

# Very early-onset psychosis/schizophrenia: Case studies of spectrum of presentation and management issues

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### ABSTRACT

Schizophrenia occurs very uncommonly in children younger than 13 years. The disease is preceded by premorbid difficulties, familial vulnerability, and a prodromal phase. The occurrence of positive psychotic symptoms such as delusions and hallucinations depends on the level of cognitive development of child. Furthermore, at times it is very difficult to differentiate the psychopathology and sustain a diagnosis of schizophrenia in view of similarities with disorders such as autism, mood disorders, and obsessive compulsive disorders. Here, we present three case studies with varying presentation of childhood-onset psychosis/schizophrenia and associated management issues.

**Keywords:** Childhood onset, psychosis, schizophrenia, very early onset

### Introduction

Schizophrenia is a chronic severe mental illness with heterogeneous clinical profile and debilitating course. Research shows that clinical features, severity of illness, prognosis, and treatment of schizophrenia vary depending on the age of onset of illness.<sup>[1,2]</sup> Hence, age-specific research in schizophrenia has been emphasized. Although consistency has been noted in differentiating early-onset psychosis (onset <18 years of age) and adult-onset psychosis (onset >18 years), considerable variation is observed with regard to the age of childhood-onset schizophrenia or very early-onset psychosis/schizophrenia (VEOP/VEOS).<sup>[2,3]</sup> Most commonly, psychosis occurring at <13 years of age has been considered to be of very early onset and that between 13 and 17 years to be of adolescent onset.<sup>[4]</sup> Furthermore, VEOS has been considered to be rare and shown to have differing clinical features (including positive and negative symptoms, cognitive decline, and neuroimaging findings), course, and outcome when compared with that of early-onset or adult-onset schizophrenia.<sup>[3]</sup> Progress in acknowledgement of psychotic

disorders in children in the recent times has led primary care physicians and paediatricians to increasingly serve as the principal identifiers of psychiatrically ill youth. In recent years, there has been substantial research in early intervention efforts (e.g., with psychotherapy or antipsychotic medicines) focused on the early stages of schizophrenia and on young people with prodromal symptoms.<sup>[5]</sup> Here, we report a series of cases with very early onset of psychosis/schizophrenia who had varying clinical features and associated management issues.

### Case Reports

#### Case 1

A 14-year-old boy, educated up to class 6, belonging to a family of middle socioeconomic status and residing in an urban area was brought with complaints of academic decline since 3 years and hearing voices for the past 2 years. The child was born out of a nonconsanguineous marriage, an unplanned, uneventful, but wanted pregnancy. The child attained developmental milestones as per age. From his early childhood, he was exposed to aggressive behavior of his father, who often attempted to discipline him and in this pursuit at times was abusive and aggressive toward

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him. Marital problems and domestic violence since marriage lead to divorce of parents when the child attained age of 10 years.

The following year, the child and the mother moved to maternal grandparents' home and his school was also changed. Within a year of this, a decline in his academic performance with handwriting deterioration, and irritable and sad behaviour was noted. Complaints from school were often received by the mother where the child was found engaged in fist fights and undesirable behavior. He also preferred solitary activities and resented to eat with the rest of the family. In addition, a decline in performance of daily routine activities was seen. No history suggestive of depressive cognitions at that time was forthcoming. A private psychiatrist was consulted who treated him with sodium valproate up to 400 mg/day for nearly 2 months which led to a decline in his irritability and aggression. But the diagnosis was deferred and the medications were gradually tapered and stopped. Over the next 1 year, he also started hearing voices that fulfilled dimensions of commanding type of auditory hallucinations. He suspected that family members including his mother collude with the unknown persons, whose voices he heard and believed it was done to tease him. He eventually dropped out of school and was often found awake till late night, seen muttering to self, shouting at persons who were not around with further deterioration in his socialization and self-care. Another psychiatrist was consulted and he was now diagnosed with schizophrenia and treated inpatient for 2 weeks with risperidone 3 mg, olanzapine 2.5 mg, and oxcarbazepine 300 mg/day with some improvement in his symptoms. Significant weight gain with the medication lead to poor compliance which further led to relapse within 3 months of discharge. Frequent aggressive episodes over the next 1 year resulted in multiple hospital admissions. He was brought to us with acute exacerbation of symptoms and was receiving divalproex sodium 1500 mg/day, aripiprazole 30 mg/day, trifluoperazine 15 mg/day, olanzapine 20 mg/day, and lorazepam injection as and when required. He was admitted for diagnostic clarification and rationalization of his medications. He had remarkable physical features of elongated face with large ears. Non-cooperation for mental state examination, and aggressive and violent behavior were noted. He was observed to be muttering and laughing to self. His mood was irritable, speech was laconic, and he lacked insight into his illness. We entertained a diagnosis of very early-onset schizophrenia and explored for the possibilities of organic psychosis, autoimmune encephalitis, and Fragile X syndrome. The physical investigations done are shown in Table 1. Further special investigations in the form of rubella antibodies (serum IgG = 64.12 U/mL, IgM = 2.44 U/mL) and polymerase chain reaction for Fragile X syndrome (repeat size = 24) were normal. His intelligence quotient measured a year ago was 90, but he did not cooperate for the same during present admission. Initially, we reduced the medication and only kept him on aripiprazole 30 mg/day and added lurasidone 40 mg twice a day and discharged him with residual negative symptoms only. However, his hallucinations

and aggression reappeared within 2 weeks of discharge and was readmitted. This time eight sessions of bilateral modified electroconvulsive therapy were administered and he was put on aripiprazole 30 mg/day, chlorpromazine 600 mg/day, sodium divalproex 1000 mg/day, and trihexyphenidyl 4 mg/day. The family was psychoeducated about the illness, and mother's expressed emotions and overinvolvement was addressed by supportive psychotherapy. Moreover, an activity schedule for the child was made, and occupational therapy was instituted. Dietary modifications in view of weight gain were also suggested. In the past 6 months, no episodes of violence came to our notice, though irritability on not meeting his demands is persistent. However, poor socialization, lack of motivation, apathy, weight gain subsequent to psychotropic medications, and aversion to start school are still unresolved. Influence of his multiple medications on bone marrow function is an impending issue of concern.

## Case 2

An 11-year-old boy, educated up to class 3, belonging to a rural family of lower socioeconomic status was brought with complaints of academic decline since 2 years, repetition of acts, irritability since a year, and adoption of abnormal postures since 6 months. He was born out of a nonconsanguineous marriage, uneventful birth, and pregnancy. He was third in birth order and achieved developmental milestones at an appropriate age. Since 2 years, he would not attend to his studies, had poor attention, and difficult memorization. He attributed it to lack of friends at school and asked for school change. There was no history of low mood, depressive cognitions, conduct problems, or bullying and he performed his daily routine like his premorbid self at that time. Since a year, he was observed to repeat certain acts such as pacing in the room from one end to another, continuously for up to 1–2 h, with intermittent stops and often insisted his mother to follow the suit, stand nearby him, or else he would clang on her. He prohibited other family members except his mother near him and would accept his meals only from her. He repeatedly sought assurance of his mother if he had spoken everything right. He also washed his hands repeatedly, up to 10–20 times at one time, and was unable to elaborate reason for the same. His mood during that period was largely irritable with no sadness or fearfulness. He mostly wore the same set of clothes, would be forced to take bath or get nails/hair trimmed, and efforts to these were often met with aggression from the patient. Eventually, he stopped going to school and his family sought faith healing. Within the next 5–6 months, his illness worsened. Fixed gaze, reduced eye blinking, smiling out of context, diminished speech, and refusal to eat food were the reasons for which he was brought to us. His physical examination was unremarkable and his mental state examination using the Kirby's method showed an untidy and ill-kempt child, with infrequent spontaneous acts, and occasional resentment for examination. He had an expressionless face, with occasional smiling to self, negativism, and mutism. No rigidity in any of the limb was observed. He was diagnosed with catatonic schizophrenia and probable obsessive compulsive disorder (vs mannerisms). We performed a battery of physical investigation to rule out organic psychosis [Table 1]. He responded to injection lorazepam with which catatonia melted away. He was also prescribed olanzapine up

**Table 1: Details of investigations done in the three children**

Investigation	Case 1	Case 2	Case 3
Hemogram and routine blood biochemistry	Hb 15.5 g% TLC 4010 mm <sup>3</sup> Platelets 5.22 lac/mm <sup>3</sup>	Hb 14.4 g% TLC 5540/mm <sup>3</sup> Platelets 3.09 lac/mm <sup>3</sup>	Hb 12.1 g/dL TLC 6980/mm <sup>3</sup> Platelet counts 4.27 lac/mm <sup>3</sup>
Liver and kidney function tests (including serum electrolytes)	Within normal limits	Within normal limits	Within normal limits
Fasting blood sugar (mg%)	76	65	72
ECG	Within normal limits	Within normal limits	Within normal limits
Serum vitamin B12 level (pg/mL)	208	328	229
Thyroid panel	FT3/FT4/TSH: 3.04 pg/mL; 1.27 ng/dL; 1.47 mIU/L	FT3/FT4/TSH: 3.85 pg/mL; 0.85 ng/dL; 2.98 mIU/L	FT3/FT4/TSH: 3.60 pg/mL; 0.90 ng/dL; 1.20 mIU/L
ASLO titres	Negative	Negative	Negative
Serum ceruloplasmin (mg/dL)	25.20	30	18.05
24-H urinary copper	45.06 µg (day 1), 42.81 µg (day 2), 41.00 µg (day 3)	22.08 µg (day1), 25.80 µg (day 2), 23 µg (day 3)	28.87 µg (day 1), 35.03 µg (day 2), 32.65 µg (day 3)
CSF panel for anti-NMDA antibodies	Negative	Not analyzed	Negative
CSF – routine and chemical	Glucose 60 mg/dL, proteins 27 mg/dL, globulin normal, cytological – no cells	Not analyzed	Glucose 48 mg/dL, proteins 22 mg/dL, globulin normal, cytological – occasional lymphocytes
Neuroimaging of brain	MRI brain showed diffuse cerebral atrophy and ventriculomegaly	NCCT head was normal	MRI of brain was normal
EEG	Evidence of mild to moderate degree of generalized cerebral dysfunction	Normal awake record	Normal awake record

Hb- Haemoglobin, TLC- Total leukocyte count, ECG- Electrocardiogram, FT3- Free tri-iodothyronine, FT4- Free thyroxine, TSH- Thyroid stimulating hormone, ASLO- Anti-streptolysin O Titres, CSF- Cerebro-spinal fluid, NMDA- N-methyl-D-aspartate, NCCT- Non-contrast computed tomography, EEG- Electroencephalography, MRI- Magnetic resonance imaging

to 15 mg/day, fluoxetine 20 mg/day, and dietary modification and lactulose for constipation. The family left against medical advice with 50%–60% clinical improvement [rating on Bush Francis Catatonia Rating scale (BFCRS) reduced from 10 to 4]. He relapsed within a month of discharge, initially with predominance of the probable obsessive compulsive symptoms. Fluoxetine was further increased to up to 60 mg/day. But within the next 2 months, the catatonic symptoms reappeared and he was readmitted. He had received olanzapine up to 25 mg/day, which was replaced with risperidone. In view of nonresponse to intravenous lorazepam, we administered him five sessions of modified bilateral Electro-convulsive therapy (ECT) (rating on BFCRS reduced from 8 to 0). The family was psychoeducated about the child's illness and the need for continuous treatment was emphasized. He was discharged with up to 80%–90% improvement. At follow-ups, he started participating at farm work of the family, took care of self, with some repetition of acts such as washing of hands, and denied any associated anxiety symptoms. However, efforts to re-enroll in school had been futile as the child did not agree for it. He has been maintaining at the same level since 6 months of discharge.

### Case 3

A 7-year-old girl, student of second class, belonging to a high socioeconomic status family living in an urban locality was brought with complaints of academic decline, irritability, and abnormal behavior for the past 9 months. The child was born out of a nonconsanguineous marriage, is first in order, and was a wanted child. Maternal health during pregnancy was normal, but the period of labor

was prolonged beyond 18 h, so a lower segment caesarean section was performed. There was no history of birth-related complications and the child's birth weight was 2.80 kg. The child attained developmental milestones as per age. The child had a temperament characterized by high activity levels, below average threshold of distractibility, average ability to sustain attention and persist, easy to warm up, adaptation to new situations, and regular bowel and bladder habits. She was enrolled in school at the age of 4 years and progressed well till 9 months back when a decline in her academic interest was observed by her class teacher. Deterioration of her handwriting skills and avoidance of group activities in school were observed. Similarly, at home persistent irritable behavior was seen and her play activities with her siblings reduced. However, her biofunctions were normal during this period.

One month prior to visiting us, she started insisting on wearing the same dress. She wore the same colored or at times the same dress which she would not take off even at bed or bath time. In addition, a change in her mood from largely irritable to cheerful was noted. Her activity levels were increased and it would be difficult to make her sit quietly in class. Her speech output was more than her usual self and she talked incessantly. Her sleep duration also decreased and she started getting up 3–4 h earlier than her usual routine. In view of these symptoms, her family made first contact with us. Her physical examination was normal and mental state examination revealed her to be cheerful, overactive, and difficult to interrupt. She sang and danced during the interview. We diagnosed her with acute mania on the basis of clinical evaluation and assessment on MINI Kid 6.0.<sup>[6]</sup> The

details of her physical examination are depicted in Table 1. She was initially treated with olanzapine 5 mg/day which was later on increased to 10 mg/day. However, no response was observed with it in the next 2 weeks, so it was cross tapered with sodium valproate which was built up to 400 mg/day. She improved by nearly 50%, but her mood still remained cheerful/irritable. She did not resume her school and was brought irregularly for the follow-up. Within the next 2 months, she also started muttering to herself and made certain abnormal gestures. She often feared staying alone, or while going to bed insisted the lights to be kept on and ask someone to accompany her in the toilet unlike her previous self. When asked, she reported seeing a lady in white clothes, with no other details. She stopped asking for food on her own and remained lost in her fantasy world. However, her interest in dressing and appreciating herself in mirror persisted. Her mood during this period was mostly labile and often changed from cheerful to sad or irritable. As per the family, the medications were continued as advised. So in view of the emerging picture, the diagnosis was revised to schizo-affective disorder, and in addition to hike in dose of sodium valproate to 500 mg/day, risperidone 2 mg/day was also added. However, even after 8 weeks of treatment with this combination with hike of risperidone to 4 mg/day, there was no relief. The child is still symptomatic, does not go to school, and has significant dysfunction. Psychosocial intervention in the form of psychoeducation, activity scheduling for the child, and occupational therapy has been instituted in addition to the existing treatment regimen, but results are yet to be seen.

## Discussion

The older concept of neurodegenerative etiology of schizophrenia has been superseded by evolving neurodevelopmental nature of this disease. The latter has been attributed to initiation of the underlying pathophysiological processes long before the onset of clinical disease and interaction of the various genetic and environmental factors. The more accommodating theorist propose schizophrenia to be of neurodevelopmental in origin which in turn speeds the process of neurodegeneration.

On clinical front, VEOS is associated with a more insidious onset, prominent negative symptoms, auditory hallucinations, poorly formed delusions which is in part due to less developed cognitive abilities.<sup>[7]</sup> The presence of history of speech and language delay as well as motor development deficits have been observed in major studies on childhood-onset schizophrenia, be it the Maudsley early-onset schizophrenia project or the NIMH study.<sup>[8,9]</sup> Premorbid deficits in social adjustments and presence of autistic symptoms have also been shown. Moreover, the early onset of psychosis is associated with poor prognosis, worse overall functioning, and multiple hospitalizations.<sup>[7]</sup> The duration of untreated psychosis in childhood-onset psychosis has been shown to be smaller in hospital-based studies<sup>[10]</sup> and larger in community settings.<sup>[11]</sup> In addition, the presence of comorbidities and an organic etiology or history of maternal illness during pregnancy is a common finding in VEOS.<sup>[10]</sup> In addition, obsessive compulsive symptoms are frequently observed in

first-episode drug-naïve schizophrenia patients and have a poorer outcome, more severe impairment of social behavior, and lower functioning.<sup>[12]</sup> However, in many instances it is very difficult to differentiate the obsessive compulsive symptoms from the motor symptoms of schizophrenia such as stereotypy and mannerisms and varying degree of insight.<sup>[13]</sup>

In the present case series, all the children had an insidious onset of illness, with initial symptom of academic decline, and poorly formed psychotic symptoms/psychotic-like experiences. All the children reported here had dropped out of school, showed a shift in their interests, withdrew from social circle, appeared to be distant, had impaired self-care, and often lacked concern for others along with a range of mood disturbances. All these symptoms fit into the classical description of prodromal symptoms of schizophrenia.<sup>[14]</sup> In contrast to available evidence, no history of motor, speech, or language delay was noted in any of the child. Furthermore, no history suggestive of autistic features or problems in social adjustments prior to onset of illness was forthcoming.

However, the diagnosis of schizophrenia could be clearly made in the first case, while the second child had predominant catatonic and probably obsessive compulsive symptoms. It is difficult to ascertain the diagnosis of schizophrenia on the basis of presence of only catatonic symptoms and no delusions and hallucinations or negative symptoms as required by Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition or International Classification of Diseases, Tenth Revision. However, it is very difficult to sustain any other diagnosis for the second child. In the third child, the illness has been evolving and the clinical picture changed from predominant mood symptoms to psychotic-like experiences at later stage. Therefore, at present a diagnosis of schizo-affective disorder is entertained. We could not find any possible organic etiology in any of the three cases with the best of our efforts.

With provision of pharmacological and psychosocial treatment in accordance to the available treatment guidelines,<sup>[15]</sup> remission was not achieved in two of the three children. Currently, the available evidence also suggests that the prognosis of childhood-onset schizophrenia is mainly poor as it disrupts the social and cognitive development and thus nearly two-third of children do not achieve remission.<sup>[16]</sup> On a positive note, we have been able to retain all the children in treatment.

Other issues faced by the families of three children and the treating team are briefly discussed below. In countries like India, where significant expenses are born by patients/family, associated stigma, limited social services, and the anti-psychotic related adverse effects raise the burden of care exponentially. In 2/3 index patients, the family bore the costs of special investigations, which was not possible in the second child and led to financial difficulties for the single mother of the first child. Adding on, the availability of rehabilitation services for children with major mental illnesses is scarce in various parts of our country. Furthermore, we successfully used ECT for management of acute disturbance in two of the three

patients prior to the notification of Mental Health Care Act, 2017 that prohibits its use in minors. The case series also put forward a strong case for strengthening and sensitizing primary care physicians and pediatricians in identifying and treating cases of VEOP, since they are more likely to be the first points of contact with patients of the discussed age group. In view of the duration of untreated psychosis being a very eloquent prognostic factor for VEOP and the symptomatology of the same showing significant heterogeneity, arming primary care physicians and pediatricians with the right skills to identify, treat, or refer patients with VEOP, especially in the prodromal period, might profoundly contribute in decreasing the morbidity and improving prognosis. Citing this lacuna which could be filled and used to our advantage, Stevens *et al.*<sup>[17]</sup> elaborated and discussed various questions which practitioners might find useful.

## Conclusion

Childhood-onset schizophrenia is a rare occurrence. The current case series highlights differing clinical presentation of VEOS/VEOP in children and adolescents. Certain other issues pertinent to the management of VEOS/VEOP are also touched upon in this article. With the early recognition of childhood mental health illnesses, we need to build and strengthen ample child and adolescent mental health services in India.

## Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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## Conflicts of interest

There are no conflicts of interest.

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