a creative treatment approach in an environment where dissociative disorder diagnoses are apparently infrequent.

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Dementia-like state in a patient treated with vigabatrin

Sir – Vigabatrin is a novel anti-convulsant drug, and structural analogue of the neurotransmitter GABA. It was first administered to man in 1979, and controlled studies proved its efficacy in refractory complex partial seizures.[1,2] A non-progressive and reversible micro-vacuolation or intramyelinic oedema localised to certain white matter tracts of the CNS has been shown in rats and dogs following long term treatment.[3] However, neurophysiological[4] and neuropathological[3] studies confirmed its safety in human beings. Most common side effects reported[5] are irritation, aggression and memory problems. We report on a case presenting with a picture similar to dementia in a patient treated with vigabatrin.

Case report

A 62 year old lady was admitted for assessment, and treatment of worsening epileptic attacks, who had epilepsy for 33 years following a left hemiparetic stroke. Her seizures were complex partial with secondary generalisation, poorly controlled on carbamazepine 600mg daily and sodium valproate 2,000mg daily. She had been living with and was cared for by her husband. She had a previous right mastectomy for carcinoma of the breast 16 years earlier, but was otherwise well. All routine biochemical and haematological investigations were normal. Sodium valproate was reduced to 1,000mg daily, and vigabatrin was started at 500mg daily then increased to 1,500mg after one week.

Two weeks later she became seizure free but was observed to be tearful and depressed believing that her husband had killed himself. She was disoriented for time and place, verbally aggressive and incontinent of urine. A CT scan showed generalised atrophy, but no focal lesion. She was referred for psychiatric opinion after two months for assessment of dementia. She was observed to be visually hallucinating and scored five on the mini mental state. Her mood was fatuous, but she denied any mood congruent or incongruent delusion. She had right/left disorientation, constructional apraxia, tactile agnosia and nominal dysphasia. Her EEG showed generalised slowing in the theta range, but no sharp waves. Serial mini-mental test scores remained at five.

Vigabatrin was reduced gradually at a rate of 500mg weekly and was accompanied by a corresponding improvement in her physical and mental state. Two weeks later there was no evidence of cognitive impairment, and her seizures recurred with a reduced frequency and severity.

Discussion

Confusion has been reported in 3.4% of patients receiving vigabatrin in controlled studies.[5] Our patient had all the signs suggestive of dementia that were clearly associated with vigabatrin treatment and remitted on its discontinuation. The most likely explanation is a prolonged acute organic brain syndrome (acute confusional state with visual hallucinations) that has been mistaken for a state of dementia. Depressive pseudodementia is unlikely considering the severity of the cognitive impairment. There is currently no published evidence that vigabatrin has any deleterious effects on indices of cognitive function.[5] However, schizophrenia-like state, depression and aggressive behaviour were reported in 4%-8% of patients receiving the drug especially in patients with a past psychiatric history.[5]

The possible pathophysiology of this adverse effect is complex and the presence of brain damage is a major contributory factor. Cortical GABA concentrations are reduced in Alzheimer's disease[6] and hence the mechanism of action of vigabatrin in epilepsy associated with increased cortical GABA is unlikely to explain the above adverse effect. It is very unlikely that forced normalisation[7] is the likely cause since the improvement in cognitive functions preceded the recurrence of seizures. However, it can only be ruled out with sequential EEG recordings before, during, and after the abnormal behavioural state. A reversible dementia like state needs to be included among possible side effects of vigabatrin especially in patients with brain damage.

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