Funny Turns in an Elderly Man

A Ali[1,2,3], D Christian[2,4], E Chung[2,4]

ABSTRACT

A 74-year old man presented with recurrent attacks of altered sensorium, sometimes with abrupt falls, against the background of a long history of chronic obstructive airways disease and ischaemic heart disease. Investigations revealed evidence of significant cardiac conduction abnormalities and this led to the insertion of a permanent pacemaker. However, he continued to have recurrent 'syncopal' attacks. He was hospitalized to clarify the nature and aetiology of these attacks. Multiple stereotyped events were observed by different medical personnel, lasting up to two hours in duration. An attack was terminated by the administration of intravenous diazepam. Subsequent initiation of anti-epileptic drugs led to the complete abolition of these episodes. He has remained event-free since then ie *two years. Because of multiple co-morbidities, the elderly present a greater diagnostic challenge in the evaluation of paroxysmal alterations in sensorium. It is essential that epilepsy, particularly with non-convulsive seizures, be included in the differential diagnosis whenever evaluating these patients.*

Alteraciones Curiosas en un Anciano

A Ali, D Christian, E Chung

RESUMEN

Un hombre de 74 años de edad se presentó con ataques recurrentes de sensorio alterado, algunas veces con caídas abruptas, antecedentes de una larga historia de enfermedad pulmonar obstructiva crónica, y enfermedad cardiaca isquémica. Las investigaciones revelaron evidencias de anormalidades significativas en la conducción cardíaca, lo cual condujo a la inserción de un marcapasos permanente. Sin embargo, el paciente continuó padeciendo ataques "sincopales" recurrentes, y fue hospitalizado con el propósito de aclarar la naturaleza y etiología de estos ataques. Episodios estereotipados múltiples que duraban hasta dos horas, fueron observados por diferente personal médico. Se le puso fin a un ataque mediante la administración de diazepán intravenoso. La iniciación subsiguiente de medicamentos antiepilépticos condujo a la abolición total de estos episodios. Desde entonces, es decir, desde hace dos años, el paciente ha permanecido libre de episodios. Debido a múltiples comorbilidades, el anciano presenta un mayor desafío diagnóstico en la evaluación de las alteraciones paroxismales en el sensorio. Es esencial que la epilepsia, en particular cuando va acompañada de ataques no convulsivos, sea incluida en el diagnóstico diferencial cuando se evalúa a estos pacientes.

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INTRODUCTION

Elderly patients are frequently afflicted with paroxysmal impairments of consciousness usually because they often have chronic medical disorders such as diabetes mellitus and hypertension and can also be on many medications. The differential diagnosis of transient impairment of sensorium in the elderly is wide and includes metabolic encephalopathies *eg* medication side effects, syncope, including cardiogenic syncope, transient ischaemic attacks and strokes, the syndrome of transient global amnesia, psychogenic fugue states and epileptic seizures. Many elderly patients may have more than one cause for this symptom. Thus, misdiagnosis and empirical (often inappropriate) treatment is common in this age group. In addition, epileptic seizures are often atypical and more subtle in the elderly, in whom the prevalence of epilepsy is higher than in any other age group (1). A logistic problem often faced is also the reluctance on the part of the patient, family or physician to allow the performance of

From: Department of Medicine, Kingston Public Hospital[1]; Department of Medicine[2], The University of the West Indies; Epilepsy Centre of Jamaica, Andrews Memorial Hospital[3] and Cardiology Associates[4], Caledonia Avenue, Kingston, Jamaica.

Correspondence: Dr A Ali, Physician's Offices, Andrews Memorial Hospital, 27 Hope Road, Kingston 10. Fax: (876) 906-5339, e-mail: amzamd@yahoo.com.

advanced and perhaps expensive investigations, perceived as unlikely to yield answers to elucidate the clinical picture.

We present the case history of an elderly man whose clinical course is instructive.

CASE HISTORY

Mr NM, a 74-year old right-handed man first came to the medical attention of the cardiologist in 1993 with a history of typical angina. His vascular risk factors included hypertension and cigarette smoking of long standing. Clinical examination revealed evidence of early chronic obstructive airway disease and an ECG revealed evidence of right bundle branch block (RBBB). He had previously seen the cardiologists in 1985 for nonspecific respiratory symptoms at which time his ECG was found to be normal.

Over the ensuing years, clinical evidence of aortic stenosis was noted, confirmed by echocardiography (valve gradient of 30 mmHg) and he continued to have stable angina. His respiratory status deteriorated slowly despite treatment with theophylline and a sleep study demonstrated evidence of significant obstructive sleep apnoea (OSA) (apnoea: hypoapnoea (A:H) index of 50). In October 1999, continuous positive airway pressure at 9cm H_2O was implemented with good effect (A:H index of 2.7).

However, in May 2000, he returned with a history of two episodes of abrupt loss of consciousness and falling. Serum glucose levels were normal and repeat echocardiography was unchanged. His medications had been unchanged except for an increase in theophylline to 250 mg twice daily, from 125 mg daily. In January 2001, he did not improve and developed sudden onset of chest pain at rest and an assessment of unstable angina was made. Despite medical management, and he was eventually admitted to hospital in February, and settling with the addition of a transdermal nitrate patch.

An ECG in June 2001 revealed the usual RBBB but also paroxysmal atrial fibrillation. In September 2001, a marked sinus bradycardia of 38/minute was noted and his digoxin was discontinued.

In May 2002, he again suddenly lost consciousness and fell, hitting his head, remaining unconscious for approximately five minutes. ECG then revealed RBBB and first degree AV block. A possible reason for this event was felt to be that he had taken tramadol for pain a short while before the loss of consciousness. He was scheduled for a 24-hr Holter monitor and pain medication was changed to non-steroidal anti-inflammatory drugs.

A brief episode of dizziness in March 2002 led to him falling but without definite loss of consciousness but in September 2002 he again had another “blackout”. An ECG revealed a heart rate of 44/min with RBBB and 2:1 AV block. In view of the continuing deterioration in cardiac electrophysiological findings and his ongoing syncopal events, a permanent demand pacemaker was implanted in October 2002, on the assumption that the episodes were due to intermittent complete heart block.

In the ensuing four months, he experienced a number of other medical problems including a pulmonary embolism, urinary tract infection (UTI) and ongoing difficulties with his COPD, necessitating almost constant usage of his CPAP machine. However, there was no definite history of altered sensorium until a few days after a routine herniorraphy in June 2003 when he had recurrences of the typical blackouts. An assessment of possible TIAs was made and aspirin and dipyridamole added without discernible benefit, as attacks continued. He was therefore admitted for assessment of the nature of these attacks, as well as for treatment of an intercurrent UTI.

On July 1, 2003 while in hospital, the medical officer on duty was called to see him because of an apnoeic attack observed by the nurses. His eyes were open and pupils were equally reactive to light but he demonstrated no spontaneous activity and was unreactive to commands. Vital signs were stable and he was not apnoeic at the time of assessment. An assessment of possible anoxic brain injury was made but he spontaneously recovered. Later that day another episode occurred and this time it was observed by the cardiologist. He was noted to be unresponsive, eyes were open with occasional eyelid fluttering and he responded with a shout to supraorbital pressure, but otherwise remained unresponsive until he spontaneously regained awareness approximately two hours later. He was amnestic of the event.

A detailed history of these odd events was then obtained from his wife. She indicated that in the preceding year he had had 6–8 similar episodes. In the week immediately before admission, she observed two such events. Initially these were brief, approximately five minutes long but recently they had become more prolonged. The attacks occurred at all times of the day and night whether he was lying or standing. If standing he would fall heavily. An attack was characterized by staring with limpness of the limbs. No tonic or clonic activity had ever been noted. After recovering he was his usual self with no confusion or physical weakness as reported by his wife.

Detailed neurological examination revealed no abnormalities except for a mild right pronator drift. An assessment of a possible recurrent complex partial seizure with non-convulsive *status epilepticus* was made, probably secondary to cerebrovascular disease. An EEG was planned but the patient had another episode observed by a nurse, lasting eight minutes. The decision was taken to initiate anti-epileptic drug (AED) treatment prior to the EEG in view of the frequency of events. Carbamazepine was started at 100 mg twice daily.

On July 4, another of his stereotyped events was noted by the nursing team. The neurologist examined him 30 minutes after the onset of the event and he was noted to be staring with occasional eye blinks to threat. No response was obtained to painful stimuli. The decision was taken to at-

tempt to terminate the episode with intravenous benzodiazepines (diazepam) as clinically the episode appeared to be one of non-convulsive *status epilepticus*. The CPAP mask was applied and after the slow administration of 5 mg of diazepam, he began moving his arms and his eyes became focussed with a change in his respiratory pattern. He then immediately had an apnoeic spell for which he required temporary manual (Ambu bag) ventilation. Peripheral pulses and blood pressure were well maintained throughout the event. After five minutes, spontaneous respiration resumed and soon after the patient spoke coherently although he was very drowsy.

The carbamazepine was rapidly increased to therapeutic dosages over the ensuing week. No further events were observed in hospital. A routine interictal electroencephalogram (EEG) done towards the end of his hospitalization revealed bitemporal delta slowing, left more than right, sometimes occurring in long runs on the left. All in-hospital baseline blood investigations were normal. Computed tomography scan of the brain had previously demonstrated features consistent with cerebral atrophy and diffuse cerebrovascular disease.

He was discharged and has remained event-free for the last two years on 200 mg of carbamazepine thrice daily. He continues to be seen for monitoring of pacemaker function and OSA for which he remains on CPAP.

DISCUSSION

Paroxysmal alteration of sensorium is a common phenomenon in the elderly. Elderly patients, unlike the young, often have multiple medical problems and are therefore frequently on multiple medications, with their own potential for causing alteration of sensorium, for example anti-diabetic agents (inducing hypoglycaemia) and anti-hypertensive agents (inducing syncope from postural hypotension).

The specific differential diagnosis of transient alterations of consciousness in the elderly is very wide. They include metabolic encephalopathies *eg* drug effects, syncope including cardiogenic syncope, vertebrobasilar insufficiency with non-epileptic drop attacks, transient global amnesia, psychogenic states including fugue states and epilepsy. Lastly but certainly not least is the well-known frequency with which the elderly will complain of non-specific symptoms of “dizziness”, “blackout” and “bad-feelings” for which aetiology is all too often either not searched for or found on cursory assessments.

The clinical characteristics of epilepsy in the elderly are more diverse than in the younger age group (2) leading to additional confusion. It is understandable therefore that the identification of an epileptic aetiology is comparatively rarely made in the midst of such a wide differential diagnosis.

A particularly important scenario is the need to make an accurate distinction between cardiac and epileptic events. In this population cardiac events are very common particularly in patients who were heavy smokers and hypertensive with established ischaemic heart disease. The clinical progress of our patient resulted in the placement of a permanent pacemaker without the anticipated resolution of these episodes, and ultimately led to appropriate admission for characterization of these events.

The ideal modality of evaluation of these paroxysmal alterations of sensorium is by admission to a video-EEG unit where the patient’s electroencephalogram, electrocardiogram and clinical state can be monitored continuously until a few typical events can be recorded. These can be subjected to analysis focussed on the determination specifically of whether the episodes are epileptic or not. The continuous ECG monitoring allows for the additional determination of whether the episodes may be a disorder of cardiac rhythm.

Due to the unavailability of this modality of evaluation at that time, the decision was taken to initiate anti-epileptic drug treatment empirically but also to utilize IV diazepam both because of the duration of the event and also as a diagnostic tool. The abrupt termination of the clinical event and his subsequent freedom from further episodes on maintenance anti-epileptic treatment confirms the epileptic nature of the events.

It is important to stress that interictal EEG recording in this age group is often of low yield in the diagnosis of epilepsy. This fact must be recognized when considering the diagnosis of an epileptic syndrome as the cause of episodic events in the elderly (3). This was the case in our patient in that although the interictal EEG was not normal it did not reveal unequivocal evidence of epilepsy *ie* sharp/spike-wave discharges. Even in centres where video-EEG is easily available, elderly patients only constitute between 1.4% and 4.3% of all patients admitted to these units for diagnostic evaluation (4,5) – therefore increasing the reliance on interictal EEG in the diagnosis of epilepsy in this age group, with the attendant hazards of missing the correct diagnosis.

Equally confusing is the fact that during some seizures, typically temporal lobe seizures, there can be a significant pre-ictal acceleration or, much less commonly, slowing of the heart rate, typically frontal lobe in onset, with irregularities of rhythm (6,7). This is felt to be due to the involvement of the limbic system especially the diencephalon, resulting in autonomic sequelae.

In some patients with an ictal bradycardia syndrome (8), the degree of bradycardia can itself lead to syncope with or without convulsive activity. In the index patient, the events were those of non-convulsive *status epilepticus* and not syncopal (ictal or otherwise) as they were prolonged and rapidly ameliorated by intravenous benzodiazepines. Of course, this patient’s heart rate was controlled by the presence of a pacemaker, therefore precluding a cardiac syncopal cause.

An important additional lesson is noted in our patient with regard to pharmacotherapy. During his treatment two drugs were exhibited to treat co-morbid conditions, both of which may have had an impact on his neurological status.

Theophylline and tramadol can both lower the seizure threshold (9) and it is interesting that the events became more frequent and severe with the increase of the theophylline dosage and also with the temporary addition of tramadol.

Although, as previously stated, it is ideal to record EEG during an episode *ie* document the epileptic nature of the event with ictal EEG, this may not always be readily available. The prominent bi-temporal focal slowing on *interictal* EEG (indicative of focal subcortical dysfunction) was suggestive but not itself diagnostic of an epileptic aetiology. However, the characteristic clinical features in the eyes of trained observers, and response to treatment, both in the acute setting and with maintenance AEDs, confirmed the epileptic nature of the events in our patient.

In summary, an elderly man with a long history of ischaemic heart disease presented with increasingly prolonged episodes of altered consciousness. The stereotyped nature of the events was recognized and intitiation of anti-epileptic drug treatment resulted in the complete cessation of events and the return of an acceptable quality of life. Epilepsy with non-convulsive seizures should always be borne in mind in an elderly patient with similar clinical presentations.

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