CASE REPORT

# Delirious Mania in a Case with Multiple Demyelinating Foci

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## Abstract

**Introduction:** A serious but underdiagnosed neuropsychiatric illness called delirious mania is described by the sudden onset of delirium, mania, and psychosis. Demyelination is the term used to describe the loss of myelin while axons are largely preserved. Depending on the site of insult, various neuropsychiatric manifestations may appear. We are discussing a case that was presented initially as mania but after a proper evaluation it was found to be a case of delirious mania along with multiple demyelinating foci in brain imaging, which had an impact on the presentation of this case.

**Case description:** This is a case of 19-year-old man presented with complaints of talkativeness, tall claims, increasingly bizarre, and disorganized behavior with intermittent restlessness for the last 2 months. After a few trials with psychotropics, the patient started showing improvement with a combination of mood stabilizers and antipsychotics.

**Conclusion:** Recent reports suggest that delirious mania may constitute up to 15% of all acute mania cases. When delirious mania is unrecognized or improperly treated, it can progress rapidly in severity and can become life-threatening. Like in our case, patients presenting with manic symptoms should also be properly evaluated for other underlying pathology which may have a significant impact on patient's well-being.

**Keywords:** Bells mania, Delirious mania, Demyelination, Psychosis.

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## Introduction

A serious but underdiagnosed neuropsychiatric illness called delirious mania is described by the sudden onset of delirium, mania, and psychosis. Intense hyperactivity, emotional instability, paranoia, strange hallucinations, and disorientation are all features of this time-limited but possibly lethal form of acute psychosis.

Mania and delirium are traditionally thought of as two distinct syndromes. But delirious mania, sometimes known as mixed mania and delirium, has a long history. Calmeil[1] reported it initially, followed by Bell,[2] who gave it the eponymous name Bell's mania. Delirious mania does not exist in the diagnostic category in contemporary nosologies like the Diagnostic and Statistical Manual of Mental Disorders (5th edition) (DSM 5)[3] and the International Classification of Diseases (10th edition) (ICD 10).[4]

Six out of twenty acutely manic individuals in a case series Carlson and Goodwin published were spatially not oriented to time and place.[5] In a group of bipolar patients who were being treated in mental hospitals, Ritchie et al.[6] reported a 35.5% rate of delirium, which is quite significant.[6]

What is more, failing to diagnose and proper treatment can have catastrophic results. Over the past century, it is potentially fatal in nature consistently.[7] Although the fatality rate is unknown, it is significant in a classic case series of Bell containing 40 patients,[8] around 70% passed away during treatment in the hospital. If the improper medication is administered, delirious mania may progress more quickly to its malignant, life-threatening form,[9] but the initiation of appropriate management of it may reduce fatality.

Here, a case has been discussed which was presented initially as mania but after a proper evaluation it was found to be a case of delirious mania along with multiple demyelinating foci in brain imaging, which had an impact on the presentation of this case.

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## Case Description

This is a case of 19-year-old unmarried man who was brought to the casualty by his parents. The patient was presented with complaints of talkativeness, tall claims, increasingly bizarre, disinhibited, and disorganized behavior, and a wandering tendency with intermittent agitation, which was the insidious onset and gradually progressive over 2 months. According to his parents, all his symptoms started with increased talkativeness and he spent money excessively chewing gutkha.

Prior to this, there was no past history of psychiatric illness or no family history of any psychiatric illness. There was no such improvement after multiple faith healing and private psychiatric consultation, so he was brought to the casualty. The patient presented with stable vitals and a normal computed tomography (CT) brain report and was admitted to the male psychiatry ward.

During the first 2 days of the hospital stay, he appeared agitated and confused at times with the very little amount of sleep. On

treatment with parenteral antipsychotics and oral mood stabilizer, he started showing gradual improvement except for episodic disinhibited behavior, exhibiting unusual behavior like remaining naked in infront of female staff (claims of having a "legend penis") and restlessness at times. His general physical and neurological examination did not reveal any abnormality.

On mental status examination rapport was partially established with inadequate eye-to-eye contact, increased psychomotor activity, increased tone and quantity of speech with decreased reaction time and presence of pressure of speech with irrelevancy at times, affect being labile with grandiose and persecutory delusion without any perceptual disturbances, his attention was drawn and concentration ill sustained, fluctuating orientation to time, place and person, with impaired judgment and grade I insight.

His routine blood investigations and CT scan brain reports were normal. Magnetic resonance imaging (MRI) brain imaging reveals that T2 flair hyperintensities foci are noted in the periventricular area deep white matter adjacent to the frontal horn of the right lateral ventricle, right partial lobe, bilateral occipital lobe, showing no diffusion restriction in diffusion-weighted imaging (DWI)/apparent diffusion coefficient (ADC) sequence and no significant enhancement on post-contrast, suggestive of demyelinating foci.

He was responding to Tab olanzapine 15 mg and Tab sodium valproate 1.2 gm in divided doses, was discharged with the same medication, and advised for neurology consultation and regular follow-up at psychiatry outpatient department (OPD).

## Discussion

Because most of the research on delirious mania is anecdotal, there is little evidence to support management. As with every delirium or organic manic episode, underlying organic causes must be addressed and rectified.[10] The use of a second antipsychotic and a mood-stabilizer in combination, as well as high-dose benzodiazepines or electroconvulsive therapy (ECT), are supported by previous reports of delirious mania.[11,12]

Demyelinating foci are one of the most common causes of white matter hyperintensities (WMH) on MRI, which may present with various neuropsychiatric symptoms like delirious mania. The differentials of bells mania include numerous neurological, medical, and toxicological causes. From various studies, it was found that WMH is a clinically important marker of increased risk of stroke, dementia, mood disorder, and other neuropsychiatric complications with high mortality.

In our case, the patient was presented initially as having mania but after a proper evaluation found to be a case of delirious mania along with multiple demyelinating foci in brain imaging, and showed marked improvement on an adequate dose of second-generation antipsychotic and mood stabilized with a low dose of benzodiazepine. The patient was advised for a neurology consultation for the treatment of contributing underlying causes which will further prevent the neuropsychiatric complications.

## Conclusion

According to recent reports, up to 15% of all acute mania episodes may be caused by delirious mania. Delirious mania can rapidly worsen and become life-threatening if it is not detected or treated effectively. Like in our case, patients presenting with manic symptoms should also be properly evaluated for other underlying pathology, which may have a significant impact on patients' well-being.

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