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Primary Hypoparathyroidism Exacerbated by Dengue with Seizures

Edras Samuel Hernandez Musus[1]*, Allan Antonio Vargas Escobar[1], Jonatan Esau Del Cid Montufar[2], Victor Enrique Roman Toro[3]

[1]Internal Medicine Resident, Guatemalan Social Security Institute (IGSS), General Hospital of Diseases, University of San Carlos of Guatemala, Guatemala City, Guatemala. [2]Head of Observation Service, Guatemalan Social Security Institute (IGSS), General Hospital of Diseases, Guatemala City, Guatemala. [3]Endocrinologist, Guatemalan Social Security Institute (IGSS), General Hospital of Diseases, Guatemala City, Guatemala.

**Corresponding author: Edras Samuel Hernandez Musus.*

Abstract

Primary hypoparathyroidism is a rare endocrine disorder 10. The first is the first in the series of 1990s films. by low parathyroid hormone (PTH) levels and serum calcium. We present the case of a 25-year-old patient who presented with generalized seizures. Initial studies revealed severe hypocalcemia (calcium 5.3 mg/ dL), low PTH (5.7 pg / mL), thrombocytopenia (68,000/ µL), and elevated liver function tests (AST 223 U/L, ALT 180 U/L), suggesting primary hypoparathyroidism exacerbated by acute dengue infection (positive antigen). The electrocardiogram showed sinus rhythm, and venous blood gas confirmed hypocalcemia without acid -base abnormalities. Both brain CT and MRI were normal, showing no calcifications typically associated with advanced hypoparathyroidism. Lumbar puncture and additional infectious tests they were negative. Electroencephalogram (EEG) revealed abnormal findings suggestive of cortical irritative activity in the left frontotemporal region with contralateral projection. Treatment included intravenous calcium gluconate and teriparatide to correct hypocalcemia, along with dengue management. This case illustrates how the inflammatory and metabolic stress of dengue can decompensate underlying endocrine conditions. Compared to a documented case with basal ganglia calcifications, our patient showed no such signs, highlighting variations in the clinical presentation of hypoparathyroidism exacerbated by acute infections.

Keywords: generalized seizures; severe hypocalcemia; primary hypoparathyroidism; dengue; cortical irritative activity endocrine decompensation

Introduction

Primary hypoparathyroidism is a rare endocrine disorder 10. The first is the first in the series of 1980s films. by impaired production of parathyroid hormone (PTH), resulting in hypocalcemia. This condition may remain asymptomatic until a stress factor, such as an infection, triggers severe clinical manifestations. Primary hypoparathyroidism can be misdiagnosed as epilepsy due to the neuromuscular symptoms associated with hypocalcemia, such as seizures and tetany [1]. Dengue, a viral infection prevalent in tropical regions, can complicate the management of hypoparathyroidism. Dengue has been shown to cause hypocalcemia through various mechanisms, including calcium loss due to capillary leakage and increased metabolic demand during acute infection [2]. This case presents the exacerbation of primary hypoparathyroidism during an acute dengue infection, leading to seizures. There are case reports where dengue is triggered the clinical demonstration of severe hypocalcemia in patients with primary hypoparathyroidism [3, 4].

Case Presentation

A 25-year-old male patient was admitted to the emergency department following a generalized tonic-clonic seizure episode that began five hours prior to presentation. The patient experienced loss of consciousness and a postictal state lasting approximately 30 minutes, without incontinence. He was transported to the hospital by paramedics. The patient had no relevant medical history and worked remotely in a call center. He reported no substance use, drugs, alcohol, or tobacco A week prior; he had experienced myalgia associated with an undocumented feverish episode, which he had dismissed. Initial physical examination recorded the following vital signs: blood pressure of 134/84 mmHg, heart rate of 76 beats per minute, respiratory rate of 21 breaths per minute, glucose level of 170 mg/ dL, and oxygen saturation of 98% in ambient air.

The patient was afebrile, weighed 75 kg, and was well hydrated Neurologically, he was alert but presented episodes of drowsiness and fluctuating consciousness, along with tongue lacerations. I have responded appropriately to simple questions but became irritable intermittently. I have obeyed simple commands, such as opening and closing his eyes and moving his limbs, and could articulate short phrases. Lung auscultation revealed good bilateral air entry with no abnormal sounds (no rhonchi or wheezes) and no use of accessory muscles. Generally, the patient was in good respiratory and neurological condition, although with episodes of irritability and drowsiness.

Admission laboratory findings showed significant abnormalities: serum calcium of 4.8 mg/ dL (severe hypocalcemia), platelet count of 69,000/mm³ (thrombocytopenia), WBC of 7.07 x 10^3/µL, LYN of 9.4%, hematocrit of 42.3%, ESR of 20 mm/h, and procalcitonin of 0.25 ng/ mL. Additional findings included total bilirubin (1.7 mg/ dL), ALT (142 U/L), and GGT (503 U/L). Other results included a total CPK of 1715 U/L (indicating muscle injury), prolactin of 12.7 ng/ mL, vitamin D of 25 ng/ mL, and parathyroid hormone (PTH) of 5.76 pg / mL, indicating primary hypoparathyroidism. Due to the severity of his condition, the patient was assessed with multiple diagnostic and therapeutic interventions. An electrocardiogram was obtained to evaluate potential alterations caused by hypocalcemia, revealing sinus rhythm without other significant abnormalities. A lumbar puncture with a meningitis panel yielded negative results, ruling out central nervous system infection. A brain CT scan showed no calcifications, and a urine toxicology screen was negative (see Figure 1).

Figure 1: Axial brain CT, showing no calcifications characteristic of advanced hypoparathyroidism.

Due to the presence of thrombocytopenia, elevated liver enzymes, and a history of fever and myalgia, dengue was suspected and confirmed with a positive NS1 antigen test. Immediate management with intravenous calcium replacement was initiated, including a bolus followed by maintenance infusion. For seizure control, a loading dose of levetiracetam was administered, followed by a regular dosage regime. After stabilization, the patient was transferred to the ward, where an electroencephalogram (EEG) was performed. The EEG showed an abnormal trace with findings suggestive of cortical irritative activity in the left frontotemporal region with contralateral projection. A brain MRI was also performed, yielding normal results (see Figure 2).

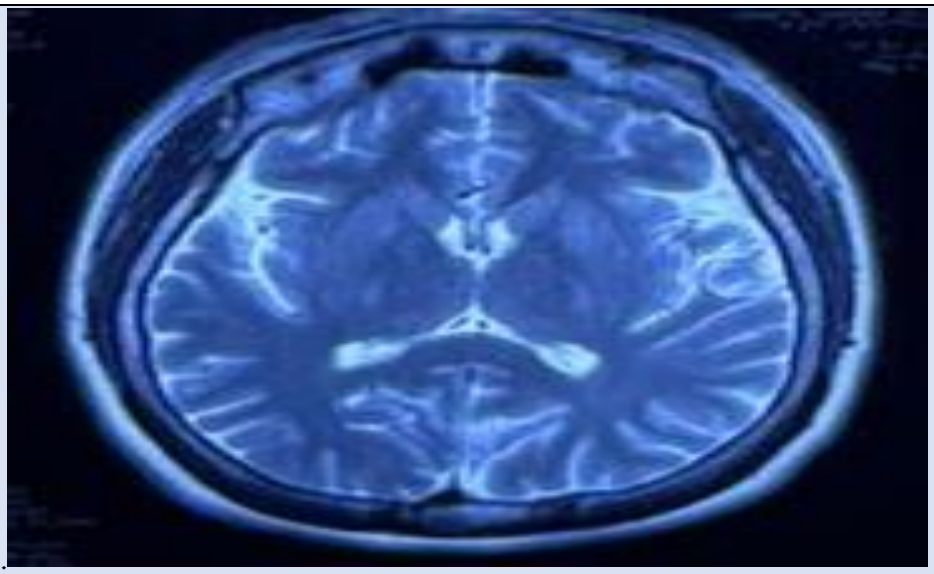

Figure 2: Axial T2-weighted MRI showing no structural abnormalities.

The patient was evaluated by neurology, where the decision was made to continue only with antiseizure medication. Subsequently, endocrinology adjusted the management of hypoparathyroidism, initiating therapy with teriparatide.

Discussion

Primary hypoparathyroidism is a rare endocrine disorder 10. The first is the first in the series of 1990s films. by low parathyroid hormone (PTH) levels and consequences hypocalcemia. In this case, we present a young patient with previously undiagnosed primary hypoparathyroidism that was exacerbated during an acute dengue infection, resulting in seizures. This case underscores the importance of considering acute infections as potential triggers for endocrine decompensations in patients with underlying conditions [5,6]. Dengue, a common viral infection in tropical regions, can complicate the management of endocrine conditions like hypoparathyroidism. Hypocalcemia in dengue may result from various mechanisms, including capillary leakage and increased metabolic demand during acute infection. These mechanisms can aggravate preexisting hypocalcemia in patients with hypoparathyroidism, leading to severe neuromuscular manifestations such as seizures and tetany [7,8].

In this case, the patient presented with generalized tonic-clonic seizures, and initial studies revealed severe hypocalcemia (calcium 4.8mg/dL) and low PTH levels (5.7pg/mL). The presence of thrombocytopenia and elevated liver function tests, along with a history of fever and myalgia, suggested dengue infection, confirmed by a positive NS1 antigen test.1,7 Initial management included correction of hypocalcemia with intravenous calcium gluconate, followed by maintenance infusion, and administration of levetiracetam for seizure control. The lumbar puncture and additional infectious tests were negative, ruling out other infectious etiologies of the central nervous system. Both the brain CT and MRI showed no calcifications typical of advanced hypoparathyroidism, consistent with a recent disease diagnosis.9 The EEG demonstrated cortical irritative activity in the left frontotemporal region with contralateral projection, suggesting a possible structural cause for the seizures. However, the brain MRI was normal, ruling out evident structural injuries [2].

The patient's management was optimized by adding teriparatide, a PTH analog, enabling better regulation of calcium levels and improving his clinical state. This case highlights the need for comprehensive evaluation and multidisciplinary management in patients with endocrine decompensations exacerbated by acute infections [1,9]. This case report contributes to the existing literature on the interaction between acute infections and endocrine decompensations, emphasizing the need to consider underlying endocrine diagnoses in patients with atypical clinical presentations during acute infections [3,5,6].

Declarations

Ethics approval and consent to participate

This clinical case report did not require review by the ethics committee, but informed consent was obtained from the patient for their participation.

Consent for publication

The patient provided consent for the publication of this clinical case and associated data.

Availability of data

The data used in these reports are available upon reasonable request from the corresponding author.

Competing interests

The authors declare that they have no competition interests related to this manuscript.

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Author Information

Hernández Musús Edras Samuel, Internal Medicine Resident, University of San Carlos of Guatemala, Guatemala City, Guatemala.

References

1. Khor, Z. X., Pua, Q. Y., & Tai, Y. T. (2023). Primary hypoparathyroidism: Hypocalcaemia misdiagnosed as epilepsy. *BMJ Case Reports*, 16(10):e257005.
2. Shivanthan, M. C., & Rajapakse, S. (2014). Dengue and calcium. *International Journal of Critical Illness and Injury Science*, 4(4):314-316.
3. Higgoda, R., Lokuketagoda, K., Poobalasingham, T., Wedagedara, V., Perera, D., et al. (2018). Dengue fever manifesting with tetany as the first presentation of primary hypoparathyroidism: A case report. *BMC Research Notes*, 11(1):588.
4. Pasieka, J. L., Wentworth, K., Yeo, C. T., Cremers, S., Dempster, D., Fukumoto, S., et al. (2020). Etiology and pathophysiology of hypoparathyroidism: A narrative review. *Journal of Bone and Mineral Research*, 37(12):2586-2601.
5. Georgakopoulou, V. E., Avramopoulos, P., Papalexis, P., Bitsani, A., Damaskos, C., et al. (2022). COVID-19 induced hypoparathyroidism: A case report. *Experimental and Therapeutic Medicine*, 23(5):346.
6. Sindhuja, T., Akash, K., Bhatia, G., Gupta, S., & Kumar, A. (2024). Primary hypoparathyroidism associated with catatonia in a patient with bipolar affective disorder: A case report. *Psychiatry Research Case Reports*.
7. Guzman, M., Gubler, D., & Izquierdo, A. et al. (2016). Dengue infection. *Nature Reviews Disease Primers*, 2:16055.
8. Umakanth, M., & Suganthan, N. (2020). Unusual manifestations of dengue fever: A review on expanded dengue syndrome. Cureus, 12(9): e10678.
9. Hans, S. K., & Levine, S. N. (2024). Hypoparathyroidism. *StatPearls*.

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