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Acute psychosis with a favorable outcome as a complication of central pontine/extrapontine myelinolysis in a middle aged man

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Abstract

Central pontine myelinolysis is a demyelinating condition affecting the pons characterized by an acute progressive quadriplegia, dysarthria, dysphagia, and alterations of consciousness. Pathologic features include prominent demyelination in the central pons with sparing of axons and neurons. This condition is usually associated with systemic disorders such as hyponatremia, chronic alcoholism, liver failure, severe burns, malignant neoplasms, hemorrhagic pancreatitis, hemodialysis, and sepsis. There are limited reports of psychosis in patients with central pontine/extrapontine myelinolysis (CPEM). We have described a case of CPEM with psychosis as a complication which recovered completely with treatment given for short duration using low dose atypical antipsychotic (quetiapine). We also discuss etiopathology and clinical outcome of psychosis in this rare neurological disorder.

Keywords: Central pontine myelinolysis, magnetic resonance imaging, quetiapine

INTRODUCTION

Central pontine myelinolysis (CPM) is a demyelinating condition affecting the pons characterized by an acute progressive quadriplegia, dysarthria, dysphagia, and alterations of consciousness. Pathologic features include prominent demyelination in the central pons with sparing of axons and neurons. This condition is usually associated with systemic disorders such as hyponatremia, chronic alcoholism, liver failure, severe burns, malignant neoplasms, hemorrhagic pancreatitis, hemodialysis, and sepsis.[1] In about 10% cases, CPM is associated with extrapontine myelinolysis (EPM) leading to the appearance of parkinsonian symptoms such as rigidity of limbs, bradykinesia, tremors, and decreased blinking.[2] Studies on CPM have mainly focussed on its etiology, neurological manifestations, radiological appearance, and outcome.[3–5] There is limited literature available on neuropsychiatric complications of central pontine/extrapontine myelinolysis (CPEM). Some of the behavioral manifestations that have been described so far are - personality change, inappropriate affect, emotional lability or incontinence, disinhibition, poor judgment, and delirium.[6,7] A few reports have also described occurrence of catatonic syndrome as a rare manifestation due to EPM.[8–10]

However, there are limited reports of psychosis in patients with CPEM.[11] We have described a case of CPEM with psychosis as a complication which recovered completely with treatment given for short

duration using low dose atypical antipsychotic (quetiapine). We also discuss etiopathology and clinical outcome of psychosis in this rare neurological disorder.

CASE REPORT

A 53-year-old male presented to emergency department with complaints of acute onset, generalized rigidity and tremors of all limbs, difficulty speaking and swallowing along with slowness of movements, masked facies, and decreased blinking. He was recently discharged from medical ICU after being managed for episodes of vomiting and decreased food intake associated with confusion, lethargy, disorientation to time and place. Also he developed two episodes of generalized tonic clonic seizures. His serum Na^+ was found to be very low (102 mEq/ml). He was also put on tab levetiracetam 1000 mg for seizure prophylaxis. At the time of discharge after 6 days of hospitalization his serum Na^+ was 131 mEq/ml. His hyponatremia had been corrected rapidly (by 13 mEq/ml in initial 8 h). He had undergone neurosurgery for non-functional pituitary macroadenoma 10 years back. He was not taking any hormone supplements. It was hypothesized that he might have been having chronic hyponatremia due to hypopituitarism. Patient developed rigidity and associated symptoms after 6 days of sodium correction. He also underwent magnetic resonance imaging (MRI) of the brain which did not reveal any abnormality except for chronic ischemic changes in the bilateral centrum semiovale.

Detailed assessment did not reveal psychiatric symptoms or any previous psychiatric illness nor was there any history of substance abuse. There was no family history of any psychiatric illness as well. From the history and review of medical records, a provisional diagnosis of CPEM was made. Neuroleptic malignant syndrome was ruled out as patient was not subjected to any antipsychotics in previous hospital as per records. Moreover, serum creatine phosphokinase (CPK) levels were normal. He was started on oral trihexyphenidyl (centrally acting anticholinergic) 1 mg TDS along with supportive treatment.

MRI brain was repeated on day 4 of admission (after 5 days of onset of symptoms) which showed pontine hyperintensities and bilateral basal ganglia (caudate and putamen) hyperintensities on T2W1 and FLAIR sequences confirming the diagnosis of CPEM (secondary to rapid correction of hyponatremia) [Figures 1 and 2].

Patient's symptoms started improving dramatically after the treatment was started. Hormone supplementation was started following reports of low Adrinocorticotrophic Hormone (ACTH), Thyroid Stimulating Hormone (TSH), Follicle Stimulating Hormone (FSH) and Leutinising Hormone (LH) levels. After 3 days of hospitalization, he developed behavioral symptoms for which psychiatric consultation was again taken. He was found to have disturbed sleep, irritability, suspiciousness toward a fellow patients in the ward and aggression toward treating resident doctors. He developed delusion of persecution and reference, claiming that a female patient on neighboring bed was passing insulting remarks to him indirectly when talking to others and was also teasing him through her gestures. He would appear restless and kept pacing in ward in anger. He was fully conscious and oriented with clear sensorium. No apparent mood symptoms could be noticed. He did not have any Schneiderian First rank symptoms. Possibility of any substance abuse in ward was ruled out. He was started on quetiapine 50 mg and dose was increased to 100 mg over a period of 3 days. He tolerated it well and his psychotic symptoms started improving over a period of 4 weeks. He was discharged from the ward after his extrapyramidal symptoms got completely resolved. During follow-up visits, trihexyphenidyl was tapered off over next 1 month and quetiapine was gradually tapered off over next 6 months without any new symptoms. The patient achieved complete remission and started attending office subsequently.

DISCUSSION

In this case, diagnosis of CPEM could be established both clinically (presence of dysphagia and dysarthria suggested pontine involvement and presence of generalized rigidity, bradykinesia, tremors, and related parkinsonian symptoms indicated extrapontine lesion) as well as radiologically. We did consider the

possibility of levetiracetam-induced psychosis but ruled it out given the fact that this drug was used in a low dose (1000 mg/day; approximately 166 mg/kg) and patient's psychotic symptoms subsided with quetiapine without the need to stop Levetiracetam.[12] Another possibility was trihexyphenidyl-induced psychosis. However, it was used at very low dose in the present case (3 mg/day). Additionally, the nature of trihexyphenidyl psychosis, as described in literature, is toxic psychosis.[13]

Thus, the psychotic symptoms that appeared could be attributed to brain lesions that are associated with CPEM. This case highlights that psychotic symptoms could occur as a complication of CPEM and can be managed effectively. These features subside shortly with resolution of neurological signs and symptoms associated with the brain lesions. The case in study hints at the possible role of basal ganglia in etiopathogenesis of psychotic symptoms given the fact that Huntington, Wilson, and Parkinson disease, which all have basal ganglia involvement are also associated with psychotic symptoms, at times. The present case also reiterates the fact that in CPEM, the outcome is not always dismal. Additionally, correction of hyponatremia should be paced appropriately and should be limited to about 25 mmol/l during the initial 24-48 h.[14]

Footnotes

Source of Support: Nil

Conflict of Interest: None declared.

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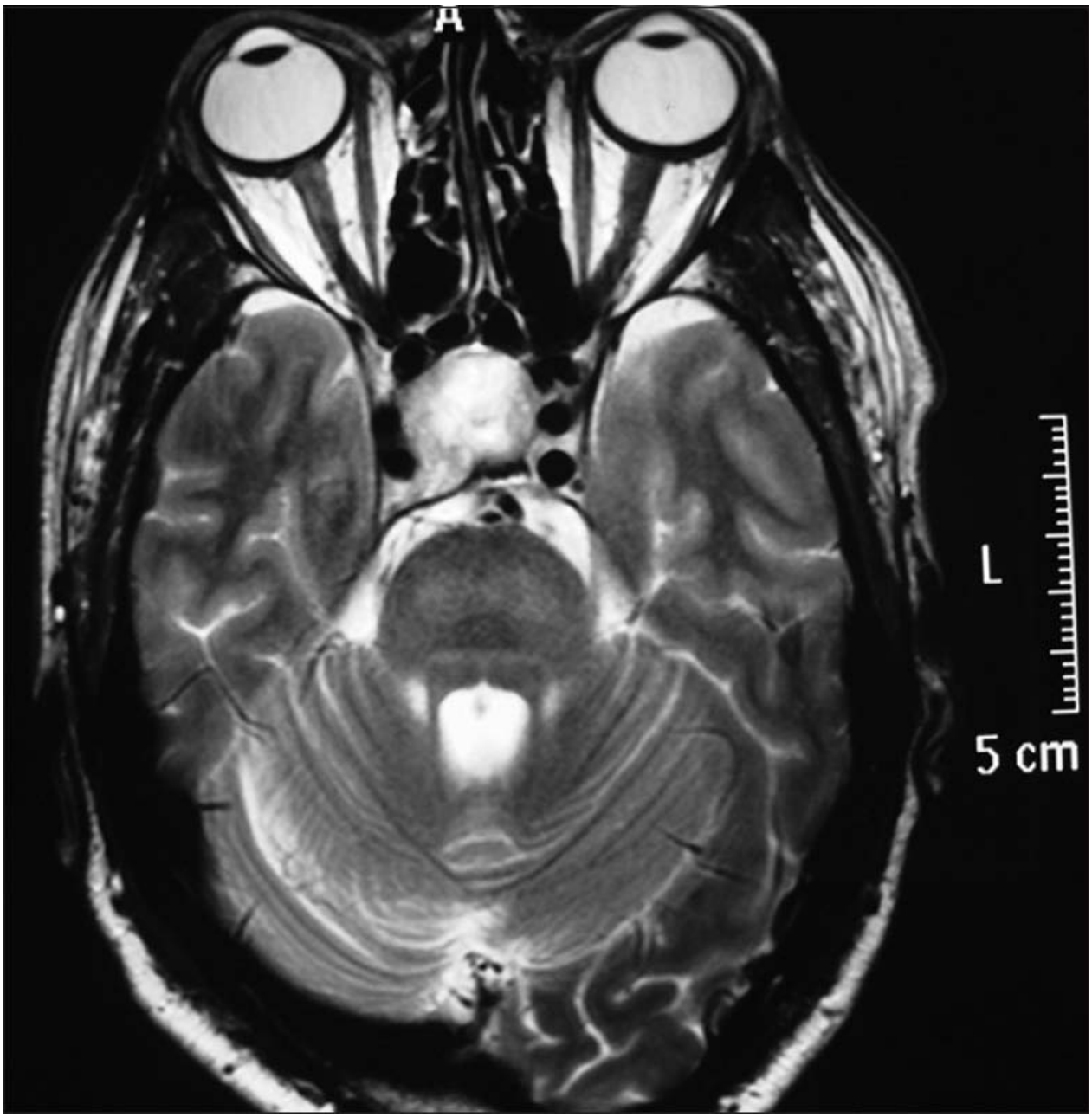
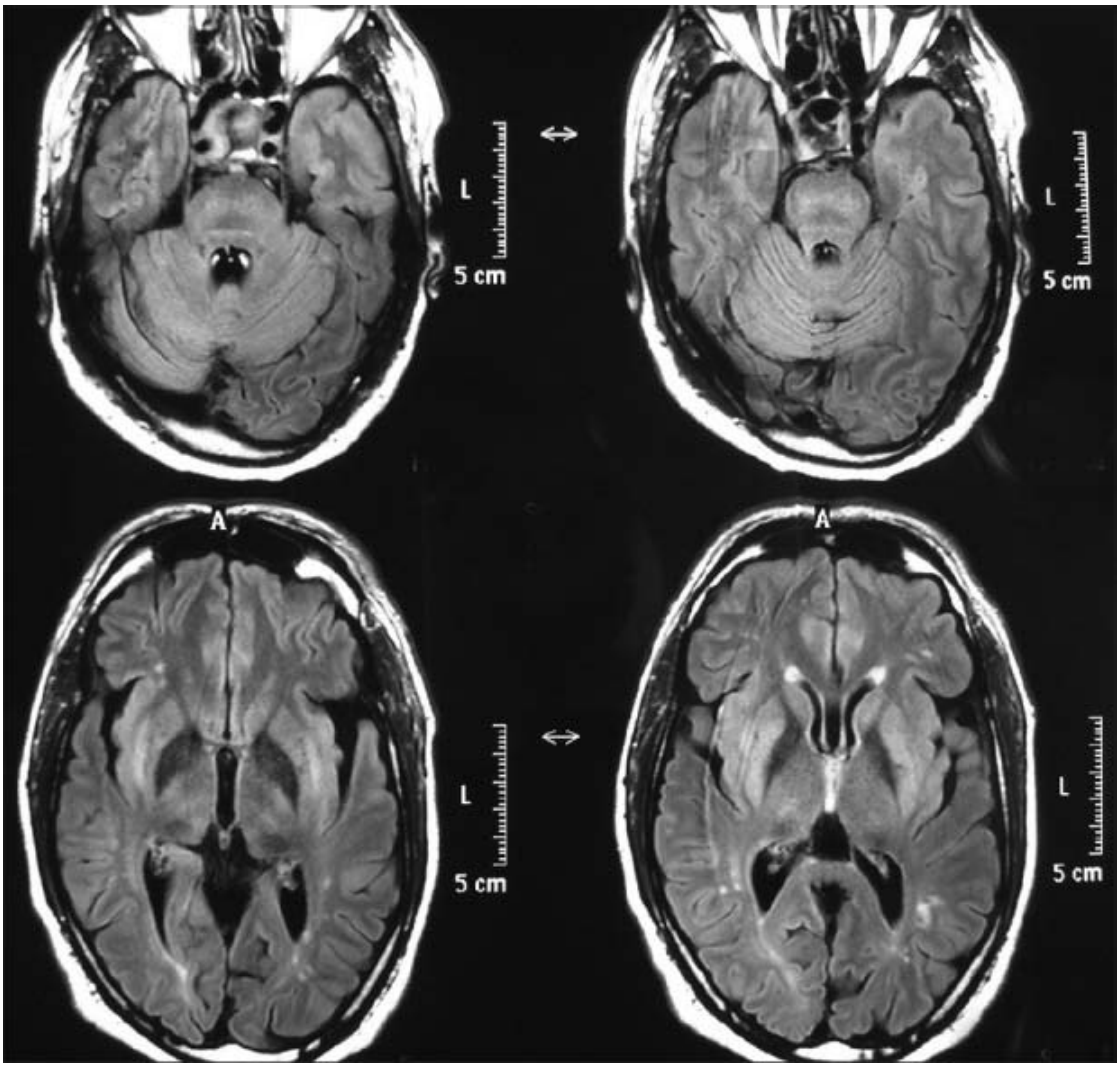
Figures and Tables

Figure 1



Pontine hyperintensities and bilateral basal ganglia (caudate and putamen) hyperintensities on T2W1 and FLAIR sequences

Figure 2



Bilateral basal ganglia (caudate and putamen) hyperintensities on T2W1 and FLAIR sequences confirming the diagnosis of central pontine/extrapontine myelinolysis