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CHRONIC POSTOPERATIVE HYPOPARATHYROIDISM DECOMPENSATED BY A RELAPSE OF THYROTOXICOSIS AND COVID-19: A CASE REPORT

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Abstract

Relevance. Chronic postoperative hypoparathyroidism is a rare complication of thyroidectomy. It needs lifelong therapy. But reports of decompensation caused by a combination of thyrotoxicosis, COVID-19, and long-term use of outdated drugs like dihydrotachysterol are few.

Objective. To describe a case of decompensated chronic postoperative hypoparathyroidism in a patient with Graves' disease - and to analyse what led to the decompensation, how we diagnosed it, and how we treated it.

Clinical Case. A 58-year-old woman presented in November 2023 with limb cramps, tachycardia, and tremor. She had a history of subtotal thyroidectomy for Graves' disease (1991), followed by hypoparathyroidism treated with dihydrotachysterol for over 30 years - without adequate control. In 2022, she had COVID-19 pneumonia (60 % lung involvement) and then a stroke, which left her with left-sided hemiparesis. On admission: total calcium 1.57 mmol/L, ionized calcium 0.61 mmol/L, phosphorus 1.64 mmol/L, PTH 6.78 pg/mL, plus decompensated thyrotoxicosis (free T4 29.45 pmol/L, TSH receptor antibodies 13.5 IU/L), a prolonged QT interval, and a cataract. We started alfacalcidol 1 μg/day, calcium carbonate 2000 mg/day, and thiamazole 30 mg/day. Over 15 months, her calcium normalised to 2.35 mmol/L, TSH receptor antibodies fell to 4.61 IU/L, and she became euthyroid. But then she stopped alfacalcidol on her own - because she felt well - and her calcium dropped again.

Conclusion. This case shows three things. First, dihydrotachysterol is outdated and should be replaced with active vitamin D metabolites. Second, COVID-19 can trigger calcium-phosphorus decompensation. Third, thorough biochemical monitoring is essential - and patients need to be warned not to stop alfacalcidol even when they feel completely well.

Keywords: *hypoparathyroidism, parathyroid hormone, hypocalcemia, hyperphosphatemia, thyroidectomy, alfacalcidol.*

Introduction

Postoperative hypoparathyroidism is a well-known complication of thyroid surgery. The parathyroid glands may be inadvertently removed, injured, or devascularised during neck surgery, particularly thyroid procedures [1]. Most clinicians see transient hypocalcemia early after surgery, but some patients go on to develop chronic disease. According to international guidelines, hypoparathyroidism is considered chronic when it persists for more than 12 months after surgery [2].

What surprised us in this case was not the diagnosis itself. Rather, it was the combination of three events unfolding over decades: chronic postoperative hypoparathyroidism with inadequate calcium replacement, a COVID-19 infection that was followed by a stroke and a relapse of thyrotoxicosis, and a severe decompensation of the chronic hypoparathyroidism.

Patients with Graves' disease are known to be at higher risk for this complication. Thyrotoxicosis increases thyroid vascularity, making surgery

more difficult and putting the parathyroid blood supply at risk [3]. Much less is known about long-term outcomes after subtotal resections - an approach that was common in the Soviet era and still affects many patients today [1].

Why does chronic hypoparathyroidism matter? Reduced bone resorption, decreased renal calcium reabsorption, impaired activation of vitamin D, and diminished intestinal calcium absorption all contribute to sustained hypocalcemia [4]. If poorly managed, this affects multiple organs. Patients with this condition have significantly higher risks of cardiovascular disease, chronic kidney disease, nephrolithiasis, seizures, and psychiatric disorders compared with matched controls. Intracranial calcifications and cataracts are also frequently reported [4]. Quality-of-life assessments using standardised instruments show substantially reduced scores in physical functioning, cognitive domains, and mental health [5].

Standard therapy relies on oral calcium supplementation and active vitamin D analogues. This approach alleviates symptoms of hypocalcemia, but it does not replicate the physiological actions of parathyroid hormone on the kidneys and skeleton. As a result, patients remain at risk for long-term complications including nephrocalcinosis, declining renal function, and disturbances in mineral metabolism [6].

Emerging evidence suggests an additional interaction between hypoparathyroidism and COVID-19. SARS-CoV-2 may affect parathyroid function through ACE-2 receptor-mediated mechanisms and cytokine-driven modulation of the calcium-sensing receptor [7]. Case reports have described both new-onset hypoparathyroidism during infection and significant destabilisation in patients with pre-existing disease [7]. Pre-existing parathyroid deficiency may also be associated with more severe clinical courses of COVID-19, including higher rates of intensive care admission and need for mechanical ventilation [8].

This case report describes a patient with chronic postoperative hypoparathyroidism who experienced a thyrotoxicosis relapse, severe COVID-19 pneumonia, and a cerebrovascular accident. We focus on the interactions between these conditions, the challenges of low treatment adherence, and the persistent use of outdated therapies in post-Soviet clinical practice. The case report has been prepared in accordance with the CAse REport (hereinafter – CARE) guidelines, and the completed CARE checklist is provided as supplementary material.

Case Report

A 58-year-old woman presented on 6 November 2023 with limb cramping, palpitations, hand tremor, anxiety, restlessness, shortness of breath (especially on exertion), fatigue, and weakness.

She was diagnosed with Graves' disease in 1991 at age 25. Due to inadequate disease control on antithyroid therapy, she underwent thyroid lobectomy and subsequently continued treatment with methimazole. In the early postoperative period, she developed persistent muscle cramps requiring repeated intravenous calcium chloride administration.

Between 2007 and 2015, the patient reduced her dose of antithyroid medication on her own. In 2015, she clinically deteriorated with weight loss, tachycardia, recurrent muscle cramps, and worsening general condition. In 2022, endocrinological evaluation led to the prescription of methimazole 10 mg, bisoprolol 2.5 mg, and dihydrotachysterol 0.3 mg/day; however, clinical improvement remained insufficient.

The patient's condition worsened significantly after she developed COVID-19 pneumonia involving approximately 60 % of lung parenchyma. In the same year, she suffered an acute cerebrovascular accident with subsequent development of left-sided hemiparesis.

Her past medical history is also notable for long-standing arterial hypertension diagnosed in 2000 (grade 3 hypertension), for which she is receiving bisoprolol 5 mg/day and candesartan 16 mg/day.

Family history: No history of thyroid or parathyroid disorders, autoimmune diseases, or early-onset cardiovascular disease in first-degree relatives.

Social history: The patient lives in an suburban area, is unemployed, and resides with her family. She has no history of smoking, alcohol abuse, or use of illicit substances. She reports a sedentary lifestyle due to residual left-sided hemiparesis.

Allergic history: No known drug or food allergies.

Physical Examination. On assessment, the

patient had a height of 160 cm, weight of 56 kg, and a body mass index of 21.9 kg/m². She appeared pale, with warm, dry skin and pale pink mucous membranes. The pulse rate was 118 beats per minute, and blood pressure was 100/70 mmHg. Hand grip strength was reduced bilaterally. A postoperative scar was observed on the anterior neck. The thyroid gland was mildly and asymmetrically enlarged (Grade I) and non-tender on palpation. Mild exophthalmos was also present. Neurological examination revealed postural instability on Romberg testing and a fine tremor of the hands on extension. A positive «telegraph pole» symptom was noted. Chvostek's and Weiss's signs were negative, whereas Trousseau's sign was positive.

Diagnostic Testing. Initial hormonal assessment on 6 November 2023 showed suppressed thyroid-stimulating hormone (hereinafter – TSH) at 0.029 mIU/L (reference range: 1.0–4.0 mIU/L), with elevated free thyroxine (fT4) at 29.45 pmol/L (10-25 pmol/L) and free triiodothyronine (hereinafter – fT3) at 7.4 pmol/L (3.1-6.8 pmol/L). Elevated anti-thyroid peroxidase antibodies and thyrotropin receptor antibodies were also detected (13.5 IU/L; reference <1.5 IU/L). Serum biochemistry on 17 November 2023 confirmed hypocalcemia, with total serum calcium of 1.79 mmol/L and ionized calcium of 0.95 mmol/L. Parathyroid hormone (hereinafter – PTH) was reduced at 0.72 pmol/L, accompanied by hyperphosphatemia (1.64 mmol/L). ECG on 9 November 2023 revealed sinus tachycardia with a heart rate of 102 beats per minute, left axis deviation, QT interval prolongation, ST-segment depression, and T-wave inversion (Figure 1).



Figure 1. ECG on 9 November 2023 showing sinus tachycardia, QT prolongation, and ST-segment depression.
Source: Original image from the patient's medical record

Thyroid ultrasound on 6 November 2023 showed a total thyroid volume of 21.1 cm³ (right lobe 4.8 cm³, left lobe 16.3 cm³) with diffuse parenchymal heterogeneity consistent with chronic thyroid disease. Eye examination on 15 November 2023 identified an incomplete complicated cataract.

Prolonged use of dihydrotachysteryl (hereinafter – DHT) without biochemical follow-up masked the chronic instability of calcium homeostasis. Thyrotoxicosis further confounded the presentation by transiently elevating serum calcium through increased bone resorption.

Final Clinical Diagnosis

Graves' disease with grade I goiter, relapse of thyrotoxicosis with decompensated course. Chronic postoperative hypoparathyroidism (since 1991), decompensated.

Treatment

The patient was treated with methimazole 30 mg/day (10 mg three times daily), alfacalcidol 1 μg/day, calcium carbonate 2000 mg/day, and bisoprolol 5 mg/day.

Follow-Up and Outcomes

One month after discharge (25 December 2023), labs (Table 1) showed partial stabilization of thyroid function and calcium–phosphorus metabolism. Clinically, the patient reported a marked improvement in general well-being, and episodes of muscle cramps and tetanic seizures had ceased.

At six months, labs indicated the development of biochemical hypothyroidism. Treatment was adjusted to include levothyroxine 50 μg/day while continuing methimazole 10 mg twice daily, al-

facalcidol 1.5 μg/day, and calcium supplementation 2000 mg/day. Regular monitoring of serum calcium, phosphorus, TSH, and free T4 was recommended.

On 18 November 2024, repeat thyroid function confirmed stable euthyroidism. Thyrotropin receptor antibody levels fell significantly (from 8.16 IU/L to 2.5 IU/L). However, hypocalcemia recurred, which was attributed to the patient's self-discontinuation of alfacalcidol. The alfacalcidol dose was subsequently increased.

In 2025, she remained euthyroid with stable serum calcium. She reported sustained improvement in her general condition. Continued therapy with alfacalcidol was strongly recommended, and long-term biochemical monitoring was advised (Table 1).

Table 1. Dynamics of Hormones and Biochemical Parameters

Indicator	06.11.23	15.11.23	25.12.23	03.06.24	15.11.24	02.02.2025	Normal Range
TSH	↓0.029		0.01	↑6.3	4	3.5	0.4-4.0 mIU/L
Free T4	↑24.3	↑29.45	17.50	↓3.6	18.9	17.01	9-19.05 pmol/L
Free T3	-	↑11.3	3.61	↓1.3	2.64	2.71	2-4.4 pg/mL
Anti-TPO	1109.37				528.09	1056	less than 5.6 IU/L
PTH		**↓6.78**	-	-	**↓9.85**	10.9	15-65 pg/mL
Anti-TSHR		13.5	8.16	-	2.5	4.61	less than 1.5 IU/L
Vitamin D		39,6			↓25.0		30-100 ng/mL
Total Calcium		**↓1.57**	**2.09**	**↓1.99**	**↓1.79**	2.35	2.25-2.75 mmol/L
Ionized Calcium		**↓0.61**	**0.97**	**0.93**	**↓0.95**	1.1	1.1-1.35 mmol/L
Phosphorus		**↑1.64**	-	-	**1.40**	1.35	0.87-1.45 mmol/L
Magnesium		0.84	0.91			0.85	0.73-1.03 mmol/L
Creatinine		48		100.2	68.9	72.78	44-97 μmol/L

**Note: Free T3 is reported in pg/mL*

Source: Table 1 is original and based on the patient's laboratory data collected from November 2023 to February 2025.

Discussion

Postoperative hypoparathyroidism is a well-known complication of thyroid surgery - in fact, the most common one [1]. What surprised us in this case was not the diagnosis itself, but the combination of three events unfolding over time: long-standing postoperative hypoparathyroidism with inadequate calcium replacement, a COVID-19 infection that was followed by a stroke and a relapse of thyrotoxicosis, and finally a severe convulsive syndrome.

Low compliance as a modifier of clinical outcomes. Between 2007 and 2015, the patient reduced her methimazole dose on her own initiative. She reported feeling well and saw no need to continue the medication at the same dose. By 2015, her condition had deteriorated significantly: she lost weight, developed tachycardia, and her muscle cramps became more frequent. After discharge in 2023, she felt well at one month. At six months, however, she developed clear symptoms of hypothyroidism. She had not reduced the methimazole dose as recommended - because she was afraid that lowering it might trigger a relapse of thyrotoxicosis. Her reasoning was understandable: she wanted to avoid the symptoms she had previously experienced.

In 2024, after euthyroidism was achieved, she stopped alfacalcidol on her own. Her reason was the same - she felt well. This led to a drop in serum calcium. Non-adherence to long-term therapy is common in chronic endocrine disorders, and this patient was no exception. In our view, this behavioural factor amplified every biological trigger: surgery, COVID-19 infection, and metabolic decompensation. Effective management of chronic hypoparathyroidism therefore requires not only correct prescriptions but also continuous patient education and follow-up.

Clinical severity and risks. What frightened us most were the seizures - occurring together with ECG changes and uncontrolled thyrotoxicosis. We

were genuinely concerned about atrial fibrillation. The mortality risk in such a situation is high [4]. In our experience, the combination of hypocalcemic seizures and thyrotoxic tachycardia creates a particularly dangerous situation that requires immediate and aggressive management of both conditions simultaneously.

Outdated therapies in post-Soviet practice. In our opinion, DHT has been obsolete for years. Yet it remains widely prescribed across the CIS countries. Why? Doctors are used to it - and so are patients. Many have been taking the same pills for decades. They do not come back for follow-up. They do not look for updated guidelines. They simply continue what they know. This patient took DHT for 30 years before anyone switched her to alfacalcidol. International guidelines no longer recommend this agent, and we strongly advocate for replacing it with alfacalcidol or calcitriol (Table 2) [2].

Table 2. Native Vitamin D and Its Active Metabolites Used in the Treatment of Hypoparathyroidism

Types of Vitamin D	Activity	Dose	Onset of Action	Duration of Action	Modern Role
Ergocalciferol	1	500-1000 IU/day	10–14 days	Week-month	Adjunct
Cholecalciferol	1	500-1000 IU/day	10–14 days	Week-month	Adjunct
Dihydrotachysterol (A.T.10)	5-10	0.3-1.0 mg/day	4-7 days	28 days	Outdated, risk of accumulation
Alfacalcidol (alpha-D3, Etalpha)	1000	0.25-2.0 mcg once/twice daily	7-8 hours	3 days	Main choice in CIS countries
Calcitriol (Rocaltrol)	1000	0.5-4 mcg/day	3-6 hours	3 days	Gold standard

Source: Compiled by the authors

COVID-19 as a trigger. The COVID-19 pandemic has highlighted the vulnerability of calcium homeostasis in critically ill patients. The virus may affect parathyroid function through several mechanisms: viral entry via ACE2 receptors, inflammatory cytokines increasing calcium-sensing receptor sensitivity, and respiratory alkalosis reducing ionized calcium levels [7; 9]. In this patient, COVID-19 pneumonia involving approximately 60 % of lung parenchyma was followed by an acute cerebrovascular accident and destabilization of her underlying endocrinopathies. Although a direct causal link is difficult to prove, the temporal sequence suggests that SARS-CoV-2 infection likely contributed to parathyroid suppression in an already vulnerable patient [7;10].

Conventional treatment and its limitations. Standard therapy with oral calcium and active vitamin D metabolites (alfacalcidol or calcitriol) requires careful titration to relieve hypocalcemic symptoms while avoiding hypercalcemia, hypercalciuria, and nephrolithiasis [2; 6]. In practice, patients often experience calcium fluctuations, persistent hyperphosphatemia, and symptoms that limit quality of life [5]. In 2015, the U.S. Food and Drug Administration approved recombinant human parathyroid hormone (rhPTH(1-84)) for patients inadequately controlled on conventional treatment, although access to this therapy remains limited in many countries [11].

Follow-up recommendations. Close biochemical follow-up is essential in chronic hypoparathyroidism. Under stable conditions, we recommend assessment of serum calcium, phosphorus, creatinine, and urinary calcium excretion every 3–6 months. During dose changes or metabolic instability - as seen in this patient after COVID-19 and during thyrotoxicosis relapse - more frequent monitoring is required. The primary goals of therapy are relief of hypocalcemic symptoms, improvement of quality of life, maintenance of serum calcium in the low-normal range, normalization of phosphate levels, and prevention of renal complications [2].

Strengths of this case report include the detailed longitudinal follow-up over 15 months and the emphasis on interactions between thyrotoxicosis, COVID-19, and chronic hypoparathyroidism. Limitations are that it is a single case, so causality cannot be proven; the patient's adherence was self-reported, and we lacked urinary calcium excretion data.

Conclusion

This case taught us several lessons. First,

thyrotoxicosis can transiently mask hypocalcemia by increasing bone resorption - and antithyroid therapy then unmasks it. Second, COVID-19 likely contributed to parathyroid suppression in this patient. Third - and most importantly in our view - low patient adherence repeatedly influenced the clinical course. The patient reduced or stopped her medication when she felt well, believing she no longer needed it. Each time, her calcium dropped and symptoms returned.

Therefore, managing chronic hypoparathyroidism is not just about prescribing calcium and vitamin D. It is also about understanding how patients think, anticipating self-discontinuation, and investing time in education and follow-up. We recommend that clinicians always verify adherence and explicitly warn patients not to stop alfacalcidol - even when they feel completely well.

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COVID-19 ЖӘНЕ ТИРЕОТОКСИКОЗДЫҢ ҚАЙТАЛАНУЫМЕН ДЕКОМПЕНСАЦИЯЛАНҒАН СОЗЫЛМАЛЫ ОТАДАН КЕЙІНГІ ГИПОПАРАТИРЕОЗ: КЛИНИКАЛЫҚ ЖАҒДАЙ

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Аңдатпа

Өзектілігі. Созылмалы отадан кейінгі гипопаратиреоз - тиреоидэктомияның өмір бойы ем қабылдауды қажет ететін сирек асқынуы. Тиреотоксикоз, COVID-19 және дигидротахистеролды ұзақ жылдар қолдану аясындағы оның декомпенсациясы жағдайлары әдебиетте бірлі-жарым.

Мақсаты. Грейвс ауруымен науқаста декомпенсацияланған созылмалы отадан кейінгі гипопаратиреоздың клиникалық жағдайын сипаттау және декомпенсация факторларын, диагностика ерекшеліктерін, терапияны түзету тәсілдерін талдау.

Клиникалық жағдай. 58 жастағы науқас 2023 жылы аяқ-қол құрысуы, тахикардия, дірілдеу шағымдарымен түсті. Анамнезде: 1991 жылы Грейвс ауруы бойынша субтотальды тиреоидэктомия,

30 жылдан астам дигидротахистерол қабылдау, 2022 жылы COVID-19 және сол жақты гемипарез. Түскенде: гипокальциемия, гиперфосфатемия, паратиреоидты гормон төмендеген, декомпенсирленген тиреотоксикоз. Альфакальцидол, кальций карбонаты, Тирозол тағайындалды. 15 айдан кейін кальций тұрақталып, эутиреоз қалыптасты.

Қорытынды. Бұл клиникалық жағдай дигидротахистеролды ұзақ уақыт қолданудың қауіптерін, COVID-19-дың кальций-фосфор алмасуының декомпенсациясындағы триггерлік рөлін және науқастарды Д-дәруменінің белсенді метаболиттеріне уақтылы ауыстырудың маңыздылығын көрсетеді.

Түйін сөздер: *гипопаратиреоз, паратгормон, гипокальциемия, гиперфосфатемия, тиреоидэктомия, альфакальцидол.*

ХРОНИЧЕСКИЙ ПОСЛЕОПЕРАЦИОННЫЙ ГИПОПАРАТИРЕОЗ, ДЕКОМПЕНСИРОВАННЫЙ РЕЦИДИВОМ ТИРЕОТОКСИКОЗА И COVID-19: КЛИНИЧЕСКИЙ СЛУЧАЙ

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Аннотация

Актуальность. Хронический послеоперационный гипопаратиреоз - редкое осложнение тиреоидэктомии, требующее пожизненной терапии. Случаи его декомпенсации на фоне тиреотоксикоза, COVID-19 и многолетнего приёма дигидротахистерола в литературе единичны.

Цель. Описать клинический случай хронического послеоперационного гипопаратиреоза с декомпенсированным течением у пациентки с болезнью Грейвса и проанализировать факторы декомпенсации, особенности диагностики и подходы к коррекции терапии.

Клинический случай. Пациентка 58 лет, поступила в 2023 году с судорогами, тахикардией, тремором и тревожностью. В анамнезе: субтотальная тиреоидэктомия по поводу болезни Грейвса, послеоперационный гипопаратиреоз с тетаническими приступами, более 30 лет приёма дигидротахистерола без компенсации, COVID-19 с поражением лёгких и острое нарушение мозгового кровообращения с левосторонним гемипарезом. При поступлении: гипокальциемия, гиперфосфатемия, низкий паратиреоидный гормон, декомпенсированный тиреотоксикоз, удлинение QT, катаракта. Назначены альфакальцидол, карбонат кальция, тирозол. За 15 месяцев наблюдения достигнута нормализация кальция, снижение антител к рТТГ, стойкий эутиреоз.

Вывод. Данный клинический случай демонстрирует риски длительного применения дигидротахистерола при хроническом гипопаратиреозе, роль COVID-19 как триггера декомпенсации кальций-фосфорного обмена и важность своевременного перевода пациентов на активные метаболиты витамина Д с тщательным биохимическим мониторингом.

Ключевые слова: *гипопаратиреоз, паратиреоидный гормон, гипокальциемия, гиперфосфатемия, тиреоидэктомия, альфакальцидол.*

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