International Archives of Medical and Health Research

Original Article

Print ISSN: 2705-1420; Online ISSN: 2705-1439
DOI: https://doi.org/10.33515/iamhr/2025.020/09

Paroxysmal Dyskinesias in Children: Diagnostic Challenges and Misdiagnosis as Epilepsy in Resource-Limited Sub-Saharan Africa - A Narrative Review

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ABSTRACT

Background: Paroxysmal dyskinesias (PD) are rare, episodic movement disorders in children characterized by sudden, transient involuntary movements with preserved consciousness. In resource-limited Sub-Saharan Africa, they are frequently underrecognized and misdiagnosed as epilepsy due to overlapping clinical features and limited diagnostic capacity, resulting in inappropriate management and avoidable morbidity. **Aim:** To provide a comprehensive narrative overview of pediatric PD, with emphasis on clinical features, diagnostic challenges, and misdiagnosis as epilepsy in resource-constrained Sub-Saharan African settings. **Methods:** A narrative review of published literature was conducted using electronic databases, including MEDLINE, ScienceDirect, Google Scholar, and World Health Organization resources. Eligible studies included descriptive, observational, and review articles addressing pediatric PD, diagnostic challenges, and health-system factors. Findings were synthesized qualitatively. **Data Synthesis:** Paroxysmal dyskinesias typically present with stereotyped, trigger-induced episodes of dystonia or choreoathetosis, preserved awareness, and normal interictal neurological function. Misdiagnosis as epilepsy is common, driven by clinical overlap, high epilepsy prevalence, limited access to EEG, neuroimaging, and genetic testing, and low clinician awareness. Diagnostic delays are frequent, leading to unnecessary long-term antiepileptic therapy, financial burden, and psychosocial stigma. However, accurate diagnosis is achievable through careful clinical assessment, recognition of triggers, and use of pragmatic tools such as caregiver-recorded videos. Management is generally effective, combining low-dose pharmacotherapy, trigger avoidance, and supportive care. **Conclusion:** Strengthening clinical awareness, improving diagnostic capacity, and adopting low-cost, context-appropriate strategies are essential to reduce misdiagnosis and improve outcomes for children with PD in resource-limited settings.

Keywords: Paroxysmal dyskinesias, Epilepsy misdiagnosis, Pediatric movement disorders, Diagnostic challenges, Sub-Saharan Africa

INTRODUCTION

Paroxysmal dyskinesias (PD) are a heterogeneous group of rare, episodic movement disorders characterized by sudden, transient attacks of involuntary movements, including dystonia, chorea, athetosis, or mixed phenomenology, occurring in the context of preserved consciousness and typically with a normal neurological examination between episodes.1 Affected children, particularly in primary (genetic) forms, generally exhibit normal cognitive development and neurological function during interictal periods, which may contribute to under-recognition of the disorder in routine clinical practice.[1,2] The condition most commonly begins in childhood or adolescence, with distinct temporal patterns across subtypes: paroxysmal kinesigenic dyskinesia (PKD) often presents between 5 and 15 years of age, paroxysmal non-kinesigenic dyskinesia (PNKD) frequently begins in early childhood, and paroxysmal exercise-induced dyskinesia (PED) typically manifests during late childhood or adolescence, particularly in the school-age years.[1,2]

The clinical recognition of PD has evolved considerably over time. Early descriptions in the late nineteenth and early twentieth centuries documented patients with recurrent involuntary movements without loss of consciousness, although such cases were frequently misclassified as "reflex epilepsy" or functional ("hysterical") disorders due to limited understanding of their underlying mechanisms.[3,4] Foundational contributions by early neurologists, including observations by Gilles de la Tourette, helped delineate episodic motor phenomena distinct from other movement disorders. A major milestone was achieved in

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Published: 24-03-2026

1940 with the detailed description of familial paroxysmal choreoathetosis, which emphasized preserved consciousness and normal neurological status between attacks, thereby distinguishing PD from epileptic seizures.[3,4]

Subsequent advances in clinical characterization led to the introduction of the term paroxysmal kinesigenic choreoathetosis by Kertesz in 1967, highlighting attacks triggered by sudden voluntary movement and initiating a classification framework based on precipitating factors.[5] This work culminated in the widely accepted classification proposed by Demirkiran and Jankovic in 1995, which categorized PD into PKD, PNKD, and PED, forming the basis of contemporary diagnostic approaches.[1,3–5]

The advent of electroencephalography (EEG) and neuroimaging further refined the distinction between PD and epilepsy, demonstrating the absence of epileptiform activity during attacks and typically normal structural imaging in primary forms. These findings supported the conceptualization of PD as functional or network disorders rather than epileptic conditions. Advances in molecular genetics between 2004 and 2011 provided critical insights into the biological basis of PD, with the identification of pathogenic variants in genes such as *PRRT2*, *PNKD*, and *SLC2A1*. These discoveries established PD as part of a broader group of genetically mediated channelopathies and synaptopathies, with notable clinical and mechanistic overlap with epilepsy and migraine within a spectrum of paroxysmal neurodevelopmental disorders.[6]

Despite these advances, the epidemiology of PD in children remains poorly defined, particularly in low- and middle-income regions. Available data from high-income countries suggest that PKD is the most common subtype in pediatric populations, with estimated prevalence ranging from approximately 1 in 150,000 to 1 in 1,000,000, while PNKD and PED are considerably rarer.[1,7,8] Both sporadic and familial forms are recognized, with autosomal dominant inheritance and variable penetrance commonly observed in genetic cases. PKD demonstrates a male predominance, whereas PNKD shows a more equal sex distribution, and PED typically affects school-aged children and adolescents.[1,7,8] Although PD has been reported across diverse ethnic groups, the predominance of published cases from Asia likely reflects greater diagnostic awareness and access to genetic testing rather than true geographic variation in disease burden. Reports from Sub-Saharan Africa, including Nigeria, remain exceedingly limited, suggesting substantial under-recognition and underreporting.[9]

The pathophysiology of PD is complex and incompletely understood, but current evidence implicates dysfunction in basal ganglia and thalamocortical circuits, abnormalities in synaptic transmission, and ion channel dysregulation.[7] Primary PD is largely genetic, with *PRRT2* mutations most commonly associated with childhood-onset PKD, while *SLC2A1* mutations are linked to PED and glucose transporter type 1 (GLUT1) deficiency syndrome.[6,7] Secondary forms arise from identifiable neurological or systemic conditions, including structural brain lesions, metabolic derangements, infections, and inflammatory disorders. PD frequently coexists with other paroxysmal neurological conditions, particularly epilepsy and migraine, reflecting shared genetic and pathophysiological mechanisms.[6,13,19] For instance, benign infantile seizures are commonly observed in individuals with *PRRT2* mutations, underscoring the close relationship between PD and epileptic disorders.[6]

Genetic heterogeneity further complicates the clinical landscape. While mutations in the *PNKD* (MR-1) gene have been identified in patients with classic PNKD phenotypes, not all cases demonstrate a clear genetic basis, highlighting limitations of current diagnostic approaches.[10–12] Environmental triggers such as caffeine and alcohol, linked to MR-1–related neurotoxicity, may provide important clinical clues in such cases.[10,11] Similarly, linkage studies in PED have yielded inconsistent findings, although associations with chromosomal loci and syndromic presentations involving epilepsy, developmental delay, and migraine suggest the existence of additional, yet unidentified genetic determinants. [14,15]

Clinically, PD is classified based on etiology, precipitating factors, and duration of attacks.[1,16] Primary forms are typically genetic and characterized by episodic symptoms with normal interictal neurological function, whereas secondary forms are associated with persistent neurological abnormalities reflective of underlying pathology.[1,20] Classification by triggers further distinguishes PKD (precipitated by sudden movement), PNKD (triggered by stress, fatigue, or dietary factors), and PED (induced by prolonged physical exertion), each with distinct clinical profiles and therapeutic implications.[7,10,14,21] Notably, PKD often responds

dramatically to low-dose anticonvulsants such as carbamazepine, a feature that paradoxically contributes to diagnostic confusion with epilepsy.[7,10]

In resource-limited settings, particularly in Sub-Saharan Africa, the diagnostic distinction between PD and epilepsy presents a significant clinical challenge. The high burden of epilepsy, coupled with limited access to specialized pediatric neurology services and diagnostic tools such as EEG, neuroimaging, and genetic testing, predisposes to frequent misclassification of PD as epileptic seizures.9 This challenge is further compounded by low clinician awareness of movement disorders, limited subspecialty training, and systemic healthcare constraints. As a result, many children with PD are inappropriately treated with long-term antiepileptic medications, often with suboptimal clinical response.

The consequences of misdiagnosis are substantial. Inappropriate treatment exposes affected children to unnecessary medication-related adverse effects, financial burden, and psychosocial stigma, while delaying accurate diagnosis and targeted management.[22,23] In certain genetic forms, such as GLUT1 deficiency syndrome, failure to recognize PD may result in missed opportunities for effective interventions, including dietary therapies.[6] Furthermore, the paucity of region-specific data on PD in Sub-Saharan Africa perpetuates gaps in clinical awareness, research, and policy development, thereby limiting improvements in diagnostic accuracy and patient outcomes.

Given these challenges, there is a critical need for a comprehensive synthesis of current evidence to enhance recognition and understanding of PD in children, particularly within resource-constrained settings. This narrative review aims to provide an in-depth overview of paroxysmal dyskinesias in the pediatric population, with specific emphasis on diagnostic challenges and the frequent misdiagnosis as epilepsy in Sub-Saharan Africa. By integrating existing knowledge with contextual realities, this review seeks to improve clinical awareness, promote accurate diagnosis, and inform more effective management strategies for affected children in the region.

METHODS

A narrative review approach was adopted to synthesize available evidence on paroxysmal dyskinesias (PD) in children, with particular emphasis on diagnostic challenges and misdiagnosis as epilepsy in resource-limited settings of Sub-Saharan Africa. This design was considered most appropriate given the rarity of PD, the predominance of descriptive and observational literature, and the marked heterogeneity in study designs, populations, and reported outcomes. The review integrates historical perspectives, clinical characteristics, epidemiological data, and health system factors to provide a comprehensive understanding of the condition in both global and African contexts.

A comprehensive literature search was conducted using electronic databases, including MEDLINE (via PubMed), ScienceDirect, Google Scholar, and the World Health Organization databases, to identify relevant full-text articles published in English through February 2026. These databases were selected for broad coverage across neurology, pediatrics, and global health literature, with particular attention to studies originating from or relevant to low- and middle-income countries. The search strategy incorporated combinations of keywords and Boolean operators derived from four core domains: the disease entity, diagnostic considerations, population group, and geographic or health-system context. Search terms included "paroxysmal dyskinesia," "paroxysmal kinesigenic dyskinesia," "paroxysmal non-kinesigenic dyskinesia," "paroxysmal exercise-induced dyskinesia," and "episodic movement disorders," combined with terms such as "misdiagnosis," "diagnostic challenge," "differential diagnosis," "epilepsy mimic," and "non-epileptic events," alongside "children," "pediatric," "adolescents," and contextual terms including "Sub-Saharan Africa," "resource-limited settings," "low- and middle-income countries," and "Africa." Boolean combinations, for example ("paroxysmal dyskinesia" AND "epilepsy" AND "misdiagnosis" AND "children"), were used to refine search outputs. Additional relevant studies were identified through manual screening of the reference lists of retrieved articles.

Studies were considered eligible if they reported on paroxysmal dyskinesias or related episodic movement disorders in pediatric populations, including descriptive, epidemiological, hospital-based, and review studies. Publications addressing diagnostic challenges, misclassification, or health system limitations were prioritized. Studies conducted globally were included to provide comparative insights, with particular attention to

those addressing African or other resource-constrained settings. Only articles published in English with an accessible full text were included. Reports focusing exclusively on adult populations without pediatric data, and studies lacking sufficient methodological detail were excluded. Titles and abstracts of retrieved records were screened for relevance, followed by full-text assessment of potentially eligible articles.

A total of 41 relevant publications met the inclusion criteria and were included in the final synthesis. Given the heterogeneity in study designs, sample sizes, and reported outcomes, quantitative meta-analysis was not feasible. Instead, data were synthesized qualitatively, with emphasis on recurring themes pertinent to pediatric PD in resource-limited settings. These included the influence of high epilepsy prevalence on diagnostic overshadowing, underreporting of PD in clinical and community settings, reliance on clinical judgment in the absence of diagnostic tools such as electroencephalography and neuroimaging, and the use of anticonvulsant therapy, reinforcing diagnostic misclassification. Additional considerations included the scarcity of trained pediatric neurologists and movement disorder specialists, as well as the emerging role of low-cost diagnostic adjuncts such as caregiver-recorded videos in improving clinical recognition.

Findings from African contexts were interpreted alongside global evidence on epidemiology, genetics, and pathophysiology to highlight disparities in diagnostic capacity and disease recognition between high-income and resource-limited settings. Case reports and small case series were included to illustrate characteristic clinical presentations and diagnostic dilemmas, particularly in resource-limited settings, but were interpreted with caution regarding generalizability. This integrative approach was intended to provide a clinically relevant and context-sensitive synthesis to inform improved recognition, diagnosis, and management of paroxysmal dyskinesias in children.

DATA SYNTHESIS

Clinical Features

Movement Phenomenology

Paroxysmal dyskinesias are characterized by sudden, brief, and recurrent episodes of involuntary hyperkinetic movements, with complete or near-complete resolution between attacks. The predominant phenomenology includes dystonia and choreoathetosis, with ballistic movements occurring less frequently. Tremor and myoclonus are generally not typical features, and their presence should prompt consideration of alternative diagnoses.[7] The episodic nature, with abrupt onset and termination, distinguishes PD from other movement disorders that exhibit a more continuous or fluctuating course over time.

Trigger Patterns

Attacks may occur spontaneously or be precipitated by identifiable triggers, often consistent within individual patients. Sudden voluntary movement or startle commonly provokes attacks in PKD, whereas PNKD episodes are typically triggered by stress, fatigue, sleep deprivation, caffeine-containing beverages, alcohol, or emotional stimuli. PED is characteristically induced by prolonged physical exertion. Additional triggers such as dehydration and, in adolescent females, menstruation have also been reported.[7,20,24] Recognition of these reproducible triggers provides an important diagnostic clue and helps differentiate PD from other paroxysmal neurological conditions.

Distribution of Movements

The distribution of dyskinetic movements varies by subtype but is often stereotyped in each patient. PKD frequently involves the upper limbs, whereas PED more commonly affects the lower limbs. Movements may be unilateral or bilateral and can extend to involve the face, neck, and trunk in more severe episodes. Although the pattern of involvement tends to be consistent, variability between attacks may occur.[20,24]

Interictal Normality

A defining feature of primary PD is the preservation of normal neurological function between episodes. Children typically demonstrate normal cognition, neurological examination, and developmental milestones during interictal periods.[1,20] This interictal normality, while diagnostically helpful, may also contribute to under-recognition, particularly in settings where episodic symptoms are not directly observed by clinicians.

Differential Diagnosis

Epilepsy Mimics

Epilepsy represents the most common and clinically significant mimic of PD. Conditions such as focal epilepsies, particularly frontal lobe seizures and focal aware seizures with motor features, may closely resemble dyskinetic episodes. Reflex epilepsies triggered by

movement or sensory stimuli further complicate differentiation. Other conditions that may be confused with PD include benign infantile seizures, nocturnal seizure disorders with dystonic features, episodic ataxias, tetany, periodic paralyses, and neuromyotonia.[20,25,26] The overlap in episodic motor manifestations and triggering factors necessitates careful clinical evaluation.

Distinguishing PD from epilepsy relies primarily on clinical semiology, including preserved consciousness, stereotyped triggers, brief duration of attacks, and normal interictal examination. Electroencephalography, where available, is useful in excluding epileptiform activity. Misdiagnosis has significant implications, including inappropriate labeling of epilepsy, unnecessary long-term treatment, and psychosocial consequences.[26]

Functional Movement Disorders

Functional movement disorders (FMD) represent another important consideration in the differential diagnosis. These disorders are characterized by variability, inconsistency, distractibility, and suggestibility of movements, often changing with attention or observation.[27,28] In contrast, PD is typically associated with consistent and reproducible triggers, stereotyped attack patterns, and preserved awareness. However, coexistence of functional symptoms and organic movement disorders may occur, further complicating diagnosis. [7,29]

Paroxysmal Non-Epileptic Events

Several non-epileptic paroxysmal conditions in children may mimic PD, including breath-holding spells, shuddering attacks, benign paroxysmal torticollis, and benign paroxysmal vertigo. These conditions share episodic presentations but lack the characteristic hyperkinetic motor phenomenology and trigger patterns observed in PD.[26,30–32] Careful history-taking and clinical observation are essential for differentiation.

Drug-Induced Movement Disorders

Drug-induced movement disorders, such as acute dystonic reactions or levodopa-induced dyskinesias, may present with abnormal movements resembling PD but are distinguished by their temporal relationship to medication use.[7] These are not primary paroxysmal disorders and should be excluded through a detailed medication history.

Diagnostic Challenges in Sub-Saharan Africa

Limited Access to Diagnostic Tools

The diagnosis of PD in Sub-Saharan Africa is significantly constrained by limited access to electroencephalography, neuroimaging, and genetic testing, which are often available only at tertiary centers.[9,41] As a result, clinicians frequently rely on clinical assessment alone, increasing the risk of misclassification, particularly in differentiating PD from epilepsy.

Delays in Diagnosis

Delayed diagnosis is common, with reported intervals of several years between symptom onset and accurate diagnosis.[9] This delay reflects a combination of limited specialist availability, low index of suspicion, and prioritization of more prevalent neurological conditions such as epilepsy and infectious diseases.

Consequences of Misdiagnosis

Misdiagnosis of PD as epilepsy carries substantial clinical and psychosocial consequences. Children are often exposed to long-term antiepileptic therapy with limited or no benefit, increasing the risk of adverse effects and financial burden. In addition, an epilepsy label may result in social stigma, educational restrictions, and reduced quality of life.[26] The failure to recognize PD also delays implementation of appropriate management strategies, including trigger avoidance and targeted therapies.

Role of Caregiver-Recorded Videos

In the absence of advanced diagnostic tools, caregiver-recorded videos using mobile devices have emerged as a valuable adjunct for diagnosis. These recordings allow clinicians to directly observe attack phenomenology, improving diagnostic accuracy and facilitating differentiation from epileptic events.[33]

Educational and Systemic Gaps

Systemic challenges, including limited training in movement disorders, scarcity of pediatric neurologists, and inadequate integration of neurology into primary healthcare systems, further contribute to under-recognition.[9,41] Addressing these gaps requires targeted educational interventions, development of simplified diagnostic algorithms, and increased use of telemedicine and regional collaborations to support clinical decision-making.[34,39]

Management

Non-Pharmacological Interventions

Management of PD in resource-limited settings emphasizes simple, effective, and low-cost strategies. Education of caregivers, teachers, and patients regarding the non-epileptic nature of the condition is essential to reduce stigma and prevent unnecessary restrictions. Trigger avoidance, including minimizing stress, ensuring adequate sleep, and avoiding known precipitants such as caffeine, is a cornerstone of management.

Dietary and Lifestyle Measures

Dietary and lifestyle modifications are particularly important in PED and metabolically mediated forms. Regular meals, adequate hydration, and avoidance of prolonged fasting are recommended. In settings where formal ketogenic diet therapy is not feasible, modified dietary approaches using locally available foods may provide benefit, particularly in suspected *SLC2A1*-related disorders.[35]

Pharmacological Treatment

Paroxysmal kinesigenic dyskinesia (PKD) demonstrates a remarkable response to low-dose anticonvulsants, particularly carbamazepine, with response rates exceeding 80–90%.[36] Effective control is often achieved at doses lower than those used for epilepsy. Oxcarbazepine is a suitable alternative, while phenytoin and valproate may be considered when first-line agents are unavailable or contraindicated.[7,37,38] In resource-limited settings, a therapeutic trial of carbamazepine may serve both diagnostic and therapeutic purposes. Paroxysmal non-kinesigenic dyskinesia (PNKD) shows variable responsiveness to pharmacotherapy. Benzodiazepines such as clonazepam may reduce attack frequency or severity in some patients, although avoidance of triggers remains the mainstay of management.[6]

Management of Paroxysmal exercise-induced dyskinesia (PED) focuses on lifestyle modification, including reducing exercise intensity, incorporating rest periods, and ensuring adequate nutrition and hydration.[7] Acetazolamide may be beneficial in selected cases, particularly when metabolic etiologies are suspected. Secondary PD requires identification and treatment of the underlying cause, such as metabolic disorders, structural brain lesions, or inflammatory conditions.[7] Targeted therapy may result in significant clinical improvement.

Prognosis

Paroxysmal dyskinesias in children generally have an excellent prognosis, particularly primary forms, which are non-progressive with preserved neurological function between attacks. PKD often shows a reduced attack frequency with age, and many patients achieve partial or complete remission by adulthood, with excellent responses to carbamazepine or related agents.[7,36] PED associated with SLC2A1 mutations frequently responds to dietary interventions,[35] while PNKD exhibits a more variable course. Early recognition and accurate diagnosis are critical to prevent misdiagnosis as epilepsy, unnecessary long-term antiepileptic use, and psychosocial stigma. Management combining pharmacological therapy, trigger avoidance, caregiver education, lifestyle modifications, and low-cost diagnostic tools such as caregiver-recorded videos or telemedicine optimizes outcomes. Strengthening clinician awareness, integrating PD into national pediatric neurology guidelines, and enhancing diagnostic capacity represent cost-effective, high-impact strategies to improve pediatric neurological care in resource-limited Sub-Saharan African settings.[9,34,35,39.41]

CONCLUSION

Paroxysmal dyskinesias in children are frequently underrecognized and misdiagnosed as epilepsy in resource-limited Sub-Saharan Africa, driven by overlapping clinical features and constrained diagnostic capacity. This review highlights that accurate diagnosis is largely clinical and achievable through recognition of characteristic triggers, preserved consciousness, and normal interictal findings. Early identification enables effective, low-cost management and avoids unnecessary exposure to antiepileptic medications. Strengthening clinician awareness, integrating movement disorder training, and leveraging practical tools such as caregiver-recorded videos and telemedicine are essential. Improving diagnostic pathways and integrating paroxysmal dyskinesias into pediatric neurology frameworks will enhance outcomes and reduce the clinical and societal burden of misdiagnosis.

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How to cite this article: Legbo J. Paroxysmal Dyskinesias in Children: Diagnostic Challenges and Misdiagnosis as Epilepsy in Resource-Limited Sub-Saharan Africa - A Narrative Review. Int Arch Med Health Res 2025; 6(1): 76-83.