

Viral encephalopathy presenting as schizophrenia

A 40-year-old married male, without any contributory past or family history, who was apparently normal one month back, had an abrupt onset of neck pain that lasted two days. Later the pain spread to his forehead and did not abate with aspirin. Seven days later he continued to have headaches, developed vomiting, and continuous fever that was initially low grade, but became high grade in a matter of days. Fever persisted despite receiving treatment from a general physician and hence the patient was hospitalised for further management. Over the next week, fever and headache persisted and he experienced four episodes of tonic seizures following which he was unable to recognise family members intermittently, heard voices in isolation, and became agitated to the extent that he had to be physically restrained. He also started believing that neighbouring patients were malevolent and harassing him; that his brother was having a sexual relationship with his wife; and that people around him were being sent by the Central Bureau of Investigation to spy on him. His symptoms showed diurnal variation with severity of hallucinations and delusions increasing at night.

Investigations revealed the following: magnetic resonance imaging (MRI) brain-hyper intensities involving the left medial temporal lobe, predominantly the parahippocampal gyrus, which was thought to be due to herpes simplex encephalitis; magnetic resonance (MR) angiography of the brain was normal; lumbar puncture showed a colourless cerebrospinal fluid (CSF) at normal pressure and further examination revealed a total cell count of 30/cumm (lymphocytes 98%, polymorphs 2%) but sugar and protein were within normal limits. CSF was negative for IgG and IgM antibodies for **herpes simplex**

virus (HSV) 1 and 2; acid-fast bacilli, adenosine deaminase and CSF culture revealed negative results. **Erythrocyte sedimentation rate** (ESR) was 40 mm; total count 23,400; differential counts showed polymorphs 85% and lymphocytes 13%. He was clinically diagnosed as having meningoencephalitis (possible herpes simplex infection) with behavioural sequelae and systemic bacterial infection. He was treated with acyclovir, Faropenem sodium 200 mg thrice daily and both linezolid 600 mg and phenytoin sodium 100 mg twice daily. Following this treatment, fever and headache reduced and did not reoccur. But disorientation (to time, place and person) that started during the onset of the illness persisted. He was admitted to our institute for further management.

At admission, he had tachycardia (110/min), stage I hypertension (155/96) (1) and brisk deep tendon reflexes. Rest of the general physical and systemic examination was unremarkable. Mental status examination revealed delusions of reference; use of neologisms; delusions of persecution; auditory hallucinations (both male and female voices discussing about him); visual hallucinations. Assessment of cognition (2) revealed that he had normal attention span and comprehension. Vigilance tests showed errors of both commission and omission. He was not able to do fist palm, fist ring and reciprocal coordination tests. He had neologisms and was unable to repeat complex sentences. Naming, reading and writing capabilities were normal. Recalling his personal information and new learning ability after 5, 10 and 30 min was normal. Paired associate learning was impaired. He was able to copy the drawings but drawing to command was impaired. Calculation, proverb interpretation and abstraction abilities were impaired. There was no right-left disorientation, finger agnosia, evidence of denial or neglect. Mini Mental State Examination (MMSE) (3) score was 22.

Positive and Negative Syndrome Scale (PANSS) (4) was administered to quantify his psychotic symptoms even though he was not a case of schizophrenia. He scored 29 on the positive scale, 12 on the negative scale and 51 on the general psychopathology scale. During his fortnight's stay, his orientation used to fluctuate on a diurnal basis. He was started on olanzapine 10–20 mg/day and amlodipine 5 mg/day. Neurologist's review showed normal electroencephalogram and brain MRI. CSF examination revealed 10 lymphocytes and negative results for CSF VDRL, ELISA for TB antigen, IgG antibodies for HSV and India ink preparation. Psychopathology was stable for about a week after hospitalization and then he started to improve. By the 10th day, his MMSE score was 29; he did not have any of the psychotic symptoms; tachycardia and hypertension normalised and there were no neologisms or irrelevant speech. Sleep-wake cycle had normalised. PANSS score reduced to 12 on positive scale, 7 on negative scale and 16 on general psychopathology scale. He was discharged on the 14th day with olanzapine 20 mg/day along with phenytoin 200 mg/day.

This case shows an extremely rare clinical phenomenon in which classical schizophrenic symptoms were part of an encephalopathy involving one temporal lobe. Usually, if there are hyper intensities in the medial temporal lobe in MRI, along with fever and seizures, we think of herpetic involvement, but antibody testing and EEG did not reveal any abnormality. Although previous reports of herpes encephalitis mention about psychotic symptoms (5,6) and Kluwer-Bucy like syndrome (7) none of them had as clear, specific and characteristic schizophrenic features. To our knowledge, this is the first report of its kind. Our patient did not have any genetic vulnerability to develop schizophrenia (family history was not contributory), but presented with well-formed, elaborate delusions of reference, persecution,

infidelity, delusional misinterpretation, bizarre delusion, neologisms and third-person auditory hallucinations preceded by fever, headache, confusion, clouding of consciousness and seizures. Although the classical symptoms of schizophrenia were present, ICD-10 does not allow us to make diagnosis of schizophrenia in this case for obvious reasons, according to which diagnosis was made as delirium due to viral encephalopathy (8). Also, the lesion had disappeared when the MRI was repeated at our institute. When the patient came to our institute, his constitutional symptoms of meningoencephalitis had stopped but he had cognitive dysfunction along with typical schizophrenic features including the rare but specific symptom of neologisms (not as part of fluent aphasia) (9). These symptoms lasted less than a month and improved almost completely. Another interesting aspect of this case is that a localised structural pathology in the temporal lobe causing such a variety of psychopathologic phenomena. This case demonstrates that encephalitic processes can present with classical schizophrenic features and present diagnostic difficulties for the clinician.

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