

Clarifying clinical findings in Hoffmann syndrome and myxedema

Jacob Draves¹, Steven Yale², Halil Tekiner³, Eileen Yale⁴

¹ Midwestern University, Downers Grove, United States of America

² University of Central Florida, Orlando, United States of America

³ Erciyes University, Kayseri, Türkiye

⁴ NOVA Southeastern University, Jacksonville, United States of America

Corresponding author: Steven Yale, University of Central Florida, Orlando, United States of America; Email: steven.yale.md@gmail.com

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Letter to the Editor

Dear Editor

Bano et al.^[1], in their paper titled “Hoffmann syndrome: a rare presentation of hypothyroid myopathy,” discuss their findings regarding a patient with this syndrome. Reports of Hoffmann syndrome remain scarce in the literature. In this letter, we build upon Hoffmann’s original observation, clarify additional signs, and interpret them in relation to the features described in this patient.

Johann Hoffmann (1857–1919) first reported the case of an 18-year-old male who underwent multiple partial thyroidectomies due to goiter, resulting in hypothyroidism.^[2] The patient exhibited rigidity of the lower limbs, impaired gait, delayed patellar reflexes, myxedema, cold intolerance, muscle spasms, and hoarseness of voice. He had delayed muscle relaxation, marked by challenges in initiating movements, as seen by his extended difficulty in opening his hand and ambulating. Notably, his condition improved with repetitive voluntary movements; however, residual muscle contraction continued to cause stiffness and a sluggish gait even after several steps. His muscles were enlarged and firm. Hoffmann concluded that the delayed muscle relaxation was not indicative of true myotonia, as residual contraction and stiffness persisted. The symptoms abated with thyroid extract, prompting Hoffmann to ascribe the low thyroid hormone levels.^[2]

In the present case, Bano et al. document delayed deep tendon reflexes—a feature consistent with Woltman sign, the delayed relaxation of muscle stretch reflexes in myxedema.^[1] Chaney (1924) quantified this sign in the Achilles tendon, although Henry Woltman (1889–1964) had earlier recognized it clinically.^[3] In their report, the authors described

right-dominant calf pseudohypertrophy confirmed by magnetic resonance imaging (MRI) and normal electromyography (EMG), markedly elevated creatine kinase (CK) and thyroid-stimulating hormone (TSH) levels, and prompt recovery following levothyroxine therapy—findings that closely mirror Hoffmann’s original description of reversible hypothyroid myopathy.^[1] The patient also displayed Gowers’ sign, described by William Richard Gowers (1845–1915), who observed that patients with pseudohypertrophic muscular paralysis place their hands on their knees to rise—a movement he attributed to Guillaume Duchenne de Boulogne (1806–1875).^[4] Gowers later characterized the maneuver now bearing his name: from a prone position, patients push backward with hands and feet apart, alternately walking the hands up the thighs until an upright stance is achieved.^[4]

Hoffmann syndrome, also known as hypothyroid myopathy or myxoedematous myotonia, is characterized by painful muscle stiffness and pseudomyotonia. Hoffmann’s original report did not describe proximal muscle weakness or hypertrophy limited to the calves. Instead, he noted myxedema, delayed reflexes, generalized muscular enlargement, stiffness, pseudomyotonia, and spasms—features that later became essential for diagnosis.

This letter corrects the historical attribution of Gowers sign to Duchenne, outlines its performance, and situates it within the context of hypothyroid myopathy. It also re-emphasizes the Woltman sign as a neurophysiological correlate of the reflex delay seen in this patient. Finally, it highlights the absence of sustained contractions or pseudomyotonia in the case, helping to distinguish adult Hoffmann syndrome from Kocher-Debré-Semelaigne syndrome, its pediatric analogue.^[5]

Ethical statements

The authors declared that no clinical trials were used in the present study.

The authors declared that no experiments on humans or human tissues were performed for the present study.

The authors declared that no informed consent was obtained from the humans, donors or donors' representatives participating in the study.

The authors declared that no experiments on animals were performed for the present study.

The authors declared that no commercially available immortalized human and animal cell lines were used in the present study.

Conflict of interest

The authors have declared that no competing interests exist.

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Use of AI

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Data availability

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Author contributions

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References

1. Bano S, Anwar SO, Ambreen S, et al. Hoffmann syndrome: a rare presentation of hypothyroid myopathy. *Folia Med (Plovdiv)* 2025; 67(5):1–4. doi: 10.3897/folmed.67.e153006. PMID: 41163611.
2. Hoffmann J. Weiterer Beitrag zur Lehre von der Tetanie [Further contribution to the doctrine of tetany]. *Dtsch Z Nervenheilkd* 1897; 9:278–90 [German].
3. Chaney WC. Tendon reflexes in myxedema: a valuable aid in diagnosis. *JAMA* 1924; 82(25):2013–6. doi: 10.1001/jama.1924.02650510013005
4. Gowers WR. Pseudohypertrophic muscular paralysis: a clinical lecture. London: J & A Churchill; 1879.
5. Mangaraj S, Sethy G. Hoffman's syndrome - a rare facet of hypothyroid myopathy. *J Neurosci Rural Pract* 2014; 5(4):447–8. doi: 10.4103/0976-3147.140025. PMID: 25288869; PMCID: PMC4173264.