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Epilepsy in Indigenous and non-Indigenous people in Far North Queensland

John Archer

Ruth Bunby

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ABSTRACT

Objective: To compare patterns of epilepsy in Indigenous and non-Indigenous people presenting to hospital.

Study design: Retrospective cross-sectional survey of individuals admitted to hospital with a diagnosis of epilepsy (1 January 2001 – 31 December 2004); presenting to the emergency department with a seizure (2004); or presenting to the epilepsy clinic (1 September 2002 – 31 March 2005).

Setting: Cairns Base Hospital, the major referral centre for Far North Queensland, including Cape York and the Torres Strait, with a population of 230 000 (13% Indigenous).

Main outcome measures: Proportion of Indigenous patients presenting for epilepsy; proportion of Indigenous and non-Indigenous groups affected by each of the main epilepsy syndromes.

Results: Of 359 patients attending the epilepsy clinic and 918 patients having electroencephalography (EEG), 11% and 13% were Indigenous, respectively (in proportion with the catchment population). However, 30% (146/486) of patients presenting to the emergency department with seizure, 31% (130/418) of inpatient admissions with epilepsy, and 44% (28/63) of patients admitted with status epilepticus were Indigenous. Indigenous patients were more likely to have an abnormal EEG result ($P = 0.025$), while non-Indigenous patients presenting to the clinic were more likely to be classified as non-epileptic (31% v 18%). In those with abnormal EEG, the frequency distribution of abnormalities was similar, and, in those with epilepsy, syndrome classification also showed similar frequencies. There was no significant difference in occurrence of epileptogenic abnormalities detected by imaging (13% non-Indigenous v 18% Indigenous) or in alcohol consumption (38% v 37%).

Conclusions: Indigenous Australians have similar epilepsy syndromes to the non-Indigenous population, but they present with more serious disease. This discrepancy may relate to inequitable health care utilisation due to cultural issues or geographic isolation.

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Australian Aboriginals and Torres Strait Islanders are burdened with higher rates of many chronic illnesses, most notably diabetes, vascular disease and renal failure.[1] However, there is limited information regarding epilepsy in these populations.

Population-based studies have shown higher incidences of epilepsy in developing countries, with the age-adjusted incidence ranging from 64 to 190 per 100 000 person-years,[2-6] while that in developed countries is lower, at 24 to 53 per 100 000 person-years.[7-10] However, no direct comparative studies have been performed. The most commonly cited hypothesis to explain this incidence difference is a higher rate of infectious diseases in developing countries (eg, those causing cerebral tuberculosis or neurocysticercosis).[11,12]

The health status of Australia's Indigenous population is in many respects more typical of that in developing countries.[13] However, in contrast to the situation in many developing nations, Indigenous Australians have a full range of health care services available to them, including a comprehensive range of neurological investigations — neurological assessment, electroencephalography (EEG), and cerebral imaging, including computed tomography (CT) and magnetic resonance imaging (MRI). However, geographic isolation and cultural issues lead to persisting inequalities in health care utilisation. Government strategies attempt to overcome this through subsidised transport from remote regions to major centres, and "outreach" medical clinics, where doctors visit remote communities by plane.

Cairns Base Hospital (CBH) in Far North Queensland serves a vast and mostly sparsely populated area about the same size as Victoria or the United Kingdom. Of the population of 230 000, of whom 135 000 live in Cairns and its immediate surrounds, 13% are Indigenous.[14] The hospital's catchment area extends to the Torres Strait, 1000 km away. The only specialist neurological services for the region are at CBH, which also provides the only EEG, CT and MRI services. These are available free of charge to all patients.

Our aim was to compare patterns of epilepsy in Indigenous and non-Indigenous people presenting to CBH.

METHODS

Data sources

Epilepsy cases were identified from several overlapping sources. We reviewed hospital discharge diagnoses, emergency department (ED) diagnoses, epilepsy clinic classifications and EEG classifications, comparing Indigenous and non-Indigenous patients. Patients of all ages were included in the study.

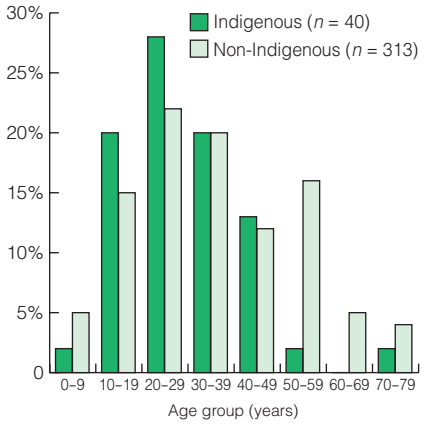
Hospital and ED data

Hospital discharge coding data were searched for all patients with an admission diagnosis of epilepsy or status epilepticus from 1 January 2001 to 31 December 2004. These figures were compared with overall hospital admission data, excluding dialysis patients (60% of whom are Indigenous), as they are admitted three times per week and would skew the data. Patients presenting to the ED are entered into a different database as only some are admitted. All patients presenting with epilepsy or seizure were recorded. Indigenous status is routinely recorded by hospital administrative staff.

EEG laboratory data

The EEG service at CBH for neonates, children and adults has been operating since 2002. It uses a database reporting system, which routinely records demographic information, including Indigenous status. To facilitate analysis, EEG diagnoses were grouped into broad categories, reflecting clinically useful classifications. Epileptiform abnormalities included definite sharp waves or sharp- and slow-wave complexes, and were grouped as: "generalised" (consistent with idiopathic generalised epilepsy), "focal frontotemporal", "focal non-temporal", or

1 Age distribution of patients presenting to the epilepsy clinic



Presentations of Indigenous people are significantly skewed towards younger ages ($P = 0.025$, unpaired t test). Children are under-represented, as this is primarily an adult clinic. ◆

"other" (including multifocal discharges, secondary generalised activity such as slow spike and wave, and electrographic status). Non-epileptiform abnormalities included focal or diffuse slowing and triphasic waves.

Epilepsy clinic data

An epilepsy clinic has been operating at CBH since 2002. All patients are seen by the same neurologist (JA). Indigenous status is checked, if not already recorded, by direct questioning of the patient. An epilepsy history is obtained from each patient, covering epileptic auras, features suggesting focality at seizure onset, eyewitness descriptions of seizures, history of head trauma causing loss of consciousness, and current alcohol intake (standard drinks per week).

At the completion of each clinic, information is entered into the database, including demographic characteristics, seizure type and frequency, and results of key investigations. The likely epilepsy syndrome is recorded according to International League Against Epilepsy (ILAE 2001) criteria. Although EEG information is used to aid classification, syndrome diagnosis is sometimes made in the absence of definitive EEG abnormalities (eg, with recent development of myoclonic jerks and seizures, which are tonic–clonic from the outset, in an intellectually normal adolescent, a diagnosis of idiopathic generalised epilepsy would be made even if EEG gave normal results).

2 Classification of patients attending the electroencephalography (EEG) service (Sep 2002 – Mar 2005)

EEG classification	Non-Indigenous	Indigenous
Normal	465 (58%)	56 (47%)
Abnormal	332 (42%)	62 (53%)
Total	797 (100%)	118 (100%)

Indigenous patients were more likely to have an abnormal EEG ($P = 0.025$, Fisher's exact χ^2 test). ◆

Statistical methods

Descriptive statistics were used for common ratios, χ^2 testing for nominal data of sufficient sample size, and unpaired Student's t tests for continuous data. Statistical analysis was performed using StatView version 5.0.1 (SAS Institute, Cary, NC, USA).

Ethical approval

The study was approved by the ethics committee of CBH, which includes an Indigenous representative.

RESULTS

Hospital admissions

Between 2001 and 2004, there were 418 admissions to CBH with a diagnosis of epilepsy; 31% (130/418) were Indigenous patients. This can be compared with the population of the hospital catchment area (13% Indigenous people) and that of general CBH admissions (20% Indigenous people). Over the same period, 44% of those admitted with status epilepticus ($n = 63$) were Indigenous. The over-representation of Indigenous people in this group is more evident in the subgroup of adults (> 18 years) with status epilepticus ($n = 40$), of whom 53% were Indigenous.

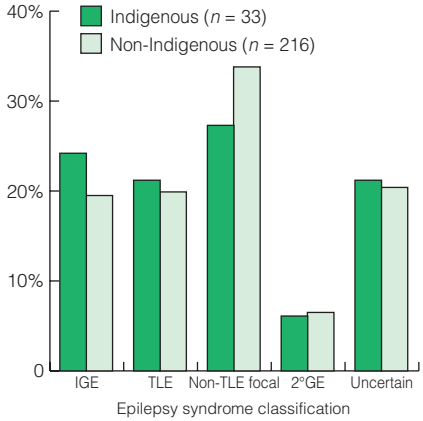
Emergency department

In 2004, 486 patients presented to the ED with an acute seizure (coded with a diagnosis of "postictal", "seizure", or "status epilepticus"), of whom 30% were Indigenous; 17% of all general admissions to the ED over the same period were Indigenous patients.

Epilepsy clinic and EEG service

From 1 September 2002 to 31 March 2005, 359 patients were seen in the epilepsy clinic, of whom 11% ($n = 40$) were Indigenous, 87% ($n = 313$) non-Indigenous, and 2% ($n = 6$) of unknown status. Over a similar period, 13% (118/918) of the EEGs performed were on Indigenous patients. These figures compare favourably with the expected proportion of Indigenous people in the catchment population. Indigenous patients attending the clinic were significantly younger ($P = 0.025$, unpaired t test), with a pronounced peak between the ages of 10 and 30 years. Non-Indigenous patients attending the clinic were spread more evenly across the age spectrum (Box 1); 53% of both Indigenous and non-Indigenous patients attending the clinic were female.

3 Classification of patients presenting to the epilepsy clinic (excluding non-epileptic presentations)



IGE = idiopathic generalised epilepsy.
TLE = temporal lobe epilepsy.
Non-TLE focal = focal epilepsy not arising from the temporal lobe.
2°GE = secondary generalised epilepsy. ◆

Non-Indigenous patients (compared with Indigenous patients) presenting to the epilepsy clinic were more likely to receive a diagnosis of a non-epileptic disorder (31% v 18%), although this difference was not significant. Non-epileptic attacks included syncope, panic attacks and pseudoseizures. However, converging results come from the finding that non-Indigenous patients were more likely to have normal EEG results (58% v 47%; $P = 0.025$; Fisher's exact χ^2 test) (Box 2).

For patients attending the epilepsy clinic who were diagnosed with epilepsy, the proportions of Indigenous and non-Indigenous patients in the main syndrome groupings were remarkably similar. In a parallel finding, Indigenous and non-Indigenous patients with abnormal EEG findings showed similar proportions of the different abnormalities (Box 3). A crude estimation of seizure burden was calculated by adding annual seizure frequency for each seizure type for each patient. This can be quite

inaccurate for some seizures, such as absences, which may not always be detected, and myoclonic seizures which may be erratic, frequent and occur in clusters. Within these limitations, no significant difference in seizure burden was noted between Indigenous and non-Indigenous patients.

Sixty per cent of Indigenous patients and 56% of non-Indigenous patients attending the clinic had cerebral imaging performed or already available. Reasons for not performing imaging included non-epileptic attacks, idiopathic generalised epilepsy, or patient non-compliance. Twenty per cent of Indigenous patients and 17% of non-Indigenous patients were found to have abnormalities on CT or MRI scans that could account for their epilepsy syndrome (Box 4). Epileptogenic abnormalities in both groups included ischaemic injury, tumour, dysplasia, hippocampal sclerosis and post-traumatic gliosis. That there was no significant difference in the rate of reported head injury (13% non-Indigenous patients v 18% Indigenous) collaborated these findings. Alcohol was consumed by 38% of non-Indigenous and 37% of Indigenous patients with epilepsy (Box 5). There was no significant difference in the amount of alcohol being consumed among those currently drinking, although subject numbers were small.

4 Proportion of patients treated in the epilepsy clinic in whom abnormalities* were detected on CT or MR imaging of the brain

Imaging result	Non-Indigenous ($n = 313$)	Indigenous ($n = 40$)
Normal	105 (34%)	15 (38%)
Epileptogenic abnormality	53 (17%)	8 (20%)
Non-epileptogenic abnormality	16 (5%)	1 (3%)
No information	139 (44%)	16 (40%)

* Epileptogenic abnormalities include hippocampal sclerosis; tumours; porencephaly from stroke, trauma or neurosurgery; and malformations of cortical development. CT = computed tomography; MR = magnetic resonance. ◆

DISCUSSION

Aboriginal and Torres Strait Islander patients presenting to CBH with epilepsy have similar epilepsy syndromes to non-Indigenous patients, but the condition in Indigenous patients at presentation is more serious. Although the proportion of Indigenous patients presenting to the epilepsy clinic or for EEG appointments is the same as in the catchment population, they are over-represented among ED presentations and inpatient admissions. This increase (about 30% of those presenting with epilepsy) exceeds the Indigenous over-representation seen at the hospital (about 20% of presentations). The situation is more marked with the most severe of epilepsy presentations — status epilepticus — where Indigenous patients comprise 44% of presentations, rising to 53% of the subgroup of adults with status epilepticus. Thus, there is a gradient of progressive over-representation, moving from the mild to more severe epilepsy presentations. However, data from the EEG laboratory, which serves both inpatients and outpatients, and the epilepsy clinic suggest that rates of basic syndromic classification are similar. The proportion of epilepsy patients with idiopathic generalised epilepsy, which probably has a largely genetic basis, was similar at about 20% in both groups. Temporal lobe epilepsy, which can have an environmental contribution through prolonged febrile convulsions of early childhood, also showed similar rates of about 20% in the Indigenous and non-Indigenous groups.

It is a commonly held belief among those working with Indigenous people that most cases of epilepsy are due to the effects of alcohol excess and head trauma. Our findings of similar rates of epileptogenic abnormalities seen on structural imaging provide no evidence to support this belief. The rate of structural abnormalities is slightly higher than that seen in series from first seizure clinics (about 13%),[15] but this is probably accounted for by the mixture of patients in our study, which includes those with refractory epilepsy in whom brain lesions are more commonly observed. A very similar percentage of Indigenous and non-Indigenous patients presenting to the epilepsy clinic consumed alcohol. It is not possible to say that alcohol does not contribute to epilepsy in Indigenous patients, as our study is not primarily designed to answer this question, but our data suggest that alcohol is relevant in a minority of Indigenous patients with epilepsy.

5 History of head injury causing loss of consciousness, or alcohol consumption in the past year in epilepsy clinic patients

	Non-Indigenous	Indigenous
Head injury		
No ($n = 293$)	261 (87%)	32 (82%)
Yes ($n = 47$)	40 (13%)	7 (18%)
Total	301 (100%)	39 (100%)
Alcohol consumption		
No ($n = 177$)	153 (62%)	24 (63%)
Yes ($n = 107$)	93 (38%)	14 (37%)
Total	246 (100%)	38 (100%)

Totals for head injury and alcohol consumption differ, as patients with inadequate documentation of these variables are not included in the analysis.◆

Of the several possible reasons for the more severe epilepsy presentations in Indigenous patients, the most likely relate to inequalities in health care utilisation. Indigenous Australians, for a variety of reasons including cultural issues and geographic isolation, are less likely to consult a medical practitioner.[16,17] Despite recent attempts to improve relationships between Indigenous Australians and government bodies, a history of mistrust leads to reluctance to seek help. Added to this are fewer doctors and often only a fly-in service in more remote regions. In some instances, there seems to be a "learned helplessness", perhaps tied in with low socioeconomic status and lowered expectations from life. Some Indigenous Australians simply put up with medical conditions, such as intermittent seizures, that their non-Indigenous compatriots would seek help for. This may also be related to reduced medication compliance, but this is a complex issue. Reduced compliance can result from inadequate explanations from medical staff, and financial or geographical difficulty in obtaining medication.

Racial variation in epilepsy has been examined in the United States, which appears to show a modestly increased (1.7 times higher) incidence of epilepsy in African American children.[18] The same study also examined the effect of socioeconomic class, showing that, after controlling for race, the incidence of epilepsy remained significantly higher in people of lower socioeconomic status.[18] Similar socioeconomic effects have been reported in the UK, where a 2.3-fold increase in epilepsy incidence was seen in the lowest fifth compared with the highest fifth of the socioeconomic spectrum.[19] It is unclear whether

this phenomenon reflects cause, effect, or an epiphenomenon.

Our study does not provide data on incidence or prevalence of epilepsy in Indigenous and non-Indigenous people in the tropical north of Australia. However, it does provide fairly robust information on epilepsy syndromes in the individuals seen. We cannot exclude the possibility that, at a community level, there is an increased rate of epilepsy among Indigenous people, and that clinic presentation rates falsely appear proportional to the population due to under-presentation. However, counteracting this effect, non-Indigenous people are more likely to seek neurology care outside the public sector. It is clear from expected incidence and prevalence data that we are not seeing all cases of epilepsy in the region. But, as I am the sole neurology practitioner for this region, providing the only EEG service, it is likely this is a reasonably representative sample of epilepsy presentations.

Our study suggests that there is a role for improved epilepsy education among Indigenous Australians, Indigenous health workers and medical practitioners working in the area. For clinicians working with Indigenous people, extra time may need to be allocated to ensure explanations and medication advice are delivered in a culturally sensitive fashion. In the longer term, there is a need to improve accessibility of epilepsy services for Indigenous Australians.

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COMPETING INTERESTS

None identified.

AUTHOR DETAILS

John Archer, FRACP, PhD, Senior Lecturer,[1] Neurologist[2]
Ruth Bunby, BAppSc, Neurophysiology Technician[2]
1 School of Medicine, James Cook University, Cairns, QLD.
2 Department of Neurology, Cairns Base Hospital, Cairns, QLD.
Correspondence: john.archer@jcu.edu.au

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