CASE REPORT

Poststroke psychosis in an 8-year-old child with moyamoya disease

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SUMMARY

Psychiatric illnesses following cerebrovascular accidents has been a popular area of research in recent times. Though poststroke anxiety and mood disorders including depression have been reported often, reports of poststroke psychosis have been considerably rarer. This case report documents an even rarer occurrence of a psychotic disorder in an 8-year-old child with moyamoya disease following a cerebrovascular accident. The child was managed with low-dose atypical antipsychotic, risperidone and the psychotic symptoms eventually were under control around 3 weeks following the initiation of psychotropic medication.

BACKGROUND

The moyamoya disease is a rare, progressive, occlusive disease of the cerebral vasculature with particular involvement of the circle of Willis.¹ This disorder increases the propensity for cerebrovascular accidents that are the third leading cause of death in developed countries.² In addition to the mortality associated with cerebrovascular accidents, the subsequent psychiatric morbidity and the associated disability in survivors have been a matter of interest worldwide. Neuropsychiatric disorders have been reported in 20–60% of such patients and the most common manifestations include depressive disorder, anxiety disorders and other cognitive disorders.³ However, there have been only a few case reports documenting the onset of a psychotic disorder following a cerebrovascular accident. The prototype patient is an elderly patient who developed psychotic symptoms within a few days of the event lasting for a maximum of a few months.⁴ There has been no case report, to the best of the author's knowledge, documenting psychotic symptoms following stroke in a young child. Here, we report an 8-year-old boy with moyamoya disease who developed psychotic symptoms following a cerebrovascular accident.

CASE PRESENTATION

An 8-year-old boy was referred from neurology outpatient department (OPD) to psychiatry OPD of our tertiary care centre with symptoms of irritability and frequent anger outbursts, suspiciousness against other children, muttering to self and reporting of hearing voices of cartoon characters accusing or scolding the child at a frequency of at least 3–4 times/day lasting for more than 20 min each. He refused feeding and was uncooperative for self-care and grooming activities. His sleep was disturbed with the total sleep duration reduced to only

3–4 h. The child would appear vigilant during the night and would turn aggressive frequently against his family members. He would often ask his family members to warn others not to trouble him and would not elaborate further when enquired by them. The onset of symptoms was acute around a fortnight ago. The symptoms had evolved over this period with initial symptoms of suspiciousness, irritability followed by frequent anger outbursts and muttering to self with report of hearing voices not heard by others. There was no family history of psychiatric or neurological illness except the history of stroke in his maternal grandfather at 65 years of age. The child is the second of the three siblings and the other two siblings are normal. The child was diagnosed with moyamoya disease 1 year ago following multiple episodes of sensory deficits that would self-resolve. The child had developed left-sided haemiparesis abruptly around a couple of days ago. On investigation, MRI revealed ischaemic changes in left middle cerebral artery region. The patient was managed on lines of an ischaemic stroke with anticoagulant agents by the neurologist. From the following day, the patient was observed to exhibit the aforementioned symptoms. This reporting of behavioural abnormalities and hearing voices immediately alerted the neurologist to refer the patient to psychiatric OPD after necessary counseling regarding the possibility of a comorbid psychiatric illness. His Brief Psychiatric Rating Scale (BPRS)⁵ score was 43 at the time of reporting to psychiatric OPD.

INVESTIGATIONS

The child was diagnosed with moyamoya disease based on classical angiographic findings including bilateral fine network of vessels at the base of the brain giving a fuzzy appearance and leptomeningeal anastomoses.

DIFFERENTIAL DIAGNOSIS

A diagnosis of organic psychosis was entertained. An independent psychotic disorder was ruled out considering the onset and course of illness; absence of family history of psychiatric illness and history of psychiatric symptoms.

The differential diagnosis for moyamoya disease included the syndromes of mitochondrial encephalomyopathy, lactic acidosis and stroke-like episodes (MELAS); homocystinemia; temporal/giant cell arteritis and mitochondrial cytopathies. These disorders were ruled out based on clinical presentation, serum homocysteine levels and brain MRI.

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TREATMENT

The child (weight 23 kg) was started on low-dose risperidone (0.5 mg/day increased to 2 mg/day in 3 weeks following minimal response), an atypical antipsychotic following which the patient gradually started to show improvement in his psychotic and behavioural symptoms. The sleep normalised initially followed by reduced hallucinatory behaviour, irritability and anger outbursts. He also started to engage himself in self-care activities. The patient was gradually more manageable by the family members. The BPRS score was 9 around 3 weeks following the initiation of psychotropic medication.

OUTCOME AND FOLLOW-UP

The patient was thereafter maintaining well during the subsequent 3-week period during which hallucinatory behaviour and anger outbursts were minimal as reported by the family members. Following this period, the patient was lost to follow-up.

DISCUSSION

Poststroke depression and anxiety symptoms have been widely reported in the literature. However, poststroke psychosis which is considerably rarer has not been adequately reported in the literature. Management of neuropsychiatric comorbidity following stroke forms an essential component of a comprehensive management plan for the patient.⁶ These disorders complicate recovery and rehabilitation of poststroke patients and predict poorer long-term prognosis. Also drug compliance and regular follow-up with the physician are variables likely to be affected by the psychiatric comorbidity. Adequate liaison between the treating neurologist and the psychiatrist forms the cornerstone of management of neuropsychiatric comorbidity which if untreated results in significant disability and worsening of quality of life. Adequate and aggressive management of psychiatric comorbidity is warranted in these cases to hasten overall recovery. This case report is the first report documenting poststroke psychotic symptoms in a young child and the aforementioned requirement of adequate management of neuropsychiatric comorbidity cannot be understated in this special population as well.

Low-dose atypical antipsychotics are generally the most widely prescribed treatment option in this clinical presentation.

Our case highlights the possibility of effective management of psychotic symptoms following antipsychotic therapy in such cases.

Learning points

- ▶ Poststroke neuropsychiatric morbidity needs to be managed effectively by adequate liaison with a psychiatrist for a better long-term outcome.
- ▶ If untreated, neuropsychiatric morbidity predicts poor quality of life in poststroke patients.
- ▶ Low-dose atypical antipsychotics would be the agents of choice in these cases.
- ▶ Sensitisation of neurologists regarding psychiatric symptomatology is essential for prompt referral and early intervention.
- ▶ The possibility of onset of psychiatric disorders secondary to even childhood neurological illnesses needs to be considered.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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