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Case Report

Suspected methimazole-associated muscle cramps in a woman with graves' disease: A case report

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ABSTRACT

Background: Methimazole is a standard treatment for Graves' disease and is generally well tolerated. Painful muscle cramps are rarely reported, while thyrotoxicosis itself may cause neuromuscular manifestations. This overlap can make causality difficult to establish.

Case presentation: A 38-year-old woman with Graves' disease and secondary hypertension was treated with Methimazole 20 mg/day and Propranolol 30 mg/day. Approximately one month after treatment initiation, she developed recurrent, sudden, severe, painful muscle cramps triggered by abrupt movements or changes in body position. Episodes lasted less than five minutes and resolved spontaneously without residual weakness. The cramps persisted despite reduction of Methimazole to 10 mg/day. Thyroid function fluctuated during follow-up. In April 2026, FT4 was 13.12 pmol/L and TSHs was 3.59 mIU/L, after which the patient discontinued her medications. The cramps completely resolved and did not recur.

Discussion: The temporal relationship and complete resolution after treatment discontinuation suggest a possible association with Methimazole. However, thyrotoxicosis, concomitant medications, absence of rechallenge, and lack of acute creatine kinase or electrolyte measurements limit definitive causal attribution.

Conclusion: Painful muscle cramps may represent an uncommon adverse effect associated with Methimazole. Awareness and longitudinal assessment may facilitate recognition and evaluation of similar events.

1. Introduction

Hyperthyroidism is a common endocrine disorder characterized by excessive synthesis and secretion of thyroid hormone (TH) by the thyroid. TH increases the rate of basal metabolic, thermogenesis of the tissue, and reduces the level of serum cholesterol and systemic vascular resistance. The untreated of hyperthyroidism causes weight loss, atrial fibrillation, cardiovascular dysfunction, osteoporosis, embolic events, fragility fractures and gastrointestinal abnormalities. The most frequent causes of hyperthyroidism are Graves' disease and toxic nodular goiter. Patients with newly diagnosed Graves' hyperthyroidism are

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usually medically treated for 12–18 months with Methimazole as the preferred drug (Kahaly et al., 2018). Beta-adrenergic blockers such as Propranolol may be used to control adrenergic manifestations, particularly during the initial phase of treatment while thyroid hormone excess is being controlled (Shah et al., 2025).

Methimazole is generally considered an effective and well-tolerated antithyroid drug (ATD), although adverse drug reactions can occur. In recent years, the identification and characterization of its adverse drug reactions remain important components of pharmacovigilance and medication safety assessment. Using real-world data, recent research has systematically evaluated adverse events associated with Methimazole exposure, applying multiple analytical approaches to identify potential safety signals. Such findings may contribute to a better understanding of the safety profile of Methimazole and support more informed and individualized treatment decisions in clinical practice (Sun et al., 2025). Commonly recognized adverse effects include cutaneous reactions and arthralgia, whereas serious reactions such as agranulocytosis, hepatotoxicity, and vasculitis are uncommon. In a prospective multicenter study of patients with Graves' hyperthyroidism receiving ATDs, adverse drug reactions occurred in approximately 10% of patients, with most reactions occurring during the first six months of treatment (Karmisholt et al., 2022). Consequently, unusual symptoms developing during Methimazole treatment require consideration of both recognized and less commonly reported adverse drug reactions.

Musculoskeletal symptoms in patients with Graves' disease are particularly challenging to interpret because thyroid hormone excess itself can affect skeletal muscle. Thyrotoxic myopathy encompasses a range of neuromuscular manifestations, including muscle weakness, muscle paralysis, and muscle pain. Proximal muscle weakness is a characteristic manifestation, although the clinical presentation can vary considerably (Cui & Zhang, 2022). In addition, myopathy and other muscle-related manifestations may occur in association with changes in thyroid hormone concentrations or other complications of thyrotoxicosis (Cui & Zhang, 2022; Setoyama et al., 2022). Therefore, the occurrence of muscle symptoms during Methimazole treatment should not automatically be attributed to the drug, as both the underlying thyroid disorder and other potential causes of muscle symptoms should be considered (Ji & Kim, 2021).

Painful muscle cramps associated with oral Methimazole appear to be particularly uncommon. Tian et al. (2022) reported two patients with hyperthyroidism who developed

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myalgia and painful muscle cramps during treatment with oral Methimazole. In both cases, the symptoms persisted despite reduction of the Methimazole dose and improved after discontinuation of oral Methimazole. The symptoms did not recur after the patients were switched to topical Methimazole, leading the authors to propose a temporal association between oral Methimazole exposure and the muscle symptoms (Tian et al., 2022). This report is particularly relevant because it suggests that painful muscle cramps may represent a rare manifestation associated with oral Methimazole, although the available evidence remains limited to case-level observations.

The present case describes a 38-year-old woman with Graves' disease who developed recurrent, severe, movement-triggered painful muscle cramps approximately one month after starting oral Methimazole. The episodes persisted intermittently during long-term treatment and disappeared completely after treatment discontinuation, with no recurrence thereafter. This case is noteworthy because the longitudinal clinical and thyroid-function data allow the temporal relationship between Methimazole exposure, thyroid status, dose adjustments, and the muscle symptoms to be described in detail.

2. Case presentation

Patient information and initial diagnosis

A 38-year-old woman was diagnosed with hyperthyroidism in July 2024. Laboratory evaluation at diagnosis showed an FT4 concentration of 41.31 pmol/L, equivalent to approximately 3.21 ng/dL, and a markedly suppressed TSH concentration of <0.0025 µIU/mL. The patient was diagnosed with Graves' disease and concomitant secondary hypertension.

At the initial evaluation, her blood pressure was 180/90 mmHg, heart rate was 152 beats/min, and body weight was 65 kg. Treatment was initiated with Methimazole 20 mg/day (10 mg twice daily) and Propranolol 30 mg/day (10 mg three times daily). The initial treatment resulted in improvement in the patient's cardiovascular parameters and thyroid function. In August 2024, her blood pressure and heart rate had decreased to 167/102 mmHg and 99 beats/min, respectively. Candesartan 8 mg/day was added for blood pressure management.

Development of painful muscle cramps

Approximately one month after initiating regular Methimazole therapy, the patient began experiencing recurrent episodes of sudden and severe muscle pain. She described the sensation using the Indonesian terms "*keseleo*" and "*otot tertarik*", which corresponded to a sudden painful sensation resembling a muscle cramp, spasm, or muscle being pulled. The

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episodes were typically triggered by sudden movements or abrupt changes in body position. Examples reported by the patient included sitting down too quickly, taking an incorrect step while climbing stairs, and reaching backward to scratch her back. The episodes could occur during otherwise ordinary daily activities.

The episodes were characterized by severe pain, occurred intermittently, and lasted for no more than approximately five minutes. The pain subsequently resolved spontaneously, and the patient returned to her usual condition without persistent pain or other residual symptoms. She did not report persistent muscle weakness following the episodes. The symptoms continued intermittently throughout the period of Methimazole treatment. Importantly, the episodes persisted despite a reduction in the Methimazole dose from 20 mg/day to 10 mg/day in November 2024.

Longitudinal clinical course

In September 2024, the patient remained on Methimazole 20 mg/day, Propranolol 30 mg/day, and candesartan 8 mg/day. Her blood pressure had improved to 120/80 mmHg, although her heart rate remained elevated at 114 beats/min. The muscle cramps continued. In October 2024, FT4 had decreased to 25.23 pmol/L and TSH remained suppressed at 0.01 µIU/mL**.** The patient continued to experience recurrent painful episodes. In November 2024, FT4 further decreased to 18.31 pmol/L**,** and Methimazole was reduced to 10 mg/day**.** Propranolol was also reduced to 20 mg/day. Despite these dose reductions, the muscle cramps persisted.

From December 2024 through January 2025, the patient continued Methimazole 10 mg/day and Propranolol 20 mg/day, with candesartan 8 mg/day. The muscle cramps continued to occur intermittently. Her body weight increased from 73.7 kg in December 2024 to 75 kg in January 2025.From February through December 2025, the patient did not attend clinical examinations but reported continuing the previously prescribed medications. The muscle cramps continued during this period.

In Januari 2026, the patient remained on Methimazole 10 mg/day and Propranolol 20 mg/day. FT4 was 31.18 pmol/L and TSH was <0.1 µIU/mL, indicating that biochemical hyperthyroidism had not yet completely resolved. The painful muscle episodes continued. In February 2026, FT4 decreased to 16.92 pmol/L, although TSH remained <0.1 µIU/mL, and the

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muscle cramps were still reported. In March 2026, the patient continued Methimazole 10 mg/day and Propranolol 20 mg/day, and the episodes were still present. By April 2026, FT4 had reached 13.12 pmol/L and TSH had increased to 3.59 µIU/mL**,** indicating biochemical normalization of thyroid function. At that point, the patient discontinued her medications. Notably, the painful muscle cramps had persisted throughout the period of treatment but subsequently resolved after treatment discontinuation. The patient's clinical course, including thyroid function, treatment regimen, and the persistence of painful muscle cramps, is summarized in Table 1.

3. Discussion

The present case describes recurrent, brief, severe, and movement-triggered painful muscle cramps occurring during treatment of Graves' disease with oral Methimazole. The most notable feature is the temporal relationship between Methimazole exposure and the onset of symptoms, followed by complete and sustained resolution after treatment discontinuation. Methimazole is generally considered an effective and well-tolerated antithyroid drug, although adverse drug reactions ranging from relatively common minor reactions to rare clinically significant events have been reported (Butt et al., 2023; Tan et al., 2021). However, because the patient also had Graves' disease with fluctuating thyroid function and received other medications, the clinical course should be interpreted as supporting a possible Methimazole-associated adverse drug reaction rather than establishing definite drug causality.

The temporal association is consistent with the limited previous literature. Tian et al. (2022) reported two patients with hyperthyroidism who developed myalgia and painful muscle cramps during treatment with oral Methimazole (Thyrozol). In one patient, muscle cramps developed after six months of treatment when thyroid function had normalized, whereas in the other, symptoms developed approximately two weeks after treatment while thyroid function remained abnormal. In both patients, the symptoms persisted despite reduction of the Methimazole dose but improved substantially after discontinuation of oral Methimazole. The symptoms did not recur after the patients were switched to topical Methimazole, leading the authors to suggest a strong temporal association between oral Methimazole exposure and the muscle symptoms (Tian et al., 2022).

The present case shares several clinically relevant features with those previously reported cases. First, the muscle symptoms developed after exposure to oral Methimazole. Second, the

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symptoms recurred during continued treatment. Third, reduction of the Methimazole dose did not result in immediate resolution. Finally, the symptoms disappeared after treatment discontinuation. These similarities provide supportive evidence for considering oral Methimazole as a potential contributor to the patient's symptoms. Nevertheless, the similarity to previous case reports should be regarded as supportive rather than confirmatory evidence because the existing literature remains limited to individual case observations.

An important feature of the present case is the absence of a clear dose-response relationship. Methimazole was initially administered at 20 mg/day and was subsequently reduced to 10 mg/day in November 2024. Despite the dose reduction, the painful muscle cramps continued for more than one year. This pattern is notable because a dose-response relationship may provide additional support for a causal association between a drug and an adverse event. However, dose-response is only one component of causality assessment, and the absence of a clear dose-response relationship does not by itself exclude a drug-related adverse reaction (Manjhi et al., 2024; Pradhan et al., 2023).

The underlying Graves' disease represents an important alternative explanation. Thyroid hormone excess can affect skeletal muscle and produce a spectrum of neuromuscular manifestations collectively described as thyrotoxic myopathy. Reported manifestations include muscle weakness, paralysis, and muscle pain, while proximal muscle weakness is considered a characteristic presentation of thyrotoxic myopathy (Cui & Zhang, 2022; Fu et al., 2023; S. Y. Lee & Pearce, 2024). Thyrotoxic periodic paralysis represents another recognized neuromuscular manifestation of hyperthyroidism; however, it is characterized predominantly by acute episodes of muscle weakness or paralysis, typically associated with hypokalemia, rather than isolated painful muscle cramps (Patel & Ladak, 2021).

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Table 1. Longitudinal clinical course of the patient: clinical parameters, thyroid function, treatment regimen, and characteristics of muscle cramps

Period	Blood Pressure (mmHg)	Heart Rate (bpm)	Body Weight (kg)	FT4 (pmol/L)	TSHs (µlu/mL)	Therapy Regimen	Muscle cramps/ spasms	Characteristics of episode	Other relevant findings
July 2024	180/90	152	65	41,31	<0,0025	Methimazole 2 x 10 mg Propranolol 3 x 10 mg	No	-	Initial diagnosis and therapy
August 2024	167/102	99	-	-	-	Methimazole 2 x 10 mg Propranolol 3 x 10 mg	Yes	Sudden, severe pain; triggered by movement/position change; ≤5 min	First episode of severe cramps/spasms
September 2024	120/80	114	67	-	-	Candesartan 1 x 8 mg Methimazole 2 x 10 mg Propranolol 3 x 10 mg	Yes	Sudden, severe pain; triggered by movement/position change; ≤5 min	Recurrent episodes
October 2024	130/90	100	68	25,23	0,01	Candesartan 1 x 8 mg Methimazole 2 x 10 mg Propranolol 3 x 10 mg	Yes	Sudden, severe pain; triggered by movement/position change; ≤5 min	Episodes persisted
November 2024	130/70	85	71	18,31	<0,05	Candesartan 1 x 8 mg Methimazole 1 x 10 mg Propranolol 2 x 10 mg	Yes	Sudden, severe pain; triggered by movement/position change; ≤5 min	Episodes persisted
December 2024	130/80	88	73,7	-	-	Candesartan 1 x 8 mg Methimazole 1 x 10 mg Propranolol 2 x 10 mg	Yes	Sudden, severe pain; triggered by movement/position change; ≤5 min	Episodes persisted
January 2025	120/70	84	75	-	-	Candesartan 1 x 8 mg Methimazole 1 x 10 mg Propranolol 2 x 10 mg Candesartan 1 x 8 mg	Yes	Sudden, severe pain; triggered by movement/position change; ≤5 min	Episodes persisted

February 2025-December 2025	-	-	-	-	-	Methimazole 1 x 10 mg Propranolol 2 x 10 mg	Yes	Sudden, severe pain; triggered by movement/position change; ≤5 min	Episodes persisted
January 2026	130/90	95	72	31,18	<0,1	Candesartan 1 x 8 mg Methimazole 1 x 10 mg Propranolol 2 x 10 mg	Yes	Sudden, severe pain; triggered by movement/position change; ≤5 min	Episodes persisted
February 2026	120/90	95	74	16,92	<0,1	Methimazole 1 x 10 mg Propranolol 2 x 10 mg	Yes	Sudden, severe pain; triggered by movement/position change; ≤5 min	Episodes persisted
March 2026	-	-	-	-	-	Methimazole 1 x 10 mg Propranolol 2 x 10 mg	Yes	Sudden, severe pain; triggered by movement/position change; ≤5 min	Episodes persisted
April 2026	-	-	-	13,12	3,59	Discontinued	No	–	Thyroid function normalized
After discontinuation	-	-	-	-	-	None	No recurrence	–	Asymptomatic at pharmacy visit

Description: FT4, free thyroxine; TSHs, sensitive thyroid-stimulating hormone. Laboratory reference ranges were based on the Provincial Government Health Laboratory of South Kalimantan: FT4, 12–22 pmol/L; TSHs, 0.45 mIU/L. Thyroid status was interpreted based on the combination of FT4 and TSHs values and the clinical context.

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The longitudinal thyroid-function data in the present case make this alternative explanation particularly important. The patient's FT4 concentration decreased substantially from 41.31 pmol/L at diagnosis to 25.23 pmol/L in October 2024 and 18.31 pmol/L in November 2024, although TSH remained suppressed. In January 2026, FT4 increased again to 31.18 pmol/L with TSH <0.1 µIU/mL, followed by a decrease in FT4 to 16.92 pmol/L in February 2026. Thus, the painful muscle symptoms occurred during a prolonged period of changing and incompletely controlled thyroid function. This temporal overlap prevents the thyroid disorder from being completely excluded as a contributor to the symptoms.

Nevertheless, the phenotype of the muscle symptoms in the present case differs from the typical presentation of thyrotoxic myopathy. The patient's episodes were sudden, intensely painful, brief, and consistently associated with movement or abrupt changes in body position. Each episode resolved spontaneously within approximately five minutes, and the patient did not report persistent muscle weakness or residual symptoms. Muscle cramps represent a distinct clinical phenomenon characterized by sudden, involuntary, and painful muscle contraction, and their evaluation should consider neurological, musculoskeletal, metabolic, medication-related, and other systemic causes (Lam et al., 2022). In contrast, classical thyrotoxic myopathy is more commonly characterized by persistent or progressive muscle weakness, particularly involving proximal muscles, although muscle pain may also occur (Cui & Zhang, 2022). Therefore, the symptom phenotype in the present case is not typical of classical thyrotoxic myopathy, although it cannot completely exclude a thyroid-related muscular manifestation.

Another relevant consideration is a Methimazole-associated myopathic or muscle-injury reaction. Musculoskeletal symptoms, including arthralgia and myalgia, are recognized adverse effects reported during antithyroid drug therapy (Lane et al., 2023). Kim et al. (2015) reported a 13-year-old girl with Graves' disease who developed myalgia approximately two weeks after initiating Methimazole, accompanied by a marked elevation of serum creatine kinase despite normalization of thyroid hormone concentrations. Her myalgia and CK elevation subsequently improved following reduction of the Methimazole dose and addition of levothyroxine (Kim et al., 2015). Similarly, Ji and Kim (2021) reported an 11-year-old girl with Graves' disease who developed myalgia and marked CK elevation after Methimazole treatment despite normal thyroid hormone concentrations. In that case, muscle symptoms and CK elevation improved after Methimazole discontinuation and switching to propylthiouracil (Ji & Kim, 2021). Other

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rare musculoskeletal reactions to Methimazole have also been reported. Kawasumi et al. (2023), for example, described antithyroid arthritis syndrome after Methimazole initiation, characterized by severe pain, arthralgia, inflammatory findings, and improvement following Methimazole withdrawal. Although these cases involved objective biochemical or inflammatory evidence and therefore differ from the present case, they support the possibility that clinically significant musculoskeletal manifestations may occur during Methimazole therapy (Kawasumi et al., 2023).

The present case, however, should not be classified as Methimazole-induced myositis or Methimazole-induced myopathy. No creatine kinase concentration was obtained during an acute episode, and there was no objective evidence of muscle injury or inflammation. In addition, the symptoms were episodic and short-lived rather than persistent, with the patient returning to her baseline condition after each episode. These clinical features differ from the cases of Methimazole-associated myalgia or myopathy previously reported, in which objective biochemical evidence of muscle injury, particularly elevated creatine kinase concentrations, was documented (Ji & Kim, 2021; Kim et al., 2015). Therefore, based on the available clinical information, the most appropriate description is suspected Methimazole-associated recurrent painful muscle cramps.

Concomitant medications also need to be considered. Candesartan was added in August 2024, around the period in which the muscle symptoms emerged, and therefore represents a potential temporal confounder. Propranolol was also administered during much of the period in which the cramps occurred. However, the available case literature provides a more specific precedent for painful muscle cramps during oral Methimazole treatment than for the concomitant medications used in this case (Tian et al., 2022). Nevertheless, a contribution from concomitant medications cannot be completely excluded because a formal dechallenge of Methimazole alone was not performed. This limitation is relevant to causality assessment, as the interpretation of an adverse drug reaction requires consideration of alternative causes, temporal relationships, and the effects of drug withdrawal (Pradhan et al., 2023).

The dechallenge in this case is particularly noteworthy. By April 2026, FT4 had decreased to 13.12 pmol/L and TSH had increased to 3.59 µIU/mL, indicating biochemical normalization of thyroid function. The patient then discontinued her medications, after which the painful muscle cramps completely resolved and did not recur during subsequent follow-up. Resolution of the symptoms following treatment discontinuation provides supportive

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evidence for a possible drug-related association. However, the interpretation of this dechallenge is complicated by the simultaneous normalization of thyroid function. Because Methimazole and Propranolol were discontinued at approximately the same time that the patient became euthyroid, it is not possible to determine whether symptom resolution resulted from withdrawal of Methimazole, resolution of thyrotoxicosis, discontinuation of another medication, or a combination of these factors. In adverse drug reaction causality assessment, positive dechallenge is considered supportive evidence, but its interpretation should take into account the natural course of the underlying condition and other potential explanations for symptom resolution (Hammad et al., 2023).

The absence of rechallenge further limits the strength of the causal inference. Recurrence of the adverse event following re-exposure to the suspected drug would provide stronger evidence for a causal relationship; however, deliberate rechallenge is generally not appropriate when re-exposure may expose the patient to an unnecessary risk, particularly when alternative explanations or treatment options are available. Therefore, in the present case, causality is supported by the temporal association and positive dechallenge, but not by rechallenge evidence (Hammad et al., 2023).

A structured causality assessment can further illustrate the strength and limitations of the available evidence. Assessment of a suspected adverse drug reaction should consider the temporal relationship between drug exposure and the event, previous evidence of similar reactions, response to drug withdrawal, the presence or absence of rechallenge, dose-response relationships, and potential alternative explanations or confounding factors (Anjum et al., 2022; Hammad et al., 2023; Manjhi et al., 2024). Structured pharmacovigilance approaches similarly emphasize the integration of case-level evidence, biological plausibility, and the overall strength of the available safety signal rather than relying on a single criterion (Sullivan et al., 2022). In addition, a scoping review of pharmacovigilance signals found that temporality, positive dechallenge or rechallenge, and exclusion of competing causes were among the main features used to support suspected drug-related associations (Sartori et al., 2023).

In the present case, the temporal sequence between Methimazole exposure and the onset of painful muscle cramps, the similarity to a previously reported reaction during oral Methimazole therapy, and complete symptom resolution after treatment discontinuation support a possible drug-related association. However, the absence of rechallenge, lack of

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objective evidence of muscle injury, absence of a clear dose-response relationship, and the presence of alternative explanations, particularly fluctuating thyroid function and concomitant medications, limit the strength of the causal inference. Overall, the available evidence supports a possible Methimazole-associated adverse drug reaction rather than a definitive causal relationship.

The case has several strengths. First, the patient was followed longitudinally from the initiation of antithyroid therapy through subsequent changes in treatment and normalization of thyroid function. Second, serial FT4 and TSH measurements allow the muscle symptoms to be considered in the context of changing thyroid status rather than relying solely on the temporal relationship with Methimazole exposure. Third, the medication history documents the reduction of Methimazole from 20 mg/day to 10 mg/day without immediate resolution of the symptoms, providing useful information regarding the absence of an obvious dose-response relationship. Finally, the complete and sustained disappearance of symptoms after treatment discontinuation provides clinically meaningful follow-up information.

Several limitations should nevertheless be acknowledged. The symptom assessment was retrospective, and no physical examination or laboratory assessment was performed during an acute muscle-cramp episode. In particular, CK and electrolyte measurements were unavailable, limiting the ability to evaluate muscle injury, electrolyte disturbances, or other potential metabolic causes of muscle symptoms. Comprehensive clinical and medication information, together with appropriate laboratory evaluation and consideration of alternative etiologies, is important for the assessment and documentation of suspected adverse drug reactions (L. Lee et al., 2022). In addition, because Methimazole and other medications were discontinued around the time thyroid function normalized, a drug-specific dechallenge cannot be clearly separated from improvement in the underlying hyperthyroid state. The introduction of candesartan around the onset of symptoms represents an additional potential confounding factor.

Despite these limitations, the present case adds to the limited literature describing painful muscle cramps occurring during oral Methimazole therapy. The clinical similarity to the previously reported cases, particularly the persistence of symptoms despite Methimazole dose reduction and their subsequent resolution after discontinuation, supports consideration of Methimazole as a possible contributor. At the same time, the case demonstrates why causal attribution should remain cautious in patients with Graves' disease, because thyroid hormone

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excess can produce a range of neuromuscular manifestations (Cui & Zhang, 2022; Fu et al., 2023; S. Y. Lee & Pearce, 2024). Furthermore, the clinical course of Graves' disease and the response to antithyroid therapy may vary over time, making longitudinal assessment important when interpreting symptoms occurring during prolonged antithyroid drug treatment (Jeong Kim, 2023). The most appropriate interpretation is therefore a suspected or possible Methimazole-associated recurrent painful muscle-cramp reaction, rather than definite Methimazole-induced myopathy.

4. Conclusion

Recurrent painful muscle cramps occurring during Methimazole treatment may represent an uncommon adverse drug reaction associated with Methimazole in patients with Graves' disease. The temporal relationship and complete resolution following treatment discontinuation support a possible association. However, causality cannot be established definitively because thyrotoxicosis and concomitant medications remain potential alternative explanations, and objective assessments during the symptomatic episodes were unavailable. Clinicians and pharmacists should be aware of this potential adverse effect and consider both drug-related and disease-related causes when evaluating new-onset muscle cramps during antithyroid therapy.

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6. Conflict of interest

The author declares no conflict of interest.

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