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Spasmophilia Grade IV in Pregnancy: Diagnosis and Conservative Management of a Rare Neuromuscular Disorder of Calcium Dysregulation — A Case Report

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Abstract

Spasmophilia is an uncommon neurovegetative disorder characterized by tissue-level calcium dysregulation and neuromuscular hyperexcitability. Its occurrence in pregnancy is rarely reported, and the physiological remodeling of calcium and magnesium homeostasis during gestation poses distinctive diagnostic and therapeutic challenges. Case Presentation. A 26-year-old primigravida at 36–37 weeks of gestation presented with a history of intermittent stiffness and cramping of the hands and feet that had been diagnosed electrophysiologically as grade IV spasmophilia prior to conception. Involuntary movements had abated during pregnancy. Physical examination revealed positive Chvostek and Trousseau signs; a nerve conduction study demonstrated the repetitive multiplet discharges characteristic of grade IV disease. Laboratory evaluation showed mild anemia (hemoglobin 6 g/dL) with calcium and magnesium within the lower reference ranges. Management and Outcome. The patient was managed conservatively with oral calcium carbonate, folic acid, ferrous sulfate, and neurotropic B vitamins, along with non-pharmacological measures, while avoiding benzodiazepine muscle relaxants, which are contraindicated in pregnancy. She delivered a healthy female neonate (3,175 g, Apgar 8/9) at term without neuromuscular complications. Spasmophilia in pregnancy can be diagnosed through a structured integration of clinical signs, electrophysiological assessment, and electrolyte assessment, and is amenable to safe conservative management centered on calcium supplementation and avoidance of teratogenic relaxants.

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Introduction

Spasmophilia is a neurovegetative disorder defined by a tissue-level dysregulation of calcium during periods of adaptive demand, in which the calcium available to cells and tissues becomes insufficient relative to the quantity required (Hedayat & Lapraz, 2019; Hedayat et al., 2020). The deficiency may arise in two principal ways: an inadequate quantity of calcium or an inadequate temporal availability of calcium relative to demand.1 The clinical consequence is a state of neuromuscular hyperexcitability in which the motor nerve exhibits abnormal sensitivity to mechanical or electrical stimulation, predisposing the patient to spasm, tetany, and cramp accompanied by characteristic clinical, electrical, and humoral findings (Julianti et al., 2017).

The clinical spectrum of spasmophilia is broad and frequently non-specific. Reported manifestations span mental symptoms (panic attacks, depression), neurological complaints (headache), neuromuscular phenomena (muscle cramp, paraesthesia, bronchospasm), digestive symptoms (abdominal colic, gastric reflux), and urinary symptoms (urgency, voiding difficulty).1 This heterogeneity, combined with the rarity of formal diagnosis, means that the condition is easily overlooked, particularly when it coincides with the physiological changes of another major condition such as pregnancy.

Pregnancy itself imposes a profound remodeling of divalent-cation homeostasis. Total serum calcium falls as plasma albumin declines with volume expansion, intestinal calcium absorption rises under the influence of 1,25-dihydroxycholecalciferol, and urinary calcium excretion increases, a state described as the absorptive calciuria of pregnancy (Ahmed, 2021; Kovacs, 2011; Hacker et al., 2012). Magnesium requirements likewise increase. Against this physiological backdrop, the boundary between adaptive change and pathological deficiency can be difficult to define, and an underlying disorder of calcium regulation such as spasmophilia may be unmasked, modified, or masked by gestation.

Reports of spasmophilia complicating pregnancy are scarce, and consensus on diagnostic confirmation and safe therapy in this population is limited. The purpose of this case report is to describe the diagnostic reasoning, electrophysiological confirmation, and conservative management of a primigravida with electrophysiologically confirmed grade IV spasmophilia, and to discuss the relationship between gestational electrolyte homeostasis and the clinical presentation, in order to provide a practical framework for clinicians who encounter this rare combination.

Materials and Methods

This study was conducted as a descriptive case report in accordance with CARE reporting recommendations. The patient was a 26-year-old primigravida with previously diagnosed grade IV spasmophilia who attended antenatal care at 36–37 weeks of gestation.

Clinical assessment included detailed history taking, physical examination, neurological evaluation, laboratory investigations, obstetric ultrasonography, and review of previous electromyographic findings. Diagnostic confirmation was based on clinical manifestations of neuromuscular hyperexcitability and electrophysiological evidence obtained through nerve conduction studies and electromyography.

Management consisted of conservative treatment adapted to pregnancy safety considerations. Maternal and neonatal outcomes were documented until delivery. Written informed consent was obtained for publication.

Results and Discussions

Case Presentation

History

A 26-year-old primigravida (gravida 1, para 0, abortus 0) presented to the fetomaternal clinic at an estimated 36 weeks of gestation with a history of intermittent stiffness of the hands and feet. The first day of her last menstrual period was 23 November 2022. She had been diagnosed with carpopedal spasm suspected to represent spasmophilia in December 2022, prior to conception, and had subsequently been confirmed electrophysiologically as having grade IV spasmophilia. Notably, the involuntary movements had not recurred during pregnancy; residual symptoms were felt predominantly at rest.

She denied hemibody weakness, facial asymmetry, dysarthria, altered consciousness, numbness, nausea, or vomiting. No uterine contractions were radiating to the back, no rupture of membranes, and no bloody show. She reported no use of medication or herbal remedies and had no history of hypertension, diabetes mellitus, cardiac or renal disease, childhood seizures, or a family history of seizures. Fetal movements were preserved. Nutritional and socioeconomic status were adequate. Menarche occurred at 13 years with regular cycles.

Examination

On general examination, the patient was alert with a blood pressure of 120/80 mmHg, pulse 84 beats/min, respiratory rate 20 breaths/min, temperature 36.5 °C, weight 62 kg, and height 160 cm (body mass index 24.2 kg/m^2, normal). Conjunctivae were not pale, sclerae not icteric, the chest was clear, and cardiac auscultation was unremarkable. There was no pretibial edema. Obstetric examination demonstrated a fundal height of 30 cm (two fingerbreadths below the xiphoid process), a longitudinal lie, right-sided fetal back, cephalic presentation, head not engaged (5/5), no uterine contractions, a fetal heart rate of 160 beats/min, and an estimated fetal weight of approximately 2,700–2,790 g.

Targeted neurological examination relevant to neuromuscular excitability revealed positive Chvostek and Trousseau signs, with preserved cranial nerve function, normal limb power and tone, and no pathological reflexes. The Chvostek sign denotes twitching or contraction of the facial musculature on percussion of the facial nerve approximately 0.5–1 cm below the zygomatic process; it is graded from +1 (reaction at the corner of the mouth) through +2 (extending to the ala nasi) to +3 (involving the entire hemiface).4 The Trousseau sign refers to carpopedal spasm provoked by suprasystolic inflation of an upper-arm cuff for several minutes, producing flexion of the wrist and metacarpophalangeal joints, hyperextension of the fingers, and adduction of the thumb—the characteristic main d'accoucheur or obstetrician's hand.

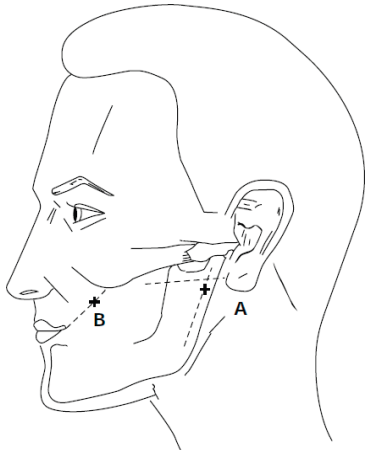

Figure 1. Anatomical landmarks for elicitation of the Chvostek sign. Adapted from Erwin & Fithrie, 2017.

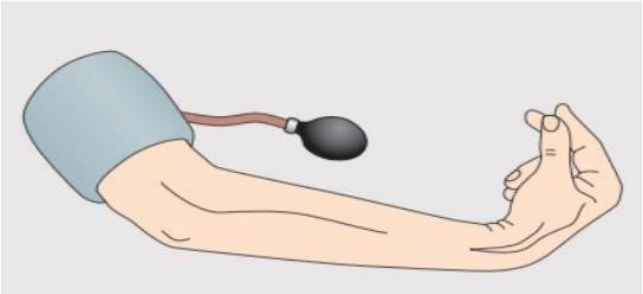

Figure 2. Schematic of the Trousseau maneuver demonstrating carpopedal spasm. *Adapted from Susanti et al., 2020.*

Figure 3. Positive Trousseau sign (obstetrician's hand) elicited in the present patient during sphygmomanometer-induced ischemia.

Investigations

Laboratory evaluation on 7 August 2023 (Table 1) demonstrated mild anemia with a hemoglobin of 9.6 g/dL and a hematocrit of 30%, while leucocyte and platelet counts were within normal limits. Serum calcium (9.2 mg/dL) and magnesium (1.80 mg/dL) lay within the lower reference range; chloride was mildly elevated (111 mEq/L), and potassium was normal. Hepatic enzymes and random blood glucose were normal, and the infectious-disease screen (HBsAg, TPHA, VDRL) was non-reactive.

Table 1

Selected laboratory findings on 7 August 2023.

Parameter	Result	Reference range	Unit
Haemoglobin	9.6	11.4–15.0	g/dL
Erythrocytes	3.65	4.00–5.70	$10^6/mm^3$
Leucocytes	8.96	4.73–10.89	$10^3/mm^3$
Haematocrit	30	35–45	%
Platelets	198	189–436	$10^3/\mu L$
Random glucose	110	<200	mg/dL
Chloride	111	96–106	mEq/L
Potassium	4.0	3.5–5.5	mEq/L

Calcium	9.2	8.6–10.3	mg/dL
Magnesium	1.80	1.8–2.6	mg/dL
SGOT / SGPT	14 / 8	0–32 / 0–31	U/L

Obstetric ultrasonography on 8 August 2023 confirmed a single live fetus in cephalic presentation. Fetal biometry yielded a biparietal diameter of 8.58 cm, head circumference of 31.09 cm, abdominal circumference of 30.52 cm, femur length of 7.12 cm, transcerebellar diameter of 4.67 cm, and an estimated fetal weight of 2,616 g, corresponding to approximately 35–36 weeks of gestation. The placenta was anterior, amniotic fluid was adequate (deepest pocket 3.34 cm), and a single nuchal cord loop was identified. The fetus was assessed as tending towards the smaller side for gestational age.

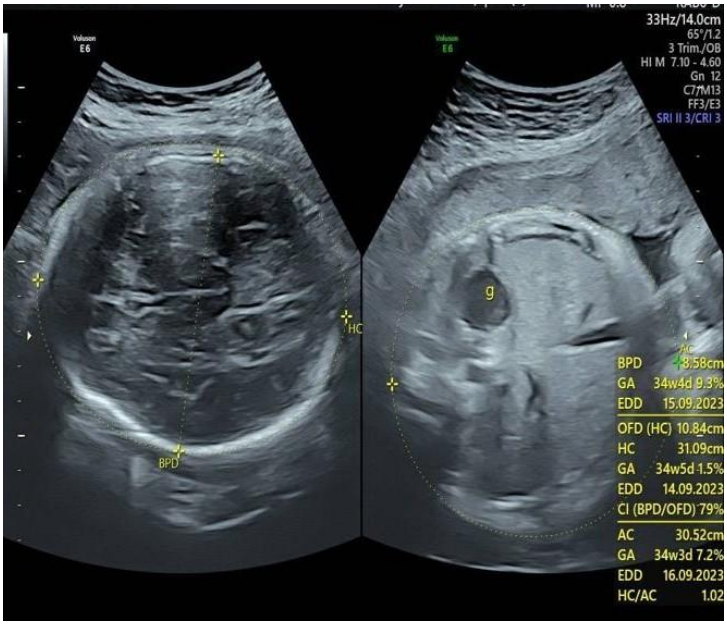

Figure 4. Obstetric ultrasonography demonstrating fetal cephalometry (biparietal diameter and head circumference) consistent with the third trimester.

A nerve conduction study performed on 2 January 2023 concluded that the electrophysiological findings were consistent with grade IV spasmophilia. Electromyography records muscle activity through needle electrodes at rest and during graded contraction; in spasmophilia, an electrical stimulus of ordinarily subthreshold intensity provokes potential amplitudes markedly above the mean.3,4 The pathognomonic finding is the appearance of doublets, triplets, or multiplets—repetitive motor-unit action potentials arising from neuromuscular hyperexcitability.4,6 A doublet is a spontaneous motor-unit action potential occurring as two linked discharges; three discharges constitute a triplet, and more than three a multiplet.

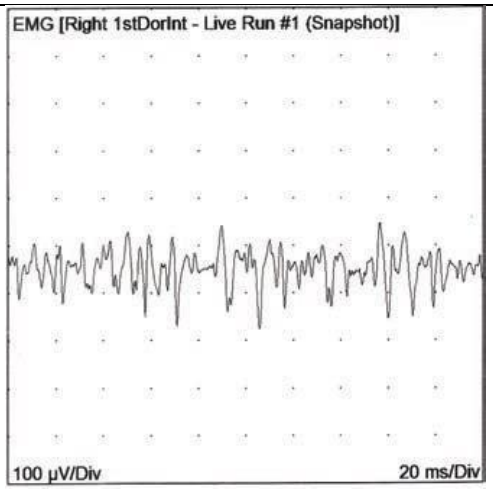

Figure 5. Electromyographic recording from the present patient showing repetitive multiplet discharges characteristic of grade IV spasmophilia. Patient record, 2 January 2023.

Based on clinical grading, electromyographic patterns are stratified into four grades: grade 1+, infrequent doublets, triplets, or multiplets after hyperventilation; grade 2+, abundant multiplets after hyperventilation without obstetrician's hand; grade 3+, abundant multiplets with obstetrician's hand after hyperventilation; and grade 4+, abundant multiplets and obstetrician's hand emerging after an ischaemic test.4,6 In the present patient, the combination of bilateral hand stiffness with paraesthesia, positive Chvostek and Trousseau signs, and a nerve conduction study reported as grade 4 established the diagnosis.

Diagnosis

The working diagnosis was a primigravida at 36 weeks of gestation, not yet in labor, with grade IV spasmophilia and a single live fetus in cephalic presentation. The maternal and fetal prognoses were each considered guarded (dubia) at presentation.

Management and Outcome

Management was conservative. The patient received oral calcium carbonate (500 mg three times daily), folic acid (400 µg once daily), ferrous sulfate (one tablet once daily), and a neurotropic B-vitamin combination (vitamins B1, B6, and B12) once daily, with continued observation of vital signs, uterine activity, and fetal heart rate. Antenatal review one week later confirmed a stable maternal condition and reassuring fetal growth on ultrasonography (estimated fetal weight 2,593–2,635 g; adequate amniotic fluid).

On 29 August 2023, the patient delivered a live female neonate weighing 3,175 g, measuring 49 cm in length and 24 cm in head circumference, with Apgar scores of 8 and 9 at 1 and 5 minutes, respectively. The neonate was assigned to rooming-in care. No neuromuscular complications occurred in the mother during the peripartum period.

Discussion

This case illustrates three issues of practical importance: the structured approach to diagnosing spasmophilia in pregnancy, the selection of a therapeutic strategy compatible with gestation, and the interpretation of clinical symptoms in the light of pregnancy-specific electrolyte physiology.

Establishing the diagnosis

Diagnosis was based on the integration of history, physical examination, and targeted investigations. The history of pre-gestational carpopedal spasm, subsequently confirmed electrophysiologically, provided anchoring evidence, while the bedside Chvostek and Trousseau signs offered simple, reproducible indicators of latent neuromuscular hyperexcitability (Erwin & Fithrie,

2017). Additional bedside maneuvers described in the literature, including hippus, glabellar tap, tongue fasciculations, and skeletal-muscle fasciculations, may further support the clinical impression (Hedayat et al., 2020). Laboratory markers that may accompany spasmophilia include increased bone alkaline phosphatase, altered osteocalcin concentrations, phosphate abnormalities, and reductions in magnesium paralleling calcium deficiency (Hedayat et al., 2020). Electromyography remains the confirmatory modality, with doublets, triplets, and multiplets constituting the electrical signature of the disorder; in the present patient, the nerve conduction study was reported as grade 4, the most severe category (Julianti et al., 2017; Erwin & Fithrie, 2017; Ginanneschi et al., 2018).

Therapeutic strategy in pregnancy

Calcium supplementation is the treatment of choice for spasmophilia, and a muscle relaxant may be added in non-pregnant patients (Erwin & Fithrie, 2017). Pregnancy alters this therapeutic approach because calcium demand rises substantially during the third trimester to support fetal skeletal mineralization (Hacker et al., 2012; Kovacs, 2011). The World Health Organization recommends daily supplementation of 1.5–2.0 g elemental calcium from approximately 20 weeks of gestation until delivery in populations with low dietary calcium intake (World Health Organization, 2013; World Health Organization, 2016). Alprazolam is classified as a category D medication and is not recommended during pregnancy because studies have demonstrated fetal risk (Gidai et al., 2008). For pregnancy-associated muscle cramps, stretching, massage, relaxation techniques, and local heat application are preferred (Luo et al., 2020).

Magnesium supplementation has been shown to benefit women with leg cramps during pregnancy (Katzberg, 2020; Araújo et al., 2020). Anemia was defined according to World Health Organization criteria as hemoglobin below 11 g/dL during pregnancy (Frayne & Pinchon, 2019). Daily supplementation with folic acid and iron improves maternal hemoglobin concentration and pregnancy outcomes (Frayne & Pinchon, 2019; Yismaw et al., 2018). Vitamins B1, B6, and B12 contribute to neuronal metabolism, neurotransmitter synthesis, remyelination, and nerve regeneration (Silviana et al., 2021; Baltrusch, 2021).

The neurotropic combination of vitamins B1, B6, and B12 normalizes nerve function by addressing metabolic disturbance. Thiamine (B1) acts as a coenzyme in carbohydrate metabolism, supplying energy to nerve fibers through thiamine pyrophosphate-dependent oxidative metabolism and ultimately ATP production. Pyridoxine (B6) is central to amino-acid and neurotransmitter metabolism, modulating glutamate release and supporting sphingolipid synthesis for the myelin sheath. Cobalamin (B12) contributes to nerve regeneration, neuronal survival, remyelination, and maintenance of the myelin sheath; the three vitamins act synergistically to support nerve regeneration more effectively than any agent alone.

Electrolyte homeostasis and the clinical picture

In pregnancy, total serum calcium declines as albumin falls with volume expansion. The recommended daily calcium allowance rises from 1,000 mg in the healthy non-pregnant adult to approximately 1,200 mg during gestation. In the non-pregnant state, calcium metabolism is governed by parathyroid hormone, calcitonin, and the active form of vitamin D (1,25-dihydroxycholecalciferol): parathyroid hormone mobilises calcium and phosphate from bone via osteoclasts, calcitonin restrains osteoclastic activity and limits renal calcium reabsorption, and active vitamin D enhances intestinal and renal calcium and phosphate handling, with parathyroid hormone driving renal conversion of vitamin D to its active form (Ahmed, 2021).

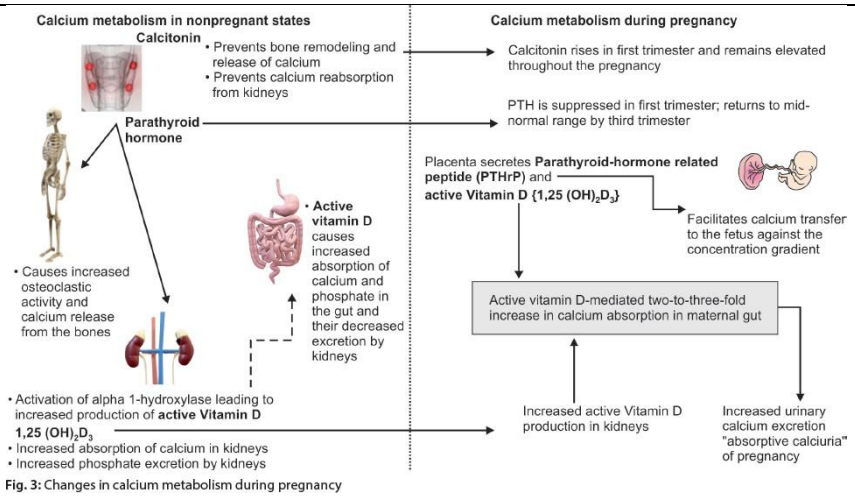

Figure 6. Changes in calcium metabolism during pregnancy. Adapted from Ahmed, 2021.

The maternal physiology that facilitates calcium delivery to the fetus centers on increased intestinal absorption of calcium and phosphorus, mediated by elevated 1,25-dihydroxycholecalciferol. The resulting rise in absorbed calcium increases renal calcium excretion—the absorptive calciuria of pregnancy—and calciuria is itself a urinary marker that may indicate spasmophilia in pregnant women. Parathyroid hormone is suppressed toward the low-normal range in the first trimester. It rises to mid-normal by the third trimester, facilitating maternal skeletal calcium accrual early in gestation for later transfer to the fetal skeleton as fetal demand peaks. Calcitonin rises in the first trimester and remains elevated, with additional secretion by the breast and placenta, which restrain maternal bone resorption (Ahmed, 2021).

The principal causes of hypocalcemia in pregnancy are inadequate oral calcium intake and vitamin D deficiency. Maternal hypocalcemia can reduce transplacental calcium transfer and fetal parathyroid stimulation, potentially producing fetal hyperparathyroidism with skeletal deformity, intrauterine fracture, long-bone bowing, and low birth weight. Maternal hypercalcemia, although rare, may present with nausea, vomiting, generalized pain, anxiety, or depression and can lead to neonatal hypocalcemia, permanent hypoparathyroidism, and fetal growth restriction (Ahmed, 2021).

Magnesium contributes to vasomotor tone, and its deficiency is linked to hypertension outside pregnancy. Serum magnesium remains unchanged or falls slightly in pregnancy because of haemodilution, and its homeostasis is incompletely understood given its predominantly intracellular distribution. The dailyThe daily magnesium requirement is 300–360 mg in healthy women, rising by an additional 40–50 mg in pregnancy, and is regulated by intestinal absorption and renal excretion.16 The leading cause of hypomagnesemia in pregnancy is inadequate maternal intake; other causes include chronic diarrhea, diabetes mellitus, certain drugs (loop diuretics, thiazides, aminoglycosides, amphotericin B, calcineurin inhibitors, prior cisplatin), and genetic disorders. Magnesium deficiency frequently coexists with other electrolyte abnormalities, may present as lethargy and weakness, and in severe form as tetany and cardiac arrhythmia (Ahmed, 2021).

In the present case, the cramping and stiffness of the hands and feet represent muscle spasm attributable to neuromuscular hyperexcitability.3 Muscle cramps are among the most common complaints reported by pregnant women, particularly in the second and third trimesters; approximately 30–50% of women experience leg cramps at least twice weekly in the third trimester.7,17 Recognized contributors include excessive ankle plantar-flexor load, over-exertion, metabolic and circulatory disturbance, underlying medical conditions, nutritional deficiency (vitamins E and D), and electrolyte imbalance involving magnesium, calcium, and sodium.17 The convergence of an established disorder of calcium regulation with these gestational influences

provides a coherent explanation for the symptomatology observed in this patient. It reinforces the rationale for a calcium-centered conservative strategy.

Conclusion

Spasmophilia is a rare neuromuscular disorder characterized by calcium dysregulation that may be encountered in pregnancy, where it overlaps with the physiological remodeling of calcium and magnesium homeostasis. This case demonstrates that the diagnosis can be established through a structured synthesis of history, the bedside Chvostek and Trousseau signs, electrolyte assessment, and electromyographic confirmation of repetitive multiplet discharges. Management in pregnancy should center on calcium supplementation, correction of coexisting anemia, and neurotropic support, while avoiding benzodiazepine muscle relaxants that carry fetal risk and favoring non-pharmacological measures for acute cramps. With this approach, the present patient achieved a favorable outcome, delivering a healthy term neonate without maternal or neonatal neuromuscular complications. Clinicians should remain alert to the possibility of an underlying calcium-regulation disorder in pregnant women presenting with cramps and tetany. They should individualize therapy in accordance with the safety constraints of gestation.

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Conflict of Interest

The authors declare that they have no conflict of interest.

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