Active convulsive epilepsy in a rural district of Kenya: a study of prevalence and possible risk factors

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Summary

Background

Few large-scale studies of epilepsy have been done in sub-Saharan Africa. We aimed to estimate the prevalence of, treatment gap in, and possible risk factors for active convulsive epilepsy in Kenyan people aged 6 years or older living in a rural area.

Methods

We undertook a three-phase screening survey of 151 408 individuals followed by a nested community case-control study. Treatment gap was defined as the proportion of cases of active convulsive epilepsy without detectable amounts of antiepileptic drugs in blood.

Findings

Overall prevalence of active convulsive epilepsy was 2·9 per 1000 (95% CI 2·6—3·2); after adjustment for non-response and sensitivity, prevalence was 4·5 per 1000 (4·1—4·9). Substantial heterogeneity was noted in prevalence, with evidence of clustering. Treatment gap was 70.3% (65·9—74·5), with weak evidence of a difference by sex and area. Adjusted odds of active convulsive epilepsy for all individuals were increased with a family history of non-febrile convulsions (odds ratio 3·3, 95% CI 2·4—4·7; p<0·0001), family history of febrile convulsions (14·6, 6·3—34·1; p<0·0001), history of both seizure types (7·3, 3·3—16·4; p<0·0001), and previous head injury (4·1, 2·1—8·1; p<0·0001). Findings of multivariable analyses in children showed that adverse perinatal events (5·7, 2·6—12·7; p<0·0001) and the child's mother being a widow (5·1, 2·4—11·0; p<0·0001) raised the odds of active convulsive epilepsy.

Interpretation

Substantial heterogeneity exists in prevalence of active convulsive epilepsy in this rural area in Kenya. Assessment of prevalence, treatment use, and demographic variation in screening response helped to identify groups for targeted interventions. Adverse perinatal events, febrile illness, and head injury are potentially preventable associated factors for epilepsy in this region.

Introduction

WHO estimates that of the 50 million people with epilepsy in the world, 80% live in developing countries,[1] but this estimate is based on data of few studies. Findings of a review of epilepsy in sub-Saharan Africa[2] suggested that small studies provide widely varying and imprecise estimates of disease burden, whereas large studies might underestimate prevalence owing to under-reporting from stigma. Furthermore, definitions of epilepsy vary across studies, as do methods for screening and data collection.[3] Door-to-door surveys are thought to be the most effective screening method in areas with poor resources for capture and maintenance of medical history. In such studies, researchers detect mainly convulsive epilepsies because of scarce resources and low awareness of non-convulsive epilepsy. Nine case-control studies aiming to ascertain possible risk factors have been reported from sub-Saharan Africa, with limited investigation of confounding effects.[2] Many people with epilepsy in this region do not access treatment.[4]
We undertook a large screening survey in a rural malaria-endemic area of Kenya. We aimed to estimate the prevalence of, treatment gap in, and possible risk factors for active convulsive epilepsy in individuals aged 6 years and older.

Methods

Study setting

We did the study in the Kilifi District, a rural area on the coast of Kenya; the Kilifi District Hospital is located in the administrative centre. An area of 891 km2 has been mapped and forms part of a demographic surveillance system. Re-enumeration of the population is done every 4 months (births, deaths, and migration). The study area is divided administratively into six divisions (Malindi, Bahari, Vitengeni, Ganze, Chonyi, and Kikambala) and within these divisions into 15 locations, 40 sublocations, and 185 enumeration zones (figure 1). Mijikenda are the indigenous people of coastal Kenya and consist of Giriama, Chonyi, and Kauma ethnic groups and some other small groups. Kilifi is the second poorest district within Kenya;[5] literacy in adults is low, access to sanitation facilities is poor, and subsistence farming is the main source of income. Life expectancy is 57 years for women and 51 years for men.[5] Malaria is endemic, with two peaks in transmission from May to August and December to January.[6] Infectious diseases such as malaria, pneumonia, and bacteraemia are typical causes of admission to Kilifi District Hospital.[7], [8]

Figure 1 Full-size image (105K) Download to PowerPoint
Study area

Enumeration zones and boundaries of administrative divisions in the Kilifi District, Kenya, showing prevalence (per 1000) of active convulsive epilepsy, adjusted for age and sex.
The national ethics review committee of Kenya and ethics committees of the Institute of Child Health and London School of Hygiene and Tropical Medicine in London, UK, approved the study. We obtained written informed consent from participants. In the case-control study, cases were consenting survey participants with active convulsive epilepsy.

Procedures

We did a three-phase door-to-door survey during population re-enumeration between August and November, 2003. In phase 1, the census team, which consisted of field workers supervised by EB and AGS, asked the head of every household two questions about every person within the household, to identify those who had had convulsions (see webpanel). When parents were interviewed in a previous study in this area,[9] the second question was 100% sensitive for detection of active epilepsy in children. In phase 2, the epilepsy field team, which consisted of field workers supervised by VMO, visited individuals identified in phase 1 and interviewed them or their guardians (see webpanel). In phase 3, people suspected of having epilepsy in phase 2 were invited to attend for formal assessment and diagnosis at Kilifi District Hospital within 1 week of the phase 2 interview. One clinician (EC) who was fluent in the local languages Kigiriama and Kiswahili obtained a detailed medical history from which to make a diagnosis and record a description and frequency of seizures. A panel of neurologists (TK, GM, BGN, LJS, and CRN) reviewed case notes to confirm diagnoses.
We defined active convulsive epilepsy as two or more unprovoked convulsions, with one convulsion taking place within 12 months before phase 3. Our definition was based on the most recent International League Against Epilepsy classification of active epilepsy at the time of study design and on criteria for offering antiepileptic drugs to patients in Kenya.[10], [11] Children younger than 6 years were excluded in phase 2 because of difficulties in differentiating between febrile seizures and epilepsy in infants.[12]
We asked individuals with epilepsy if they were currently taking or had previously taken any antiepileptic drug, and we showed them actual tablets to aid recognition. We took a blood sample (from those who gave consent) to test for phenobarbital—the only antiepileptic drug available in peripheral clinics. Samples were also screened for phenytoin if use of this agent was reported. Drug amounts were measured with a fluorescence polarisation immunoassay (TDxFLx; Abbott Laboratories, Abbott Park, IL, USA), which can detect concentrations of at least 10 mg/L of phenobarbital and phenytoin. For study reasons, optimum concentrations of phenobarbital and phenytoin were delineated as 10—30 mg/L and 10—20 mg/L, respectively.[13] We defined seizure treatment gap as the proportion of people with active epilepsy whose seizures were not being treated appropriately (including diagnosis and therapeutic treatment).[10]
To control for confounding and to minimise recall bias, we randomly selected controls from the demographic surveillance system and frequency-matched them to people with active convulsive epilepsy by age, in five strata: 6—12 years, 13—17 years, 18—28 years, 29—49 years, and 50 years or older. Data for children were usually obtained from parents. Recall of previous events can decrease with age, and ascertainment of true age can be difficult for old adults in a rural population. For people who did not know their date of birth, age was related to major national or local events.

We asked participants and guardians about convulsive seizure history in their first-degree relatives (siblings and parents) and extended relatives, febrile seizures in all relatives, and serious head injury. We classified early seizures with fever and full recovery as febrile seizures. For children younger than 18 years, mothers were questioned about adverse perinatal events (history of prolonged labour and postnatal difficulties such as convulsions in the first week of life or difficulties in establishing breathing or breastfeeding).[14] We recorded sociodemographic factors, including sex, ethnic group, and location of residence.

Statistical analysis

We double-entered data and verified them with Visual FoxPro version 9.0 (Microsoft, Reading, UK). Statistical analyses were undertaken with STATA version 8 (StataCorp, College Station, TX, USA). We calculated unadjusted prevalence and exact binomial 95% CIs overall and by age and sex, per 1000 people. We used the $\chi 2$ test to measure associations between sociodemographic characteristics and overall prevalence, and with adherence to antiepileptic drugs in diagnosed cases. Adjusted prevalence by geographical area was derived at enumeration zone, sublocation, and location level from a binomial regression model, adjusted for age and sex, to examine evidence of heterogeneity. We estimated sensitivity and specificity[15] of phase 2 screening questions and self-reported antiepileptic drug use to ascertain current use of antiepileptic drugs.

We used logistic regression to investigate possible individual risk factors for active convulsive epilepsy, after adjustment for frequency-matched age variable.[15] Individuals with complete data were included in a multivariable regression model, using a forward stepwise strategy (inclusion $p<0{\cdot}1$ and exclusion $p>0{\cdot}1$) for factors relating to medical histories. Adjustment was also made for underlying sociodemographic variation (identified by overall prevalence analyses) to enhance model fit. We did a subgroup analysis in children (age <18 years) to investigate the effect of perinatal events, using the same multivariate modelling strategy. Regression models were compared with the likelihood ratio test.

Role of the funding source

The sponsor of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

In phase 1, household heads provided responses for 151 408 residents age 6 years and older. Non-response in phase 2 was not associated with age ($p=0{\cdot}7321$) or sex ($p=0{\cdot}2526$) but was higher in non-coastal ethnic groups and Malindi, Bahari, and Kikambala divisions ($p<0{\cdot}0001$). Non-response in phase 3 was associated weakly with age ($p=0{\cdot}0751$) and strongly with division ($p<0{\cdot}0001$; higher in Bahari and Kikambala), but it was not related to sex ($p=0{\cdot}9854$) or ethnic origin ($p=0{\cdot}5627$). A diagnosis of active convulsive epilepsy was made in 445 individuals, 442 directly and in three people after detection of antiepileptic drugs in a blood sample. Figure 2 shows the numbers of people screened during the three-phase survey.

Figure 2 Full-size image (25K) Download to PowerPoint
Screening survey flow
*Individuals 6 years and older resident during the previous census round. †Defined as two or more unprovoked seizures, with the most recent within past 12 months. ‡History of epileptic seizures. ¶Confirmed by testing of blood sample.

Of 1283 participants deemed negative after phase 2, 1174 were re-interviewed; 70 were identified as positive and attended phase 3 assessment. In addition to 442 diagnoses made by phase 3 assessment of phase 2-positive individuals before blood testing, a further 24 people with active convulsive epilepsy were identified who were originally phase 2 negative. Sensitivity and specificity estimates were 94·8% (442/466; 95% CI 92·4—96·7) and 52.3% (46/88; 41·4—63·0), respectively.

Overall prevalence of active convulsive epilepsy was 2·9 per 1000 people (95% CI 2·6—3·2). Unadjusted prevalence varied with age (p=0·0002) and sex (p=0·0684), with a higher prevalence in males and in people age 13—28 years (table 1). Little evidence was seen for a difference by ethnic origin (p=0·1586). Based on a 5-year cut-off for date of last seizure, unadjusted prevalence of active epilepsy was estimated as 3.1 per 1000 (95% CI 2·8—3·4). Taking non-response and phase 2 sensitivity into account, the estimated prevalence of active convulsive epilepsy was 4·5 per 1000 (4·1—4·9), assuming 100% sensitivity in phase 1 (see webappendix).

Table 1Table image
Unadjusted prevalence of active convulsive epilepsy by age and sex

The median number of eligible individuals screened in phase 1, per enumeration zone, was 959 (IQR 679—1275). After adjustment for age and sex, strong evidence was seen of heterogeneity in prevalence of active convulsive epilepsy across zones within the large study area (p<0·0001). Heterogeneity was also present at location and sublocation levels (p<0·0001). Figure 1 shows adjusted prevalence by enumeration zone.

Of 445 people with active convulsive epilepsy, 408 (92%) gave consent for blood samples to be taken. Detectable amounts of antiepileptic drugs were recorded in 132 samples and optimum concentrations in 63, providing minimum treatment gap estimates of 70·3% (95% CI 65·9—74·5) and 85·8% (82·3—88·9), respectively. Age was associated with non-consent for blood samples, with children younger than 12 years less likely to provide a sample (p<0·0001). Sex (p=0·4312), ethnic origin (p=0·3292), and location (p=0·3529) were not associated with consent. Current use of any antiepileptic drug was self-reported by 127 people for phenobarbital and 121 for phenytoin. Self-reported and blood sample results were concordant in 307 individuals—76 for antiepileptic drugs and 231 without drug use. Sensitivity and specificity of self-reporting were 57·8% (95% CI 48·7—66·1) and 86·7% (78·8—87·9), respectively. Evidence was weak for an association between male sex and adherence to antiepileptic drugs (p=0·0568) and between non-adherence and division (p=0·0735), and no evidence was noted for a link with age (p=0·1267), ethnic origin (p=0·3907), or seizure frequency (p=0·3271).

Recruitment of controls was based on immediate results from phase 2. As a result, the number of controls exceeded the number of cases; however, frequency matching was upheld (table 2).

Analyses of potential individual risk factors were adjusted for age. A slight geographical association with odds of active convulsive epilepsy was recorded. Data suggested increased odds of active convulsive epilepsy with previous head injury and with a family history of febrile seizures or convulsions in individuals of all ages (table 2). An interaction was recorded between history of febrile convulsions and convulsive seizures (p=0·0076), when assessed in four categories (table 3). The final multivariable model was adjusted for these factors and underlying demographic characteristics (sex, division, and ethnic origin), to obtain a better model fit to the data.

Table 2Table image
Age-adjusted odds ratios for sociodemographic and medical history associated factors

Table 3Table image
Possible risk factors for active convulsive epilepsy from final multivariate models

In the final multivariate model in children younger than 18 years, adverse perinatal events and mother's marital status were identified as possible risk factors, after adjustment for seizure histories and sociodemographic factors (table 3), with higher odds of active convulsive epilepsy if mothers were widowed rather than married. After adjustment for perinatal event, head injury was no longer significant in children (p=0·1610). In both final models, family history of convulsions was collapsed into a binary variable for increased power and the interaction term was included, with results suggesting increased odds of active convulsive epilepsy in people with a history of either febrile or non-febrile convulsions, or both types, compared with no history of either.

Discussion

Estimates from our large community survey of epilepsy in a rural Kenyan district show overall prevalence of active convulsive epilepsy to be 4·5 per 1000, adjusted for non-response and screening sensitivity. Findings of a previous study, undertaken when the demographic surveillance survey was a third the size it is today, estimated a similar prevalence of this disorder and suggested that 3·5% of deaths in a 2-year period were epilepsy related.16 We also noted strong evidence of heterogeneity of prevalence across small geographical areas, suggesting clustering of people with active convulsive epilepsy. Our findings highlighted differences within small divisions in response rate and awareness of epilepsy—outcomes that were suspected by other researchers but not supported by available data until now.2 Heterogeneity of prevalence could be attributable to clustered exposure to environmental or genetic associated factors. Access to medical care could also account for clustering, since people living closer to the Kilifi District Hospital are treated more frequently than are those living further away,17 although visual inspection of the prevalence map indicated no gradient surrounding the hospital.

Findings of other large studies in sub-Saharan Africa, surveying more than 15 000 people, have noted the prevalence of active convulsive epilepsy to range from 5·2 to 12·5 per 1000.18—20 In a further three studies that included non-convulsive epilepsy, prevalence was 10·2—18·2 per 1000.20, 21 Estimates from small studies are especially variable.2 Cross-sectional prevalence

estimation is thought to underestimate the frequency of life-time epilepsy by more than 75%.[22] Non-convulsive epilepsy can constitute 50% of all epileptic disorders in community-based studies.[22] Therefore, the life-time prevalence of epilepsy in the Kilifi District could be more than double the prevalence of active convulsive epilepsy measured in this study.

Our findings also indicated a negligible difference in prevalence estimates based on a 1-year or 5-year cut-off for most recent seizure, justifying cautious comparison of findings of large published studies using either definition. Complexities of comparisons between studies are probably affected more by difficulties in capture of individuals at risk during early screening phases and identification of causes than by differences in definitions of active epilepsy.

We recorded high prevalence of active convulsive epilepsy in adolescents and young adults. Findings of studies from Europe and the USA have shown peaks in incidence of epilepsy in young children and elderly people (>65 years).[22, 23] Inter-regional comparisons without comparable age standardisation are not so informative since only 3% of our study population was age 65 years or older. However, variation in prevalence by age and sex in the Kilifi District—with high prevalence in adolescents and young adults and low prevalence in adult women—is similar to that seen in other studies in rural sub-Saharan Africa.[2, 24] A fairly low prevalence of active convulsive epilepsy during adult life could be attributable to spontaneous remission, although it might also indicate that people with epilepsy die prematurely, as reported in other developing countries.[16, 25]

The treatment gap was substantial in the group of people we studied, but it was slightly lower than that reported in other areas of sub-Saharan Africa.[4, 26] The treatment gap based on self-reported antiepileptic drug use would have been similar to published results, but the low sensitivity of self-reported use highlights a need for intervention studies to increase awareness of epilepsy and treatment options. Phenytoin use could have been underestimated in our study since we only tested blood samples in 2% of people who reported its use because of financial limitations, although phenytoin is not widely available in Kenya. Few children gave consent for blood testing and parents reported low use of antiepileptic drugs.

We only detected convulsive epilepsies in our study; identification of these epileptic disorders is a priority in sub-Saharan Africa because they are associated with more comorbidity, injury, and mortality than are non-convulsive epilepsies. Detection of non-convulsive epilepsy is difficult and would need detailed medical anthropological studies to ascertain symptoms such as staring episodes and hallucinations.

In our three-phase survey, phase 1 aimed to identify individuals having convulsions with high sensitivity, thereby efficiently screening a large population with only limited interview time available during the census enumeration. With phase 2 we aimed to confirm that people identified in phase 1 had had convulsions and ascertain the timing of recent seizures with high specificity. The questions in both surveys (see webpanel) should be sufficient for us to identify any people having convulsions, provided individuals wish to divulge the relevant information. Negative responses should only be attributable either to lack of awareness of convulsions in family members on the part of the household head or to stigma-related bias, which is regarded as the most likely reason for negative responses. An assumption of 100% phase 1 sensitivity leads us to the most conservative prevalence estimate (see webappendix). Stigma is known to be widespread within communities in our study area[12] and even within health-care workers in developing countries.[12, 27] An important reason for high non-response could be beliefs that epilepsy is caused by bewitchment so treatment with modern medicine is inappropriate.[12, 27] The high sensitivity for phase 2 suggests that, in those reporting convulsions, the questions we

asked are very effective for detection of people with convulsive epilepsies, although these results are also susceptible to stigma-related bias.

After controlling for underlying demographic variation, a family history of febrile or non-febrile convulsions and previous head injury in adults were important, preventable, associated factors. In children, perinatal events and their mother being a widow were possible risk factors. We postulated that mother's marital status as a sociodemographic variable could be a marker for death of the child's father, which in turn could be a marker for increased poverty. The nature and possible causality—based on which status came first—of the relation with active convulsive epilepsy clearly needs further investigation, since currently to interpret this finding with any conclusive meaning is difficult.

Of nine case-control studies in sub-Saharan Africa, only two used multivariate methods to control for confounding; findings of these two studies identified febrile and convulsive family seizure history and perinatal events as possible risk factors for epilepsy.[2] In our study, interviewers tried to distinguish between convulsive epilepsy and febrile convulsions in family histories, but there was probably dependence in many cases owing to long recall. This factor could account for the unusual interaction noted in which, compared with no history of either febrile or non-febrile convulsions, odds of active convulsive epilepsy were higher for people with a history of only febrile convulsions than for those with a history of both types, when we might have expected that an increase in odds would be highest in individuals exposed to both types of convulsive history. Also, low power for investigating these effects in our sample led to low precision. Estimates from a case-control study in Indian children noted similar odds ratios to ours for age-adjusted effects of febrile illness and first-degree family history of convulsive seizures.[28]

Epilepsy is most prevalent in adolescents and young adults in this rural area of Kenya, with considerable geographical variation in prevalence. Intervention is needed to increase awareness of epilepsy as a treatable disorder and to augment treatment-seeking in rural areas to detect people in need. Identification of adverse perinatal events, febrile illness leading to seizures, and head injury as possible risk factors suggests that much epilepsy is preventable.

Contributors

AGS, VMO, TK, LWS, BGN, and CRN contributed to the idea and design for the study. GM, VMO, EC, EB, and CRN implemented the study. GM, VMO, and CRN managed the study. TK, AGS, and CRN analysed data. TK, BGN, and CRN drafted the report. AGS, GM, VMO, TK, and LWS reviewed the report. All authors read and approved the final version of the report.

Conflict of interest statement

We have no conflicts of interest.

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Web Extra Material

Webappendix

PDF (58K)
Formulae for adjusted prevalence estimate and corresponding standard error
Webpanel

PDF (35K)
Screening questions

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