The African Journal of MEDICAL SCIENCES

Volume 4

1973

BLACKWELL SCIENTIFIC PUBLICATIONS
Oxford London Edinburgh Melbourne

Afr. J. med. Sci. (1973) **4**, 223–225.

Cerebral Tumours and Epilepsy in Nigeria

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(*Received* 29 *May* 1972)

Summary. The role of cerebral tumour in the pathogenesis of epilepsy in Nigeria is reviewed. The peculiarities and associated dangers of unorthodox medical treatment of epilepsy in Nigeria are stressed in so far as they affect early diagnosis and management of epilepsy.

Symptomatic epilepsy simulating centrencephalic epilepsy is shown by two patients who had frontal lobe meningiomas.

Résumé. Le rôle de la tumeur cérébrale dans la pathogénie de l'épilepsie au Nigéria est revu. Les particularités et les dangers asseciés au traitement non-orthodoxe de l'épilepsie sont soulignés dans la mesure où ils intéressent le diagnostic et le traitement précoces de l'épilepsie.

L'épilepsie symptomatique simulant l'épilepsie centrocéphalique est démontrée par deux malades atteints de mémingiome du lobe frontal.

The prevalence of epilepsy in most African countries has not been accurately assessed but at a conference on the epidemiology of epilepsy in Africa in Marseille (1968) it was estimated to be between 8 and 13 per thousand. Epilepsy, cerebrovascular diseases and infections of the central nervous system continue to be the main neurological problems encountered in Africa.

Epilepsy is one of the common presentations of cerebral tumour. However, the role played by cerebral tumour in the pathogenesis in Africa needs careful evaluation. Lack of trained medical personnel and medical facilities cannot but affect any medical statistical figures obtained from most parts of Africa. Even in University centres, specialist medical personnel are few and the equipment with which they have to work is even less. In any series of epileptic patients studied, the proportion of symptomatic cases determined depends on the availability of medical personnel and equipment. In a series of 234 epileptic patients studied in Ibadan by Dada & Odeku (1966) 26·7% were symptomatic epilepsies. In another study in Lagos, Dada (1968) found fifty-five of 117 patients (47%) with epilepsy to be symptomatic. Out of these fifty-five patients, aetiological diagnosis was made in thirty-three patients (60%). The analysis of the thirty-three patients is as follows:

1. Hemiconvulsion-Hemiplegia-Epilepsy syndrome (HHE) — Eleven cases (33%)

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2. Cerebral palsy	Seven cases (20%)
3. Meningo-encephalitic scars (three post-traumatic and two-post-infective)	Five cases (15%)
4. Brain tumours (all meningioma)	Three cases (9%)
5. Mental retardation (in-born errors of metabolism)	Three cases (9%)
6. Tuberculoma	Two cases (6%)
7. Sturge–Weber syndrome	Two cases (6%)

Accordingly, about 10% of epilepsies in this small series is due to cerebral tumours. A larger series of 639 patients with epilepsy studied in Lagos and Ibadan (Dada, Osuntokun & Odeku, 1969) showed 13% of the proved symptomatic cases to be due to cerebral tumour.

The presentation of epilepsy in the three patients with cerebral tumours shown above is of interest. The first patient was an 8-year-old girl with a 4 month history of generalized convulsive seizures who was referred from another hospital. There did not appear to be an aura before the attacks which resulted in loss of consciousness. She was having these fits once or twice weekly. Several 'native' herbalists and practitioners were consulted. A month before her admission to the referring hospital she was observed to have left hemiparesis with hypotonia and dysphasia. There was bilateral papilloedema and hydrocephalus. Right carotid angiogram revealed a massive right temporo-parietal tumour. The child died within a few hours of the removal of the tumour. Late diagnosis caused by a number of factors, among which is ignorance of the general public about epilepsy, undoubtedly produced the massive size of this tumour and the fatal outcome.

The remaining two patients illustrate the peculiar presentation of symptomatic epilepsy which is occasionally encountered.

A 36-year-old cook–steward presented with a 3 year history of convulsive seizures with varying frequencies of once every 1–3 months. He had received both orthodox and unorthodox forms of treatment. He never had an aura before the attack, which simulated grand mal epilepsy. There was no lateralization during the fit, but the referring doctor observed that his left pupil was larger than the right after one of his attacks. Plain skull radiographs showed rather large frontal sinuses and thickened diploe. The calcified falx cerebri was in the mid-line. Cerebrospinal fluid examination was normal. A carotid angiogram was not carried out at this stage as he was thought to have centrencephalic epilepsy. He had been in the habit of alternating the drugs he received from the neurology clinic with those he got from the unorthodox 'native' practitioners. However, the fits persisted in spite of adequate medication. Further investigations, including a left carotid angiogram showed a left frontal tumour which proved to be a meningioma on histology. The patient is alive and well.

Symptomatic epilepsy may occasionally mimic centrencephalic epilepsy if the organic lesion is situated in one of the so-called 'silent areas' of the brain, as in this case from the frontal lobe. This type of presentation is sometimes described as 'secondary' grand mal.

The third case was similar to the second in every aspect. A 42-year-old housewife presented with what appeared to be grand mal epilepsy which started 3 years previously. The fits recurred under usual anticonvulsant management. Neurological and general medical examination failed to reveal any abnormality. She was, accordingly, thought to be a probable case of 'secondary' grand mal epilepsy and she was admitted for further investigations after

1 year of follow-up at the neurology clinic. The cerebrospinal fluid examination was then abnormal, with a protein content of 70 mg/100 ml. Her electro-encephalograph showed poorly-formed alpha rhythm over the right hemisphere with intermittent bursts of slow activity over the right temporal and parietal regions. A right fronto-parietal mass was revealed on carotid angiography. The mass proved to be a meningioma.

A high percentage of defaulters is seen among epileptic patients. This often aggravates the difficulties in the management of epilepsy, as shown by the second and third patients. However, when a tumour is in evolution the clinical features produced tend to vary with time and it is this variation in presentation as well as failure to achieve drug control that points to the organic nature of the attack. During one of the attacks suffered by the third case, she had aphasic arrest and a short period of 'absence' whilst she was chairman of a ladies' meeting. She did not convulse nor did she fall.

The protean clinical manifestations of cerebral tumour are well recognized. Whilst epilepsy accounts for one of the common presentations, the form of the epilepsy may itself be varied.

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