

Psychomotor Epilepsy with Rare Psychiatric Manifestations

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Summary

A rare case of complex partial seizure (psychomotor epilepsy) with varying shades of psychiatric manifestations is presented. The highlights are multiplicity of its manifestation in the same case, its interchanging pattern and the variable durations. This gray area of psychiatry and neurology is discussed with special reference to diagnosis and management.

Key Words: Complex partial seizure, Confusional, Non confusional psychotic episodes, Carbamazepine

Introduction

The interrelationship of epilepsy and psychiatric illness is still not very clear. The prevalence of psychosis in epilepsy ranges from 0.6% to 19% and is 4-12 times more frequently associated with temporal lobe epilepsy¹. It is now clear that they are mostly ictal or post ictal confusional states including "Fugue" and "twilight state"^{1,2}. The EEG feature of these conditions is unknown. In addition, there are also brief, self limiting, non-confusional psychotic episodes which are neither ictal nor post ictal events and resemble schizophrenia or affective psychosis².

Case Report

Mr. M.S.B. a 27-year-old single, Malay male from the army with past history of febrile seizure at the age of 18 months was having attacks of generalised tonic clonic seizure since 1989. The history collected from his relatives and friends revealed that there was usually a distinct change in his behaviour 1-2 days prior to these attacks. It started with withdrawal for hours followed by wandering around his camp, aggressiveness, social disinhibition of undressing before the public or entering the army canteen completely naked at odd hours. This lasted for about 2-6 hours followed by generalised tonic

clonic seizure. On the other occasions it would start with headache, epigastric "discomfort" or "fullness", involuntary chewing and a feeling of changing into a tiger or elephant which lasted from a few minutes to an hour and either subsided spontaneously or was followed by a tonic clonic seizure. The memory of these preceding events was variable. Following the generalised tonic clonic seizure, the patient either went into a deep slumber or remained confused for a certain period of time. During this confusional states which generally lasted for few hours, he was either found to be aggressive and vividly hallucinating or overtly paranoid, restless with behavioural oddities like saluting his friends inappropriately and smiling inappropriately. There was also history of abnormal behaviour without any confusion and temporal relation with "fits" characterised by excessive happiness, hyperactivity, excessive talk and grandiose claims that the P.M. will give him the power to rule the country and he was the only one who can save the country. He also had visual hallucinations of the prime minister, aeroplanes flying around, a blazing fire and commanding auditory hallucination. The duration of each such episode was from 1 to 10 days. In one of such episodes observed in the ward he was found to be absolutely withdrawn, mute with a vacant look, walking around like a robot but eating, dressing and sleeping well and was subtly irritable.

When interviewed in this phase he denied being depressed, anxious, fearful or happy, denied any hallucinatory experiences and there was no evidence of confusion. The EEG recording at this phase did not show active ictal discharge and was essentially normal with mild generalised slowing. The preictal and post ictal behavioural profile were not stereotyped and were changing from one pattern to the other. There was no evidence of any psycho-social stressors, either at home or at work, which could have contributed to the genesis or maintainance of this abnormal behaviour. EEG showed - bilaterally synchronous, high voltage (150 - 200 Mv) centripetal discharges suggestive of generalised epilepsy, C.T. - showed mild cerebral atrophy with ventricular dilatation and all other base line investigations including VDRL & TPHA were normal. The patient was initially treated with carbamazepine 800 mg daily but it had to be discontinued due to leukopenia and was substituted with sodium Valproate 2.4 gm daily in divided doses with an optimum blood level of 69.65 mg/ml. Haloperidol 5-10 mg was added on a short and need to continue basis. The seizure frequency came down to an irregular pattern of one in 6 to 9 months and so do the behavioural abnormality. The patient subsequently defaulted follow up and so the current status is unknown.

Discussion

The present case has many interesting as well as atypical features. The pre-ictal changes did not come with all the attacks and the ictal manifestation was sometimes in abnormal behaviour form and sometimes in visceral form. The post ictal abnormal behaviour usually started 5 to 7 hrs after the seizure; the ratio of "fits" to abnormal behaviour was generally 10:2; many attacks of abnormal behaviour started after the patient awoke from post ictal sleep; some seizures were followed by confusional psychosis and some by non-confusional psychosis and the nature of the psychosis was not stereotyped but varying at different points of time ranging from schizophrenia like psychosis to

atypical psychosis to affective psychosis. In short it had a varied presentation with changing clinical profile in all the pre-ictal, ictal as well as post ictal phases, all of which were clearly seizure related with no functional overlay. The preictal changes were probably non-convulsive statuses preceding the usual convulsive attacks which unfortunately could not be confirmed by a E.E.G. recording as none of these attacks occurred during his hospital stay. The postictal confusional type presented either as aggressive hallucinatory states or as paranoid state. The non-confusional type at various points of time presented as atypical psychosis and affective disorder either in manic or apparently depressive (stupor) phase. These atypical psychoses are described as at times schizophrenia and at times affective disorder, more typically combined and often alternating between one and the other and remitting fast without residual deficits¹. Though the symptom profile is clearly suggestive of a complex partial seizure, the EEG failed to detect any specific focal discharge from either of the temporal lobes and instead showed a generalised seizure pattern which is but not uncommon. The C.T. finding of mild cerebral atrophy with ventricular dilatation is difficult to explain without any history of neurological problem and basic investigations all being normal. Probably it points toward a degenerative process and might be causally related to his current problems. Carbamazepine although is the drug of choice for its anti-kindling properties³, it had to be discontinued in our case due to leukopenia. Sodium valproate along with a short course of Haloperidol brought down both the seizure and the episodic abnormal behaviour to a great extent. Unfortunately, its complete cessation could not be achieved as the patient defaulted follow up.

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References

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