HERPES SIMPLEX ENCEPHALITIS PRESENTING WITH CATATONIA

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Introduction

Herpes simplex has been incriminated in 20 per cent of cases of severe sporadic encephalitis [1]. Herpes simplex Type I virus is of particular interest to the psychiatrist because of its propensity to destroy limbic cortex in the frontal and temporal lobes. This propensity is explained on grounds of proximity of these structures to the olfactory pathways or the meningeal branches of the trigeminal nerve. Alternately the neuroanatomical, neurochemical and neuroimmunological properties of the limbic cortices are hypothesised to permit the virus to manifest its destructive behaviour [2]. Virus as a cause of psychoses remains a tantalizing possibility and a recent study [3] revealed a striking temporal co-relation between severity and resolution of psychopathology and causally significant viral infection in 20 out of 35 cases of acute psychoses. We report an interesting case of herpes simplex encephalitis (HSE) manifesting as catatonic schizophrenia.

Case Report

A 16-year-old girl was referred to the psychiatry outpatient department with complaints of being relatively mute, unco-operative and neglecting food and personal care of 1 week duration. There was no past or family history of viral infection, exanthemata or psychiatric illness.

General examination revealed mild fever (temp - 99.2°F), tachycardia (pulse - 100/min), dry tongue and emaciation (body weight 33 kg). Central nervous system examination was normal with intact cranial nerves, normal gait and co-ordination and absence of meningeal signs. Fundus examination of both eyes undilated was normal. Cardio vascular system, respiratory and gastro intestinal system evaluation was within normal limits. Psychologically she was unkempt, unco-operative and had to be

physically prodded to the examination couch. There was an uniform rigidity, waxy flexibility and she was actively negativistic. She remained mute and uttered a single word during the course of 30 minutes of clinical and psychiatric evaluation. She was fully conscious, grossly oriented and lacked insight. Routine urine and blood counts, ECG and radiograph skull was within normal limits.

With these findings a provisional diagnosis of catatonic schizophrenia was made and she was given oral antipsychotics and electroconvulsive therapy thrice. Clinical response was indifferent and patient repeatedly manifested oculogyric crisis and hence a neurophysician's review was requested nearly 10 days after initial presentation. Neurophysician confirmed absence of objective neurological signs but detailed genital examination carried out for the first time at this stage revealed herpetic vesicles on both labia majora. Electroencephalogram revealed periodic lateralizing epileptiform discharges (PLEDS) and computerized tomographic scan revealed hypodensity in left frontotemporal region. Cerebrospinal fluid analysis was equivalent.

Her diagnosis was modified as HSE and she was treated with acyclovir (1000 mg/day in 5 divided doses) with which she made a quick recovery. Within a week she became co-operative, started speaking spontaneously, took personal care and catatonic features resolved. However she was amnesic for the period of catatonia. EEG showed recovery from PLEDS. Detailed psychological testing however revealed intellectual deficit (IQ-70), reduced memory quotient (Memory quotient-41 on PGI memory scale) and persisting left fronto-temporal and right temporal dysfunction on Luria - Nebraska neuro-psychological test batter (LNNB). She is under periodic review and latest scores after 6 months indicate absence of localized brain dysfunction on LNNB and improved IQ and memory scores (Figs. 1 and 2).

Discussion

Predominant neurocognitive defects are well recorded in cases of HSE due to the proclivity of the virus to destroy the fronto-temporal structures which are presumed to be the seat of psychotic behaviour. In a series of 52 cases of HSE followed

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Fig. 1 :

by Protas II [4] et al a large majority manifested with memory deficit and unmotivated actions in the absence of neurological signs. Review of cases over last 15 years do not record any case presenting with catatonic features alone in the absence of neurological signs.

The role of acyclovir in preventing long term cognitive sequelae following HSE is controversial. Shimada [5] et al have reported a 57 year old man who suffered HSE and was not cured by antiviral agents for 3 years, earlier presenting with complete loss of memory for events subsequent to the encephalitis and retrograde amnesia for several years leading to encephalitis. MRI in this case revealed total destruction of medial temporal lobes bilaterally. Gorden et al [6] followed 4 cases of HSE who received acyclovir in the early stages of illness for 1.5 to 4 years and reported long lasting neuropsychological residue despite early administration of acyclovir. Counsell et al [7] however, reported good clinical and neuropsychological recovery in cases of HSE who received 10 day course of acyclovir in the early stages. Our patient showed significant neuropsychological deficits on detailed psychometry despite apparent clinical recovery. Follow-up over 6 months has demonstrated total recovery of localized brain dysfunction on LNNB though intelligence and memory

Fig. 2 :

scores remain in the strictly average range. Though, we do not have baseline intelligence and memory score pertaining to her premorbid period, her current average scores are a reflection of the damage inflicted by the virus.

We report this case to recommend more frequent EEG investigation in psychotic cases.

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