



# Delirious mania: Presentation, pathogenesis, and management

## Rapid recognition is critical to avoid initiating inappropriate treatment

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### Acknowledgments

The authors express great gratitude to Professor Max Fink for his characterization of delirious mania. We also thank Dr. Fink for his continued guidance and support during the preparation of this manuscript.

### Disclosures

The authors report no financial relationships with any companies whose products are mentioned in this article, or with manufacturers of competing products.

doi: 10.12788/cp.0414

**D**elirious mania is a syndrome characterized by the acute onset of severe hyperactivity, psychosis, catatonia, and intermittent confusion. While there have been growing reports of this phenomenon over the last 2 decades, it remains poorly recognized and understood.<sup>1,2</sup> There is no widely accepted nosology for delirious mania and the condition is absent from DSM-5, which magnifies the difficulties in making a timely diagnosis and initiating appropriate treatment. Delayed diagnosis and treatment may result in a detrimental outcome.<sup>2,3</sup> Delirious mania has also been labeled as lethal catatonia, specific febrile delirium, hyperactive or exhaustive mania, and Bell's mania.<sup>2,4,5</sup> The characterization and diagnosis of this condition have a long and inconsistent history (*Box*,<sup>1,6-11</sup> *page 28*).

Delirious mania continues to be met with incertitude in clinical practice, and numerous inconsistencies have been reported in the literature. For example, some cases that have been reported as delirious mania had more evidence of primary delirium due to another medical condition or primary mania.<sup>12,13</sup> Other cases have demonstrated swift improvement of symptoms after monotherapy with antipsychotics without a trial of benzodiazepines or electroconvulsive therapy (ECT); the exclusion of a sudden onset questions the validity of the diagnosis and promotes the use of less efficacious treatments.<sup>14,15</sup> Other reports have confirmed that the diagnosis is missed when certain symptoms are more predominant, such as a thought disorder (acute schizophrenia), grandiosity and delusional ideation (bipolar disorder [BD]), and less commonly assessed catatonic signs (ambitendency, automatic obedience). These symptoms are mistakenly attributed to the respective



## Delirious mania

### Clinical Point

Patients with delirious mania typically develop symptoms extremely rapidly

### Box

## Delirious mania: A brief history

Delirious mania was originally recognized in 1849 by Luther Bell in McLean Hospital after he observed 40 cases that were uniquely distinct from 1,700 other cases from 1836 to 1849.<sup>6</sup> He described these patients as being suddenly confused, demonstrating unprovoked combativeness, remarkable decreased need for sleep, excessive motor restlessness, extreme fearfulness, and certain physiological signs, including rapid pulse and sweating. Bell was limited to the psychiatric treatment of his time, which largely was confined to physical restraints. Approximately three-fourths of these patients died.<sup>6</sup>

Following Bell's report, this syndrome remained unexplored and rarely described. Some researchers postulated that the development of confusion was a natural progression of late-phase mania in close to 20% of patients.<sup>7</sup> However, this did not

account for the rapid onset of symptoms as well as certain unexplained movement abnormalities. In 1980, Bond<sup>8</sup> presented 3 cases that were similar in nature to Bell's depiction: acute onset with extraordinary irritability, withdrawal, delirium, and mania.

For the next 2 decades, delirious mania was seldom reported in the literature. The term was often reserved to illustrate when a patient had nothing more than mania with features of delirium.<sup>9</sup>

By 1996, catatonia became better recognized in its wide array of symptomology and diagnostic scales.<sup>10,11</sup> In 1999, in addition to the sudden onset of excitement, paranoia, grandiosity, and disorientation, Fink<sup>1</sup> reported catatonic signs including negativism, stereotypy, posturing, grimacing, and echo phenomena in patients with delirious mania. He identified its sensitive response to electroconvulsive therapy.

disease.<sup>1,16</sup> This especially holds true when delirious mania is initially diagnosed as a primary psychosis, which leads to the administration of antipsychotics.<sup>17</sup> Other cases have reported that delirious mania was resistant to treatment, but ECT was never pursued.<sup>18</sup>

In this review, we provide a more comprehensive perspective of the clinical presentation, pathogenesis, and management of delirious mania. We searched PubMed and Google Scholar using the keywords "delirious mania," "delirious mania AND catatonia," or "manic delirium." Most articles we found were case reports, case series, or retrospective chart reviews. There were no systematic reviews, meta analyses, or randomized control trials (RCTs). The 12 articles included in this review consist of 7 individual case reports, 4 case series, and 1 retrospective chart review that describe a total of 36 cases (*Table*,<sup>1,2,5,17,19-26</sup> page 30).

### Clinical presentation: What to look for

Patients with delirious mania typically develop symptoms extremely rapidly. In virtually all published literature, symptoms were reported to emerge within hours to

days and consisted of severe forms of mania, psychosis, and delirium; 100% of the cases in our review had these symptoms. Commonly reported symptoms were:

- intense excitement
- emotional lability
- grandiose delusions
- profound insomnia
- pressured and rapid speech
- auditory and visual hallucinations
- hypersexuality
- thought disorganization.

Exquisite paranoia can also result in violent aggression (and may require the use of physical restraints). Patients may confine themselves to very small spaces (such as a closet) in response to the intense paranoia. Impairments in various neurocognitive domains—including inability to focus; disorientation; language and visuospatial disturbances; difficulty with shifting and sustaining attention; and short-term memory impairments—have been reported. Patients often cannot recall the events during the episode.<sup>1,2,5,27,28</sup>

Catatonia has been closely associated with delirious mania.<sup>29</sup> Features of excited catatonia—such as excessive motor activity, negativism, grimacing, posturing, echolalia, echopraxia, stereotypy, automatic

obedience, verbigeration, combativeness, impulsivity, and rigidity—typically accompany delirious mania.<sup>1,5,10,19,27</sup>

In addition to these symptoms, patients may engage in specific behaviors. They may exhibit inappropriate toileting such as smearing feces on walls or in bags, fecal or urinary incontinence, disrobing or running naked in public places, or pouring liquid on the floor or on one's head.<sup>1,2</sup>

Of the 36 cases reported in the literature we reviewed, 20 (55%) were female. Most patients had an underlying psychiatric condition, including BD (72%), major depressive disorder (8%), and schizophrenia (2%). Three patients had no psychiatric history.

### Physical examination

On initial presentation, a patient with delirious mania may be dehydrated, with dry mucous membranes, pale conjunctiva, tongue dryness, and poor skin turgor.<sup>28,30</sup> Due to excessive motor activity, diaphoresis with tachycardia, fluctuating blood pressure, and fever may be present.<sup>31</sup>

Certain basic cognitive tasks should be assessed to determine the patient's orientation to place, date, and time. Assess if the patient can recall recent events, names of objects, or perform serial 7s; clock drawing capabilities also should be ascertained.<sup>1,2,5</sup> A Mini-Mental State Examination is useful.<sup>32</sup>

The Bush-Francis Catatonia Rating Scale should be used to elicit features of catatonia, such as waxy flexibility, negativism, gegenhalten, mitgehen, catalepsy, ambitendency, automatic obedience, and grasp reflex.<sup>10</sup>

### Laboratory findings are nonspecific

Although no specific laboratory findings are associated with delirious mania, bloodwork and imaging are routinely investigated, especially if delirium characteristics are most striking. A complete blood count, chemistries, hepatic panel, thyroid functioning, blood and/or urine cultures, creatinine phosphokinase (CPK), and urinalysis can be ordered. Head imaging such as MRI and CT to rule out intracranial pathology are typically performed.<sup>19</sup> However, the

diagnosis of delirious mania is based on the presence of the phenotypic features, by verification of catatonia, and by the responsiveness to the treatment delivered.<sup>29</sup>

### Pathogenesis: Several hypotheses

The pathogenesis of delirious mania is not well understood. There are several postulations but no salient theory. Most patients with delirious mania have an underlying systemic medical or psychiatric condition.

**Mood disorders.** Patients with BD or schizoaffective disorder are especially susceptible to delirious mania. The percentage of manic patients who present with delirious mania varies by study. One study suggested approximately 19% have features of the phenomenon,<sup>33</sup> while others estimated 15% to 25%.<sup>34</sup> Elias et al<sup>35</sup> calculated that 15% of patients with mania succumb to manic exhaustion; from this it can be reasonably concluded that these were cases of misdiagnosed delirious mania.

**Delirium hypothesis.** Patients with delirious mania typically have features of delirium, including fluctuation of consciousness, disorientation, and/or poor sleep-wake cycle.<sup>36</sup> During rapid eye movement (REM) and non-REM sleep, memory circuits are fortified. When there is a substantial loss of REM and non-REM sleep, these circuits become faulty, even after 1 night. Pathological brain waves on EEG reflect the inability to reinforce the memory circuits. Patients with these waves may develop hallucinations, bizarre delusions, and altered sensorium. ECT reduces the pathological slow wave morphologies, thus restoring the synaptic maintenance and correcting the incompetent circuitry. This can explain the robust and rapid response of ECT in a patient with delirious mania.<sup>37,38</sup>

**Neurotransmitter hypothesis.** It has been shown that in patients with delirious mania there is dysregulation of dopamine transport, which leads to dopamine overflow in the synapse. In contrast to a drug effect (ie, cocaine or methamphetamine) that acts by inhibiting dopamine reuptake, dopamine overflow in delirious mania is caused by the loss of dopamine transporter regulation. This results in a dysfunctional

### Clinical Point

Of the 36 cases in the literature we reviewed, 100% presented with severe forms of mania, psychosis, and delirium



## Delirious mania

### Clinical Point

Diagnosis is based on the presence of phenotypic features, verification of catatonia, and responsiveness to the treatment

### Table

## Case studies, case series, and retrospective studies of delirious mania

Study	Cases, N	Gender	Psychiatric history	Symptoms
Fink <sup>1</sup> (1999)	3	Male: 3/3	Bipolar disorder: 3/3	Mania: 3/3 Psychosis: 3/3 Delirium: 3/3
Karmacharya et al <sup>2</sup> (2008)	16 <sup>a</sup>	Female: 13/16 Male: 3/16	Bipolar disorder: 10/16 Major depressive disorder: 2/16 Anxiety disorder: 1/16 PTSD: 1/16 Psychotic episode: 1/16 No psychiatric history: 1/16	Mania: 16/16 Psychosis: 16/16 Delirium: 16/16
Detweiler et al <sup>5</sup> (2009)	3	Male: 2/3 Female: 1/3	No psychiatric history: 1/3 Schizophrenia: 1/3 Bipolar disorder: 1/3	Mania: 3/3 Psychosis: 3/3 Delirium: 3/3
Vasudev et al <sup>19</sup> (2010)	1	Female	Bipolar disorder	Mania Psychosis Delirium
Danivas et al <sup>17</sup> (2010)	2	Male: 1/2 Female: 1/2	Bipolar disorder: 2/2	Mania: 2/2 Psychosis: 2/2 Delirium: 2/2
Lee et al <sup>22</sup> (2012)	5	Male: 4/5 Female: 1/5	Bipolar disorder: 5/5	Mania: 5/5 Psychosis: 5/5 Delirium: 5/5
Bipeta et al <sup>23</sup> (2012)	1	Male	Bipolar disorder	Mania Psychosis Delirium
Jacobowski et al <sup>20</sup> (2013)	1	Female	Major depressive disorder	Mania Psychosis Delirium
Nunes et al <sup>24</sup> (2014)	1	Female	Bipolar disorder	Mania Psychosis Delirium
Tegin et al <sup>25</sup> (2017)	1	Male	Bipolar disorder	Mania Psychosis Delirium
Melo et al <sup>26</sup> (2020)	1	Female	Bipolar disorder	Mania Psychosis Delirium
Reinfeld et al <sup>21</sup> (2021)	1	Male	No psychiatric history	Mania Psychosis Delirium

<sup>a</sup>Retrospective chart analysis

ECT: electroconvulsive therapy; PTSD: posttraumatic stress disorder

dopaminergic state that precipitates an acute state of delirium and agitation.<sup>39,40</sup>

Serotonin plays a role in mood disorders, including mania and depression.<sup>41,42</sup> More specifically, serotonin has been implicated in impulsivity and aggression as shown by reduced levels of CSF 5-hydroxyindoleacetic acid (5-HIAA) and depletion of 5-hydroxytryptophan (5-HTP).<sup>43</sup>

Alterations in gamma-aminobutyric acid (GABA) transmission are known to occur in delirium and catatonia. In delirium, GABA signaling is increased, which disrupts the circadian rhythm and melatonin release, thus impairing the sleep-wake cycle.<sup>44</sup> Deficiencies in acetylcholine and melatonin are seen as well as excess of other neurotransmitters,

Catatonias?	Treatment
Yes	2 cases had partial response to lorazepam, with complete resolution with bilateral ECT. One case went straight to bilateral ECT, with improvement
Catatonias was not assessed	6/16: Dramatic improvement with 1 to 4 ECT sessions 12/16: Showed benefit from lorazepam as well
Yes	2/3: Remitted with ECT
Yes	12 ECT sessions until remission
Yes	2/2: Complete resolution with ECT
Yes, 3/5	2/5: Remitted with 6 sessions of bilateral ECT 3/5: Responded to benzodiazepine therapy
Yes	Improved with lorazepam; however, complete resolution after 4 bilateral ECT sessions
Yes	Treatment was not published
Yes	Six sessions of bilateral ECT
Yes	Six sessions of bilateral ECT with complete remission
Yes	Complete resolution with 8 sessions of ECT
Yes	Failed benzodiazepines but complete resolution after 6 bilateral ECT sessions

including norepinephrine and glutamate.<sup>45</sup> Conversely, in catatonias, functional imaging studies found decreased GABA-A binding in orbitofrontal, prefrontal, parietal, and motor cortical regions.<sup>46</sup> A study analyzing 10 catatonic patients found decreased density of GABA-A receptors in the left sensorimotor cortex compared to psychiatric and healthy controls.<sup>47</sup>

Other neurotransmitters, such as glutamate, at the N-methyl-D-aspartate receptors (NMDAR) have been hypothesized to be hyperactive, causing downstream dysregulation of GABA functioning.<sup>48</sup> However, the exact connection between delirious mania and all these receptors and neurotransmitters remains unknown.

**Encephalitis hypothesis.** The relationship between delirious mania and autoimmune encephalitis suggests delirious mania has etiologies other than a primary psychiatric illness. In a 2020 retrospective study<sup>49</sup> that analyzed 79 patients with anti-NMDAR encephalitis, 25.3% met criteria for delirious mania, and 95% of these patients had catatonic features. Dalmau et al<sup>50</sup> found that in many cases, patients tend to respond to ECT; in a cases series of 3 patients, 2 responded to benzodiazepines.

**COVID-19 hypothesis.** The SARS-CoV-2 virion has been associated with many neuropsychiatric complications, including mood, psychotic, and neurocognitive disorders.<sup>51,52</sup> There also have been cases of COVID-19-induced catatonias.<sup>53-55</sup> One case of delirious mania in a patient with COVID-19 has been reported.<sup>21</sup> The general mechanism has been proposed to be related to the stimulation of the proinflammatory cytokines, such as tumor necrosis factor-alpha and interleukin-6, which the virus produces in large quantities.<sup>56</sup> These cytokines have been linked to psychosis and other psychiatric disorders.<sup>57</sup> The patient with COVID-19-induced delirious mania had elevated inflammatory markers, including erythrocyte sedimentation rate, C-reactive protein, ferritin, and D-dimer, which supports a proinflammatory state. This patient had a complete resolution of symptoms with ECT.<sup>21</sup>

### Management: Benzodiazepines and ECT

A step-by-step algorithm for managing delirious mania is outlined in the *Figure* (page 32). Regardless of the underlining etiology, management of delirious mania consists of benzodiazepines (lorazepam and diazepam); prompt use of ECT, particularly for patients who do not improve with large doses of lorazepam; or (if applicable)

### Clinical Point

**Patients with bipolar disorder or schizoaffective disorder are especially susceptible to delirious mania**



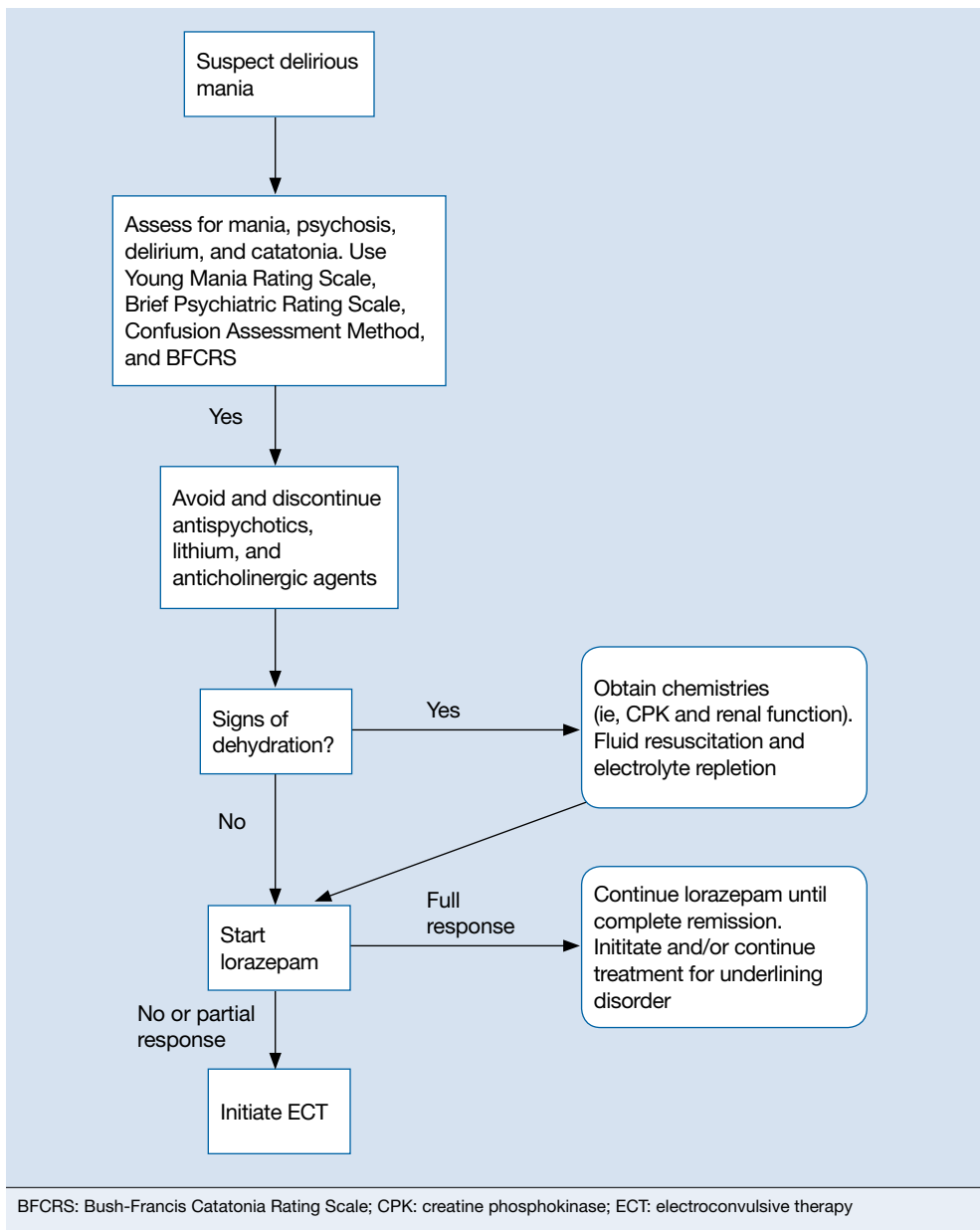
## Delirious mania

### Clinical Point

Management consists of benzodiazepines, ECT, and continued treatment of the patient's underlying medical condition, if applicable

Figure

## Algorithm for the management and treatment of delirious mania



continued treatment of the underlying medical condition, which does not preclude the use of benzodiazepines or ECT. Recent reports<sup>27,58</sup> have described details for using ECT for delirious mania, highlighting the use of high-energy dosing, bilateral electrode placement, and frequent sessions.

Knowing which medications to avoid is as important as knowing which agents to administer. Be vigilant in avoiding

high-potency antipsychotics, as these medications can worsen extrapyramidal symptoms and may precipitate seizures or neuroleptic malignant syndrome (NMS).<sup>28</sup> Anticholinergic agents should also be avoided because they worsen confusion. Although lithium is effective in BD, in delirious mania, high doses of lithium and haloperidol may cause severe encephalopathic syndromes, with symptoms that can include lethargy, tremors, cerebellar

dysfunction, and worsened confusion; it may also cause widespread and irreversible brain damage.<sup>59</sup> While positive outcomes have been documented when using a combination of antipsychotics and lithium,<sup>8,60</sup> this approach should be considered carefully and tailored to individual cases, taking into account the severity of manic and psychotic symptoms in addition to the level of catatonia.

Due to long periods of hyperactivity, withdrawal, and diaphoresis, patients with delirious mania may be severely dehydrated with metabolic derangements, including elevated CPK due to rhabdomyolysis from prolonged exertion, IM antipsychotics, or rigidity. To prevent acute renal failure, this must be immediately addressed with rapid fluid resuscitation and electrolyte repletion.<sup>61</sup>

**Benzodiazepines.** The rapid use of lorazepam should be initiated when delirious mania is suspected. Doses of 6 to 20 mg have been reported to be effective if tolerated.<sup>5,20</sup> Typically, high-dose lorazepam will not have the sedative effect that would normally occur in a patient who does not have delirious mania.<sup>2</sup> Lorazepam should be titrated until full resolution of symptoms. Doses up to 30 mg have been reported as effective and tolerable.<sup>62</sup> In our literature review, 50% of patients (18/36) responded or partially responded to lorazepam. However, only 3 case reports documented a complete remission with lorazepam, and many patients needed ECT for remission of symptoms.

ECT is generally reserved for patients who are not helped by benzodiazepine therapy, which is estimated to be up to 20%.<sup>5</sup> ECT is highly effective in delirious mania, with remission rates ranging from 80% to 100%.<sup>1</sup>

## Related Resources

- Arsan C, Baker C, Wong J, et al. Delirious mania: an approach to diagnosis and treatment. *Prim Care Companion CNS Disord.* 2021;23(1):20f02744. doi:10.4088/PCC.20f02744
- Lamba G, Kennedy EA, Vu CP. Case report: ECT for delirious mania. *Clinical Psychiatry News.* December 14, 2021. <https://www.mdedge.com/psychiatry/article/249909/bipolar-disorder/case-report-ect-delirious-mania>

### Drug Brand Names

Diazepam • Valium	Lithium • Eskalith, Lithobid
Haloperidol • Haldol	Lorazepam • Ativan

ECT is also effective in acute nondelirious mania (comparable to depression); however, it is only used in a small minority of cases (0.2% to 12%).<sup>35</sup> In our review, 58% of cases (21/36) reported using ECT, and in all cases it resulted in complete remission.

A dramatic improvement can be seen even after a single ECT session, though most patients show improvement after 4 sessions or 3 to 7 days.<sup>1,2,5</sup> In our review, most patients received 4 to 12 sessions until achieving complete remission.

No RCTs have evaluated ECT electrode placement in patients with delirious mania. However, several RCTs have investigated electrode placement in patients with acute nondelirious mania. Hiremani et al<sup>63</sup> found that bitemporal placement had a more rapid response rate than bifrontal placement, but there was no overall difference in response rate. Barekattain et al<sup>64</sup> found no difference between these 2 bilateral approaches. Many of the delirious mania cases report using a bilateral placement (including 42% of the ECT cases in our review) due to the emergent need for rapid relief of symptoms, which is especially necessary if the patient

## Clinical Point

**Avoid using high-potency antipsychotics because they can worsen EPS and precipitate seizures or NMS**

## Bottom Line

Delirious mania is routinely mistaken for more conventional manic or psychotic disorders. Clinicians need to be able to rapidly recognize the symptoms of this syndrome, which include mania, psychosis, delirium, and possible catatonia, so they can avoid administering toxic agents and instead initiate effective treatments such as benzodiazepines and electroconvulsive therapy.





## Delirious mania

### Clinical Point

Many delirious mania case reports include use of bilateral ECT due to the emergent need for rapid relief of symptoms

is experiencing hemodynamic instability, excessive violence, risk for self-harm, worsening delirium, or resistance to lorazepam.

### Prognosis: Often fatal if left untreated

Patients with delirious mania are at high risk to progress to a more severe form of NMS or malignant catatonia. Therefore, high-potency antipsychotics should be avoided; mortality can be elevated from 60% without antipsychotics to 78% with antipsychotics.<sup>4</sup> Some researchers estimate 75% to 78% of cases of delirious mania can be fatal if left untreated.<sup>3,6</sup>

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## Delirious mania

### Clinical Point

**Patients with delirious mania are at risk to progress to a more severe form of NMS or malignant catatonia**

### Delirious mania continued from page 34

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