation and Holter monitoring could help improve the risk stratification of such patients [4,12]. This case reinforces the need for increased clinical awareness and early testing for thyroid dysfunction in patients presenting with unexplained QTc interval changes, arrhythmias. Prompt diagnosis and appropriate treatment are life-saving in hypothyroid patients at risk for lethal cardiac arrhythmias. Further studies are warranted to better characterize at-risk groups and define optimal monitoring protocols and thyroid targets. Still more enhanced understanding of arrhythmia pathophysiology in hypothyroidism can help refine treatment and prevention strategies.

Limitations

(1) Cardiac MRI was not performed due to financial constraints.
(2) Since the patient responded favorably to the initial treatment and no further episodes of arrhythmias occurred after initiating levothyroxine replacement therapy, the clinical team decided that additional investigative procedures were not warranted.
(3) Advanced metabolic testing, including markers of oxidative stress and lipid profiling beyond

routine assessments, was not conducted, limiting the ability to evaluate the full role of dyslipidemia in arrhythmogenesis.

Conclusion

In our patient, severe hypothyroidism presented as life-threatening VT without overt cardiac abnormalities or complications like pericardial effusion or myxedema. This case illustrates the potential for thyroid dysfunction, compounded by dyslipidemia, to cause unexplained VT in apparently healthy individuals. In our experience, timely recognition and treatment of hypothyroidism prevented the recurrence of the arrhythmia and normalized cardiac function. While our findings cannot be generalized, they suggest that consideration of thyroid function in cases of unexplained ventricular arrhythmias may be warranted.

Learning points/take home messages

(1) Hypothyroidism most commonly causes bradyarrhythmias, but it can rarely precipitate VT in patients with no prior cardiac history.
(2) The mechanism of VT in hypothyroidism involves dysregulation of cardiac ion channels, leading to prolonged ventricular repolarization, and triggered activity can precipitate VT.
(3) Dyslipidemia, a common feature of hypothyroidism, can exacerbate arrhythmic risk by promoting oxidative stress and destabilizing cardiac ion channel function.
(4) Unexplained QTc interval prolongation, arrhythmias, or ECG changes, especially without cardiac risk factors, should prompt screening for thyroid dysfunction.

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Patient consent was obtained.

Conflicts of interest

There are no conflicts of interest.

References

1 Jabbar A, Pingitore A, Pearce SH, Pearce S, Zaman A, Lervasi G, Razvi S. Thyroid hormones and cardiovascular disease. *Nat Rev Cardiol* 2017; **14**:39–55.
2 Unnikrishnan A, Kalra S, Sahay R, Bantwal G, John M, Tewari N. Prevalence of hypothyroidism in adults: an epidemiological study in eight cities of India. *Indian J Endocrinol Metab* 2013; **17**:647–652.
3 Klein I, Ojamaa K. Thyroid hormone and the cardiovascular system. *N Engl J Med* 2001; **344**:501–509.
4 Aravind GN, Archita R. Evaluation of the QTc interval and QT dispersion in patients with newly detected clinical hypothyroidism. *Int J Adv Med* 2019; **6**:1416–1420.
5 Kannan L, Kotus-Bart J, Amanullah A. Prevalence of cardiac arrhythmias in hypothyroid and euthyroid patients. *Horm Metab Res* 2017; **49**:430–433.
6 Yamakawa H, Kato T, Noh J, Yuasa S, Kawamura A, Fukuda K, *et al*. Thyroid hormone plays an important role in cardiac function: from bench to bedside. *Front Physiol* 2021; **12**:606931.
7 Zeppenfeld K, Hansen J, Riva M, Winkel BG, Behr E, Blom N, *et al*. 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. *Eur Heart J* 2022; **43**:3997–4126.
8 Tzivoni D, Banai S, Schuger C, Benhorin J, Keren A, Gottlieb S, Stern S. Treatment of torsade de pointes with magnesium sulfate. *Circulation* 1988; **77**:392–397.
9 Schenck JB, Rizvi AA, Lin T. Severe primary hypothyroidism manifesting with Torsades de Pointes. *Am J Med Sci* 2006; **331**:154–156.
10 Goyal B, Yadav V, Jhanwar S, Patel S, Kaushik A. Severe primary hypothyroidism presenting as polymorphic ventricular tachycardia. *Rev Colomb Cardiol* 2022; **29**(Suppl 4):34–37.
11 Kandan SR, Saha M. Severe primary hypothyroidism presenting with Torsades de Pointes. *BMJ Case Rep* 2012; **2012**:bcr1220115306.
12 Lee B, Lee WF, Lim BL. Rare case of Torsades de Pointes in severe hypothyroidism: literature review and challenges in management. *Int J Emerg Med* 2022; **15**:11.
13 Dillmann WH. Cellular action of thyroid hormone on the heart. *Thyroid* 2002; **12**:447–452.
14 Kabadi UM, Kumar SP. Pericardial effusion in primary hypothyroidism. *Am Heart J* 1990; **120**:1393–1395.
15 Dutta D, Garg A, Khandelwal D, Kalra S, Mittal S, Chittawar S. Thyroid symptomatology across the spectrum of hypothyroidism and impact of levothyroxine supplementation in patients with severe primary hypothyroidism. *Indian J Endocrinol Metab* 2019; **23**:373–378.
16 Velez-Oquendo G, Kilaru V, Ye K, Ashfaq S. Large pericardial effusion secondary to generalized myxedema from undiagnosed Hashimoto's thyroiditis. *Am J Case Rep* 2023; **24**:e940631.