



## Epilepsy, poverty and early under-nutrition in rural Ethiopia

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### ARTICLE INFO

#### Article history:

Received 14 March 2012

Received in revised form 29 July 2012

Accepted 9 August 2012

#### Keywords:

Epilepsy  
Malnutrition  
Africa  
Anthropometry  
Education

### ABSTRACT

**Purpose:** The incidence of epilepsy in Ethiopia is high compared with industrialised countries, but in most cases the cause of epilepsy is unknown. Childhood malnutrition remains widespread. We performed a case–control study to determine whether epilepsy is associated with poverty and markers of early under-nutrition.

**Methods:** Patients with epilepsy ( $n = 112$ ), aged 18–45 years, were recruited from epilepsy clinics in and around two towns in Ethiopia. Controls with a similar age and gender distribution ( $n = 149$ ) were recruited from patients and relatives attending general outpatient clinics. We administered a questionnaire to define the medical and social history of cases and controls, and then performed a series of anthropometric measurements. Unconditional logistic regression was used to estimate multivariate adjusted odds ratios. Multiple linear regression was used to estimate adjusted case–control differences for continuously distributed outcomes.

**Results:** Epilepsy was associated with illiteracy/low levels of education, odds ratio = 3.0 (95% confidence interval: 1.7–5.6), subsistence farming, odds ratio = 2.6 (1.2–5.6) and markers of poverty including poorer access to sanitation ( $p = 0.009$ ), greater overcrowding ( $p = 0.008$ ) and fewer possessions ( $p < 0.001$ ). Epilepsy was also associated with the father's death during childhood, odds ratio = 2.2 (1.0–4.6). Body mass index was similar in cases and controls, but patients with epilepsy were shorter and lighter with reduced sitting height ( $p < 0.001$ ), bitrochanteric diameter ( $p = 0.029$ ) and hip size ( $p = 0.003$ ). Patients with epilepsy also had lower mid-upper arm circumference ( $p = 0.011$ ) and lean body mass ( $p = 0.037$ ).

**Conclusion:** Epilepsy in Ethiopia is strongly associated with poor education and markers of poverty. Patients with epilepsy also had evidence of stunting and disproportionate skeletal growth, raising the possibility of a link between early under-nutrition and epilepsy.

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## 1. Introduction

Malnutrition early in life has irreversible effects on the development of the brain, and it has been suggested that early under-nutrition may increase susceptibility to seizures later in life.<sup>1</sup>

There is some support for this hypothesis from animal studies. Neuronal loss has been reported in the cerebral cortex of adult rats exposed to mild early malnutrition and subsequently rehabilitated.<sup>2</sup> Further work has shown that the hippocampal formation

seems particularly vulnerable to early malnutrition in rats with reductions in density of dentate granule, hilar, CA1 and CA3 pyramidal cells.<sup>3</sup> Malnutrition has also been shown to influence neurogenesis in the dentate gyrus of immature rats.<sup>4</sup> Such structural changes could potentially increase susceptibility to seizures, and indeed protein malnutrition during development in rats has been linked with an increase in susceptibility to seizures in adulthood.<sup>5</sup> Furthermore, early malnutrition increases susceptibility to seizures in several animal models of epilepsy including hippocampal kindling,<sup>6</sup> kainic acid,<sup>7</sup> and pentylenetetrazol induced seizures.<sup>8</sup>

Although there is a considerable amount of evidence from animal studies which supports a link between early malnutrition and seizures, data from clinical studies are limited, and the relationship between malnutrition and epilepsy remains inadequately explored. In 1997 Hackett reported that a sample of 26

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children with epilepsy had a lower body mass index (BMI) compared with controls.<sup>9</sup> Subsequently a more detailed study performed in West Africa found that the prevalence of malnutrition, defined as BMI < 18.5 kg/m<sup>2</sup>, was higher in people with epilepsy compared with controls.<sup>10</sup> This study was, however, performed in a small rural area in Benin, and it is not clear that this association applies elsewhere. In addition, BMI is primarily a marker of nutritional status at the time of measurement and it was, therefore, not possible to determine whether malnutrition preceded or was a consequence of the epilepsy. A longitudinal cohort study might answer this question but would not be feasible in a resource poor setting lacking good quality medical records. As there is frequently a latent period between a cerebral insult and the onset of epilepsy, an alternative approach is to look for evidence of early malnutrition in patients with epilepsy. Malnutrition in early life leads to abnormal skeletal growth, and stunting and skeletal disproportion are potential markers of early under-nutrition.<sup>11</sup>

The incidence of epilepsy is high in Ethiopia compared with industrialised countries, and in most cases the cause of the epilepsy is unknown. Excluding family history, risk factors for epilepsy are found in only 10–14% of patients.<sup>12,13</sup> Pigs are not reared in Ethiopia and neurocysticercosis, the most common cerebral infection causing epilepsy in low income countries, is thought to be rare. Ethiopia is one of the poorest countries in the world and childhood malnutrition remains widespread. The purpose of this study was to determine whether there is a link between poverty, markers of early under-nutrition and epilepsy in Ethiopia.

## 2. Materials and methods

We performed a case–control study to investigate possible associations between poverty, markers of early malnutrition and epilepsy in Ethiopia. The study was based in Gondar and Jimma, 750 km north-west and 330 km south-west of the capital Addis Ababa, respectively. These two Ethiopian towns were chosen as they have well established epilepsy treatment programmes in which patients attend both the university hospitals and satellite rural health centres.

Patients aged 18–45 years with a history of two or more epileptic seizures in a period of more than 24 h were recruited from epilepsy clinics in the university hospitals in each town as well as from rural epilepsy clinics associated with these hospitals. The diagnosis of epilepsy was based on the history from the patient and a witness, and was confirmed by a physician with expertise in epilepsy.<sup>14</sup> All had a history of tonic–clonic seizures but, due to lack of facilities for EEG, accurate classification of seizure type and syndromic classification were not possible and all types of epilepsy were included. Consecutive attendees were recruited to avoid selection bias.

Controls were recruited on the basis of age and gender distribution from patients or relatives of patients attending the general walk-in clinics. Controls were recruited at each hospital and health centre that cases were obtained from so that they would be likely to be from the same geographical area and to have reached the health facility through similar referral processes. Fifty of the controls were patients attending outpatient clinics with minor medical complaints: gastrointestinal (14), respiratory (11), musculoskeletal (4), otolaryngology (4), genito-urinary (3), and miscellaneous (14). Ninety-nine of the controls were people accompanying patients to the outpatient clinics.

Cases and controls were only included if they were able to give informed consent, thus excluding patients with significant learning difficulties. We also excluded cases and controls with any impairment of either speech or gait, so as to avoid including subjects with significant neurological disability that might impair

feeding. Pregnant women were excluded from the study because of the impact that pregnancy would have on anthropometric measurements. Controls were excluded if there was any history of seizures.

Verbal and written consent was obtained by a local nurse in each study site, who spoke either Amharic or Oromifa, the local languages. Each study participant was asked to complete a structured verbal questionnaire in either Amharic or Oromifa and then undergo a series of anthropometric measurements. The interviewers were not blinded to the condition of cases and controls. The methods used were similar to those described in a previous study in patients with diabetes.<sup>15</sup>

Cases were asked about their epilepsy and treatment history, and controls were asked about the reason for attendance at the hospital and questioned regarding their medical history. Detailed information about education, occupation, housing, source of drinking water and sanitation was recorded. The participants were asked about ownership of 13 common household items (electric stove, bicycle, clock, motorcycle, cart, plough, bed net, table, sofa, spring mattress, foam mattress, grass mattress, chair/stool) and each person was given a score of between one and 13 depending on the number of items they owned. All the study participants were asked if they had been treated for malnutrition in childhood and whether their parents had died when they were children.

Standing and sitting height were measured with a portable stadiometer. Weight was recorded on a digital weighing scale to the nearest 0.1 kg. Biacromial and bitrochanteric width were measured with a Harpenden anthropometer (Holtain Ltd., Crymch, Pembrokeshire, UK). Waist and hip circumference, mid-upper arm circumference and head circumference were measured with a nylon tape measure. Whole-body bioelectric impedance was measured using a Bodystat meter (Bodystat Ltd., Douglas, Isle of Man, UK) and gel-coated aluminium foil electrodes which were attached to the non-dominant hand and foot. The scales and measuring equipment were calibrated before use and regularly checked during the study. To assess inter-observer variability, every tenth person that was examined was measured by two investigators.

The study protocol was approved by the research ethical review boards of the London School of Hygiene and Tropical Medicine, Jimma University, and Gondar University.

A target sample size of 100 cases and 100 controls was based on a power of at least 80% to detect a 5% difference in anthropometric measurements at a 5% significance level. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m<sup>2</sup>). Leg length was calculated by subtracting sitting height from standing height. Bioimpedance measurements were used to calculate percentage body fat and lean body mass using equations developed for non-Caucasian populations.<sup>16</sup> Non-normal variables were transformed appropriately. Unconditional logistic regression was used to estimate multivariate adjusted odds ratios and 95% confidence intervals. Multiple linear regression was used to estimate adjusted case–control differences and 95% confidence intervals for continuously distributed outcomes.

## 3. Results

A total of 112 cases and 149 controls were recruited. Fifty-six cases and 72 controls were recruited in Gondar, and 56 cases and 77 controls in Jimma. The characteristics of the cases and controls from Gondar and Jimma were similar and the data from the two centres were combined.

Fifty-four percent of the cases and 54% of the controls were male. Their ages ranged from 18 to 45 years. The mean age (s.d.) in the cases was 25.4 (6.1) years and in the controls 25.3 (6.7) years.

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