

Hypovitaminosis A - associated neurologic syndrome with concurrent babesiosis in a captive African lion cub (*Panthera leo*), Kano, Nigeria

Shaarani Habibu Musa¹ Aminu Bello Hanga^{1*} Auwal Usman Nagoma² Caleb Ayuba Kudi³
Paul Ayuba Abdu³ Musa Ibrahim Waziri³ Haruna Baba Galadima⁴ Yusuf Aminu Yusuf⁵
Muhammed Mikail^{6,7*}

Abstract

Neurological disorders in captive wild felids presents diagnostic and therapeutic challenges, particularly in juveniles animals maintained under suboptimal nutritional transitions. This case report describes the synergistic effects of dietary deficiencies and a concurrent hemoparasitic infection in a seven-month-old male African lion cub (*Panthera leo*) weighing 30 kg, housed at Audu Bako Zoological Garden in Kano, Nigeria. Following premature weaning onto an unsupplemented muscle-meat diet, the cub exhibited progressive neurological symptoms, including ataxia, hindlimb paresis, star-gazing, and opisthotonos. Hematological analysis revealed a mild normocytic, normochromic anemia (packed cell volume; 23%) and the presence of erythrocytes rouleaux formation, while Giemsa-stained blood smear confirmed low-grade *Babesia* spp parasitemia. Treatment involved oral vitamin A supplementation administered via cod liver oil, which resulted in a rapid, marked, and sustained resolution of neurological signs, thus confirming a primary diagnosis of hypovitaminosis A-associated neurological syndrome complicated by concurrent babesiosis. Concurrently, oral doxycycline was administered, and acaricides were applied throughout the enclosure to address the tick-borne infestation. This case highlights the importance pathophysiological interplay between precise husbandry nutrition and vector management in prematurely weaned captive lion cubs. Furthermore, it demonstrates the clinical efficacy of treatment-based diagnostic strategies in wildlife medicine, particularly within resource-limited zoological settings.

Keywords: African lion, babesiosis, captive wildlife, hypovitaminosis a, Kano Zoo, neurological disease, Nigeria

¹Gwale Veterinary Reference Laboratory, Department of Veterinary Services, Ministry for Livestock Development, Kano State, Nigeria

²Faculty of Veterinary Medicine, Bayero University Kano, P.M.B 3011, Kano, Nigeria

³Department of Medicine, Faculty of Veterinary Medicine, Ahmadu Bello University Zaria, Kaduna, Nigeria

⁴Department of Medicine, Faculty of Veterinary Medicine, University of Maiduguri, P.M.B 1069, Maiduguri, Borno, Nigeria

⁵Audu Bako Zoological Garden, Kano 700103, Nigeria

⁶Department of Veterinary Clinical Studies, Faculty of Veterinary Medicine Universiti Malaysia Kelantan, Kota Bharu 16100, Malaysia

⁷Wildlife Medicine and Conservation Research Group, Faculty of Veterinary Medicine, Universiti Malaysia Kelantan, Kota Bharu 16100, Malaysia

*Correspondence: mikail@umk.edu.my, aminubh23@gmail.com (M. Mikail, A.B. Hanga)

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Introduction

Nutritional disorders are recognized as significant contributors to morbidity in captive wild felids, particularly among the juveniles reared on imbalanced diets. (Hartley *et al.*, 2005; De Risio *et al.*, 2010; Wanker and Robert, 2012). In free ranging African lions (*Panthera leo*), essential micronutrients are acquired through the consumption of whole prey, including organ tissues such as liver that are rich in vitamin A (McDowell, 2000; Gharehaghajlou *et al.*, 2025). In captive environments, reliance on unsupplemented muscle meat diets can lead to deficiencies of fat-soluble vitamins and minerals, predisposing animals to risk of growth abnormalities, neurologic disease, and impaired immune function (Howard and Allen, 2007).

Vitamin A deficiency has been observed in captive lions and other felids, manifesting as neurological symptoms such as ataxia, paresis, seizures, star-gazing, opisthotonos, stillbirth and blindness (Wenker and Robert, 2012; Kaiser, 2014; Beers *et al.*, 2024; Gharehaghajlou *et al.*, 2025). These clinical signs are often associated with abnormal cranial bone remodeling and compression of neural structures in growing animals (Wenker and Robert, 2012; Schmidt *et al.*, 2021; Siedenburger *et al.*, 2024). Furthermore, the occurrence of these symptoms in captive felids is linked to deficiencies in copper and vitamin A, which result from diet consisting of meat that is not properly supplemented nor based on mixed, or whole carcass approach (Maratea *et al.*, 2006; Claudia *et al.*, 2024). Studies of captive big cats, including lions, indicates that hand-reared cubs fed inappropriate diets frequently develop nutrition-related disorders, this highlights the risks of premature separation and inadequate supplementation (Wilson and Phillips, 2021). Diets composed solely of muscle meat without proper supplementation often do not provide sufficient levels of fat-soluble vitamins and balanced minerals, such as vitamin A, calcium, and phosphorus, which are essential for normal growth and skeletal health (Merck Vet Manual, 2023). In captive lions, vitamin A deficiency has been linked to compromised immune function, reduced resistance to gastrointestinal and hemoparasites infections, and increased disease severity (Miller and Fowler, 2015). Consequently, lions with vitamin A deficiency may exhibit higher prevalence or intensity of parasitic infections which are frequently reported in both captive and free-ranging lions (Penzhorn, 2000; Birkenheuer *et al.*, 2008).

In addition to nutritional stressors, captive lions in sub-Saharan Africa are often exposed to tick-borne hemoparasitic infections, such as *Babesia* spp. and *Anaplasma* spp. (Hanga *et al.*, 2025). These infections can contribute to anemia, systemic inflammation, and can worsen existing nutritional or metabolic disorders resulting in clinical deterioration (McDermid *et al.*, 2017).

In zoological institutions with limited access to advanced diagnostic tools, disease management often depends on clinical history, physical examinations, basic laboratory findings and the response to empirical treatment (Miller *et al.*, 2022).

Reports of neurologic disease in captive African lion cubs from West Africa are rare. This case report describes a nutritional neurologic syndrome with concurrent babesiosis in a prematurely weaned captive African lion cub in Kano Zoo Nigeria and highlights practical diagnostic and therapeutic considerations within a resource limited zoological setting.

Case Report

Three African lion cubs were housed at Kano Zoological Garden, Kano, Nigeria. These cubs were acquired at approximately four months of age after being prematurely separated from the lioness. The index case involved a seven-month-old male cub that weighed approximately 30 kg and had a body condition score (BCS) of 4/9. The dietary management primarily involved un-supplemented muscle-meat diets. Initially, the regimen included 10 kg of beef per day, provided over a 16-h period followed by an 8-h fasting window. Later, this was changed to 20kg of fresh chicken carcass per day, still provided within the 16-h feeding window and accompanied by the same 8-h fasting period, all without any routine vitamin or mineral supplementation.

Shortly after acquisition, the cub exhibited cutaneous lesions consistent with *Rhipicephalus* tick infestation. The cub was with ivermectin at 0.2mg/kg, PO and the lesions resolved gradually. Several weeks later, the cub developed intermittent neurologic signs characterized by ataxia, generalized weakness, and reduced appetite. Injectable vitamin B complex was administered at 1.5 mL, IM, q 24h for three days and dietary calcium supplementation using bone meal was initiated, resulting in evident clinical improvement.

In the following weeks, the neurologic signs progressed to include acquired ataxia, hindlimb dragging and paresis as shown in Figures 1 and 2, stargazing, opisthotonos, and episode of transient recumbency following a physical assessment of the gait, posture, mentation and behavioural responses (Constable *et al.*, 2017; Mayhew and Mackey, 2022). Rectal temperature was 38.2°C and no overt systemic illness was detected on physical examination.

Differential diagnosis: Differential diagnosis considered included metabolic bone disease, congenital neurologic abnormalities, trauma and toxicities.

Result

The hematological analysis indicated a packed cell volume of 23%, consistent with Acute Normocytic Normochromic Hemolytic Anemia secondary to an acute babesiosis infection, as shown in Table 1 (Salakij *et al.*, 2010; Maas *et al.*, 2013). Giemsa-stained blood smear revealed the presence of rouleaux formation in erythrocytes along with low-grade *Babesia* spp. parasitemia as shown in Figures 3 and 4. Fecal examination showed no evidence of gastrointestinal parasites. However, the dietary history, characteristic neurological signs, and laboratory findings, a nutritional deficiency accompanied by concurrent tick-borne hemoparasitism was the tentative diagnosis.

Table 1 Showing the hematological parameters of the lion cub.

	Parameter (unit)	Results	References	Remarks
1	RBC($\times 10^{12}/L$)	12.6	7.2 - 25.6	Acute Normocytic Normochromic Haemolytic Anaemia
2	Haemoglobin (g/L)	6.2	5.1 - 8.3	
3	PCV (%)	23	26.8 - 44.1	
4	MCV (fL)	48	46.6 - 55.9	
5	MCH (pg)	18.2	14.8 - 18.5	
6	MCHC (g/L)	31.0	29.6 - 35.5	
7	MPV (fL)	8.6	8.4 - 10.6	

**Figure 1** Photomicrograph showing photo of the lion cub dragging its hind limb.**Figure 2** Photomicrograph showing the lion cub displaying paresis of the left hindlimb.

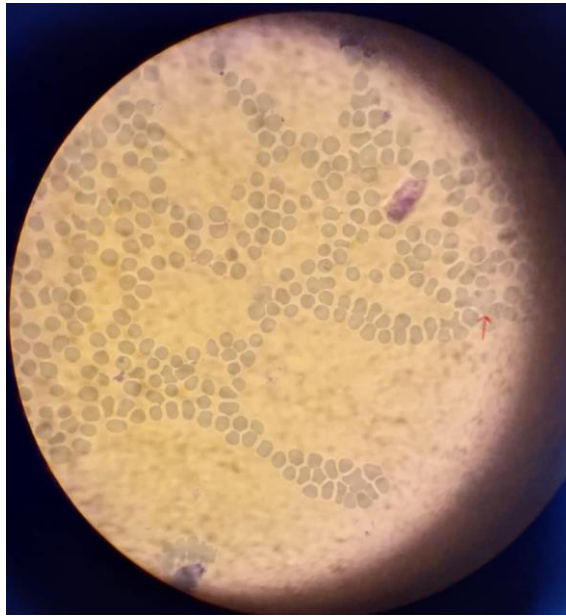


Figure 3 Photomicrograph showing the erythrocytes rouleaux formation following Geimsa-stained blood smear.

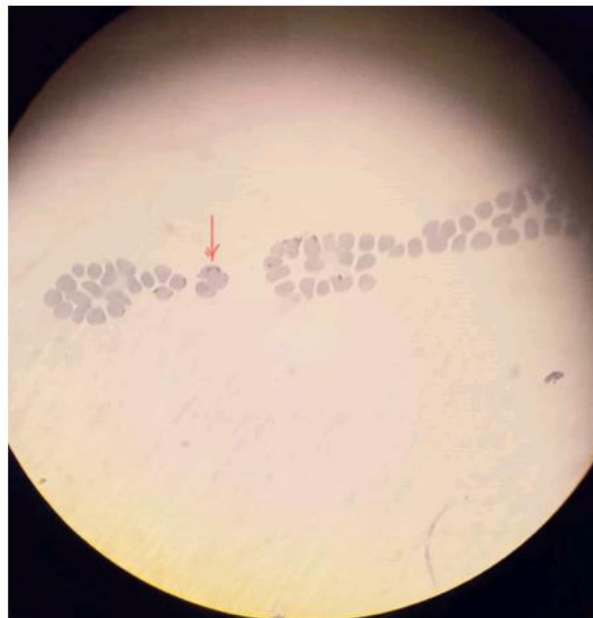


Figure 4 Photomicrograph showing the *Babesia* spp. following Giemsa blood smear.

Treatment and outcome: Oral vitamin A supplementation was initiated using cod liver oil mixed with feed, estimated at a dose of approximately 3,000 IU/kg to account for anticipated oral dosing loss (Puls, 1994; McCain *et al.*, 2008; Wenker and Robert, 2011; Siedenburger *et al.*, 2024). The supplementation followed a structured tapering regimen over a period of 51 days, as shown in Table 2. It is noteworthy that injectable vitamin A was not administered. Calcium and phosphorus supplementation were maintained through the use of whole bone meal and whole chicken carcass provisions, specifically utilizing femur bones from cows slaughtered at Kano Abattoir.

To address concurrent hemoparasitism and tick exposure, oral doxycycline was administered at 8 mg/kg PO SID for 21 days, and the enclosure was treated with an acaricide. A previous fatal case of

anaplasmosis in a cage mate indicated significant tick burden within the enclosure.

Clinical improvement was notable within two weeks of initiating vitamin A supplementation. There was a marked improvement in gait and posture, resolution of hindlimb dragging, and episodes of opisthotonos and star-gazing became infrequent and mild. Appetite and activity levels returned to near normal, as shown in Figure 5. No adverse effects were observed during the supplementation period. Sustained neurologic improvement during the tapering of the dosage supported a diagnosis of primary nutritional neurologic syndrome with concurrent mild babesiosis.

Table 2 Showing the vitamin A supplement treatment regimen.

Date Range	Supplement	Dose	Frequency	Note
11 Jul – 31 Jul 2025	Cod liver oil	5 capsules/day (~3,000 IU/kg)	Daily	Initial intensive phase
1 Aug – 15 Aug 2025	Cod liver oil	3 capsules/day (~1,800 IU/kg)	Every 2 days	Start tapering
16 Aug – 31 Aug 2025	Cod liver oil	2 capsules/day (~1,200 IU/kg)	Every 3 days	Tapering continues; monitor clinical signs
After 31 Aug 2025	Cod liver oil	Twice weekly		

**Figure 5** Showing the lion cub after treatment with vitamin A.

Discussion

The management history of the African lion cubs at the Kano Zoological Garden reflects the structural and nutritional challenges faced in captive wildlife environments, particularly when operating with limited resources. The pathogenesis of the neurologic syndrome observed in this case appears to be multifactorial, with chronic hypovitaminosis A identified as the primary underlying disorder. Factors such as premature separation, lack of organ tissues in the diet, and prolong feeding of unsupplemented muscle meat likely contributed to progressive depletion of hepatic vitamin A stores during a critical growth phase (Siedenburg *et al.*, 2024).

Vitamin A deficiency in growing felids disrupts with normal osteoclastic bone remodelling, leading to excessive cranial bone deposition and narrowing of foramina through which cranial nerves and spinal structures pass (Maratea *et al.*, 2006; Siedenburg *et al.*, 2024; Hazewinkel, 2023; Kalundia, 2025). This progressive neural compression subsequently manifests clinically as ataxia, pyrexia, star gazing, and opisthotonos (Chandra, *et al.*, 1999; McCain *et al.*, 2008; Wenker and Robert, 2012). Additionally, concurrent babesiosis may have compounded systemic stress through anaemia (Mikail *et al.*, 2026), and inflammatory responses, potentially worsening the overall neurologic and metabolic status of the animal.

Maternal care and milk are essential for early growth, immune system development, and the transfer of micronutrients in felids (Dierenfeld, 1997). In the wild, lioness typically wean their cubs much later, between 7 and 12 months (González González, 2025). Early maternal deprivation, exposes these vulnerable immature carnivores to opportunistic infections and metabolic deficiencies.

This baseline vulnerability was critically accelerated by the subsequent post-acquisition diet.

The prolonged reliance on an un-supplemented muscle-meat diet, initially comprising of beef and later transitioning to chicken, poses a severe, well-documented risk for nutritional imbalances in captive carnivores (Depauw *et al.*, 2013). Raw muscle meat lacks the necessary micronutrient diversity and balanced calcium to phosphorus ratios found in a natural, whole-prey diet. In wild settings, apex felids preferentially consumes metabolic organs, particularly the liver, which serve as the primary storage site for active retinoids (Gharehaghajlou *et al.*, 2025). Critically, young captive felids lack the necessary metabolic enzymes required to cleave carotenoid precursors (like beta-carotene) into active vitamin A, rendering them entirely dependent on dietary preformed vitamin A (Miller *et al.*, 2022).

Consequently, an exclusive all-meat diet rapidly exhausts systemic vitamin reserves during periods of rapid growth. The multi-systemic clinical progression of this seven-month-old male cub provides an informative window into sequential pathogenesis of mixed nutritional deficiencies.

The cutaneous lesions observed shortly after acquisition indicate a compromised epithelial barrier. Vitamin A is essential for maintaining epithelial tissue integrity and modulating cell-mediated immunity; its deficiency leads to squamous metaplasia of glandular structures and reduces mucosal immunity, thereby predisposing the skin to opportunistic ectoparasitic infestations, such as ticks and scabies mites (Ulrey D, 1991; Miller and Buss, 2003; Miller *et al.*, 2022). The gradual resolution of these lesions following the administration of oral ivermectin confirms a parasitic component, and indicate a severely compromised immune status. A packed cell volume of 23%, along with findings of mild normocytic, normochromic anemia, is consistent with chronic disease processes commonly reported in carnivores, including

nutritional insufficiencies and hemoparasitism (Thrall *et al.*, 2012). Normocytic, normochromic anemia is often associated with anemia of chronic disease or mild hemolysis and has been documented in felids and canine infected with tick-borne hemoparasites such as *Babesia* spp. (Irwin, 2009; Mikail *et al.*, 2026). The observation of mild leukocytosis further supports the possibility of a low-grade inflammatory or infectious process, potentially linked to hemoparasitism. The identification of *Babesia* spp. on Giemsa-stained blood smears, albeit mild, is clinically significant, as babesiosis in felids has been associated with anemia, weakness, and reduced appetite, even in the absence of pronounced fever (Irwin, 2009; Solano-Gallego *et al.*, 2016).

The diagnosis of hypovitaminosis A associated neurologic syndrome in this cub was supported by the combination of dietary history, characteristic neurologic manifestations, exclusion of major differential diagnoses, and the marked therapeutic response to vitamin A supplementation. Although advanced imaging modalities such as computed tomography or magnetic resonance imaging were unavailable, the manifestations of star gazing, opisthotonos, hindlimb paresis, and ataxia closely resemble previous documented cases of hypovitaminosis A in captive lions and other felids (Heartlet, *et al.*, 2005; Wanker and Robert, 2012; Kaiseret *et al.*, 2014).

Oral vitamin A supplementation, administered via cod liver oil at an estimated dosage of approximately 3,000 IU/kg was chosen as a safe and practical intervention, particularly in a zoological context (Miller *et al.*, 2003; Hartley *et al.*, 2005). Cod liver oil is a traditional and widely recognized source of preformed vitamin A (retinol) in carnivore nutrition. Oral administration facilitates gradual absorption and physiological regulation, thereby reducing the risk of acute toxicity when appropriately managed (Hand *et al.*, 2011). A gradual reduction of dosage following initial repletion is effective in restoring tissue stores while minimizing the risk of skeletal abnormalities and soft tissue mineralization associated with excessive vitamin A intake (Ullrey *et al.*, 1991; Dierenfeld and Graffam, 1996). The rationale for continuing calcium and phosphorus supplementation alongside vitamin A therapy is well-justified, as vitamin A plays an important role for bone remodeling and closely interacts with calcium-phosphorus homeostasis (Shastak *et al.*, 2024). Correcting mineral imbalance is therefore essential to support normal skeletal development and to reduce the risk of secondary metabolic bone disease in rapidly growing lion cubs (Edward *et al.*, 2010).

The rapid and sustained clinical improvement observed within two weeks of initiating vitamin A supplementation strongly suggests that the neurological disorder in this lion cub was primarily of nutritional origin. Notable improvement in gait and posture, the complete resolution of hindlimb dragging, and a significant reduction in the frequency and severity of opisthotonos and star-gazing agreed with previously reported responses in captive lion cubs and other felids affected by hypovitaminosis A (Hartley *et al.*, 2005; Schmitt *et al.*, 2009). Concurrent mild

babesiosis, likely acted as a secondary stressor. Tick-borne hemoparasites infections are endemic in sub-Saharan Africa (Hanga *et al.*, 2025). While babesiosis can induce anemia, lethargy and weakness, it is not typically associated with the severe and progressive neurological signs. (Penzhorn, 2006; Irwin, 2009). The rapid and sustained neurologic improvement following Vitamin A supplementation, despite concurrent parasitemia, support a primary nutritional etiology.

In conclusion, the clinical sign and therapeutic response observed in this case support a diagnosis of primary hypovitaminosis A-associated neurologic syndrome, concurrent with mild babesiosis, in a prematurely weaned captive African lion cub. These findings highlight the potential for reversibility in early-stage nutritional neurologic disorders with timely dietary intervention and emphasize the importance of balanced nutrition, routine supplementation, and integrated parasite control programs in captive wildlife management. Preventive nutritional strategies remain important for reducing morbidity and mortality in hand-reared lion cubs particularly in resource limited zoological institutions.

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