Original articles

Epilepsy in rural Ugandan children: seizure pattern, age of onset and associated findings

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Abstract

Introduction: Much information on childhood epilepsy in sub Saharan Africa is hospital based. A survey was considered necessary before integrating epilepsy management into a Ugandan community programme.

Method: Using an 'outreach' method, children with recurrent seizures were offered assessment at 19 sites in Rukungiri District. A brief history and neurological and developmental assessment was carried out on each child. A clinical diagnosis of epilepsy, including seizure type, was given to 440 of 618 children <18 years with 178 exclusions.

Results: The age-specific prevalence of epilepsy in children < 15 years was 2.04‰ (95% CI 1.94 ‰ to 2.24 ‰) based on 395 cases in an <15 years population of 193,126 . Percentage distribution by seizure type was:-generalised tonic-clonic (53%), complex partial seizures CPS (27%), simple partial and miscellaneous seizures (6% each), with some diagnostic overlap between seizure types.

Cerebral palsy, evident or evolving, was most strongly associated with CPS. A positive perinatal or infantile history was noted in 12 and 6% respectively, and 50.2% of seizures began in infancy.

Conclusions: The prevalence of epilepsy is similar in Gambian children. The high contribution from early-onset CPS, resembles Kenyan reports of malaria- associated CPS, suggesting a causal association with malaria.

Key words: seizure type, associated findings, age–specific prevalence, possible malaria association

African Health Sciences 2010; 10(3): 218 - 225

Introduction

The challenges facing those developing countries now involved in the global campaign to bring epilepsy 'out of the shadows' [1] were reviewed by Scott et al [2] .Whereas at the national level, scarce resources are often directed towards 'structural adjustment' while infectious diseases remain a major priority for health planners at the community level, health seeking behaviour , especially in rural areas, indicates little awareness of the advantages which might follow treatment with specific antiepileptic drugs AED. Comparative studies indicate that the prevalence of active epilepsy is reasonably similar in most parts of the world , though incidence is higher in developing countries. The disparity between these two measures is likely to be due to higher mortality in poorly resourced regions rather than spontaneous improvement in the condition[2] .

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Estimates of epilepsy prevalence range in Asia from 5‰ to 10‰ and in Africa from 2‰ to 58 ‰ [1,3] , the wider disparity in Africa being explained by differences in method and sample size .

Epilepsy in sub-Saharan Africa SSA is mainly secondary , reflecting persistently high risks at birth, and the adverse neurological sequelae of viral, bacterial, malarial and other parasitic infections during and beyond childhood[1] . The lack of regional information on this important condition is noted by Preux and Druet Cabanac[3]. Even when epilepsy is recognised and treatment sought, the 'treatment gap'[4] is exacerbated by the sparsity of trained health personnel, the cost and difficulty of access to technical investigation and poorly sustained drug treatment.

The burden of epilepsy , especially heavy for the poor , is also borne by the families of those affected [2]. This survey, of children living in an isolated district in western Uganda , used empirical sampling methods and clinical seizure classification in order to quantify the local problem and to plan for appropriate and sustainable anti-epilepsy treatment via an existing programme. Falciparum malaria is seasonal in this highland area, and the study was carried out in dry season when transmission is low.

Onchocerciasis is endemic in some riverine areas of western Uganda. The first section of this report describes the prevalence and pattern of seizures in a virtually untreated child community.

Method

The survey was undertaken in Rukungiri District of Western Uganda , a volcanic region bordering the Democratic Republic of Congo , with a population density of <50 to 300 per square kilometre. Rukungiri's population < 15 years approximated 193,000 at the time [5], and the district was served by one government and two mission hospitals (no paediatrician). Epilepsy was thought to be widespread and under-treated, and it was hoped that a survey would clarify the situation, prior to integrating anti-epileptic treatment into an ongoing programme for disabled children (Christian Blind Mission CBM).

Visits to each of the 18 sub-districts were made at a date and time previously publicised by community leaders e.g. pastors, catechists, mosque leaders, primary school head teachers, chair-persons of sub-county and local councils, and community volunteers in the CBM programme. Local community leaders were visited and requested to announce at a religious or other meeting, the date and time of a conveniently sited assessment for children with epilepsy. Parents were encouraged to bring any children with recurrent seizures as well as children with movement difficulties, who were separately assessed. Vernacular terms for convulsions and episodic behaviour disturbance were used in these explanations. Assessment sessions were held in 19 government health centres or church halls from 18th August to 10th September 1997. Four hundred and forty of the 600 children who presented for assessment were given a diagnosis of epilepsy.

The assessment was directed at ascertainment of the frequency, duration and pattern of seizures, complication rate, and to estimate the number likely to benefit from regular and specific antiepileptic treatment. Further information on the adverse effects of epilepsy in these children will be separately reported (Duggan in preparation). Permission for the study was obtained from the acting Medical Officer of Health, and the majority of children have subsequently been treated by the outreach programme.

Each session began with a short talk on the home management of convulsions, including discussion of common misunderstandings e.g. that epilepsy is contagious etc. At the individual assessment, a vernacular (Rukiga) history was given by a parent, older sibling, or guardian in the case of orphans, while a few teenagers gave their own accounts. The person who accompanied the child and provided information is hereafter termed 'informant'. An experienced field worker translated and facilitated communication with the paediatrician/author . The history focused on a clear description of a typical seizure and its duration and frequency, plus information on the age of onset, progression or change in seizure pattern.

At the end of each day, information from each subject's paper record was reviewed to obtain a 'best fit' seizure classification (using the descriptive classification favoured by the Commission on Classification and Terminology of the ILEA 1981[6]) which was entered on a spreadsheet.

Criteria for the diagnosis of epilepsy were a clear description of recurrent and paroxysmal time-limited changes in motor activity or behaviour. Seizures types were classified and grouped according to the 1981 International Classification of Seizures[6,7] and summarised in Table 1. Neither EEG nor other investigations were available. State of consciousness was a major discriminator. When consciousness was preserved, seizures were described as partial (PS) except when accompanied by sensory, autonomic or psychic phenomena with impaired awareness. The latter, classified as temporal lobe syndrome (n=12), were grouped with complex partial seizures (CPS). CPS were otherwise characterised by partial onset followed by loss of consciousness at some time during the episode. As indicated in Table 1 it was sometimes difficult to distinguish between seizures which began locally or unilaterally and later became generalised (with definite loss of consciousness during each episode: termed partial to general) and a group (also classified as CPS) which remained localised or unilateral despite loss of consciousness. Problems with classification will be discussed later. The term 'generalised seizure' as used here was defined by loss of consciousness, accompanied by bilateral motor manifestations[6], and akinetic general seizures were grouped with miscellaneous seizures. Subtle phenomena such as automatic behaviour or colour change, and seizures occurring during sleep may have passed unnoticed. Short lasting febrile GTCS (n= 178) in children aged 6m to < 5 years were initially regarded as benign , fever associated seizures FAS.

Direct questions were later introduced concerning access to treatment, use of traditional medicine, and perception by informants of intellectual deterioration (some parents had spontaneously mentioned 'failure of understanding'). Some of these findings are separately reported . Family history was not specifically sought. After inspection for dysmorphia , the neurological examination included checking of tone and reflexes and coordination. Gait, hand-clapping and finger-nose touching were checked on older children and truncal and head control in infants. Of 618 children with recurrent fits, 600 were examined (18 had been diagnosed previously) and 440 given a diagnosis of epilepsy; the 178 children with presumed benign FAS were excluded from analysis.

Results

Prevalence and distribution by age and type of seizure

The 440 children ranged in age from < 1 to 18 years, though age was uncertain in 10. The majority (68% of all, and 64% and 71% respectively of boys and girls) were less than 10 years old. A modest male preponderance (58% boys and M:F ratio of 1:07) was reversed in children < 3years old (48% boys and M:F ratio of 1:1.08)

Table 1: Grouping and classification of seizures in Ugandan children according to the Commission for Classification and Terminology of the International League against Epilepsy ILAE 1981

Seizure Type	Summary of typical description on which the clinical classification was based
General Seizures :	Loss consciousness, plus one of the following:- a to e
a)Tonic-clonic	bilateral tonic-clonic seizures
b)Clonic	persistent stiffness of limbs constant feature
c)Akinetic	General 'weakness' or flaccidity described
d)Absence	Child unaware of surroundings but did not fall
e)Myoclonic seiz./ or West syndrome	One example only, typical single flexion spasm (blitzkrämpfe) , duration < 1 month, in a 6 month boy with history of excessive screaming at immunisation.
Partial Seizures	No loss of consciousness plus one of the following :-
Simple Partial Seizures	No loss of consciousness; typically involved a unilateral tonic-clonic seizure, sometimes accompanied by fever
Complex partial seizures	Despite localised onset, does involve loss of consciousness. The seizures were typically localised tonic –clonic which, although consciousness was reportedly lost, were not described as generalised. Cases of affective disturbance were classified as TLS (see below)
Seizures of TLS type classified with CPS	Loss of awareness, though consciousness maintained; features typical of temporal lobe seizures, typically with short episode of garbled speech or bizarre behaviour, sometimes starting with staring.
Partial to general seizures also classified with CPS	Started with localised tonic –clonic movement which progressed to general tonic clonic seizures (level consciousness not always specified)
Uncertain types	Seizures which varied in type from time to time or were difficult to classify

In table 2, thirty eight (8.6 %) presented with a similarly affected sibling. Information from the 1991 census for Rukungiri district (193,126 children < 15 years) together with our figure of 395 epileptic children < 15 years results in an age specific estimated prevalence of 2.04‰ (95% CI 1.94 ‰ to 2.24 ‰) .The geographical distribution of these 440 children suggests no clustering around the district capital.

The age distribution at presentation of children in different seizure groups is compared in Table 2, using the ILAE classification 1981[6] mentioned above. Nearly two thirds of children presented with generalised seizures of which 235 (53%) were typical GTCS.The CPS group (n=119) included 6 children (5 girls) with partial progressing to generalised TCS. The unexpectedly small number in this group will be discussed.

Thirty children (18 with GTCS, and 12 with SPS) were reported to be typically febrile during seizures > 15 minutes. The miscellaneous group (n=27) included 18 children with akinetic GS, 4 with absence seizures, 1 with possible infantile spasms, and 4 uncertain types.

Imprecision of histories and inability to investigate certainly limited diagnostic precision [7], see later discussion. The sex distribution pattern varied with seizure type, boys being overrepresented among the GTCS, and girls among the GTS (M:F ratio 1:0.49 and 1:1.67 respectively). The significance of these differences when expressed as a proportion of all children is given by Chi^2 = 47.7 and 6.76, $p<0.01$ and $P<0.05$, respectively).

Table 2: Age of presentation and age of onset of major syndromes of epilepsy in 440 rural Ugandan children (258 boys and 181 girls)

Age	**General Seizures Tonic-clonic, tonic:** includes fever associated GTCS		**Complex Partial Seizures:** includes partial to general and temporal lobe seizures		**Simple Partial Seizures:** includes fever associated SPS		**Miscellaneous Seizures:** myoclonic, akinetic, absence, breath-holding & unclassified	
	Presentation	Onset	Presentation	Onset	Presentation	Onset	Presentation	Onset
< 1year	2	125	5	57	2	9	2	12
1 to <5 yrs	95	91	38	38	15	10	4	3
5 to< 10 yrs	82	17	40	10	5	4	3	2
10 to< 15 yrs	56	11	27	3	5	2	7	0
15 to < 18yrs	25	14	6	3	0	0	3	3
age of presentation or onset not known	7	9	3	0	0	2	8	7
Total	267	267	119	111	27	27	27	27
(% of total)		61%		27%		6%		6%

Age of onset of seizures

The parents of 422 children remembered the approximate age of onset of seizures, giving 'rounded' years for children older than one year. Half (212 or 50.2%) had presented during infancy; a similar pattern being seen in most seizure types (115/230 for GTCS, 53/99 for CPS) except for GTS of which 10/28 (36%) presented in the first year (Table 4). The atypically early age[8] of onset of seizures defined as CPS will be discussed . There was no obvious gender difference in age of onset.

Associated conditions

Associated findings included clinical signs of cerebral palsy, global and motor developmental delay, and reported learning difficulties and behavioural disturbances. In the absence of documented past history, little can be inferred about the direction or causality of these associations. Of 28 children with definite signs of cerebral palsy, 18 had CPS, 3 GTCS, the remainder being distributed between GTS, SPS and akinetic S. Cerebral palsy (non-athetoid) was significantly more common in children with CPS (18/101 compared with 7/339 , Chi^2 =5095 with Yates Correction, $p<0.01$). The trend towards increased prevalence of developmental delay and learning difficulty in boys was not analysed due to small numbers and possible reporting bias (Table 3). Gross protein energy malnutrition was evident in 7 children, and moderate underweight confirmed by anthropometry in 5/68 (7%) girls and 26/106 (24%) boys <10 years. While significantly more boys were underweight (Chi^2 = 8.55 with Yates correction $p=<0.05$), selection bias due to haphazard access to weighing equipment could not be excluded.

Information about illness in early life was recorded for 74 children, of whom 41 had suffered from perinatal and 29 from severe illness later in the first year (Table 3) . Illness at the time of the first seizure had not always been treated in hospital. Illness in infancy was recorded for only 3 of the 28 children with signs of cerebral palsy.

On inspection of the data, but without structured enquiry, it was noted that 38/440 children had a similarly affected sibling. Three others had recognised syndromes :- Seckel's , San Filippo's and Down's syndrome respectively, and two others were dysmorphic with unilateral microphthalmos and bilateral small ears.

Table 3: Associated clinical findings or past history in 440 Ugandan children with epilepsy

Clinical Observations		
Cerebral palsy	Hypertonic hemi-or quadriplegia	25
	Signs indicative of evolving CP	7
	Athetotic CP	3
Other neurological problems		
	Global developmental delay	9
	Motor developmental delay	15
	Perceived learning difficulty	64
	Deafness	7
	Gross visual impairment	3
Past History available on 74 children		
Positive neonatal history	Prolonged labour	10
	Small size at birth	11
	Symptoms suggestive of hypoxic ischaemic brain damage	10
	Miscellaneous neonatal problems	7
	Neonatal jaundice ?kernicterus	3
Description of severe illness at time of first seizure		
	Coma accompanying measles, malaria or meningitis	11
	Pneumonia or other severe febrile illness	15
	Oedematous malnutrition	2
	Possible adverse reaction to immunization	1

The changing pattern of seizures

The seizure pattern had not always remained constant over time. For example, although no one was currently suffering from infantile spasms, the past history in seven , now exhibiting a range of general and partial seizure types, was strongly suggestive. Two children currently suffering from GTCS had a history of absence seizures, 2 had histories suggestive of temporal lobe syndrome , while in 2 others, there was a past history of day-time terror attacks.

Discussion

To be effective, strategies to combat (secondary) epilepsy in sub-Saharan Africa should be three-pronged. It is necessary to quantify both prevalence and also those infectious or other exposures amenable to prevention , as well as to embark on effective and sustainable treatment for those already affected Estimates of prevalence, important for advocacy, should note geographical variations in prevalence which might indicate local differences in quality and access to maternal and child health services, or different risks of exposure to infection or parasitosis. Thirdly, the effective treatment of those already affected will be facilitated by precise diagnosis of seizure type. Some of these issues are addressed below[3].

The house to house census (door to door survey) is regarded as the 'gold standard' for estimating the community prevalence of epilepsy, though an even higher estimate is obtained when other methods of case 'capture' are used in conjunction. The validity of the relatively unstructured method used in the present survey depended on the families of affected subjects having access to information about the assessment, and on

their capability and motivation to attend at the right place and time. Such factors , together with the stipulation of 'recurrent' seizures, would favour inclusion of the more seriously affected. Nevertheless, the 395 children definitely <15 years represented 2.04‰ (95% CI 1.94 ‰ to 2.24 ‰) of the estimated < 15 years population . Though this is much lower than a recent age-specific estimate in Uganda[9,] (16.8 ‰; 95% CI 16.8 ‰ to 18.02‰ , calculated from the authors' data) and incidence data on young Kenyan children[10] , it is close to the age specific estimate (2.4‰) for epilepsy prevalence in a large Gambian series [11], which used a modification of the census method.

There were no facilities to investigate disease associations which might have given useful clues to aetiology, and historical enquiry for risks in perinatal and early life was incomplete. A positive correlation between the prevalence of epilepsy and filariasis (specifically onchocerca volvulus), has been reported elsewhere [9,12] , but without pathological findings , or a plausible mechanism, evidence of causality remains circumstantial[3]. Neurocystocercosis is known to result in secondary epilepsy , and pig-rearing methods in some parts of SSA might favour transmission of T solium. But, when unconfirmed by CT or specific brain pathology, evidence is inconclusive[3]. A lack of association was reported in western Uganda between positive serology for T solium and epilepsy[9]. Though pigs were reared locally, the early age of onset (mean age 2.7 years and onset in infancy in 50.2 %), does not support a causal role for neurocystocercosis. Nor did we observe the bi-modal distribution of age of onset thought to be a marker for epilepsy associated with parasites, particularly onchocerciasis[13]

These children would , however, have been at risk of seasonal malaria, which causes seizures via several mechanisms. The diffuse encephalopathy of cerebral malaria is characterised by coma or repeated seizures, and survivors commonly exhibit signs of permanent brain damage[10]. The fever of uncomplicated malaria is often accompanied by short GTCS in young children , similar to but arguably less benign than classical 'fever associated seizures' . A high prevalence of CPS, often local in onset, has been reported in young, sometimes afebrile, Kenyan children with malarial parasitaemia[10]. While little mention was made of the recurrence rate of these 'malaria associated CPS', they resemble the 'complex febrile seizures', common in SSA, known to progress to later epilepsy [3] . Notwithstanding the absence of an evidential link with malaria in the Rukungiri children, it is reasonable to postulate that some CPS may have had an aetiology similar to the Kenyan series , especially in view of their similar partial onset in very young children in an area endemic for malaria.

On reviewing records of the 178 children excluded with 'benign' FAS, whose parents had been sufficiently convinced of the gravity of the illness to seek help, we now consider that such a 'categorical' exclusion may not be appropriate in areas endemic for malaria. Furthermore, these 178 records demonstrated considerable overlap with those of 30 children with FAS, included by virtue of the frequency and severity of seizures. In retrospect we would now reclassify these 208 children , since complex or (malaria associated) febrile seizures carry a heightened risk of progression to long-term epilepsy.

Gender ratio, suspectibility to primary epilepsy

The M:F ratio for all seizures was 1:0.7, similar to that reported in African adults[3]. The predominant seizure type was GTCS, in common with reports elsewhere in SSA[3,14,15] and beyond [8] . Classification was based on the current and most common seizure type, although inconstant seizure pattern or progression from one to another seizure type were both noted (see below) . More structured history might have revealed evidence of familial susceptibility (we merely recorded 43 children , of whom 38 had similarly similarly affected siblings, while 3 showed definite and 2 suggestive dymorphia). Until genetic technology is available and accessible , the importance of primary epilepsy is unknown.

Perinatal and infantile risk factors

With respect to a perinatal or infantile precipitating cause of secondary epilepsy, we found circumstantial evidence in less than a fifth of children:- a perinatal problem in 41(9.3 %) and severe illness in the first year in 29 (6.5%) . By comparison Leary et al [16] identified a precipitating cause in 43% of peri-urban South African children. The number of relevant early life exposures was probably underestimated due to methodological inadequacy and to 'recollection bias'[3] .

Lack of access to EEG and or other investigations hampers both syndromic diagnosis and ability to demonstrate secondary aetiology .

Availability of relatively simple resources such as EEG, occasional CT and past hospital records facilitated clarification of the pattern of epilepsy in the Western Cape[16]. However, clinical skills may still be put to use. Whereas WHO[2] emphasises the inequality of access to investigations in SSA, Dekker[14] optimistically lays out a logical investigation strategy for resource poor situations, emphasising the role of history in building up a syndromic diagnosis rather than simply defining seizure type. This problem-oriented approach facilitates decisions about drug treatment which are not entirely driven by questions of availability and affordability[15] . The simplicity of the method also favours its use by paramedical cadres, although the possible misclassification of some partial to general seizures in the present series, suggests that misreporting may be an issue in community studies.

Strategies to prevent secondary epilepsy coincide with other strategies to improve maternal and child health e.g. improved access to medically supervised childbirth[1] and prevention of mosquito bites[18]. Even though perinatal causes did not feature strongly in this series, they may have played a role in early onset seizures. The possible link with malaria needs more structured epidemiological study. Wherever possible attention should be paid to 'coincidental' malarial parasitaemia in (non febrile) children with seizures, and follow up of children with malaria associated seizures should become standard practice . The implications of 'FAS' in areas endemic for falciparum malaria should also be reviewed.

The third suggested prong to an effective strategy for combating epilepsy is appropriate and sustainable treatment. Very few of these children had received other than symptomatic treatment for convulsions, often with traditional medicines (78% of the 79 specifically questioned). Strategies for AED treatment will be easier to plan when management of childhood epilepsy is integrated into child health care.

Conclusion

The estimated age-specific prevalence is lower than some Ugandan estimates, but the pattern of childhood epilepsy in Rukungiri is similar to previous reports from SSA. Historical inaccuracies probably led to underreporting of partial to general seizures or to their inclusion with other CPS. The likelihood of a causal association between malaria and CPS and other FAS seizures of early onset is discussed . Associations with cerebral palsy and parinatal and early life illness are noted. Few children had previously received antiepileptic treatment . While emergency treatment of convulsions is integrated into the management of childhood illness [17], the same is not true for the long-term management of epilepsy, too often regarded as a psychiatric responsibility. Until a sub-specialty of paediatric neurology is sufficiently established , should childhood epilepsy not come under the umbrella of child health?

Acknowledgements

The study was carried out on a voluntary basis and a report was submitted to Christian Blind Mission in 1997, via Miss Pat Gilmer of South Kigezi Diocese Rukungiri , at whose request the project was undertaken and whose skill in planning and organising the clinical sessions is gratefully acknowledged.

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