CASE REPORT

Psychosis as Initial Clinical Presentation for Cerebral Venous Thrombosis: A Case Report

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Abstract

Cerebral venous sinus thrombosis (CVT) is a rare cause of stroke affecting the venous circulation. It typically presents with the features of raised intracranial pressure (ICP) or cerebral edema caused by the thrombus obstructing the blood drainage from the brain. The presentation of CVT with behavioral change amounting to psychosis has rarely been reported. We aim to report the case of a young female, who presented and was admitted for the acute onset psychotic symptoms, and was later found to have CVT on brain imaging. The presentation, management and the outcome of the case have been discussed. We conclude that the physicians and psychiatrists must have a high index of suspicion for the organic lesion in the cases presenting with acute psychosis.

Keywords: Case report, Cerebral venous thrombosis, Neuroimaging, Psychosis.

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Introduction

Cerebral venous sinus thrombosis (CVT) is a rare cause of stroke accounting for less than 1% of the cases.[1] Unlike arterial strokes it is commoner in the younger age group and females.[2] The usual site of thrombosis in CVT is the junction of a cerebral vein with the large sinuses.[3] This thrombus obstructs blood drainage from brain tissue and increases venous and capillary pressure leading to cerebral edema.[3] The consequent decrease in the cerebrospinal fluid (CSF) absorption due to the occlusion of dural sinuses may result in elevated intracranial pressure (ICP).[4]

The risk factors for CVT include hypercoagulable state, dehydration, adjacent infections, use of hormone replacement therapy or oral contraceptives, pregnancy and puerperium. This diagnosis of CVT is often delayed, because of a highly variable clinical presentation.[5] It frequently presents with headache, craniofacial pain, focal seizures with or without secondary generalization or unilateral or bilateral paresis.[6] The clinical presentation of CVT with psychiatric manifestations such as psychosis, mood disturbances or severe anxiety is relatively uncommon.[7] We aim to report a rare case of CVT in a young woman who presented with the clinical features of acute onset psychosis.

Case Description

The index case, a 22-year-old married woman living in a village in a central Indian district, was brought by her husband to the psychiatry clinic at a rural tertiary health care center with complaints of altered behavior for eight days. As per the history, the change in her behavior started with the reduction in her interaction with the family members. She was found talking to herself and gesturing in the air with nobody around. She had stopped doing the household activities such as cooking or cleaning. Over the next few days, her food intake, as well as self-care, reduced drastically. The patient had no previous history of known psychiatric illness, trauma, loss of consciousness, headache or visual disturbances. The patient was evaluated by the psychiatry consultant in the outpatient department and was provisionally diagnosed with acute onset psychotic disorder. The routine laboratory investigations and the plain computed tomographic (CT) scanning of the brain done after the admission did not reveal any abnormalities. The patient was started on a low dose of risperidone and lorazepam to treat the behavioral symptoms. However, on the daily mental status examinations, the patient showed minimal improvement over the next eight days. One week after the admission, she started having additional complaints of headache, vomiting and showed confused behavior and a perplexed affect. She was referred to a physician to rule out organic involvement. The detailed neurological examination of the patient revealed altered sensorium, disturbed orientation to time and place and person, decreased attention span and memory impairment. Her speech was spontaneous, irrelevant and incoherent. There was no abnormalities on cranial nerves examination. Sensory examination was normal. Motor examination revealed paresis in right upper limb with normal tone and nutrition. Superficial and deep tendon reflexes as well as cerebellar examination did not reveal any abnormalities.

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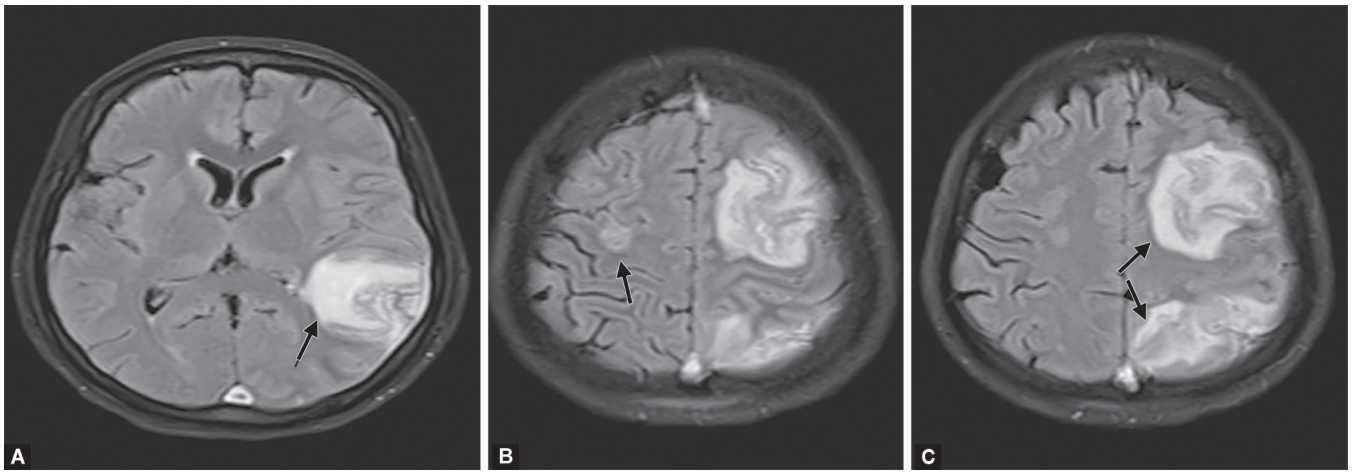
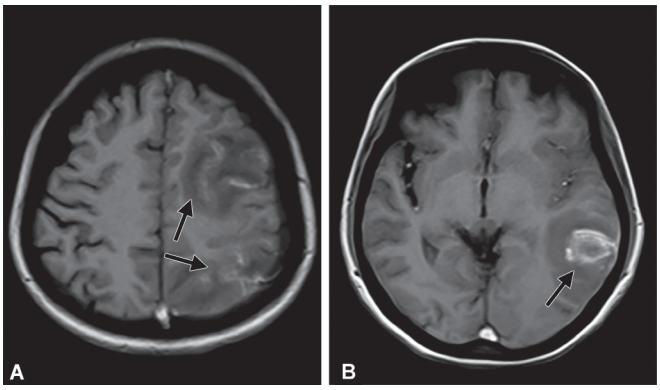
Patient consent statement: The author(s) have obtained written informed consent from the patient for publication of the case report details and related images.



Figs 1A to C: (A) T2W image suggesting venous infarct in left temporo-parietal lobe; (B) T2W image suggesting venous infarct in right frontal lobe; (C) T2W image suggesting venous infarct in left fronto-temporal lobe



Figs 2A and B: (A) FLAIR image of fronto-temporal lobe; (B) FLAIR image of temporo-parietal lobe

Hence suspecting organic lesion, the patient was transferred to the medical intensive care unit for further evaluation. Cerebrospinal fluid examination was done which revealed normal glucose and proteins with normal cytology. Fundus examination, electrocardiogram and 2D ECHO were normal.

The contrast-enhanced CT scan of the head revealed venous hemorrhagic and non-hemorrhagic infarcts in left fronto-temporo-parietal lobes secondary to cerebral venous sinus thrombosis in left transverse, left sigmoid, superior sagittal sinuses, torcular heterophili and cortical veins in left fronro-parietal lobes. There was a subtle hypodense area in the right temporal lobe likely to be an acute infarct. The magnetic resonance (MR) imaging brain revealed hemorrhagic infarcts in left temporo-parietal lobe and left fronto-parietal lobes; along with non-hemorrhagic infarct in right frontal lobe, likely to be secondary to CVT. The MR venography showed enhancing altered signal intensity filling defect noted within the lumen of superior sagittal sinus, torcular herophili, left transverse sinus, left sigmoid sinus, right sigmoid sinus, right jugular vein and cortical veins along superior sagittal sinus in fronto-parietal regions appearing heterogeneously hypointense on T1W/T2W/FLAIR (Figs 1 to 3). The diagnosis of CVT was thus confirmed. She was managed using injectable anticoagulants (enoxaparin) and was discharged after two weeks when she clinically improved.

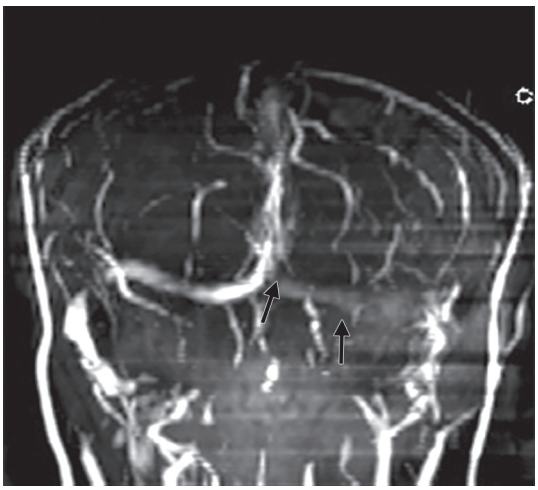


Fig. 3: Magnetic resonance venography suggestive of altered signal intensity filling defect

Discussion

Venous thrombosis differs from arterial thrombosis in the fact that the former usually affects younger individuals and female sex.[2] Our patient being a 22-year-old woman matched the demographic characteristics of commonly affected individuals. She did not have any of the risk factors of CVT such as coagulopathy, dehydration, use of thrombogenic medicines and was not pregnant. The origin of CVT in the absence of any of the risk factors has been reported in less than 10% of the cases.[6] The onset of symptoms in CVT is abrupt (less than 48 hours) in nearly half of the reported cases and acute or subacute (two to four weeks) in the remaining half.[8] Our patient belonged to the latter category with the onset of altered behavior over nearly two weeks followed by the development of intense headache and altered sensorium.

The clinical presentations of CVT can be broadly categorized into those due to increased ICP, or effect of mass lesion on the localized area of the brain or both.[6] In the present case, the ICP was normal and the venous infarct was largely localized to the frontal lobe. The dorsolateral prefrontal cortex mediates the executive functions whereas the ventromedial prefrontal cortex regulates

emotions.[9,10] The presentation of withdrawn social behavior and diminished functionality in the present case could be attributable to the pressure on the prefrontal cortex due to the thrombus. The early venous obstruction in CVT is temporarily compensated by the dilation of cerebral veins via collaterals. However, if the obstruction is not relieved, the pathology may further evolve to raise the ICP and present with headache and focal neurological deficit.[11] Similar picture was noted in our case with initial presentation as behavioral change for about two weeks. This was followed by intense headache and right upper limb paresis when she was transferred to the intensive care unit.

There are some case reports in the literature where psychotic symptoms like delusions and hallucinations are present in cases with CVT.[12,13] One case report of catatonia as presenting picture of CVT has been reported.[14]

Cerebral venous thrombosis has a better prognosis and a lower mortality rate than arterial strokes. Early diagnosis due to improved diagnostic technologies and treatment with anticoagulants has been instrumental in the improved outcomes.[15] In our case, the patient was immediately diagnosed within two weeks and was started on injectable enoxaparin. Her symptoms remitted completely on treatment and no residual neural deficit was observed by the physician when she came for follow-up examination after 15 days. Previous research suggests that a majority (80%) of CVT patients have difficulty in returning to previous work because of residual neural deficits.[15] Hence we conclude that the physicians, as well as psychiatrists, must bear in mind the possibility of organic lesions in the patients having acute onset behavioral change.

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