Clinical Neurology and Neuroscience
2026, Vol. 10, No. 1, pp. 1–4
https://doi.org/10.11648/j.cnn.20261001.11

Case Report

Corticosteroid-Induced Myopathy in a 10-Year-Old: A Case from Kara (Togo)

Lehleng Agba[1, *], Kokou Mensah Guinhouya[2], Komla Nyinèvi Anayo[3], Adama Ephoevi-Ga[3], Komi Apetse[4], Vinyo Kumako[1], Damelan Kombaté[5], Komi Assogba[2], Mofou Belo[2], Agnon Ayelola Balogou[3]

[1]Department of Neurology, University Hospital of Kara, University of Kara, Kara, Togo
[2]Department of Neurology, CHU Sylvanus Olympio, University of Lomé, Lomé, Togo
[3]Department of Neurology, CHU Campus, University of Lomé, Lomé, Togo
[4]Department of Neurology, Atakpamé Regional Hospital, University of Lomé, Lomé, Togo
[5]Department of Neurology, Kara Regional Hospital, University of Kara, Kara, Togo

Abstract

Introduction: Corticosteroid-induced myopathy (CIM) remains underdiagnosed, particularly in children. Its acute form can emerge rapidly after the initiation of corticosteroid therapy, sometimes within the first few days. Identification relies on a constellation of clinical-chronological and biological arguments, together with observation of the course after steroid withdrawal. We report an acute CIM in a 10-year-old girl from Kara (Togo) to illustrate a pragmatic diagnostic approach in a resource-limited setting. *Case presentation*: A 10-year-old schoolgirl referred for right-sided visual loss received an initial corticosteroid course with methylprednisolone 240 mg/day for 5 days, with partial improvement. Two weeks later, following a relapse, she received a second course of methylprednisolone 1 g/day for 5 days. Four days after completing this treatment, she developed diffuse myalgias and proximal weakness with inability to raise the lower limbs and walk. Beyond the clinical picture, laboratory tests showed creatine kinase (CK) 876 U/L (≈7.6× the upper limit of normal [ULN]) and aspartate aminotransferase (AST) ≈2.5× ULN, supporting a diagnosis of CIM. Other muscle enzymes were unavailable, as was electromyography. Management consisted of steroid withdrawal, analgesics and anti-inflammatory agents, and physiotherapy. Clinical improvement occurred within 72 hours; on day 7, CK was 430 U/L (≈3.7× ULN), followed by normalization to 97 U/L at 3 months, with complete functional recovery. *Conclusion*: This observation illustrates that early-onset proximal weakness occurring soon after corticosteroid pulses should prompt consideration of CIM, even in the absence of EMG. In resource-limited contexts, the trajectory of CK and the response to dechallenge are decisive elements that help optimize prognosis in a timely manner.

Keywords

Myopathy, Corticosteroid, Child, Togo, Sub-Saharan Africa

*Corresponding author: thierrielle@gmail.com (Lehleng Agba)

Received: 1 October 2025; **Accepted:** 7 January 2026; **Published:** 16 January 2026

1. Introduction

Myopathies comprise a heterogeneous group of skeletal muscle disorders with both hereditary and acquired etiologies [1]. Among the acquired causes, drug-induced myopathies are notable and include corticosteroid-induced myopathy (CIM), a complication related to exogenous or endogenous glucocorticoid excess [2]. Two patterns are typically described: a chronic form and an acute form, the latter often occurring within days of high-dose steroid pulses. Although rare overall and difficult to quantify precisely, acute forms remain underrecognized, even in high-income settings that benefit from registries, EMG/MRI, and broader laboratory panels; consequently, they are frequently published as single cases or small series [3]. Historically, the index description of acute CIM was reported in 1980 in *The British Medical Journal* (BMJ) [4]. In Africa, a four-case series was published in Tunisia (North Africa) in 2017 [5]. In sub-Saharan Africa—and particularly in pediatrics—reports remain scarce. To our knowledge, no case has been published from Kara (Togo). We therefore report an acute CIM in a 10-year-old girl occurring a few days after methylprednisolone pulses.

2. Methods

This is a single-patient case report conducted at the Department of Neurology, University Hospital of Kara (Togo). The approach consisted of a detailed clinical description complemented by routine laboratory testing available on site. Electromyography and muscle MRI were not accessible at the time of presentation. The case is presented in accordance with good reporting practices for clinical observations; the patient's data were de-identified, and consent for publication was obtained.

3. Case Presentation

A 10-year-old schoolgirl, residing in Kara, was referred from Ophthalmology to Neurology for optimal management of right-sided visual loss suggestive of retrobulbar optic neuritis (RBON). Symptom onset dated to 12 January 2022, with a rapidly progressive decline in visual acuity of the right eye, without trauma or fever. She was initially managed in Lomé (Ophthalmology), where the examination supported RBON. A corticosteroid course with methylprednisolone 240 mg/day for five days was initiated, resulting in partial improvement in right-eye vision. Brain CT was performed and showed no abnormalities to account for the symptoms.

Two weeks after completing this first course (back in Kara), the patient experienced relapse of visual symptoms, prompting re-evaluation. Summary of the ophthalmologic findings: normal acuity in the left eye (10/10) and light perception only in the right eye; right optic disc pallor with sharp margins. The mother reported that the child no longer distinguished objects clearly and perceived only light with the right eye, with intense retro-orbital pain, especially on ocular movement. There were no headaches, vomiting, altered consciousness, or seizures, and no motor, sensory, or sphincter disturbances were reported. On 10 March 2022, the clinical examination found preserved general condition with body weight 63 kg. The neurological exam was unremarkable (normal tone, strength, and tendon reflexes; no limb motor/sensory deficit; no cerebellar, pyramidal, or meningeal signs), and neuropsychomotor development was age-appropriate. RBON remained the leading working diagnosis at that stage.

Given the relapse, a second course of methylprednisolone 1 g/day for five days was administered. Four days after completing pulse therapy, the patient developed diffuse myalgias and proximal weakness with inability to raise the lower limbs and walk. On neurological examination performed at admission, there was symmetrical proximal muscle weakness predominantly affecting the shoulder and pelvic girdle muscles, preserved distal strength, normal deep tendon reflexes, and no sensory impairment. The muscles were painful to palpation but without focal tenderness or swelling. Cranial nerve examination was normal. Laboratory tests showed elevated serum creatine kinase (CK) 876 U/L (≈7.6 × upper limit of normal for girls in the local laboratory) and aspartate aminotransferase (AST) ≈2.5× upper limit of normal (ULN). Aldolase and lactate dehydrogenase (LDH) were unavailable; anti-aquaporin-4 antibodies were negative; and electromyography was not accessible. The diagnosis of acute drug-induced myopathy attributable to corticosteroids was made. Management was conservative, consisting of steroid withdrawal, analgesics/anti-inflammatories, and physiotherapy (passive, then active). Clinical improvement occurred within 72 hours; by day 7, CK had decreased to 430 U/L (≈3.7× ULN), and normalized to 97 U/L at three months, with complete functional recovery.

4. Discussion

Corticosteroid-induced myopathy (CIM) belongs to the group of toxic, non-inflammatory myopathies. The present case is one such myopathy, with the particularity of an acute onset in a 10-year-old girl. Some series suggest a female predominance (2:1) [6] but this remains uncertain. The pediatric frequency of CIM is sparsely documented; CIM can occur at any age. An even younger age than ours was reported by Fang et al. in Shanghai in 2021: a 4-year-old developed CIM following corticosteroid therapy for diffuse alveolar hemorrhage. Given the limited size of existing series, a robust sex ratio cannot presently be established. The acute mode of onset, as in our patient, is reported by Haran et al. (2018), who note that manifestations may occur within 72 hours of high-dose corticosteroid therapy, even after a single dose [3, 7].

The main mechanism by which glucocorticoids induce CIM is well detailed by Gupta et al. and Surmachevska [2, 8]. CIM

results from an imbalance of muscle-fiber metabolism with accelerated catabolism and slowed anabolism. Glucocorticoids such as methylprednisolone, used in our case, are thought to trigger increased proteolysis by synergistic activation of the three major intracellular proteolytic systems, chiefly the ubiquitin-proteasome pathway. Anabolism is slowed by depriving muscle fibers of the amino acids required for protein synthesis and by blunting hormonal signals that regulate this process. Other mechanisms are also implicated according to Gupta, including inhibition of growth factors and mitochondrial bioenergetic dysfunction, creating a deleterious environment for skeletal muscle [2, 8-11].

Clinically, CIM classically presents with a myogenic pattern—that is, symmetric proximal limb weakness. Distal involvement and, more rarely, respiratory muscle involvement are possible [3, 12-14]. Our patient exhibited this classical presentation, with associated myalgia, likely related to the rapidity of onset. Confirmation of a myopathy relies on several tests. Electromyography (EMG) is useful, but in most cases tracings are normal or only show occasional reduction in motor unit action-potential amplitude [2]. Shanina et al. reinforce this point by explaining why EMG is normal or only mildly abnormal in CIM: type IIb fibers are preferentially affected, whereas needle EMG mainly samples activity from type I fibers [15]. EMG was unavailable in our setting and therefore could not characterize this patient's profile. Whereas in chronic CIM the serum creatine kinase (CK) is usually normal, it may be elevated in acute CIM [16, 17]. In acute forms, the literature reports elevations without a universally accepted threshold [2, 18]. In our case, the CK rose to 7.6× the upper limit of normal, while Sun et al. (2017) reported 25-fold [18, 19]. However, several authors also emphasize that a normal CK does not exclude CIM [17, 20]. In our patient, CK elevation was the most decisive laboratory argument supporting the diagnosis. Beyond CK, other muscle enzymes may rise concomitantly, including LDH, aldolase, and aminotransferases (AST/ALT), as reported in myopathies and rhabdomyolysis [21-23]. The temporal relationship between symptom onset and the acute phase of high-dose treatment was pivotal in favoring CIM while ruling out other causes of myopathy. These causes include inflammatory myopathies (such as juvenile dermatomyositis and polymyositis), congenital myopathies, and critical illness myopathy. Inflammatory myopathies are usually accompanied by marked elevation of CK, muscle tenderness, and cutaneous or systemic features [3, 12], none of which were present in our patient, except for the elevated CK. Congenital myopathies typically manifest earlier in life with hypotonia, delayed motor milestones, and a chronic, non-progressive course, which were inconsistent with the abrupt onset observed here. Critical illness myopathy occurs mainly in intensive care settings, often associated with sepsis, multiorgan failure, or neuromuscular blocking agents; our patient had no such conditions. The temporal relationship with high-dose corticosteroid exposure, the absence of inflammatory signs [3], and the rapid improvement after drug withdrawal were therefore decisive in confirming corticosteroid-induced myopathy. In our case, management was essentially conservative, based on withdrawal of the offending agent and symptomatic treatment. The decline in CK, in parallel with clinical improvement, provided additional support for steroid imputability. This strategy is consistent with the literature, which underscores that CIM management is primarily dose reduction and/or discontinuation, or substitution with a non-fluorinated glucocorticoid, combined with functional rehabilitation, with no specific pharmacotherapy proven to accelerate recovery [2, 11, 24]. The evolution of CIM is generally favorable once corticosteroids are stopped, but the recovery kinetics vary. Across series and reviews, clinical improvement can begin within days to a few weeks, while complete recovery most often unfolds over weeks to a few months [25]; consistent with our case. In pediatric reports, recovery tends to be even better [2]. Recent endocrinology literature stresses that, despite expected reversibility, recovery can be prolonged, sometimes over several months, and that recovery quality depends on comprehensive care—not only stopping steroids but also symptomatic management and structured rehabilitation [26].

5. Conclusion

Corticosteroid-induced myopathy is an often underrecognized iatrogenic condition whose presentation ranges from insidious proximal weakness to acute forms occurring shortly after corticosteroid exposure. An elevation in plasma creatine kinase interpreted in light of the exposure chronology is highly suggestive of an acute form, but a normal CK does not exclude the diagnosis. EMG is often of limited utility because it is frequently normal or nonirritative. In resource-limited settings, the priority is to reason clinically and to consider CIM systematically in any patient with proximal weakness while on corticosteroids, thereby avoiding unnecessary therapeutic escalation and improving prognosis, especially when diagnosis is timely and drug dechallenge is prompt.

Abbreviations

CIM	Corticosteroid-Induced Myopathy
CK	Creatine Kinase
AST	Aspartate Aminotransferase
ALT	Alanine Aminotransferase
LDH	Lactate Dehydrogenase
EMG	Electromyography
MRI	Magnetic Resonance Imaging
RBON	Retrobulbar Optic Neuritis
ULN	Upper Limit of Normal

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Selva-O'Callaghan A, Pinal-Fernandez I, Trallero-Araguás E, et al. Classification and management of adult inflammatory myopathies. *Lancet Neurol* 2018; 17: 816-828.

[2] Surmachevska N, Tiwari V. Corticosteroid Induced Myopathy. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing, http://www.ncbi.nlm.nih.gov/books/NBK557731/ (accessed 29 August 2025).

[3] Haran M, Schattner A, Kozak N, et al. Acute steroid myopathy: a highly overlooked entity. *QJM* 2018; 111: 307-311.

[4] Van Marle W, Woods KL. Acute hydrocortisone myopathy. *Br Med J* 1980; 281: 271-272.

[5] Garbaa S, Frikha F, Loukil H, et al. Corticosteroid myopathy: an overview through four cases [in French]. La Revue de Médecine Interne 2017; 38: A151.

[6] Pasnoor M, Barohn RJ, Dimachkie MM. Toxic Myopathies. *Neurol Clin* 2014; 32: 647-670.

[7] Kumar S. Steroid-induced myopathy following a single oral dose of prednisolone. *Neurology India* 2003; 51: 554-556.

[8] Gupta A, Gupta Y. Glucocorticoid-induced myopathy: Pathophysiology, diagnosis, and treatment. *Indian J Endocrinol Metab* 2013; 17: 913-916.

[9] Schakman O, Gilson H, Thissen JP. Mechanisms of glucocorticoid-induced myopathy. *J Endocrinol* 2008; 197: 1-10.

[10] Schakman O, Kalista S, Barbé C, et al. Glucocorticoid-induced skeletal muscle atrophy. *Int J Biochem Cell Biol* 2013; 45: 2163-2172.

[11] Pereira RMR, Freire de Carvalho J. Glucocorticoid-induced myopathy. *Joint Bone Spine* 2011; 78: 41-44.

[12] Bowyer SL, LaMothe MP, Hollister JR. Steroid myopathy: incidence and detection in a population with asthma. *J Allergy Clin Immunol* 1985; 76: 234-242.

[13] Hollister JR. The untoward effects of steroid treatment on the musculoskeletal system and what to do about them. *J Asthma* 1992; 29: 363-368.

[14] Khaleeli AA, Edwards RH, Gohil K, et al. Corticosteroid myopathy: a clinical and pathological study. *Clin Endocrinol (Oxf)* 1983; 18: 155-166.

[15] Shanina E, Smith RG. Electrodiagnostic Evaluation of Myopathy. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; 2025 - (cited 2025 Sep 3). Available from: http://www.ncbi.nlm.nih.gov/books/NBK564383/

[16] Chawla J. Stepwise Approach to Myopathy in Systemic Disease. *Front Neurol*; 2. Epub ahead of print 5 August 2011. https://doi.org/10.3389/fneur.2011.00049

[17] Wu M, Liu C, Sun D. Glucocorticoid-Induced Myopathy: Typology, Pathogenesis, Diagnosis, and Treatment. *Horm Metab Res* 2024; 56: 341-349.

[18] Sun L-Y, Chu X-L. Acute myopathy following intra-muscular injection of compound betamethasone. *Medicine (Baltimore)* 2017; 96: e7474.

[19] Mammen AL. Toxic Myopathies. *Continuum (Minneap Minn)* 2013; 19: 1634-1649.

[20] Manoj M, Sahoo RR, Hazarika K, et al. Drug-Induced Myopathy. *Indian Journal of Rheumatology* 2019; 14: S27-S36.

[21] Lundberg IE, Tjärnlund A, Bottai M, et al. EULAR/ACR Classification Criteria for Adult and Juvenile Idiopathic Inflammatory Myopathies and their Major Subgroups. *Ann Rheum Dis* 2017; 76: 1955-1964.

[22] Benveniste O, Goebel H-H, Stenzel W. Biomarkers in Inflammatory Myopathies—An Expanded Definition. *Front Neurol* 2019; 10: 554.

[23] Soontrapa P, Shahar S, Eauchai L, et al. Disease spectrum of myopathies with elevated aldolase and normal creatine kinase. *Eur J Neurol* 2023; 31: e16117.

[24] He A, Koszegi B, Uzun S, et al. Autoimmune blistering diseases treated with glucocorticoids: An international study of steroid‐induced myopathy. *Acad Dermatol Venereol* 2025; 39: 340-349.

[25] Hu Y, Lu C, Lin H. Concurrence of osteonecrosis and steroid myopathy secondary to oral steroid therapy in a patient with ABCB1 gene polymorphisms: A case report. *Front Endocrinol*; 13. Epub ahead of print 7 October 2022. https://doi.org/10.3389/fendo.2022.1016687

[26] Pofi R, Caratti G, Ray DW, et al. Treating the Side Effects of Exogenous Glucocorticoids; Can We Separate the Good From the Bad? *Endocr Rev* 2023; 44: 975-1011.