VIRAL ENCEPHALITIS PRESENTING AS CATATONIC SCHIZOPHRENIA (A Case Report)

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Abstract

A rare case of viral encephalitis in an 18 year old recruit who presented with signs of catatonic schizophrenia is reported.

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KEY WORDS : Viral encephalitis; Catatonic schizophrenia

Introduction

Patients with encephalitis are known to present with psychiatric symptoms in the initial stages. However, the presence of clouding of consciousness and positive neurological findings help in differentiating them from cases of acute functional psychosis [1]. Occasionally such patients manifest only with abnormal behaviour without evidence of impairment of consciousness and without physical signs, producing diagnostic dilemma (2-6). Because of the comparative rarity of such cases we report one patient with viral encephalitis who was referred as a case of catatonic schizophrenia.

CASE REPORT

This 18 years old recruit having about ten days of service was admitted on 4th May for psychiatric evaluation to a peripheral hospital with acute onset of abnormal behaviour of four days duration manifesting with initial irritability, repeatedly coming to attention and stand-at-ease in the unit lines, irrelevant talk followed by social withdrawal, not reporting for parade, not answering questions, not eating food and not sleeping properly. Unit report mentioned that he had just reported to training centre but recruit training had not commenced. His behaviour in the unit initially was normal. There was no past or family history of psychiatric disorder, fits, suicidal attempts or systemic illness.

Initial examination by medical specialist showed average built & nourishment, temperature 99°F (maximum temperature during hospitalisation was 99.6°F), pulse - 104/min, regular, blood pressure - 110/70mm of Hg. Patient was mute, unresponsive, maintaining uncomfortable and awkward posture for long periods and waxy flexibility. There was no pallor, cyanosis, icterus, edema or generalised lymphadenopathy. Systemic examination was within normal limits. There were no focal neurological deficits. Routine hemogram, urinalysis, blood sugar, blood urea, serum creatinine, LFT, serum electrolyte, STS, funduscopy and skull radiograph were within normal limits. CSF examination on 05 May showed WBC 15/cumm, (predominantly lymphocytes), RBC 180/cumm, protein 80 mg/dl, sugar 50 mg/dl (blood sugar 135 mg/dl).

With a provisional diagnosis of catatonic schizophrenia patient was treated in ICU, with Tab Chlorpromazine 600 mg daily in divided doses along with tube feeding for 3 days and other supportive measures. Patient was asymptomatic after 2 weeks of treatment. During this period the treating physician on one occasion noted that patient was in coma, plantar reflexes were mute and abdominal reflexes were absent.

A repeat CSF examination on 15 May showed WBC 2/cumm, RBC 10/cumm, protein 40 mg/dl, sugar 50 mg/dl, CSF smear & culture for pyogenic organisms on both occasions revealed no organisms. CT Scan brain and EEG (done later) were normal.

During psychiatric interview on 24 May patient complained of tremulousness, stiffness of body and restlessness. Physical examination showed digital tremors. Mental status examination revealed a well kempt individual who was fully in touch with reality. Talk was relevant. Affect was appropriate and adequate. There were no

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hallucinations or delusions. Patient did not remember the events of the first week of hospitalisation. Otherwise memory, orientation and insight were unimpaired. Antipsychotic drug was withdrawn after which patient remained symptomfree.

Discussion

Encephalitis has long been noted to present with behavioural abnormalities. Economo (2), writing of encephalitis lethargica, mentioned that mental changes might be the only symptoms of the disease. He emphasized the initial similarity of these cases to acute schizophrenia or mania, and described how the catatonia of dementia praecox could be simulated. Other clinicians have noted that occasionally the early course of encephalitis specifically simulates that of a schizophrenic psychosis (3-5).

In this setting, subtle neurologic signs or mild disturbance of sensorium should be carefully looked for once the diagnosis of psychiatric illness is made, the significance of subtle neurological signs may be minimised by the clinicians [5]. Moreover specialised investigations (EEG, CT Scan, MRI) should be undertaken at an early stage to prevent inappropriate therapy.

Weinstein *et al* [6] described six patients with encephalitis in whom the initial symptoms were very similar to those of schizophrenia. In four of the six cases, severe emotional stress preceded the onset of the illness. Similarly, in our patients, recruit training appeared to be an important stressor. The notion that the onset of physical illness is related to physical agent, whereas the onset of psychiatric illness is related to psychological stress or life change is not always true. There is increasing and compelling evidence which shows that both psychiatric and physical illnesses become manifest during major life changes, disturbing life events or emotional state such as depression. Schmale [7]

reported that 41 of 42 unselected hospitalised patients with medical illness had evidence of loss (either actual, threatened or symbolic) and feeling of hopelessness and helplessness prior to onset of the disease while Greene *et al* [8] found that a high percentage of persons who died suddenly and unexpectedly, had been depressed. It is obvious that the presence of stressful life event does not necessarily indicate a functional psychosis.

The case highlights the fact that at times a diagnosis of schizophrenia is made for an acutely psychotic patient without careful considerations of alternative explanations. Diagnostic errors can be minimised if in addition to cross-sectional view the longitudinal criteria (now incorporated in ICD-10) [9] are also considered while arriving at the psychiatric diagnosis.

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