International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

ARTICLE TITLE	DIAGNOSTIC AND TREATMENT IN TETANY: EXPLORING NON-HYPOCALCEMIC CAUSES AND CLINICAL CHALLENGES
DOI	https://doi.org/10.31435/ijitss.4(48).2025.4358
RECEIVED	17 October 2025
ACCEPTED	19 December 2025
PUBLISHED	23 December 2025
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

DIAGNOSTIC AND TREATMENT IN TETANY: EXPLORING NON-HYPOCALCEMIC CAUSES AND CLINICAL CHALLENGES

Zofia Laska *(Corresponding Author, Email: zofia.laska00@gmail.com)*
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0009-0001-0326-8251

Marianna Latour
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0000-0002-2568-5080

Zuzanna Kudlińska
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0009-0003-4422-7784

Honorata Juniewicz
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0009-0001-0060-992X

Patryk Heryć
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0009-0004-9121-2670

Karolina Kananowicz
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0009-0009-1229-6797

Ryszard Łagowski
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0009-0007-8441-054X

Julia Kosęda
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0009-0008-7742-2814

Anna Jędrasiak
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0009-0001-3739-2066

Jakub Piotrowski
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0009-0007-8889-713X

ABSTRACT

Normocalcemic tetany is a clinical condition characterized by neuromuscular hyperexcitability in the absence of overt hypocalcemia, resulting from disturbances in ionized calcium, magnesium, potassium, or acid–base balance. Although tetany is most commonly associated with hypocalcemia, cases with normal total calcium levels pose diagnostic challenges and are frequently underrecognized. This review consolidates current knowledge regarding the etiology, clinical manifestations, diagnostic evaluation, and management of normocalcemic tetany. The etiology includes hereditary renal tubulopathies such as Gitelman and Bartter syndromes, hyperventilation-related alkalosis, magnesium deficiency, and rare autoimmune disorders involving antibodies against the voltage-gated potassium channel complex (CASPR2). Clinical presentations range from overt tetanic seizures with characteristic carpopedal spasms to latent tetany detectable via Chvostek and Trousseau signs or ischemia-induced EMG abnormalities. Case reports highlight unusual scenarios, including tetany triggered by vomiting, postoperative parathyroidectomy, and hereditary neuromuscular hypersensitivity, emphasizing that serum total calcium may remain normal while ionized calcium or other electrolytes fluctuate. Management strategies focus on prompt recognition, correction of underlying electrolyte disturbances—particularly calcium, magnesium, and potassium—and supportive care. Early administration of intravenous calcium, alongside addressing precipitating factors, is critical for rapid symptom relief. Latent or recurrent tetany in the context of normal electrolytes warrants consideration of genetic, neurologic, or autoimmune etiologies, and may require specialized testing including EMG, antibody panels, or genetic analysis. Increased awareness among clinicians, timely evaluation of ionized calcium and other electrolytes, and early intervention are essential to prevent severe complications, including life-threatening muscle spasms, rhabdomyolysis, and cardiovascular instability. This review underscores the need for further population-based studies to define the prevalence, at-risk groups, and optimal management of normocalcemic tetany.

KEYWORDS

Normocalcemic Tetany, Latent Tetany, Electrolyte Imbalance, Hypomagnesemia, Hyperventilation-Induced Tetany, Gitelman Syndrome, Neuromuscular Hyperexcitability

CITATION

Zofia Laska, Marianna Latour, Zuzanna Kudlińska, Honorata Juniewicz, Patryk Heryć, Karolina Kananowicz, Ryszard Łagowski, Julia Kosęda, Anna Jędrasiak, Jakub Piotrowski (2025) Diagnostic and Treatment in Tetany: Exploring Non-Hypocalcemic Causes and Clinical Challenges. *International Journal of Innovative Technologies in Social Science*. 4(48). doi: 10.31435/ijitss.4(48).2025.4358

1. Introduction:

Tetany is a consequence of neuromuscular dysfunction characterized by a lowered threshold of neuromuscular excitability, resulting from disturbances in electrolyte balance and acid–base homeostasis. These abnormalities—most commonly hypocalcemia, hypomagnesemia, or alkalosis—lead to a reduction in the concentration of ionized, biologically active calcium. Several clinical manifestations of tetany can be distinguished: overt tetanic seizures, latent tetany, and tetany equivalents.

A tetanic seizure typically presents with tingling and numbness followed by symmetric contractions of the hands (“obstetrician’s hand”), and subsequently of the facial muscles, leading to eyelid spasm and the characteristic “fish mouth” appearance. Spasms may also involve the forearms, chest muscles, and lower limbs, resulting in equinovarus positioning of the feet. Latent tetany is defined as a condition in which clinical signs such as Chvostek’s sign, Trousseau’s sign, and Lust’s sign can be elicited, or in which a tetanic episode can be provoked by hyperventilation. According to *Interna Szczeklika 2021*, tetany equivalents include: “eyelid spasm, photophobia, diplopia, laryngospasm, bronchospasm, spasm of the coronary, abdominal, or peripheral arteries (pseudo–Raynaud phenomenon), and cerebral vasospasm (migraine attack or transient loss of consciousness).”

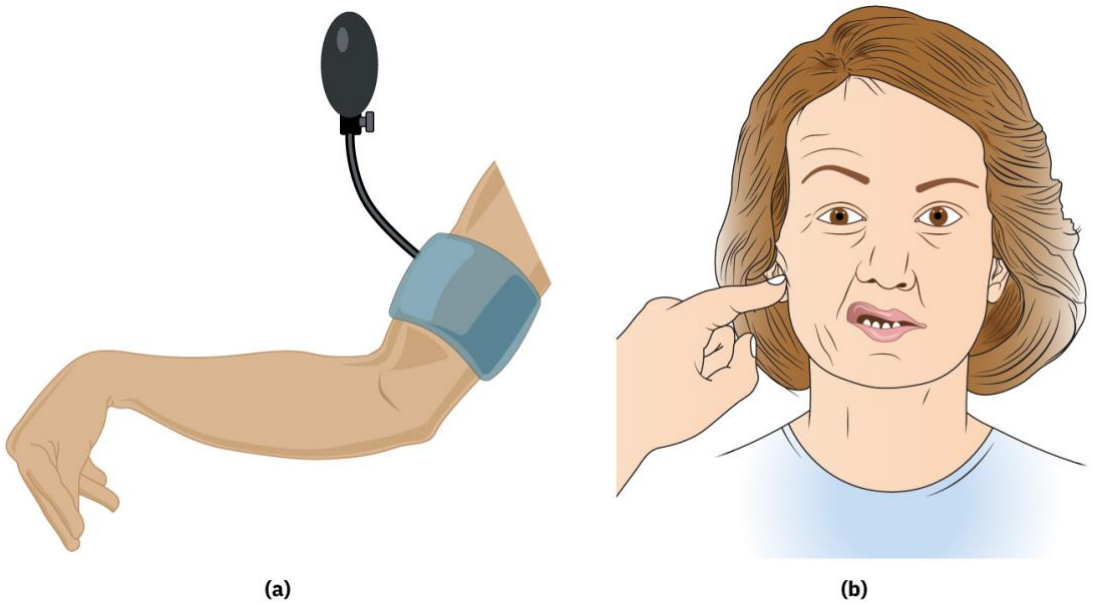

***Fig. 1** [1]*

In a 2017 study, Gouranga Santra and Himanshu Barman identified several causes of tetany, including Gitelman syndrome—a renal salt-wasting disorder resulting from a defect in the thiazide-sensitive sodium–chloride cotransporter in the distal convoluted tubule, and characterized by hypokalemia, metabolic alkalosis, hypomagnesemia, hypermagnesuria, and hypocalciuria[2] and Bartter syndrome, a tubulopathy with renal salt loss presenting with hypokalemia and hypochloremic metabolic alkalosis. Additional etiologies include recurrent vomiting leading to electrolyte depletion and metabolic alkalosis[3], anxiety-related hyperventilation, vitamin D deficiency, idiopathic hypoparathyroidism, postoperative hypoparathyroidism, acute pancreatitis, tumor lysis syndrome, and hypomagnesemia.

Recurrent tetany most commonly occurs in patients with Gitelman syndrome, Bartter syndrome, recurrent vomiting, and vitamin D deficiency, whereas overt tetany is characteristic of hyperventilation[4]. Although tetany is primarily associated with hypocalcemia, documented cases exist in which no abnormalities are present in the standard electrolyte panel; deviations become evident only upon measurement of ionized calcium, or involve other electrolytes, often leading to delayed diagnosis[5,6].

Studies investigating causes of tetany other than postoperative complications following thyroidectomy are scarce and, in our opinion, insufficient to meet clinical needs. Awareness regarding the diagnosis, etiology, and management of this condition remains inadequate.

For this reason, we sought to consolidate current knowledge on the potential causes of recurrent tetany and on diagnostic studies that may improve clinical evaluation, as well as to characterize the cases reported to date.

2. Methodology:

In order to prepare our literature review we focused on current knowledge about different tetany cases and clinical presentation.

2.1. Source selection

Included were original research articles as well as systematic reviews published between 1940 - 2025.

2.2. Inclusion criteria

Articles were included if they: -Adressed the topic of tetany with atypical course or normal calcium levels,- Described etiology of tetany or possible etiology of unsolved cases,- Contained information about patients reaction to different kinds of treatment.

2.3 Content Analysis and data synthesis

The review directly utilized 41 publications. Data from the analyzed articles were grouped thematically and then described accordingly.

2.4 Methodological limitations

The data may be affected by methodological diversity and population heterogeneity.

3. Case reports of tetany with normal calcium levels

3.1 Case 1

The authors describe the case of a boy who, from the neonatal period, suffered from recurrent seizure episodes. The seizures resolved at the age of eight; however, the information regarding whether treatment with phenytoin, acetazolamide, and phenobarbital had been continued remains inconsistent. At the age of twelve, the boy began to complain of symptoms resembling tetany attacks—paresthesias in the fingers and toes, followed by carpopedal spasms presenting as the characteristic "obstetrician's hand" posture. The attacks occurred on average once a month and resolved within 10–30 minutes, while oral calcium administration did not lead to improvement.

No disturbances in calcium homeostasis were found (serum calcium levels were normal, parathyroid hormone levels and responsiveness were normal, and no respiratory alkalosis was present). The authors also noted the presence of latent tetany symptoms in the boy's brother and mother, suggesting a hereditary component of the disorder, likely associated with a tendency toward abnormal excitability of neuronal cell membranes[7].

3.2 Case 2

This is a case report of a young 33-year-old woman whose episodic tetany symptoms began after the birth of her second child, ten years earlier. It is notable that similar symptoms were also present in other members of her family: her mother, her mother's sister, the patient's siblings, and one of her children. The episodes resolved only after intravenous calcium administration; other methods of terminating tetanic attacks—such as rebreathing into a paper bag—proved ineffective in this patient.

On physical examination, Chvostek's and Trousseau's signs were observed, with no additional abnormalities. Laboratory tests, including serum electrolyte levels (total and ionized calcium), thyroid hormones, parathyroid hormone, androgen levels, and a glucose tolerance test, all remained within normal limits. Hyperventilation did not provoke symptoms in this patient.

Electromyography (EMG) was performed. Baseline tracings were normal; however, when ischemia of the upper limb was induced, abnormalities characteristic of tetany were observed. Serum ionized calcium levels and pH were continuously monitored and remained within the normal range even during ischemia-induced spasm. While EMG recordings were still being monitored, the patient was administered intravenous calcium, which resulted in the cessation of ischemia-induced spasms after 45 minutes[8].

3.3 Case 3

In the publication by F. S. Fowweather et al., five cases of young women suffering from tetany attacks were described. In the first two cases, the attacks developed as a result of hyperventilation initially triggered by chest pain. Another patient suffered from thyrotoxicosis and was undergoing treatment with radiation, while the last patient experienced episodes of loss of consciousness without significant injury or sequelae, as well as tetany attacks induced by hyperventilation.

The investigators assessed calcium and phosphate levels both immediately after the attacks and during asymptomatic periods. In all patients, calcium levels immediately following the attacks remained at the upper limit of normal but decreased between episodes. In contrast, phosphate levels in every case showed a marked reduction during attacks compared to asymptomatic periods[9].

3.4 Case 4

A case involved a 24-year-old woman admitted to the hospital with gastroenteritis and severe vomiting. Flexion of the joints was the first sign of emerging tetany; additionally, she presented positive Chvostek and Trousseau signs. Initial blood tests and electrolyte panel showed no abnormalities other than hypophosphatemia. After admission, she experienced another tetanic episode, during which laboratory samples were successfully collected. Arterial blood gas analysis revealed respiratory alkalosis and hypophosphatemia, and hypokalemia, with ionized calcium levels within the normal range in other laboratory tests. The patient improved following administration of calcium gluconate, and she additionally received potassium, phosphate, and magnesium infusions. Ultimately, the most likely underlying cause was considered to be a panic attack associated with concomitant tachypnea [10].

3.5 Case 5

This case report describes two male patients (aged 25 and 48 years) who developed tetany after undergoing surgery for parathyroid adenoma, with symptoms persisting for 4 weeks in one patient and 3 weeks in the other. The manifestations did not resolve despite normal levels of total calcium, ionized calcium, and magnesium, as well as normal pH values.

Tetany developed despite preoperative and postoperative supplementation with alfacalcidol and postoperative administration of calcium gluconate. The authors attribute the condition to the advanced stage of disease at the time of treatment and the substantial relative decrease in calcium levels following surgery. They emphasize the crucial importance of measuring ionized calcium and magnesium levels in similar clinical scenarios[11].

Table 1. [7,8,9,10,11]

Summary Table of Case Reports

Author / Year	Patient (age/sex)	Symptoms	Laboratory Abnormalities	Proposed Cause
Isgreen WP, 1976	Adolescent, Male	Recurrent tetany episodes, carpopedal spasms	Normal Ca and Mg; no respiratory alkalosis	Congenital neuromuscular hyperexcitability
Day JW & Parry GJ, 1990	Adult female	Tetany, paresthesias	Normal total and ionized Ca; normal pH	Hereditary neuronal hyper-irritability
BMJ, 1940	Adult	Tetany triggered by spontaneous hyperventilation	Respiratory alkalosis; hypophosphatemia	Hyperventilation → alkalosis → reduced ionized calcium
Alanazi et al., 2023	Female, 24 years	Tetany, hand spasms	Normal Ca; hypophosphatemia; metabolic alkalosis	Electrolyte disturbances + metabolic alkalosis
Fonseca et al., 1987	Adults post-parathyroidectomy	Postoperative tetany, carpopedal spasms	Normal ionized Ca and Mg; no respiratory alkalosis	Neuromuscular hypersensitivity after chronic hypercalcemia

4. Potential causes of tetany with normal calcium levels

4.1 Hyperventilation

One of the cause of tetany described in the case reports cited was hyperventilation. The underlying etiology of this disorder may vary, with one of the contributing factors being anxiety disorders and stressful situations. A hyperventilation episode can be triggered by a panic attack[12], pain, asthma[13], or the use of stimulant drugs[14]. One scenario that can provoke severe stress in a patient, leading to hyperventilation and subsequently a tetany episode, and which warrants particular attention in anesthesiology practice, is an acute anxiety episode associated with a surgical procedure, such as may occur upon emergence from general anesthesia[15].

The mechanism leading to the development of tetany during hyperventilation episodes involves a simultaneous increase in partial oxygen pressure and a decrease in carbon dioxide pressure in the blood, resulting in respiratory alkalosis.

Positively charged hydrogen ions compete with calcium ions for binding sites on negatively charged plasma proteins; thus, a decrease in hydrogen ion concentration promotes increased binding of calcium ions to plasma proteins, thereby reducing the concentration of free (ionized) calcium in the blood, which represents the biologically active form[16].

4.2. Bartter syndrome and Gitelman syndrome

As previously mentioned, the hereditary basis for disorders that may lead to tetany includes renal tubulopathies causing salt wasting, such as Bartter syndrome and Gitelman syndrome. In both conditions, patients present with metabolic alkalosis, hypokalemia, hypochloremia, increased plasma renin activity, and elevated aldosterone levels.

Bartter syndrome was first described in 1962. Several forms of this disorder have been identified, caused by mutations in various genes, including SLC12A1, KCNJ1, CLCNKA, CLCNKB, BSND, and MAGED2. The clinical phenotype may include polyhydramnios, prematurity, and nephrocalcinosis; however, the classical form (type III) is characterized by variable age of presence, hypokalemia, hypochloremia, metabolic alkalosis, and the occurrence of spontaneous mutations[17].

Fujihara et al. reported a case of a 37-year-old man in whom hyperventilation rapidly triggered tetany attacks. Laboratory findings included low potassium levels, metabolic alkalosis, hyperuricemia, and increased plasma renin activity. Notably, the exacerbation of alkalosis due to hyperventilation did not result in a decrease in ionized calcium concentration; the authors therefore suggested that alkalosis itself was the cause of tetany. The patient was successfully treated with indomethacin[18].

Another case report described siblings affected by Bartter syndrome. The authors highlighted the occurrence of normocalcemic tetany induced by hypokalemia, which was effectively treated with spironolactone. They also noted personality disturbances as a potential feature of Bartter syndrome, as one patient developed depressive symptoms that resolved with hypokalemia treatment[19].

Gitelman syndrome is a rare tubulopathy caused by mutations in the SLC12A3 gene encoding the thiazide-sensitive sodium-chloride cotransporter (NCC)[20]. It was first described in 1966 by H.J. Gitelman and colleagues[21]. It is also referred to as familial hypokalemia–hypomagnesemia. As the name indicates, the condition is characterized by hypokalemia and hypomagnesemia accompanied by metabolic alkalosis and hypocalciuria[22]. The disease usually manifests during adolescence or even in adulthood. Clinical manifestations can vary widely, from asymptomatic cases to severe forms that significantly impair daily functioning. Such electrolyte disturbances can lead to the development of normocalcemic tetany. As described by Gandhi Kunal et al., in some patients, hypocalcemia may also occur alongside the aforementioned deficiencies, contributing to tetany[23]; in such cases, achieving therapeutic goals requires careful correction of other electrolytes, particularly magnesium and potassium[24].

Hardeep Singh Kalsi et al. reported a case of a 25-year-old patient with Gitelman syndrome who developed a tetany attack due to dehydration resulting from diarrhea and vomiting. Laboratory investigations revealed reduced potassium and magnesium levels, mild metabolic alkalosis, and normal serum calcium levels[25].

4.3 Hereditary Chronic Tetany

At present, only theoretical explanations—proposed by the authors of the above-mentioned case reports—exist to account for recurrent tetany with a normal electrolyte profile in multiple members of the same family. This raises suspicion of an inherited disorder; however, due to the lack of confirmed data, the authors referred to it as *congenital neuromuscular hypersensitivity* or *hereditary neuronal hypersensitivity[26]*. Later publications have suggested genetically inherited channelopathies as a plausible cause of similar presentations.

Several case reports describe mutations in the **KCNA1** gene as the underlying cause of hereditary tetany. Initially, this mutation was associated exclusively with episodic ataxia type 1[27], but subsequent findings demonstrated that it reduces magnesium reabsorption in the distal renal tubule, contributing to recurrent, hereditary tetany[28]. Interestingly, in one reported case, the affected patient exhibited normal magnesium levels, and magnesium supplementation worsened the tetanic symptoms, whereas improvement occurred only after calcium administration—raising questions regarding the precise role of **KCNA1** in this condition[29].

4.4 Tetany Induced by Magnesium Deficiency

Magnesium deficiency is one of the most frequently identified electrolyte disturbances—second only to hypokalcemia—in patients presenting with tetany. Numerous clinical conditions, such as the renal syndromes and channelopathies described above, and more commonly malnutrition, increased physiological demand, or lactation, can lead to hypomagnesemia as the key precipitating factor. It is also important to consider medications (e.g., diuretics, proton pump inhibitors)[30] and substances such as alcohol[31], which reduces renal magnesium reabsorption. Magnesium exerts a stabilizing effect on neuronal cell membranes; thus, its deficiency lowers the threshold for axonal excitability, thereby explaining tetanic manifestations[32].

Additionally, magnesium deficiency may cause hypocalcemia by impairing PTH secretion or inducing tissue resistance to PTH[33], making it difficult at times to identify a single dominant cause.

Tetany resulting from hypomagnesemia and hypocalcemia is clinically indistinguishable, and there are cases in which only magnesium administration leads to resolution of symptoms—highlighting the importance of evaluating both electrolytes[34]. Conversely, in the cases mentioned above, despite a history of hypomagnesemia, only calcium gluconate proved effective. However, this effect may have been transient, resulting from an acute rise in serum calcium; its stabilizing properties would not have been sustained without correction of the underlying magnesium deficit[35].

4.5 Less Common Causes Potentially Presenting as Normocalcemic Tetany

When more common causes of tetany have been excluded, it is worth considering rare autoimmune conditions. Although these disorders do not typically manifest as classic tetany and are often accompanied by additional neurological abnormalities, diseases associated with antibodies against the voltage-gated potassium channel complex (such as Isaacs syndrome and Morvan's syndrome) exhibit a broad clinical spectrum based on neuromyotonia and, in their early stages, may present as pseudotetany[36].

The antibodies most relevant in this context are those directed against CASPR2, a protein associated with the potassium channel complex. These antibodies induce peripheral nerve hyperexcitability, resulting in neuromyotonia and myokymia[37].

In some settings, a condition referred to as cryptopyrroluria has been proposed as a possible cause of tetany. It is purported to involve increased urinary excretion of pyrroles that bind vitamin B6 and zinc, leading to deficiencies of zinc and pyridoxal. However, according to the authors of the statement issued by the "Methods and Quality Assurance in Environmental Medicine" commission of the German Robert Koch Institute, there is no evidence supporting this theory, and they do not recommend testing patients for urinary pyrroles[38].

5. Tetany Masked by Other Conditions: Case Illustrations

Because the initial symptoms of tetany are often nonspecific and there is no pathognomonic sign, they may be masked by other conditions or overlooked and mistaken for disease exacerbations. We present several cases in which earlier consideration of tetany in the differential diagnosis would have had a significant impact on patient management.

One such example involves a 38-year-old woman who, secondary to a flare of long-standing Crohn's disease triggered by a change in therapy and cessation of parenteral nutrition, developed life-threatening tetany manifested as diffuse muscle spasms, ultimately leading to rhabdomyolysis. The episode was accompanied by hemodynamic instability, including hypotension and tachycardia; thus, empirical antibiotic therapy was initially started due to suspected septic shock, and electrolyte testing was not performed immediately. Subsequent analysis of the patient's ECG changes, along with the identification of hypokalemia, hypocalcemia, hypomagnesemia, and metabolic alkalosis, and clinical improvement after administration of calcium-containing Ringer's solution, led to the recognition of tetany as the cause of the episode[39].

Another scenario warranting suspicion of tetany is the presentation of ischemic heart disease–like symptoms with a normal echocardiogram. Chest pain, throat constriction, and palpitations are common to both conditions. Consequently, a Cardiology Clinic in Senegal conducted EMG studies on 228 patients with the above symptoms and found latent tetany in 50% of them[40].

6. Discussion:

Currently, there is a lack of large population-based studies needed to determine the true prevalence of tetany, characterize at-risk groups, and its clinical presentation.

Based on an analysis of reported cases, we conclude that in any situation with even minimal suspicion of tetany, it is crucial to promptly perform a complete electrolyte panel (including all electrolytes and ionized calcium) and arterial blood gas analysis, so that results accurately reflect the patient's serum status during the episode. Additionally, it is important not to dismiss tetany even when it appears as a manifestation of panic with hyperventilation, and to administer calcium gluconate as early as possible. This intervention, although not always causative, is the most effective in rapidly relieving muscle spasms.

It is also essential to consider tetany during exacerbations of chronic diseases that may cause electrolyte disturbances, such as Crohn's disease. In cases of recurrent muscle spasms with a normal electrolyte panel,

neurological causes should be considered, and EMG, antibody panels, or genetic testing for renal channelopathies may be warranted[41].

Available literature, primarily consisting of individual case reports, illustrates the challenges in rapid diagnosis and management—particularly in situations where tetany may be life-threatening, and recognition occurs only after other possibilities are excluded. This underscores the need to raise awareness of this condition, which may be far more prevalent in the general population than currently appreciated.

Author's contributions:
Conceptualization: Marianna Latour, Zofia Laska
Methodology: Julia Kosęda, Anna Jędrasiak
Software: Ryszard Łagowski, Honorata Jurkiewicz
Check: Anna Jędrasiak, Jakub Piotrowski,
Formal analysis:, Karolina Kananowicz, Patryk Heryć
Investigation: Jakub Piotrowski,
Resources: Julia Kosęda, Ryszard Łagowski
Data curation: Julia Kosęda, Ryszard Łagowski
Writing - rough preparation: Marianna Latour, Zofia Laska
Writing - review and editing: Zuzanna Kudlińska, Karolina Kananowicz
Visualization: Patryk Heryć, Zuzanna Kudlińska
Supervision: Anna Jędrasiak, Honorata Jurkiewicz
Project administration: Jakub Piotrowski, Julia Kosęda

All authors have read and agreed with the published version of the manuscript.
Conflict of Interest Statement: Authors have declared no conflict of interests.
Declaration of the use of generative AI and AI - assisted technologies in the writing process: In preparing this work, the authors used ChatGPT for the purpose of improving language and readability. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

REFERENCES

1. OpenStax. (2024). *Positive Chvostek's sign and Trousseau's sign*. In *Medical-Surgical Nursing* (Ch. 10.3). LibreTexts. https://med.libretexts.org/Bookshelves/Nursing/Medical-Surgical_Nursing_(OpenStax)/10%3A_Fluid_Electrolyte_and_Acid-Base_Imbalances/10.03%3A_Electrolyte_Imbalance
2. Sikora, P., Zaniew, M., Zawadzki, J., & Drabczyk, R. (2025). Zespół Gitelmana. W Interna Szczeklika. Medycyna Praktyczna. https://www.mp.pl/interna/chapter/B16.II.14.5.6
3. Sikora, P., Zaniew, M., Zawadzki, J., & Drabczyk, R. (2025). Zespół Barttera. W Interna Szczeklika. Medycyna Praktyczna. https://www.mp.pl/interna/chapter/B16.II.14.5.11
4. Santra, G., & Barman, H. (2017). *Etiological spectrum of tetany in a teaching institution of western part of West Bengal – a cross-sectional study*. *Journal of Current Research in Scientific Medicine, 3*(1), 45–50. https://doi.org/10.4103/jcrsm.jcrsm_20_17
5. Gryglas, A., Dudkowiak, R. K., & Śmigiel, R. (2015). *Tetany as a frequent cause of an emergency consultations – etiology, symptoms and cure*. *Przegląd Lekarski, 72*(1), 20–24. https://pubmed.ncbi.nlm.nih.gov/26076573/
6. Sehgal, V., Pathak, A., Singh, J., Singh, A., & Bansal, V. (2011). Normocalcaemic tetany. *European Journal of Emergency Medicine*, 18(5), 159-161. https://doi.org/10.1097/MEJ.0b013e32834eeda0
7. Isgreen, W. P. (1976). Normocalcemic tetany: A problem of erethism. *Neurology, 26*(9), 825–834. https://doi.org/10.1212/WNL.26.9.825
8. Day, J. W., & Parry, G. J. (1990). Normocalcemic tetany abolished by calcium infusion. *Annals of Neurology, 27*(4), 438-440. https://doi.org/10.1002/ana.410270413
9. Fowweather, F. S., Davidson, C. L., & Ellis, L. (1940, September 21). *Spontaneous hyperventilation tetany*. *British Medical Journal*, 2(4159), 373-376. https://doi.org/10.1136/bmj.2.4159.373
10. Alanazi, M., Alabdulgader, A., Alotaibi, A., Bin Ahmed, I., & Maskati, M. (2023). *Tetany in a Young Female Not Resulting From Hypocalcemia*. *Cureus, 15*(8), e43521. https://doi.org/10.7759/cureus.43521
11. Fonseca, V. A., Bloom, R. D., Dick, R., & Dandona, P. (1987). Tetany despite normocalcaemia and normomagnesaemia following parathyroidectomy. *Postgraduate Medical Journal, 63*(744), 885-886. https://doi.org/10.1136/pgmj.63.744.885

12. Boulding, R., Stacey, A., Niven, R., & Fowler, S. J. (2016). Dysfunctional breathing: A review of the literature and proposal for classification. *European Respiratory Review, 25*(141), 287-294. https://doi.org/10.1183/16000617.0074-2016
13. Gardner, W. N., Bass, C., & Moxham, J. (1992). *Recurrent hyperventilation tetany due to mild asthma. Respiratory Medicine, 86*(4), 349-351. https://doi.org/10.1016/S0954-6111(06)80036-3
14. McGillicuddy, C., & Molins, C. (2023). *The effect of hyperventilation syndrome on ionized and serum calcium: A case presentation in the emergency department.* Cureus, 15(7), e42310. https://doi.org/10.7759/cureus.42310
15. Williams, A., Liddle, D., & Abraham, V. (2011). *Tetany: A diagnostic dilemma*. *Journal of Anaesthesiology Clinical Pharmacology*, 27(3), 393–394. https://doi.org/10.4103/0970-9185.83691
16. Vazhakalayil, S. T. J., Veer, S., Badam, B., & Mane, J. (2025). *The domino effect: hyperventilation, hypocapnia, and tetany in airway management*. *Journal of Clinical & Diagnostic Research, 19*(7), UD04-UD06. https://doi.org/10.7860/JCDR/2025/78982.21167
17. Fulchiero, R., & Seo-Mayer, P. (2019). *Bartter syndrome and Gitelman syndrome. Pediatric Clinics of North America, 66*(1), 121-134. https://doi.org/10.1016/j.pcl.2018.08.010
18. Fujihara, K., Miyoshi, T., Yamaguchi, Y., Araki, T., & Tanaka, K. (1990).*Tetany as a sole manifestation in a patient with Bartter's syndrome and a successful treatment with indomethacin Rinsho shinkeigaku = Clinical neurology*, *30*(5), 529–532. https://pubmed.ncbi.nlm.nih.gov/2401113/
19. Suitsu, N., Kita, S., Okamoto, S., Kimura, S., & Shiozaki, Y. (1984).*Development of tetany in siblings suffered from Bartter's syndrome. No to shinkei = Brain and nerve*, *36*(8), 749–754. https://pubmed.ncbi.nlm.nih.gov/6498022/
20. Blanchard, A., Bockenhauer, D., Bolignano, D., Calò, L. A., Cosyns, E., Devuyst, O., Ellison, D. H., Karet Frankl, F. E., Knoers, N. V., Konrad, M., & Lin, S.-H. (2017). *Gitelman syndrome: Consensus and guidance from a Kidney Disease: Improving Global Outcomes (KDIGO) controversies conference. Kidney International, 91*(1), 24–33. https://doi.org/10.1016/j.kint.2016.09.046
21. Gitelman, H. J., Graham, J. B., & Welt, L. G. (1966). *A new familial disorder characterized by hypokalemia and hypomagnesemia. Transactions of the Association of American Physicians, 79*, 221-235. https://doi.org/10.1111/j.1749-6632.1969.tb13015.x
22. Knoers, N. V. A. M., & Levtchenko, E. N. (2008). *Gitelman syndrome*. *Orphanet Journal of Rare Diseases*, 3, 22. https://doi.org/10.1186/1750-1172-3-22
23. Gandhi, K., Prasad, D., Malhotra, V., & Agrawal, D. (2016). Gitelman's syndrome presenting with hypocalcemic tetany. *Saudi Journal of Kidney Diseases and Transplantation, 27*(5), 1026–1028. https://doi.org/10.4103/1319-2442.190881
24. Desai, M., Kolla, P. K., & Reddy, P. L. V. P. (2013). *Calcium unresponsive hypocalcemic tetany: Gitelman syndrome with hypocalcemia. Case Reports in Medicine*, 2013, 197374. https://doi.org/10.1155/2013/197374
25. Kalsi, H. S., Kim, M., & Alkhamesi, A. (2013). Localised normocalcaemic tetany secondary to dehydration in an individual with Gitelman syndrome. *BMJ Case Reports*, 2013, bcr2013008926. https://doi.org/10.1136/bcr-2013-008926
26. Lance, J. W., Burke, D., & Pollard, J. (1979). *Hyperexcitability of motor and sensory neurons in neuromyotonia. Annals of Neurology, 5*(6), 523-532. https://doi.org/10.1002/ana.410050605
27. Imbrici, P., Accogli, A., Blunck, R., Altamura, C., Iacomino, M., D'adamo, M. C., Allegri, A., Pedemonte, M., Brolatti, N., Vari, S., Cataldi, M., Capra, V., Gustincich, S., Zara, F., Desaphy, J. F., & Fiorillo, C. (2021). Musculoskeletal Features without Ataxia Associated with a Novel de novo Mutation in KCNA1 Impairing the Voltage Sensitivity of Kv1.1 Channel. Biomedicines, 9(1), 75. https://doi.org/10.3390/biomedicines9010075
28. Glaudemans, B., van der Wijst, J., Scola, R. H., Lorenzoni, P. J., Heister, A., van der Kemp, A. M. W., Knoers, N. V., Hoenderop, J. G., & Bindels, R. J. (2009). *A missense mutation in the Kv1.1 voltage-gated potassium channel-encoding gene KCNA1 is linked to human autosomal dominant hypomagnesemia. The Journal of Clinical Investigation, 119*(4), 936-942. https://doi.org/10.1172/JCI36948
29. Bianchi, F., Simoncini, C., Brugnoni, R., Ricci, G., & Siciliano, G. (2020). Neuromuscular tetanic hyperexcitability syndrome associated to a heterozygous Kv1.1 N255D mutation with normal serum magnesium levels. *Acta Myologica, XXXIX*(1), 36–39. https://doi.org/10.36185/2532-1900-007
30. Famularo, G., De Filippis, V., & Minisola, G. (2012). Tetany, hypomagnesemia, and proton-pump inhibitors. *The American Journal of Medicine*, 125(10), 1015–1017. https://doi.org/10.1016/j.amjmed.2012.00351
31. Smets, Y. F., Bokani, N., de Meijer, P. H., & Meinders, A. E. (2004). Tetanie bij overmatig alcoholgebruik: mogelijk een magnesiumtekort [Tetany due to excessive use of alcohol: a possible magnesium deficiency]. *Nederlands tijdschrift voor geneeskunde*, *148*(14),641–644. https://pubmed.ncbi.nlm.nih.gov/15106311/
32. Gragossian, A., Bashir, K., Bhutta, B. S., & Friede, R. (2023). Hypomagnesemia. In *StatPearls*. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK500003/
33. Ahmed, F., & Mohammed, A. (2019). *Magnesium: The forgotten electrolyte—A review on hypomagnesemia. Medical Sciences, 7*(4), 56. https://doi.org/10.3390/medsci7040056
34. Warren E. C. Wacker, Moore, F. D., Ulmer, D. D., et al. (1962). *Normocalcemic magnesium deficiency tetany. JAMA, 180*(2), 161–163. https://doi.org/10.1001/jama.1962.030501500000018b

35. Estep, H., Shaw, W. A., Watlington, C., Hobe, R., Holland, W., & Tucker, S. G. (1969). Hypocalcemia due to hypomagnesemia and reversible parathyroid hormone unresponsiveness. *The Journal of Clinical Endocrinology & Metabolism*, *29*(6), 842–848. https://doi.org/10.1210/jcem-29-6-842
36. Plantone, D., Renna, R., & Koudriavtseva, T. (2016). *Neurological diseases associated with autoantibodies targeting the voltage-gated potassium channel complex: Immunobiology and clinical characteristics.* Neuroimmunology and Neuroinflammation, 3(3), 69-78. https://doi.org/10.4103/2347-8659.169883
37. Moura, J., Antenucci, P., Coutinho, E., Bhatia, K. P., Rocchi, L., & Latorre, A. (2025). *Neuromyotonia and CASPR2 antibodies: Electrophysiological clues to disease pathophysiology. Biomolecules*, 15(9), 1262. https://doi.org/10.3390/biom15091262
38. Die (Krypto-)Pyrrolurie in der Umweltmedizin: eine valide Diagnose? Mitteilung der Kommission "Methoden und Qualitätssicherung in der Umweltmedizin" [The (crypto-)pyrroluria in environmental medicine: a valid diagnosis? Report of the "Methods and Quality Assurance in Environmental Medicine" Committee]]. (2007). *Bundesgesundheitsblatt, Gesundheitsforschung, Gesundheitsschutz*, *50*(10), 1324–1330. https://doi.org/10.1007/s00103-007-0340-5
39. Johnson, M. M., Patel, S., & Williams, J. (2019). Don't Take It 'Lytely': A Case of Acute Tetany. Cureus, 11(10), e5845. https://doi.org/10.7759/cureus.5845
40. Marie, B. D., Sow, M. S., Diack, A., Diallo, C. O., Barkire, I., Dieng, F. K., & Fall, M. D. (2017). *Spasmophilia in the cardiological outpatient department: A retrospective study of 228 sub-Saharan Africans over 5 years*. *Journal of Thrombosis and Circulation: Open Access, 3*(3), 121. https://doi.org/10.4172/2572-9462.1000121
41. Deecke, L., Müller, B., & Conrad, B. (1983). Zur Standardisierung des elektromyographischen Tetanietests in der Diagnostik der normokalzämischen Tetanie: 10minütiger Trousseau bei Patienten und Gesunden [Standardization of the electromyographic tetany test in the diagnosis of normocalcemic tetany: the 10-minute Trousseau test in patients and healthy humans]. *Archiv fur Psychiatrie und Nervenkrankheiten*, *233*(1), 23–37. https://doi.org/10.1007/BF00540035