Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

# Recorded epilepsy among children with autism spectrum disorder at St Francis Hospital Nsambya, Uganda: a retrospective cross-sectional study

**Nassanga Maimounah**[1]
Uganda Martyrs University
**Owiny Augustine Okejomoe**[2]
owinyaugustine2018@gmail.com

Mulago Specialized Women and Neonatal Hospital
**Nerima Caroline**[3]
Uganda Martyrs University
**Sebunya Robert**[3]
Uganda Martyrs University

**Research Article**

**Keywords:** autism spectrum disorder, epilepsy, children, Uganda, prevalence, modified Poisson regression

**Posted Date:** November 16th, 2026

**DOI:** https://doi.org/10.21203/rs.3.rs-11272722/v1

**License:** This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

**Additional Declarations:** No competing interests reported.

## Abstract

# Background

Epilepsy is an important comorbidity in autism, but clinic-specific evidence from Uganda is limited. This study aimed to estimate the prevalence of recorded epilepsy and examine associated clinical characteristics among children with autism spectrum disorder (ASD) attending a specialist neurology clinic in Kampala, Uganda.

# Methods

This retrospective cross-sectional study examined an extract of 221 electronic clinical records for children aged 2–17 years attending St Francis Hospital Nsambya during December 2020–December 2025. Seventeen unavailable or nonbinary epilepsy outcomes were treated as missing. Analysis of 204 records used Wilson 95% confidence intervals (CIs) and modified Poisson regression with robust standard errors, adjusting for sex, both age variables, and developmental category.

# Results

Recorded epilepsy was present in 59/204 children (28.9%; 95% CI, 23.1–35.5). Higher recorded epilepsy prevalence was associated with female sex (adjusted prevalence ratio [aPR], 1.62; 95% CI, 1.04–2.54), ASD diagnosis at 6–17 versus 2–5 years (1.88; 1.10–3.21), and a global developmental delay label versus language delay/impairment (2.23; 1.22–4.06). Estimates for regression and intellectual disability were imprecise. Extreme assumptions about all 17 missing outcomes yielded prevalence bounds of 26.7%–34.4% across all 221 entries.

# Conclusions

Recorded epilepsy affected approximately three in ten children with documented status. Referral selection, missing outcomes, and retrospective documentation limit causal interpretation and population generalizability.

## Background

Epilepsy is an important co-occurring condition in children with autism spectrum disorder (ASD), a neurodevelopmental condition characterized by differences in social communication and restricted or repetitive patterns of behavior [1]. Seizure history is therefore an important part of the clinical assessment of children with ASD. Although shared biological pathways have been proposed, the clinical expression of both conditions is heterogeneous, and their coexistence does not establish a common cause in an individual child [2].

Published estimates give different pictures of how often epilepsy occurs in autistic populations. A meta-analysis reported a pooled prevalence of approximately 10%, while a systematic review reported a median study-level prevalence of 12.1% [3, 4]. The same meta-analysis estimated a pooled prevalence of 7% in autistic children and 19% in clinical sample-based cross-sectional studies [3]. Variation in age, developmental functioning, diagnostic ascertainment, and referral setting complicates comparisons [3, 4]. A neurology clinic may see more children with suspected seizures or complex neurological needs than a community survey, which can affect the prevalence observed.

Differences in age, sex, and developmental functioning also matter when interpreting these estimates. A meta-analysis associated epilepsy with intellectual disability and female sex [5]. In a large study of autistic children, older age and lower cognitive ability remained independently associated with epilepsy in the adjusted analysis [6]. A systematic review identified cognitive impairment or intellectual disability as a comparatively consistent correlate, with less consistent evidence for several other characteristics [7]. However, a selected clinical label such as language impairment, regression, or global developmental delay is not equivalent to a standardized cognitive assessment. A child may have more than one of these difficulties. When a record provides only one developmental label, the comparison must be interpreted in light of what that label captures.

African studies offer relevant comparisons, although their study populations differ. A South African clinic series reported epilepsy in 23% of 86 autistic children [8]. Ugandan research has evaluated neurodevelopmental screening [9] and ASD symptoms among children attending a pediatric neurology clinic [10]. A community study in Northern Uganda also documented epilepsy among conditions co-occurring with autism in children aged 2–9 years [11]. These studies address different populations and ascertainment pathways. The starting population matters: a study of ASD symptoms among children with neurological disorders addresses a different question from a study of epilepsy among children already diagnosed with ASD.

We examined clinical records from the Neurology Clinic at St Francis Hospital Nsambya, Kampala, Uganda, to estimate recorded epilepsy prevalence among children with ASD and assess associations with sex, current age, age at ASD diagnosis, and the extracted developmental category. Secondary objectives were to describe epilepsy labels, medication mentions, and other clinical documentation. We distinguished missing epilepsy status from recorded negative responses to make the outcome denominator and documentation gaps explicit. The study describes the children seen at this clinic; it cannot establish national prevalence or causal determinants.

## Methods

# Study Design, Setting, and Records

We conducted an analytical cross-sectional study of retrospective clinical records from the Neurology Clinic at St Francis Hospital Nsambya in Kampala, Uganda. The clinical attendance window was

December 2020 through December 2025. The investigator abstracted data from the hospital's electronic clinical system into a structured study extract during the reported January–February 2026 period, after administrative clearance dated January 22, 2026. No new patient interviews, examinations, or investigations were performed. The original study procedures described consecutive selection of records for children aged 2–17 years with a documented specialist diagnosis of ASD. Diagnoses were taken from the existing clinical record; ASD was not independently reassessed using a standardized diagnostic instrument for this study.

The study size was determined by the 221 patient entries available in the record extract after exclusion of nonpatient separator rows. Each patient entry was the intended unit of analysis. The workbook did not contain a complete screening log, sufficient attendance-date detail to independently reconstruct the eligible sampling frame, or a documented audit of repeat attendances. Consequently, the analysis describes the available extract and cannot establish that it captured every eligible child attending during the study window. No national or catchment-population denominator was available. An audit found no identical full rows. Six groups of repeated names involved 13 entries; the investigator confirmed that these entries represented different patients rather than repeat records. All entries were retained.

## Outcome and Clinical Variables

The primary outcome was recorded epilepsy status. An explicit "YES" was classified as epilepsy and an explicit "NO" as absence of recorded epilepsy. Six "UNKNOWN" entries, ten blank entries, and one entry containing an epilepsy-syndrome label rather than a binary response were treated as missing outcome data. These 17 entries were excluded from the primary prevalence and regression analyses. A blank field was not interpreted as a negative diagnosis, and medication use alone was not used to establish epilepsy.

The International League Against Epilepsy definition provides a clinical framework for diagnosing epilepsy [12]. However, the extract did not provide sufficient seizure histories, electroencephalography findings, or diagnostic assessments to independently apply that definition to every child. The study endpoint therefore represents epilepsy documented in the source record, rather than newly adjudicated epilepsy. An explicit negative flag similarly reflects the recorded classification rather than proof that seizures never occurred.

Age values were analyzed in years. Age groups were chosen to describe preschool, school-age, and adolescent records; diagnosis age was divided at 6 years to compare recognition before and from school age. These thresholds are descriptive and do not establish clinical cutoffs. Current age was categorized as 2–5, 6–11, and 12–17 years, implemented as ages below 6, from 6 to below 12, and from 12 to below 18 years. Age at ASD diagnosis was categorized as 2–5 or 6–17 years. Recorded diagnosis age did not exceed recorded current age in the available entries. The extract did not identify a uniform index date for measuring current age. Sex was analyzed as recorded in the workbook.

The developmental field contained one extracted label per entry. Language delay and language impairment were combined as the language category. Other categories were global developmental delay (GDD), developmental regression, intellectual disability, and a heterogeneous “other” group containing neurological or syndrome labels. These were mutually exclusive analytic categories because of the structure of the extract; they do not imply that the underlying clinical conditions were mutually exclusive. In particular, the comparison of GDD with the language category is not a comparison of confirmed GDD with confirmed absence of GDD.

Perinatal history, family history of epilepsy, behavioral labels, and the combined investigation field were summarized as documented; the completeness of these fields did not establish whether the corresponding clinical history had been systematically assessed. Explicit “NONE” in the perinatal field was distinguished from an empty cell and “UNKNOWN.” The combined irritability/attention-deficit/hyperactivity disorder label was retained without separating its components. Investigation entries distinguished electroencephalography (EEG), magnetic resonance imaging (MRI), “NOT DONE,” and missing documentation; the field did not establish independent histories of both tests. A separate free-text result field contained brief clinical or investigation narratives. Entries beginning with “NORMAL” were summarized as a normal label, other nonblank entries as other result narratives, and empty entries as undocumented. These categories describe the text and do not constitute independent EEG or MRI interpretation.

Among children with recorded epilepsy, the type field was summarized as generalized, focal, an epilepsy-syndrome label, or undocumented. Syndrome labels were preserved as a separate documentation category because they are not at the same classification level as focal or generalized epilepsy. Therapy mentions were identified in free text, allowing multiple therapy types per child. Distinct recognized antiseizure medication names were counted after mapping identifiable brand names and spelling variants to generic names. This count describes names mentioned in the record and does not establish a concurrent regimen, adherence, seizure control, or drug resistance.

# Statistical Analysis

Categorical variables were summarized using counts and percentages with explicit denominators. Continuous ages were summarized by medians and interquartile ranges (IQRs). The primary epilepsy proportion used the 204 records with an explicit binary outcome, with a Wilson 95% confidence interval (CI). Missing outcomes were not imputed, and missing covariate documentation was retained in descriptive summaries rather than coded as absence. Sex, both age variables, and the developmental category were available for all 204 records in the primary regression sample.

We fitted univariable and multivariable modified Poisson models with a log link and robust sandwich standard errors to estimate crude and adjusted prevalence ratios (PRs) [13]. A finite-sample variance multiplier of $n/(n - k)$ was applied, where $k$ was the number of model coefficients, including the intercept. CIs and two-sided P values used the normal Wald approximation. Analyses were exploratory, and no

adjustment was made for multiple comparisons; effect sizes and confidence intervals were emphasized over significance thresholds. The multivariable model included sex, current age category, age-at-ASD-diagnosis category, and developmental category. Covariates were retained for their clinical relevance and availability rather than selected through univariable significance screening. Reference groups were male sex, current age 2–5 years, diagnosis age 2–5 years, and language delay/impairment. The model included nine coefficients, including the intercept. Current age and diagnosis age were entered together to distinguish their conditional associations; this adjustment cannot resolve uncertainty about the order of clinical events.

Perinatal history, family history, behavioral labels, investigations, and therapy mentions were not included as additional adjusted covariates because of sparse categories, missing or ambiguous documentation, and limitations in temporal interpretation. The “other” developmental category was retained to preserve the extracted classification, but its estimate was treated as exploratory because it contained six children with disparate conditions. The model was used for association estimates, not individual probability prediction; the largest fitted mean was 1.17, a known possible feature of the unconstrained Poisson approach to a binary outcome.

Two sensitivity analyses examined outcome uncertainty. First, extreme assumptions classified all 17 missing outcomes as negative or all as positive, giving descriptive bounds across the full extract. These bounds are not confidence intervals. Second, an exploratory label-based analysis classified two nonbinary records with a generalized-epilepsy or epilepsy-syndrome label as possible positive cases, while retaining the remaining 15 outcomes as missing. This scenario was not used to replace the primary classification. A further exploratory analysis excluded the six records in the heterogeneous “other” developmental category and refitted the adjusted model with the remaining four developmental categories (Additional file 1). No interaction tests, sampling weights, or clustered analyses were used. The investigator reported use of Stata version 18 (StataCorp LLC, College Station, TX, USA) for the original statistical analysis. The revised estimates presented here were recalculated from the supplied Excel extract in Python 3.12.14 using NumPy 2.3.5 and SciPy 1.17.0; regression coefficients were also checked by a separate numerical optimization. Executable Python reanalysis code and aggregate verification outputs are provided as supplementary materials. A Stata 18 reproduction script is also available for author verification; it was not executed during this revision.

## Declaration of AI assistance

OpenAI ChatGPT was used to assist with manuscript drafting, language editing, and preparation of Python code for reanalysis and independent numerical verification, and drafting of Stata reproduction code. Numerical outputs were checked against the source workbook and a separate optimization of the regression model. AI assistance was not used to collect clinical observations, create patient data, or assign new patient diagnoses. ChatGPT is not an author and cannot accept accountability for the work. The human authors remain responsible for the accuracy, originality, interpretation, references, and final content of the manuscript.

## Results

# Analytic Sample and Clinical Characteristics

Of the 221 entries, 59 had an explicit positive epilepsy flag, 145 an explicit negative flag, and 17 (7.7%) an unavailable or nonbinary flag. The primary sample therefore comprised 204 children. Among the 17 excluded entries, 12 were male and 5 were female; median current age was 10.0 years (IQR, 9.0–12.0), and median age at ASD diagnosis was 4.0 years (IQR, 3.0–6.0). Their developmental labels were language delay/impairment in 9, regression in 4, intellectual disability in 3, and another condition in 1. These summaries describe outcome missingness and do not establish its mechanism.

In the primary sample of 204 children, median current age was 7.8 years (IQR, 5.1–9.8), and median age at ASD diagnosis was 4.0 years (IQR, 3.0–6.0). Males accounted for 163/204 children (79.9%). Most children were aged 6–11 years (110/204; 53.9%), and 142 (69.6%) had an ASD diagnosis recorded before age 6 years (Table 1).

Table 1
Characteristics of the Primary Analytic Sample (N = 204)

| Characteristic | Children, n (%) | Epilepsy, n/N (%) |
|---|---|---|
| Sex | | |
| Male | 163 (79.9) | 42/163 (25.8) |
| Female | 41 (20.1) | 17/41 (41.5) |
| Current age | | |
| 2–5 years | 64 (31.4) | 19/64 (29.7) |
| 6–11 years | 110 (53.9) | 31/110 (28.2) |
| 12–17 years | 30 (14.7) | 9/30 (30.0) |
| Age at ASD diagnosis | | |
| 2–5 years | 142 (69.6) | 35/142 (24.6) |
| 6–17 years | 62 (30.4) | 24/62 (38.7) |
| Extracted developmental category | | |
| Global developmental delay | 50 (24.5) | 22/50 (44.0) |
| Language delay/impairment | 76 (37.3) | 13/76 (17.1) |
| Developmental regression | 46 (22.5) | 15/46 (32.6) |
| Intellectual disability | 26 (12.7) | 4/26 (15.4) |
| Other neurological/syndrome label | 6 (2.9) | 5/6 (83.3) |

Percentages in the middle column use N = 204; epilepsy percentages are within each category. Median current age: 7.8 years (IQR, 5.1–9.8); median ASD diagnosis age: 4.0 years (IQR, 3.0–6.0). Developmental categories represent one extracted label per child and do not establish absence of overlapping clinical conditions. ASD, autism spectrum disorder; IQR, interquartile range.

Language delay/impairment was the extracted developmental label in 76 children (37.3%), GDD in 50 (24.5%), regression in 46 (22.5%), intellectual disability in 26 (12.7%), and other conditions in 6 (2.9%). Recorded epilepsy proportions differed across these labels: 13/76 (17.1%) in the language category, 22/50 (44.0%) in GDD, 15/46 (32.6%) in regression, 4/26 (15.4%) in intellectual disability, and 5/6 (83.3%) in the other category (Table 1).

Additional clinical documentation is summarized in Table 2. Perinatal history was explicitly coded as none in 111/204 records (54.4%), while 57 (27.9%) were blank or unknown. Family history of epilepsy was unknown in 66 records (32.4%). All five children with a positive recorded family history of epilepsy had recorded epilepsy, but this small subgroup did not support a stable adjusted estimate. The combined irritability/attention-deficit/hyperactivity disorder label appeared in 144 records (70.6%). EEG

was the investigation label in 88 records (43.1%), MRI in 13 (6.4%), and “NOT DONE” in 97 (47.5%). These categories describe one combined field and cannot determine whether a child ever received both EEG and MRI. The separate result field was undocumented in 137/204 records (67.2%). Among 88 EEG entries, 34 had a normal label, 28 another result narrative, and 26 no documented result. Among 13 MRI entries, 3 had a normal label, 1 another result narrative, and 9 no documented result. One entry coded “NOT DONE” nevertheless contained a result narrative; this inconsistency was retained as a documentation limitation rather than reassigned to a test category. Speech therapy was mentioned for 192 children (94.1%), occupational therapy for 172 (84.3%), and physiotherapy for 3 (1.5%); these overlapping mentions do not establish treatment attendance or completion.

Table 2
Additional Clinical Documentation in the Primary Sample (N = 204)

| Recorded field/category | Children, n (%) |
|---|---|
| Perinatal history | |
| Explicitly none | 111 (54.4) |
| Birth complications | 10 (4.9) |
| Hypoxic-ischemic encephalopathy | 2 (1.0) |
| Prematurity | 21 (10.3) |
| Central nervous system infection | 1 (0.5) |
| Post-term birth | 2 (1.0) |
| Blank/undocumented | 21 (10.3) |
| Unknown | 36 (17.6) |
| Family history of epilepsy | |
| No | 133 (65.2) |
| Unknown | 66 (32.4) |
| Yes | 5 (2.5) |
| Behavioral label | |
| Blank/undocumented | 14 (6.9) |
| Combined irritability/ADHD label | 144 (70.6) |
| Mannerisms | 46 (22.5) |
| Combined investigation field | |
| Not done | 97 (47.5) |
| Electroencephalography | 88 (43.1) |
| Magnetic resonance imaging | 13 (6.4) |
| Blank/undocumented | 6 (2.9) |

Blank and unknown entries are not negative clinical findings. The combined behavioral label does not identify separate frequencies of irritability and ADHD. The investigation field does not establish independent EEG and MRI histories. Separate brief result narratives are described in the text. ADHD, attention-deficit/hyperactivity disorder; EEG, electroencephalography; MRI, magnetic resonance imaging.

## Prevalence and Associated Characteristics

Recorded epilepsy was present in 59/204 children, giving a prevalence of 28.9% (95% CI, 23.1–35.5). The proportion was 17/41 (41.5%) among females and 42/163 (25.8%) among males. It was 24/62 (38.7%) among children diagnosed with ASD at 6–17 years and 35/142 (24.6%) among those diagnosed at 2–5 years. Proportions across current-age categories were similar: 29.7%, 28.2%, and 30.0% for ages 2–5, 6–11, and 12–17 years, respectively (Table 1).

In the adjusted model, female sex was associated with higher recorded epilepsy prevalence than male sex (adjusted PR [aPR], 1.62; 95% CI, 1.04–2.54; P = 0.035). Diagnosis at 6–17 rather than 2–5 years was also associated with higher prevalence (aPR, 1.88; 95% CI, 1.10–3.21; P = 0.021). Children labeled with GDD had higher prevalence than those labeled with language delay/impairment (aPR, 2.23; 95% CI, 1.22–4.06; P = 0.009). Adjusted estimates for regression (1.76; 0.91–3.39) and intellectual disability (0.87; 0.29–2.61) were imprecise, as were the current-age contrasts. The other category had an aPR of 4.98 (95% CI, 2.66–9.32), but its five events among six heterogeneous records precluded a specific clinical interpretation. Crude and adjusted estimates are presented in Table 3.

Table 3
Crude and Adjusted Associations With Recorded Epilepsy (N = 204; 59 Events)

| Contrast | Crude PR (95% CI) | Adjusted PR (95% CI) | Adjusted P |
|---|---|---|---|
| Female vs male | 1.61 (1.03–2.52) | 1.62 (1.04–2.54) | 0.035 |
| Current age 6–11 vs 2–5 years | 0.95 (0.58–1.54) | 0.77 (0.43–1.37) | 0.374 |
| Current age 12–17 vs 2–5 years | 1.01 (0.52–1.97) | 0.58 (0.25–1.35) | 0.209 |
| ASD diagnosis age 6–17 vs 2–5 years | 1.57 (1.02–2.41) | 1.88 (1.10–3.21) | 0.021 |
| GDD vs language delay/impairment | 2.57 (1.42–4.65) | 2.23 (1.22–4.06) | 0.009 |
| Developmental regression vs language delay/impairment | 1.91 (0.99–3.67) | 1.76 (0.91–3.39) | 0.092 |
| Intellectual disability vs language delay/impairment | 0.90 (0.32–2.55) | 0.87 (0.29–2.61) | 0.808 |
| Other vs language delay/impairment | 4.87 (2.63–9.04) | 4.98 (2.66–9.32) | < 0.001 |

Modified Poisson regression with log link and robust sandwich variance; finite-sample correction n/(n − k). All adjusted estimates come from one model including sex, current age, diagnosis age, and developmental category. References: male, current age 2–5 years, diagnosis age 2–5 years, and language delay/impairment. “Other” includes five events among six heterogeneous records and is exploratory. CI, confidence interval; GDD, global developmental delay; PR, prevalence ratio.

## Epilepsy Documentation and Sensitivity Analyses

Among the 59 children with recorded epilepsy, 30 (50.8%) had a generalized label, 21 (35.6%) a focal label, 4 (6.8%) an epilepsy-syndrome label, and 4 (6.8%) no documented type. One recognized antiseizure medication name was mentioned for 32 children (54.2%), two for 17 (28.8%), and three for 6 (10.2%); no recognized name was identified for 4 (6.8%) (Table 4). These summaries describe documentation rather than confirmed treatment regimens.

Table 4
Epilepsy Labels and Antiseizure Medication Mentions Among Recorded Cases (N = 59)

| Documentation category | Children, n (%) |
|---|---|
| Recorded type/label | |
| Generalized | 30 (50.8) |
| Focal | 21 (35.6) |
| Epilepsy-syndrome label | 4 (6.8) |
| Undocumented | 4 (6.8) |
| Distinct recognized medication names mentioned | |
| None identified | 4 (6.8) |
| One | 32 (54.2) |
| Two | 17 (28.8) |
| Three | 6 (10.2) |

Percentages use the 59 primary cases. Syndrome labels are retained as a documentation category and are not equivalent to focal/generalized classifications. Recognized names included levetiracetam, carbamazepine, clobazam, lamotrigine, valproate, phenobarbital, diazepam, and clonazepam. Counts refer to distinct names mentioned, not verified concurrent regimens, adherence, or drug resistance. “None identified” does not prove that no medication was prescribed.

Classifying all 17 missing outcomes as negative yielded 59/221 (26.7%); classifying all as positive yielded 76/221 (34.4%). In the exploratory label-based scenario, 61/206 children were classified as possible cases (29.6%; 95% CI, 23.8–36.2). Adjusted estimates remained similar for female sex (aPR, 1.59; 95% CI, 1.05–2.41), diagnosis age 6–17 years (1.88; 1.12–3.15), and GDD versus language delay/impairment (2.22; 1.22–4.04). The primary analysis continued to treat all 17 nonbinary or unavailable flags as missing. Excluding the six “other” developmental records left 198 records and 54 epilepsy events. Adjusted estimates remained similar for female sex (aPR, 1.63; 95% CI, 1.04–2.54), diagnosis age 6–17 years (1.95; 1.07–3.54), and GDD versus language delay/impairment (2.22; 1.22–4.05) (Additional file 1, Table S1). This exploratory analysis assessed the influence of that category; it did not resolve the underlying developmental coding limitations.

# Discussion

Recorded epilepsy was present in 28.9% of children with an explicit epilepsy status in this specialist clinic sample. After adjustment for the specified covariates, higher recorded prevalence was associated with female sex, ASD diagnosis at age 6 years or later, and a global developmental delay (GDD) label compared with language delay/impairment. The associations apply to the children represented in these clinic records. Because seizure-onset dates were unavailable, we could not determine whether the associated characteristics preceded epilepsy.

The prevalence estimate was descriptively higher than the approximately 10% pooled estimate and 12.1% median study-level estimate in international reviews [3, 4], and the 23% reported in a South African clinic series [8]. Within the meta-analysis, prevalence was 7% (95% CI, 4–11) in autistic children and 19% (95% CI, 6–35) in clinical sample-based cross-sectional studies [3]. The child and clinical-sample estimates are particularly relevant to this clinic population. Even so, a higher observed prevalence here does not establish a geographic difference in susceptibility. Neurology referral can select children with seizures, complex developmental presentations, or established neurological diagnoses. Age distributions, cognitive profiles, diagnostic criteria, and opportunities for recognition also differ between studies. The Wilson interval describes statistical uncertainty around the observed proportion; it does not account for referral selection or diagnostic misclassification.

The Ugandan studies also need to be read in relation to whom they enrolled. Arinda et al assessed ASD symptoms among children attending a neurology service [10], whereas this analysis begins with a documented ASD diagnosis and assesses recorded epilepsy within that group. The Northern Uganda community study used screening and specialist assessment to identify autistic children and their co-occurring conditions at younger ages [11]. This study adds a description of a referred population aged 2–17 years, including conditional clinical associations and outcome-documentation uncertainty. It cannot estimate the excess epilepsy prevalence attributable to autism or quantify community-to-clinic differences.

Although females made up a smaller part of the sample, a greater proportion had recorded epilepsy. Earlier literature has linked epilepsy in autism to sex and intellectual disability [5]. In this study, the confidence interval was wide and its lower limit was close to one. Diagnostic and referral processes may select females with different unmeasured developmental characteristics from males. Standardized cognitive function and reasons for referral were unavailable, so biological differences and selection cannot be separated. Sex alone should not be used as a prediction rule on the basis of these results.

Later recorded ASD diagnosis was associated with epilepsy after adjustment for current-age category. Delayed recognition of developmental difficulties, differences in service access, or referral prompted by seizures could contribute to this pattern. The extract did not establish whether epilepsy began before or after ASD diagnosis, and both age variables reflect opportunities for clinical recognition. Their conditional association therefore does not establish that delayed ASD diagnosis causes epilepsy or that

earlier diagnosis prevents seizures. Prospective records with dated developmental concerns, diagnoses, referral, and seizure onset would be needed to investigate these pathways.

The higher prevalence in the GDD category is broadly consistent with literature linking epilepsy to greater cognitive or developmental impairment [5–7]. This finding needs to be interpreted against a key feature of the data: each record contributed only one developmental label. Children in the language category may also have had other developmental difficulties, so this estimate cannot isolate an effect of GDD. GDD terminology may also persist in older children's records despite changes in developmental assessment. The lower and imprecise estimate for intellectual disability should consequently not be interpreted as evidence of protection or as contradicting standardized cognitive findings in other studies. Misclassification between labels, overlapping conditions, and small category sizes could influence these contrasts.

For developmental regression, the confidence interval included both a small reduction and a substantial increase in epilepsy prevalence. The records also lacked the timing and cause of the developmental loss, limiting interpretation of this association. The heterogeneous other category contained five cases among six children. Although the estimated association was large, these disparate diagnoses cannot be interpreted as a single clinical exposure or used to guide screening. These exploratory contrasts, assessed without multiplicity adjustment, require replication with more precise developmental measurements.

Current-age proportions were similar across the three categories, but the adjusted estimates were imprecise and only 30 children were aged 12–17 years. International evidence has identified age as a relevant correlate [6]. Similar proportions in these age groups do not mean that epilepsy occurrence remains unchanged throughout childhood. A cross-sectional comparison combines different referral histories, observation periods, and opportunities for diagnosis and cannot estimate new epilepsy diagnoses over time.

The missing outcomes affect how confidently the findings can be interpreted. The 17 excluded children were descriptively older than those analyzed, and none had a GDD label in the extracted field. Restricting the primary analysis to children with documented outcomes therefore changed the age and developmental composition of the sample. We cannot assume that missing epilepsy status was unrelated to epilepsy or its correlates. Excluding unavailable flags avoids inventing negative outcomes, but does not eliminate missing-data bias. The 26.7%–34.4% extreme-assumption bounds describe uncertainty in the full-extract proportion under opposite binary classifications; they are not confidence intervals and do not bound bias in adjusted associations. Including the two records with epilepsy-related labels produced similar estimates, but the status of the other missing records remains unresolved.

Treatment and investigation entries showed what had been documented, but provided a less complete account of clinical care. Generalized and focal labels accounted for most recorded cases. Although some brief EEG and MRI result narratives were available, the combination of missing findings, absent full reports, and limited seizure histories prevented independent reclassification. A result narrative paired

with “NOT DONE” also illustrates uncertainty in the extracted testing history. Epilepsy-syndrome labels were therefore preserved separately. Multiple medication names can reflect concurrent prescriptions, historical changes, or rescue treatment, and cannot establish drug resistance or treatment success. Similarly, therapy mentions show that developmental therapies appeared in documentation, not that children accessed or completed them. Investigation labels may follow clinical suspicion of epilepsy and should not be treated as antecedent causes.

The analysis uses a clearly defined outcome denominator, keeps unknown entries separate, and estimates prevalence ratios directly. Reporting the small category counts and testing alternative classifications of missing outcomes makes the limits of the estimates more visible. Other limitations arise from the records themselves. Neither ASD nor epilepsy was independently reassessed; a uniform observation date and follow-up duration were unavailable. A complete eligibility log and audit of repeat attendances could not be reconstructed from the extract. The sample was from one referral clinic, and unmeasured cognitive functioning, genetic diagnoses, socioeconomic circumstances, and service access may confound the associations. The modest number of events and sparse categories limit the reliability of Wald intervals and multiple exploratory contrasts. Some fitted Poisson means exceeded one, reinforcing that the model estimates associations and is unsuitable for individual probability prediction.

For this clinic, the clearest practical implication is to document seizure histories consistently alongside developmental assessments and record epilepsy status as positive, negative, or unknown. They do not justify routine EEG or MRI for every autistic child or treatment decisions based on the regression estimates. Future multicenter studies should capture unique children, dated diagnoses, overlapping developmental domains, standardized cognitive assessments, and longitudinal seizure outcomes. These improvements would help distinguish referral-related patterns from associations that extend beyond specialist care.

## Conclusions

Approximately three in ten children with ASD and an explicit epilepsy status had recorded epilepsy in this Ugandan neurology-clinic sample. Female sex, later recorded ASD diagnosis, and GDD compared with language delay/impairment were associated with higher prevalence in the specified exploratory model. Older age among children with missing outcomes, referral selection, and nonstandardized chart labels limit interpretation. These findings describe the children represented in the clinic records and highlight gaps in documentation. Further studies would be needed to estimate population prevalence, establish causal relationships, or evaluate treatment outcomes.

## Abbreviations

aPR, adjusted prevalence ratio; ASD, autism spectrum disorder; CI, confidence interval; EEG, electroencephalography; GDD, global developmental delay; IQR, interquartile range; MRI, magnetic

resonance imaging; PR, prevalence ratio.

## Declarations

# Ethics approval and consent to participate

The study protocol was approved by the St Francis Hospital Nsambya Research Ethics Committee (UG-REC-020), which also waived the requirement for informed consent for this retrospective clinical-record review, under reference SFHN-2026-2217, dated January 10, 2026. The investigator confirmed that this REC letter covers both protocol approval and the consent waiver. Hospital authorization to conduct the study and access records was granted by St Francis Hospital Nsambya, a teaching hospital affiliated with Uganda Martyrs University, under reference NH/ADDCS/RESCH/146/26, dated January 22, 2026. Electronic record abstraction began after hospital authorization was obtained. All study procedures involving human participants and human data were performed in accordance with relevant guidelines and regulations, including the Declaration of Helsinki.

# Consent for publication

Not applicable. The manuscript presents aggregate findings and contains no individually identifying patient information, patient images, or case descriptions.

# Availability of data and materials

The deidentified data supporting the findings of this study are available from the corresponding author on reasonable request. Identifiable patient records will not be shared.

# Competing interests

The authors declare that they have no competing interests.

# Funding

The study was funded by Nassanga Maimounah, who also undertook conceptualization, investigation, and writing of the dissertation.

# Authors' contributions

Nassanga Maimounah (NM) conceptualized the study, conducted the investigation, wrote the dissertation, and provided funding. Owiny Augustine Okejomoe (OAO) performed the formal analysis and

data curation, contributed to writing, and prepared visualizations. Nerima Caroline (NC) and Sebunya Robert (SR) supervised the study.


# Acknowledgements

The authors thank the management and clinical staff of St Francis Hospital Nsambya for facilitating access to the clinical records and supporting the study.

**Clinical trial number**: not applicable.


# References


1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed. American Psychiatric Association; 2013. 10.1176/appi.books.9780890425596.
2. Lee BH, Smith T, Paciorkowski AR. Autism spectrum disorder and epilepsy: disorders with a shared biology. Epilepsy Behav. 2015;47:191–201. 10.1016/j.yebeh.2015.03.017.
3. Liu X, Sun X, Sun C, et al. Prevalence of epilepsy in autism spectrum disorders: a systematic review and meta-analysis. Autism. 2022;26(1):33–50. 10.1177/13623613211045029.
4. Lukmanji S, Manji SA, Kadhim S, et al. The co-occurrence of epilepsy and autism: a systematic review. Epilepsy Behav. 2019;98(Pt A):238–48. 10.1016/j.yebeh.2019.07.037.
5. Amiet C, Gourfinkel-An I, Bouzamondo A, et al. Epilepsy in autism is associated with intellectual disability and gender: evidence from a meta-analysis. Biol Psychiatry. 2008;64(7):577–82. 10.1016/j.biopsych.2008.04.030.
6. Viscidi EW, Triche EW, Pescosolido MF, et al. Clinical characteristics of children with autism spectrum disorder and co-occurring epilepsy. PLoS ONE. 2013;8(7):e67797. 10.1371/journal.pone.0067797.
7. Zarakoviti E, Shafran R, Skuse D, et al. Factor associated with the occurrence of epilepsy in autism: a systematic review. J Autism Dev Disord. 2023;53(10):3873–90. 10.1007/s10803-022-05672-2.
8. Lamb GV, Green RJ, Olorunju S. Tracking epilepsy and autism. Egypt J Neurol Psychiatr Neurosurg. 2019;55:55. 10.1186/s41983-019-0103-x.
9. Kakooza-Mwesige A, Ssebyala K, Karamagi C, et al. Adaptation of the ten questions to screen for autism and other neurodevelopmental disorders in Uganda. Autism. 2014;18(4):447–57. 10.1177/1362361313475848.
10. Arinda A, Nakasujja N, Odokonyero R. Prevalence of autism spectrum disorder symptoms in a paediatric neurology clinic at a tertiary hospital in Uganda. S Afr J Psychiatr. 2021;27:1548. 10.4102/sajpsychiatry.v27i0.1548.
11. Simple O, Estlin EJ, Okello KK, et al. Prevalence, distribution, co-occurring neurodevelopmental conditions, and associated factors of autism among children in Northern Uganda. Autism. 2026;30(9):2366–80. 10.1177/13623613261468802.

12. Fisher RS, Acevedo C, Arzimanoglou A, et al. ILAE official report: a practical clinical definition of epilepsy. Epilepsia. 2014;55(4):475–82. 10.1111/epi.12550.
13. Zou G. A modified Poisson regression approach to prospective studies with binary data. Am J Epidemiol. 2004;159(7):702–6. 10.1093/aje/kwh090.