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Case Report

A rare case of early onset childhood schizophrenia with predominant negative symptoms

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ABSTRACT

Early onset childhood schizophrenia (<13 years) is often a rare disorder with an estimated prevalence of 1 in 30,000 children. The disease is characterized by hallucinations, delusions and cognitive impairments. It tends to present insidiously with a premorbid period of developmental delay or regression with declining academics. Often, the diagnosis of early onset schizophrenia is missed or sometimes misdiagnosed as pervasive developmental disorders. We here present a rare case of 10-year-old boy who showed behavioural abnormalities suggestive of psychosis.

Keywords: Early onset childhood Schizophrenia, Cognitive impairments, Developmental regression, Pervasive developmental disorders, Psychosis

INTRODUCTION

Early onset childhood schizophrenia is chronic, severe mental illness. At least 5 percent of individuals with schizophrenia become symptomatic before 14 years, and up to 40% do become symptomatic prior to 18 years of age. As the early onset disease progresses, it tends to follow more severe and debilitating course with worst outcomes than the adult onset (>18 years) and adolescent onset schizophrenia (13-18 years). Due to its variation in clinical picture by age, the diagnosis often missed or misdiagnosed, thereby leading to increased duration of untreated psychosis with negative symptoms in the patients. This attributes to limited treatment options, with poor clinical outcomes. The current research in a tertiary care centre explored the comprehensive approach for early diagnosis of the illness and prompt initiation of therapy.

CASE REPORT

A 10 year old boy, student of second standard, belonging to low socioeconomic status family residing in urban locality, was brought by his mother to adolescent health clinic with chief complaints of poor scholastic performance and strange behaviour for the past two years. The child was born out of non-consanguineous marriage and was first in birth order.

The child was apparently normal until 8 years of age, to

start with, mother noticed a gradual decline in his academic performance over a period of two years. He started receiving complaints from the class teacher that his child was not actively participating in school activities along with reduced interaction with his peers. At home, he wouldn't allow anyone except his mother near him. He refused to eat food along with family members and prefered to be lonely most of times, laughing and muttering to himself. When the child was confronted upon his reduced interaction with his friends at school and home, he appeared to be apathetic and lost in his thoughts most of the time. His mother also noticed that he was not responding to call, not maintaining any eye contact. The child appeared to have lost his language skills which he attained previously evidenced by terse speech. His teachers also complained that occasionally he picks up fights with the peers, became aggressive for no reason, that had become uncontrollable over the past few months. There was also history of bullying at school. The boy's mood frequently changed from cheerful to sad and irritability. Upon further questioning the mother, she expressed her child's sleep latency was increased with a decreased duration. She also added that the boy heard some voices conversing, commenting and shouting with someone who was not around him.

Mother expressed her difficulty in providing him bath, trimming his hair, cutting nails and feeding. The boy looked untidy most of times. There was no history of visual hallucinations, repetitive behaviours, catatonia and seizures. Bowel and bladder habits were regular.

Child had multiple hospital admissions and school absenteeism in the past for the current illness. Child suffered paternal loss at 6 years of age. Later, he was exposed to harsh treatment from step father for his behavioural abnormalities.

Mother had fever with rash during her 1st trimester. She was immunized with TT, took iron and folic acid supplementation as per norms, but had poor weight gain during her pregnancy. She had no history of substance abuse or drug intake during her course of pregnancy.

The child was born at term with a birth weight of 1.8 kilograms and cried immediately after birth. He was admitted in NICU in view of symmetric intra uterine growth retardation. There was no significant family history of psychiatric disorders. The boy was immunized regularly as per IAP schedule.

Child development was normal until 8 years of age, later significant regression was noted in the language domain. Upon general examination, child was conscious and coherent. He had no dysmorphic facies. There were no neurocutaneous makers over the body. Microcephaly was present, with normal neck and spine. Limbs and posture were normal. Vitals were stable.

Child's anthropometry revealed weight and height for age at 50th percentile. Body mass index was normal. Head circumference was below the 3rd percentile.

Cardiovascular, respiratory and gastrointestinal systems were normal. A detailed CNS examination was done that showed significant impairments in higher mental functions; as evidenced by poor attention, concentration, memory, judgement, rapport, insight, speech and language. IQ assessment (needed multiple assessments as child was inattentive, not cooperative) revealed a borderline mental retardation (IQ-70). Appearance was poor, abnormal behaviour with apathetic, dull mood. Auditory hallucinations were present. No abnormalities were found in the cranial nerve, motor, sensory systems, cerebellum and autonomic nervous systems. Also, there were no meningeal signs of irritation.

Investigations

The child received a comprehensive workup, that revealed normal parameters in hemogram, metabolic panel, liver, renal and thyroid function tests, CSF examination and EEG. However, neuroimaging of brain revealed bilateral frontal cortical atrophy with corpus callosum thinning and ventriculomegaly (Figure 1 A and B). In view of therapy for severe IUGR during neonatal period and positive maternal antenatal history of fever with rash, poor weight gain during pregnancy, serum IgG and IgM rubella antibodies were done, that showed significantly elevated titres.

Differential diagnosis

In view of similarity of schizophrenia with pervasive developmental disorders, neurodegenerative disorders, Mood disorders and attention deficit hyperactivity disorders, it was difficult to differentiate the diagnosis.

Treatment

The child was diagnosed as early onset schizophrenia with predominant negative symptoms probably due to intrauterine infection and early childhood stressors. He was started on therapy with risperidone 2 mg/day initially and the dose was gradually increased to 4 mg/day along with psychosocial interventions.

Follow-up and outcome

At follow-up, child showed remarkable improvement in his behaviour. He became more engaged with his family members and also at school with peers. His mood improved. However, cognitive impairments continued, probably due to cortical loss. Studies demonstrated worse clinical outcomes in early onset schizophrenia than in adult onset displaying higher global disabilities with deteriorating course.[1]

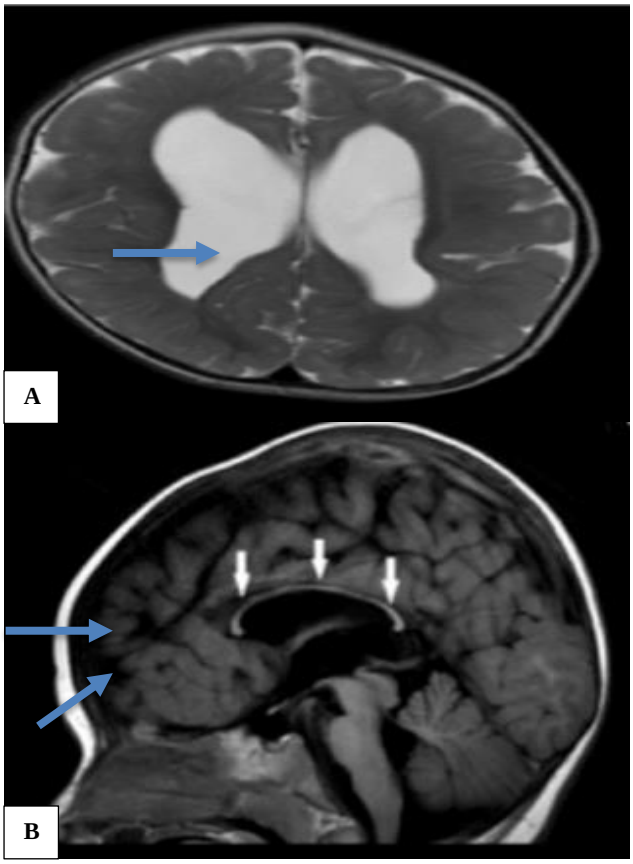

Figure 1 (A and B): MRI brain images in a 10-year-old boy with early onset schizophrenia with bilateral frontal cortical atrophy with corpus callosum thinning and ventriculomegaly; axial T2 weighted MRI showing ventriculomegaly (pointed by blue arrow), sagittal T1 weighted MRI showing frontal cortical atrophy (pointed by blue arrow) with corpus callosum thinning (pointed by white arrows).

DISCUSSION

Schizophrenia in childhood is extremely rare, sometimes undetected and its frequency was up to 50 times lower than in adulthood.[2] The 75% of children had a gradual development of the disease. When an individual had early-onset schizophrenia, they often have subtle mental delays such as language impairments, motor problems, and lack of social skills.[3] Typical positive symptoms included simple auditory hallucinations, while more frequent negative ones included flat or inappropriate mood and delusions that were less complicated than those of teenagers.[4] Most of the children had seen a significant decline in their capacity to execute at their previous level. In addition to genetic factors, environmental factors such as prenatal maternal infections may also play a role in schizophrenia pathogenesis. Schizophrenia in childhood can be dependably diagnosed using that criterion as that of the grownups and was predictors of schizophrenia or schizophrenia spectrum illnesses in adult years. Schizophrenics have significantly smaller frontal lobes, as well as smaller cerebrums and craniums, and these findings are consistent with some type of early developmental abnormality associated with prominent negative symptoms and cognitive impairment, as reported by Andreasen et al in their study of 38 schizophrenics and 49 healthy controls.[5] Computerized axial tomography of the brain, as reported by Johnstone et al showed increased cerebral ventricular size as a highly significant predictor of cognitive impairment.[6] In children with schizophrenia, a poor outcome is often linked to more severe developmental problems, which underlines necessity of long-term pharmacological and behavioural therapy.[7-10]

CONCLUSION

According to the neurodevelopmental theory of schizophrenia, environmental or genetically programmed events in utero disrupt the development of brain structure and function, and this case report underscores the importance of prenatal maternal infection prevention. Studying patients like these might help us better understand how schizophrenia arises. However, further study is required to determine the underlying causes of early onset schizophrenia.

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