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REVIEW

A Pragmatic Framework for Risk-Based Vitamin D Supplementation in Epilepsy Care in Sub-Saharan Africa

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Abstract: Epilepsy remains a major public health challenge in sub-Saharan Africa, where treatment options are often limited to older, low-cost enzyme-inducing antiseizure medications such as phenobarbital, carbamazepine, and phenytoin. Although these medications are essential for seizure control in resource-constrained settings, prolonged use may accelerate vitamin D metabolism and contribute to hypovitaminosis D, impaired bone mineralization, reduced bone mineral density, osteomalacia, and increased fracture risk. Despite these potential complications, vitamin D assessment is rarely integrated into epilepsy care in the region, and evidence regarding the burden of deficiency among people with epilepsy remains limited. This paper proposes a pragmatic framework for risk-based vitamin D supplementation in epilepsy care adapted to sub-Saharan African contexts. Given the limited availability and affordability of serum 25-hydroxyvitamin D testing, routine laboratory-guided screening is often not feasible. We therefore advocate for a clinically oriented risk-stratification approach prioritizing preventive supplementation among patients at highest risk of deficiency and skeletal complications. High-risk groups may include individuals receiving long-term enzyme-inducing antiseizure medications, children and adolescents, older adults, pregnant women, and patients with malnutrition, reduced sunlight exposure, or physical disability. The framework emphasizes integration of low-cost empirical vitamin D supplementation into routine epilepsy services, particularly at the primary-care level. It also highlights the need for context-adapted clinical algorithms, healthcare provider awareness, patient education, and implementation research to evaluate feasibility, safety, and cost-effectiveness in African settings. Integrating bone health into epilepsy management may represent a feasible and scalable strategy to reduce preventable morbidity and improve long-term outcomes among people living with epilepsy in sub-Saharan Africa.

Keywords: vitamin D supplementation, epilepsy, sub-Saharan Africa

1 Introduction

Epilepsy remains one of the most common neurological disorders worldwide, affecting nearly 50 million people globally [1]. Approximately 80% of individuals living with epilepsy reside in low- and middle-income countries, with a substantial proportion concentrated in sub-Saharan Africa [1, 2]. Despite the availability of effective antiseizure medications, epilepsy in this region is associated with a disproportionate burden of morbidity and mortality due to treatment gaps, limited diagnostic capacity, and constrained health systems [1, 3]. Many patients remain untreated or receive suboptimal therapy, contributing to preventable complications including recurrent seizures, injuries, and premature death [2, 4].

Beyond the well-recognised structural and socioeconomic barriers to epilepsy care, emerging evidence suggests that nutritional factors may also influence disease outcomes. Among these, vitamin D deficiency is increasingly recognised as a potentially important yet underappreciated issue in epilepsy management [5]. Vitamin D plays critical roles in neurophysiology, immune regulation, and bone health [6]. However, patients with epilepsy—particularly those receiving enzyme-inducing antiseizure medications—are at increased risk of vitamin D depletion [7, 8].

In sub-Saharan Africa, where older antiseizure medications remain widely used and nutritional

deficiencies are common, this interaction may represent a silent contributor to poor clinical outcomes [2, 4]. This commentary highlights the overlooked intersection between epilepsy treatment and vitamin D deficiency in sub-Saharan Africa and proposes practical strategies to integrate nutritional considerations into epilepsy care.

2 The relationship between vitamin D and epilepsy

Vitamin D is traditionally recognised for its role in calcium homeostasis and bone metabolism, but growing evidence also suggests broader involvement in neurological function [9, 10]. Vitamin D receptors and activating enzymes are widely expressed in several brain regions implicated in seizure generation and neuronal regulation, including the hippocampus and cerebral cortex [9]. Through these pathways, vitamin D may influence neuronal excitability via modulation of calcium signalling, neuroinflammatory responses, oxidative stress, and neurotrophic pathways [9, 10].

Experimental studies have suggested possible biological links between vitamin D status and neuronal excitability. Vitamin D deficiency has been associated with increased neuronal hyperexcitability in preclinical models, potentially influencing seizure susceptibility [5]. Similarly, some animal studies have reported reduced seizure susceptibility following vitamin D supplementation [11]. However, translation of these findings into clinical practice remains uncertain.

Clinical evidence evaluating the relationship between vitamin D and seizure control in humans remains limited and heterogeneous. Observational studies have consistently reported a high prevalence of hypovitaminosis D among people living with epilepsy [5]. Nevertheless, although some small interventional studies have suggested possible reductions in seizure frequency after correction of vitamin D deficiency, current evidence remains insufficient to conclude that vitamin D supplementation exerts a direct antiseizure effect [12]. Larger, well-designed clinical trials are still needed to clarify whether vitamin D status has a clinically meaningful influence on seizure outcomes.

Importantly, the principal established benefit of identifying and correcting vitamin D deficiency in people with epilepsy remains the prevention of bone-related complications. Chronic vitamin D deficiency contributes to impaired bone mineralisation and may increase susceptibility to osteopenia, osteoporosis, osteomalacia, and fractures—complications of particular concern in individuals at risk of falls and trauma during seizures [5, 13].

3 Antiseizure medications as an aggravating factor

Antiseizure medications themselves can contribute to vitamin D deficiency, particularly enzyme-inducing antiseizure drugs (EIASDs). Commonly used agents such as phenytoin, carbamazepine, and phenobarbital induce hepatic cytochrome P450 enzymes, accelerating the metabolism of vitamin D into inactive compounds [5, 14]. This process reduces circulating levels of 25-hydroxyvitamin D and impairs calcium absorption.

Long-term use of enzyme-inducing antiseizure medications has been associated with reduced bone mineral density and increased risk of osteomalacia and fractures [14]. The effect appears to be cumulative, particularly among patients receiving prolonged treatment or polytherapy [14]. Children and adolescents may be particularly vulnerable, as vitamin D deficiency during critical growth periods can impair skeletal development and contribute to growth delays [12].

Evidence from observational studies consistently demonstrates lower vitamin D levels in patients treated with enzyme-inducing antiseizure medications compared with those receiving non-inducing agents [5]. In some cohorts, more than half of patients on long-term therapy exhibit vitamin D insufficiency or deficiency [5]. These findings suggest that the pharmacological management of epilepsy can inadvertently contribute to a secondary metabolic vulnerability.

While newer non-enzyme-inducing antiseizure medications—such as levetiracetam, lamotrigine, and valproate—may have less pronounced effects on vitamin D metabolism [14], access to these drugs remains limited in many low-resource settings. As a result, older medications with known metabolic consequences continue to constitute the backbone of epilepsy treatment across much of sub-Saharan Africa.

4 Specific challenges in sub-Saharan Africa

The interaction between antiseizure therapy and vitamin D deficiency may be particularly relevant in Sub-Saharan Africa for several structural, clinical, and health-system-related reasons.

First, despite abundant sunlight across much of the region, vitamin D deficiency remains surprisingly prevalent. Urbanisation, indoor lifestyles, darker skin pigmentation, nutritional limitations, and chronic illnesses may all contribute to reduced vitamin D levels [15]. Recent epidemiological analyses suggest that vitamin D deficiency affects a substantial proportion of African populations. A systematic review including 21,474 participants from 23 African countries reported pooled prevalence estimates of 34.2% for serum 25(OH)D concentrations below 50 nmol/L and nearly 60% for levels below 75 nmol/L, underscoring the widespread nature of suboptimal vitamin D status across the continent despite abundant sunlight [15].

Importantly, very few studies have specifically evaluated vitamin D status among people living with epilepsy in sub-Saharan Africa. Despite the widespread use of enzyme-inducing antiseizure medications in the region, data on vitamin D status among people with epilepsy remain extremely scarce. Existing epilepsy research in Africa has largely focused on treatment access, stigma, seizure control, and the epilepsy treatment gap, whereas metabolic and nutritional complications remain poorly documented [16]. This absence of evidence should itself be considered a major research priority. Moreover, although antiseizure medication-associated bone disease is well documented in high-income settings, African evidence regarding bone mineral density, osteomalacia, fractures, and vitamin D deficiency among people with epilepsy remains remarkably limited.

Second, access to laboratory testing for vitamin D is extremely limited in many African health systems. Measurement of serum 25-hydroxyvitamin D—the gold standard marker of vitamin D status—is rarely available outside major referral laboratories and is often prohibitively expensive [15]. Consequently, routine monitoring of vitamin D status in patients with epilepsy is seldom performed in routine clinical practice.

Third, the therapeutic landscape of epilepsy management in sub-Saharan Africa continues to rely heavily on older enzyme-inducing antiseizure medications. Phenobarbital remains the most widely used antiseizure medication in many sub-Saharan African countries because of its low cost, broad availability, and inclusion in national essential medicines lists [16–18]. Carbamazepine and phenytoin also remain widely prescribed in many low-resource settings [16, 17]. While these medications are effective for seizure control, their hepatic enzyme-inducing properties may accelerate vitamin D catabolism and contribute to skeletal complications, including osteomalacia, reduced bone mineral density, and fractures [14]. This evidence gap is particularly concerning given the widespread long-term use of enzyme-inducing antiseizure medications such as phenobarbital in sub-Saharan Africa [16, 18].

Fourth, current epilepsy management guidelines in many low-resource settings rarely incorporate systematic nutritional assessment, bone health evaluation, or vitamin supplementation, and internationally accepted recommendations for routine screening of vitamin D deficiency in people with epilepsy remain limited [8]. As a result, vitamin D deficiency frequently remains undetected and untreated among individuals living with epilepsy.

Figure 1 summarises the conceptual pathway linking the high burden of epilepsy in sub-Saharan Africa, the widespread reliance on enzyme-inducing antiseizure medications, and the biological mechanisms leading to vitamin D depletion and downstream clinical consequences. The framework illustrates how structural health-system constraints, therapeutic practices, and metabolic effects converge to increase vulnerability to vitamin D deficiency among people living with epilepsy in the region, ultimately contributing to adverse neurological and skeletal outcomes.

The clinical consequences of this overlooked burden may be substantial. In addition to increased fracture risk and impaired bone health, vitamin D deficiency may contribute to fatigue, proximal muscle weakness, and impaired skeletal development in children. Whether correction of deficiency improves seizure control remains uncertain, although vitamin D deficiency has been suggested as a potential contributor to poorer neurological outcomes in some patients with epilepsy [5]. These complications add to the already considerable burden experienced by people living with epilepsy in sub-Saharan Africa, where stigma, delayed diagnosis, treatment interruptions, and limited access to specialised neurological care remain common [16]. Taken together, these factors suggest that vitamin D deficiency represents a largely neglected but potentially modifiable contributor to adverse epilepsy outcomes in the region.

5 A pragmatic implementation framework for vitamin D management in epilepsy care in sub-Saharan Africa

Addressing vitamin D deficiency in people with epilepsy in sub-Saharan Africa requires an approach that is clinically relevant, operationally simple, and aligned with the realities of resource-

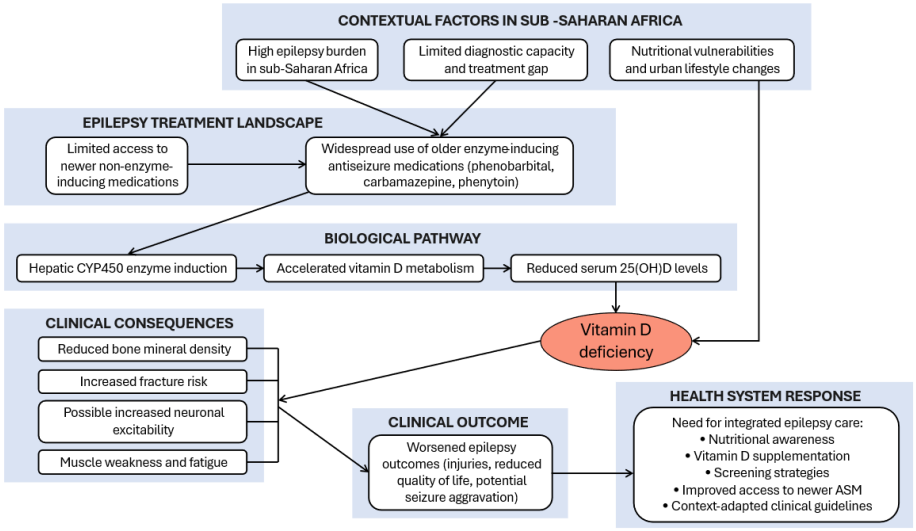

Figure 1 Conceptual framework linking antiseizure medication use, vitamin D deficiency, and epilepsy outcomes in sub-Saharan Africa.

constrained health systems. Because routine measurement of serum 25-hydroxyvitamin D is unavailable in many district and provincial hospitals, a risk-based strategy centered on empirical supplementation may be more feasible than laboratory-guided management [8,12]. This approach is supported by evidence showing that patients receiving enzyme-inducing antiseizure medications have higher vitamin D requirements and are more frequently supplemented to maintain adequate 25-hydroxyvitamin D levels, reflecting altered vitamin D metabolism and increased risk of deficiency. The principal objective of this strategy is to prevent bone-related complications associated with long-term use of enzyme-inducing antiseizure medications, including osteopenia, osteomalacia, osteoporosis, and fractures [7,8,19]. Any effect on seizure control remains uncertain (Table 1).

Table 1 Pragmatic approach to vitamin D management in people with epilepsy in sub-Saharan Africa

Clinical Situation	Recommended Action
Child or adolescent receiving long-term enzyme-inducing antiseizure medication	Prioritise preventive vitamin D supplementation and assess growth and bone health during follow-up
Pregnant or breastfeeding woman with epilepsy	Consider preventive vitamin D supplementation and reinforce nutritional counseling
Older adult receiving antiseizure medication	Consider preventive supplementation and assess fracture risk and mobility
Phenobarbital, carbamazepine, or phenytoin use for more than 6–12 months	Initiate risk-based vitamin D supplementation, particularly when laboratory testing is unavailable
Polytherapy with two or more antiseizure medications	Consider preventive supplementation because of potentially increased metabolic risk
History of fragility fracture	Initiate supplementation and assess for underlying bone disease
Persistent bone pain, proximal muscle weakness, or difficulty walking	Initiate supplementation and evaluate for osteomalacia or other skeletal complications
Malnutrition or low dietary calcium intake	Provide vitamin D supplementation and dietary counselling; consider calcium supplementation when appropriate
Limited mobility or minimal sun exposure	Consider preventive vitamin D supplementation
Serum 25-hydroxyvitamin D testing unavailable	Use clinical risk stratification and provide empirical supplementation for high-risk patients
Serum 25-hydroxyvitamin D testing available	Reserve testing for patients with severe symptoms, recurrent fractures, or poor response to supplementation
Tertiary referral hospital	Integrate bone health assessment into routine epilepsy follow-up
Primary healthcare facility	Apply a simplified algorithm based on antiseizure medication type, duration of treatment, and clinical risk factors
National epilepsy guideline development	Incorporate recommendations on bone health monitoring and risk-based vitamin D supplementation
Research setting	Prioritise studies on vitamin D deficiency prevalence, skeletal outcomes, and implementation strategies

5.1 Priority groups for intervention

In settings where universal screening is not feasible, preventive efforts should focus on patients at the highest risk of vitamin D deficiency and skeletal complications. Priority groups include children and adolescents during periods of rapid bone growth, pregnant and breastfeeding women, older adults, individuals receiving enzyme-inducing antiseizure medications (particularly phenobarbital, carbamazepine, and phenytoin) for more than 6–12 months, patients treated with multiple antiseizure medications, and those with a history of fractures, chronic bone pain, proximal muscle weakness, malnutrition, or limited mobility [8, 20]. Identifying these groups requires only routine clinical assessment and medication review and can be incorporated into standard follow-up visits.

5.2 Management when vitamin D testing is unavailable

Serum 25-hydroxyvitamin D measurement remains the reference standard for assessing vitamin D status. Still, access is often restricted to a small number of urban laboratories and may be unaffordable for many patients. In most healthcare facilities in sub-Saharan Africa, management decisions must therefore rely on clinical risk stratification rather than biochemical confirmation. For high-risk individuals receiving long-term enzyme-inducing therapy, empirical vitamin D supplementation is a pragmatic and reasonable strategy. Existing reviews suggest that vitamin D supplementation may improve biochemical markers of bone health in patients receiving antiseizure medications, particularly enzyme-inducing therapies, although evidence for fracture reduction remains limited [8, 12, 13, 20]. Where testing is available, it may be reserved for patients with recurrent fractures, severe bone pain, suspected osteomalacia, or persistent symptoms despite supplementation.

5.3 Practical supplementation strategies

Vitamin D3 (cholecalciferol) is inexpensive, widely available, and generally safe at standard preventive doses. A practical regimen in low-resource settings is daily supplementation with 800–2,000 IU, adjusted according to age, physiological status, and local clinical protocols. Similar preventive dosing ranges have been proposed in bone-health reviews and epilepsy-related supplementation studies [12, 13]. Calcium supplementation may be considered when dietary calcium intake is inadequate, particularly in children, pregnant women, and older adults. In settings where daily adherence is challenging, intermittent dosing schedules may be considered if supported by national guidelines and local availability. As with any intervention, clinicians should remain attentive to contraindications and monitor for symptoms suggestive of excessive supplementation, although toxicity is uncommon at preventive doses.

5.4 Integration into routine epilepsy care

Vitamin D prevention strategies can be incorporated into existing epilepsy services without substantial additional infrastructure [7, 8]. During routine follow-up visits, healthcare providers can review the type and duration of antiseizure therapy, identify clinical risk factors, provide counselling on dietary sources of calcium and vitamin D, and prescribe supplementation when indicated. Counselling should also address safe sun exposure and adherence to treatment. These activities can be integrated into neurology clinics, pediatric services, maternal and child health programs, and primary healthcare settings where epilepsy is managed. Standardised checklists and simple treatment algorithms may facilitate consistent implementation by physicians, nurses, and other frontline providers.

5.5 Positioning within resource-limited health priorities

In settings where healthcare resources are constrained, interventions must be evaluated based on feasibility, affordability, and potential impact. Vitamin D supplementation compares favourably on these criteria. The cost of preventive supplementation is modest relative to the clinical and economic consequences of fractures, reduced mobility, and long-term disability. Long-term exposure to older enzyme-inducing antiseizure medications has consistently been associated with impaired bone health and increased fracture risk in people with epilepsy [21]. In the interim, risk-based vitamin D supplementation offers a practical and scalable strategy to mitigate one of the most predictable adverse effects of older antiseizure therapies.

5.6 Research and policy priorities

The near absence of epilepsy-specific data on vitamin D status in sub-Saharan Africa represents a major evidence gap. Priority research areas include determining the prevalence and severity of

deficiency among people with epilepsy, identifying local risk factors, evaluating adherence to supplementation, and assessing effects on bone health outcomes such as fractures and impaired growth [8]. National and regional epilepsy guidelines should incorporate recommendations on bone health assessment and vitamin D supplementation, particularly for patients receiving long-term enzyme-inducing antiseizure medications. Several expert reviews and clinical guidance documents already advocate periodic evaluation of bone health and vitamin D status in people receiving long-term antiseizure therapy [20]. Embedding these measures into clinical protocols would support more comprehensive and preventive epilepsy care across the region.

6 Conclusion

The intersection between epilepsy, long-term antiseizure medication use, and vitamin D deficiency represents an underrecognised public health challenge in Sub-Saharan Africa. Although the relationship between vitamin D and seizure control remains uncertain, the impact of chronic vitamin D deficiency on bone health is well established. People living with epilepsy may be particularly vulnerable to osteopenia, osteomalacia, osteoporosis, and fractures, especially in settings where enzyme-inducing antiseizure medications such as phenobarbital remain widely used.

In sub-Saharan Africa, this challenge is compounded by limited access to laboratory testing, constrained healthcare resources, and the absence of routine nutritional and bone-health monitoring in epilepsy care. Under these conditions, a pragmatic framework for risk-based vitamin D supplementation may represent a feasible, low-cost, and scalable strategy to reduce preventable skeletal complications among high-risk patients receiving long-term antiseizure therapy.

Generating region-specific evidence on vitamin D status, bone health outcomes, and implementation strategies in African epilepsy populations should be considered an important research and public health priority. Integrating bone-health assessment, nutritional awareness, and pragmatic vitamin D supplementation approaches into routine epilepsy care may contribute to more comprehensive and equitable long-term management for people living with epilepsy across sub-Saharan Africa.

Author Contributions

Hilaire Abwa Lisimo and Olivier Mukuku conceived the main idea of the article and are responsible for the overall content as guarantor. Hilaire Abwa Lisimo, Olivier Mukuku, François Maheshe Polepole, Archippe Muhandule Birindwa, Célestin Kaputu-Kalala-Malu, and Stanislas Okitotsho Wembonyama contributed to content development, writing, and iterative revisions of the manuscript. All authors reviewed and approved the final version of the paper.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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