on the 3rd day of treatment and subsequently had a dramatic recovery.

After 4 weeks, a repeat MRI demonstrated reduced size of the cavity. DWI revealed no hyperintensity of abscess cavity (Figure 2a) or ventricles. The ADC maps (Figure 2b) were normal. Post contrast T1-weighted images showed enhancement of collapsing abscess and mild enhancement of the right ventricular wall indicating persistent ventriculitis. After 8 weeks, the abscess cavity had significantly reduced in size with no abnormal signal intensity in the ventricles. ADC values of the ventricles were similar to that of the general CSF spaces. Post contrast scans showed mild enhancement in the region of the abscess cavity only.

At 2 years the patient has no neurological signs or symptoms and is on follow-up.

**Discussion**

Intraventricular rupture is often a fatal complication of an intracerebral pyogenic abscess and is associated with a high mortality rate of 85%.1 The management of brain abscesses is controversial, although the uniform opinion is that early diagnosis and prompt surgical intervention is associated with a better outcome.1 The present case was successfully managed with conservative medical treatment with regular imaging follow-up.

DWI of brain has recently been studied with great interest in the characterization of brain abscess and its differentiation from other similar appearing lesions on conventional MR imaging.4,5 The abscesses are easily distinguished on diffusion weighted images from a cystic tumor by their high signal intensity and decreased ADC as compared to low signal intensity areas with high ADC in cases of cystic tumors.4,5

DWI has also been used in serial follow-up of a pyogenic abscess which demonstrated changing ADC values during treatment, but the duration of follow-up was only for 3 weeks.6 In the present case, the patient was followed up for 8 weeks and was on conservative management. At 8 weeks, there was no hyperintensity in the abscess cavity and ventricles suggesting resolution of the purulent contents.

In conclusion, DWI and ADC maps best demonstrate intraventricular rupture of an abscess. The presence of pus elsewhere can also be detected by DWI. Serial DWI with ADC maps during the management of cerebral abscess helps in assessing the response to treatment.

**References**


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**Vitamin B12 deficiency presenting with an acute reversible extrapyramidal syndrome**

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Vitamin B12 deficiency usually presents with pernicious anemia or various neuropsychiatric manifestations. Commonly seen neuropsychiatric manifestations include large fiber neuropathy, myelopathy (subacute combined degeneration of the spinal cord), dementia, cerebellar ataxia, optic atrophy, psychosis and mood disorders. The present report highlights an unusual presentation of vitamin B12 deficiency- acute onset extrapyramidal syndrome in a 55-year-old man. The patient presented with a 10-day history of slowness of all activities including a slow gait, mild tremors of hands and low volume speech. On examination, he had features of mask-like facies, reduced blink rate and cogwheel rigidity. He was investigated for the possible causes and was found to have laboratory features of vitamin B12 deficiency. Other causes for acute onset parkinsonism were excluded by appropriate investigations. He showed a dramatic improvement following treatment with intramuscular vitamin B12 injections. At a five-year follow up, he was found to be functionally independent with no neurological deficits.

**Key Words:** Vitamin B12, Neuropsychiatric manifestations, Acute extrapyramidal syndrome, Parkinsonism

### Introduction

Vitamin B12 deficiency resulting in various neuropsychiatric abnormalities such as neuropathy, myelopathy, myeloneuropathy, dementia, cerebellar ataxia, optic atrophy, psychosis and mood disturbances is well known. In addition, recurrent seizures have been reported as a rare manifestation of vitamin B12 deficiency. Extrapyramidal system involvement in the form of involuntary movements is common in infants, whereas similar involvement in adults has been described only recently. However, acute onset parkinsonism has not been reported and this report describes one such patient.

### Case Report

55-year-old man presented with bradykinesia and tremors of hands of ten days duration. There was no history of seizures or altered sensorium. On examination, he had expressionless face and reduced blink rate. Higher mental functions were normal. Speech was hypopneic and saecolic eye movements were slow. He had cogwheel rigidity of all limbs and resting tremors of both hands. Tendon reflexes were normal except for sluggish ankle jerks. Plantar reflexes were extensor. Sensory, cerebellar and autonomic systems were normal. His Hoehn and Yahr score was stage III.

There was no preceding history of encephalitis or prior use of antipsychotic or antieptic medications. He had no risk factors for atherosclerosis. There was no history of exposure to carbon monoxide or organophosphate compounds. He consumed non-vegetarian diet.

Investigations were as follows: Hemoglobin was 10.2 G%, reticulocyte count was 0.1% and mean corpuscular volume (MCV) was 118 fl. Blood picture showed hypersegmented neutrophils and bone marrow showed features of severe megaloblastic anemia. Serum vitamin B12 level was 3 pg/ml and folate was 98 ng/ml. Anti-intrinsic factor antibody was positive and antirn bile biopsy showed features of atrophic gastritis. Work up for malabsorption was negative. Serum parathormone, ceruloplasmin and thyroid function tests were normal. HIV (ELISA) was negative. CT scan of the brain and cerebrospinal fluid analysis were normal.

A diagnosis of acute onset parkinsonism with mild myeloneuropathy secondary to vitamin B12 deficiency was made. He was started on vitamin B12 injections at a dose of 1000 mg/day with which he showed improvement within three days. At the time of discharge at seven days, he had mild bradykinesia, no tremors and was functionally independent. He was advised to continue injections once a week for four weeks and monthly thereafter. He has been followed up periodically since then and at the last follow up at five years, he had no neurological deficits.

### Discussion

This patient presented with acute onset parkinsonism secondary to vitamin B12 deficiency. Typical laboratory features supported the diagnosis and other causes of acute parkinsonism (Table 1) were excluded. Marked improvement with vitamin B12 therapy further strengthened the diagnosis.

Extrapyramidal involvement due to vitamin B12 deficiency in adults is rare and previously reported features include myoclonus-like involuntary movements and chorea and focal dystonia. There are no previous reports of acute parkinsonism due to vitamin B12 deficiency. The mechanism of extrapyramidal involvement in vitamin B12 deficiency is incompletely understood. However, indirect evidence may be obtained from two facts. Firstly, methylmalonic acidemia (MMA), an inborn error of metabolism, usually presents with acute extrapyramidal syndrome in infants, and some cases respond to vitamin B12 therapy. Brain imaging and autopsy studies in MMA have shown symmetrical involvement of basal ganglia.

In conclusion, acute onset extrapyramidal syndrome can be

<table>
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<tr>
<th>Cause</th>
<th>Investigation/diagnosis</th>
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<tr>
<td>Drugs</td>
<td>Prior antipsychotic or antieptic usage</td>
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<tr>
<td>Basal ganglia infarcts</td>
<td>CT/MRI scan of brain</td>
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<td>Acute viral encephalitis</td>
<td>Brain imaging, CSF viral titers</td>
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<td>Wilson’s disease</td>
<td>K-F ring in cerea, serum ceruloplasmin</td>
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<td>Carbon monoxide toxicity</td>
<td>History of exposure</td>
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<td>Organophosphorus toxicity</td>
<td>History of exposure</td>
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<td>(K-F ring=Kayser-Fleischer ring)</td>
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a rare manifestation of vitamin B12 deficiency, which is reversible with therapy. Serum B12 levels should be checked in patients who do not have an obvious cause for an acute extrapyramidal syndrome.

References


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