Letters to the Editor

Two Cases of Somnambulism Induced by Quetiapine

Sir: Somnambulism reflects an impairment in the normal mechanisms of arousal from sleep in which motor behaviors initiated during deep, non–rapid eye movement or slow-wave sleep are activated without full consciousness. Somnambulism, a previously unreported side effect of quetiapine, is described in 2 cases. Both cases described here involved individuals with attention-deficit/hyperactivity disorder (ADHD). The possible significance of this will be discussed.

Case 1. Mr. A, a 52-year-old white man with no history of somnambulism, was undergoing treatment for DSM-IV panic disorder and schizoaffective disorder. He reported attention problems and hyperactivity since childhood and restless legs syndrome for 35 years. He was admitted to the medical unit in February 2006 to rule out myocardial infarction after falling off his porch while sleepwalking.

The patient reported that somnambulism had begun 18 months previously, after his quetiapine dose was increased to 200 mg at bedtime. This dose was later titrated to a maximum of 1000 mg during the month prior to admission. At that time, mirtazapine 30 mg at bedtime was added. This combination further aggravated the patient's somnambulism, which occurred almost nightly. The patient was witnessed by his roommate to wander in a confused state, manipulate various belongings, open the refrigerator and eat, and visit the bathroom. He was easily redirected back to bed, but did not appear to awaken.

A polysomnogram done when Mr. A was taking 800 mg of quetiapine showed no significant respiratory obstruction. Electroencephalogram (EEG) showed no epileptiform activity. Leg electromyogram findings were significant for frequent leg jerks. Quetiapine treatment was discontinued, and methylphenidate and clonazepam were started. Quetiapine 25 mg nightly was reinitiated later. No recurrence of somnambulism was reported at 8-month follow-up.

Case 2. Mr. B, an 18-year-old white man with DSM-IV diagnoses of ADHD and pervasive developmental disorder, sought consultation for episodes of somnambulism and nocturnal combativeness. At the time of presentation in June 2003, he was receiving quetiapine 400 mg nightly and dextroamphetamine sulfate 35 mg daily in divided doses.

The patient gave a history of starting quetiapine 8 months previously for onset of command auditory hallucinations. Shortly thereafter, he began to have nightmares associated with shouting, jumping from bed, property destruction, and assaults on family members. Mr. B awakened in the mornings with a headache but had no recollection of the events of the night before.

Neurologic examination findings were unremarkable, and results of a 24-hour EEG study were normal. The patient's quetiapine dose was reduced to 350 mg nightly, resulting in the return of auditory hallucinations. Dextroamphetamine sulfate was then tapered over a period of 4 months and discontinued with good result and resolution of hallucinations. The patient continued to have nocturnal outbursts, though these were less frequent and less intense, occurring approximately once every 2 weeks. Quetiapine was tapered by 50-mg decrements over several months. These symptoms resolved below a dose of 150 mg nightly. Quetiapine was discontinued, with no recurrence of somnambulism during 1 year of follow-up.

These cases suggest that quetiapine, like many other psychotropic medications, may induce somnambulism in certain individuals, possibly due to its impact on central serotonin activity.[1,2] This effect may be dose dependent. Both individuals had ADHD, which is interesting in view of recent evidence linking ADHD to polymorphism in the serotonin transporter gene and the genes for tryptophan hydroxylase and various serotonin receptors.[3,4]

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Obsessive-Compulsive Disorder After Body Dysmorphic Disorder: A Report of 2 Cases (a man and his mother)

Sir: Body dysmorphic disorder (BDD), or dysmorphophobia, is a mental disorder characterized by preoccupation with an imagined defect in one's appearance. Alternatively, BDD may involve a minor physical abnormality, but the concern is regarded as grossly excessive.[1] Most patients with BDD show repetitive behaviors, such as mirror checking, requests for reassurance, and skin picking, which resemble obsessive-compulsive disorder (OCD) compulsions. Studies using psychometric scales found that dysmorphophobic patients were more "obsessoid" and reported higher scores on the Leyton Obsessional Inventory than healthy controls.[2]

High lifetime OCD rates (34%–78%) have been found in several samples of BDD patients.[3,4] Conversely, lifetime BDD rates in patients with OCD also appear to be high, with reported rates ranging from 8%[5] to 37%.[4] OCD has been found to be the most common disorder in relatives of patients with BDD.[4] Hypochondriasis and pathologic grooming occurred more frequently in relatives of probands with OCD, whether or not probands also had the same diagnosis.[6] With respect to a familial relationship between OCD and obsessive-compulsive spectrum disorders, re-

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search suggests a shared etiology between OCD and hypochondriasis, body dysmorphic disorder, and grooming disorders.

The obsessive-compulsive spectrum was proposed in response to observations that a number of disparate disorders (for example, body dysmorphic disorder, hypochondriasis, some eating disorders, and some impulse-control disorders) share obsessive-compulsive features—that is, they are marked by obsessive thinking and/or compulsive behavior.[7–9] As regards treatment outcome, BDD, like OCD, appears to respond to selective serotonin reuptake inhibitors and clomipramine[10,11] and to exposure and response prevention.[12,13] Similarities in patient characteristics, course, comorbidity, neurobiology, and treatment response provide further support for the notion that these disorders may have a special relationship and thus should be conceptualized as a spectrum of related disorders.

Some important differences between BDD and OCD, however, have been reported. First and foremost, it seems that beliefs about appearance that underlie BDD preoccupations generally involve poorer insight than observed in beliefs underlying OCD obsessions. BDD preoccupations frequently lose their ego-dystonic character, become more similar to overvalued ideas than obsessions, and may even develop into full-blown delusional thinking.[14] Dysmorphic concerns are experienced as more natural than intrusive and are accepted and held with a significant degree of conviction rather than regarded as senseless, and patients often acquiesce to them without much resistance.[15]

Herein, we report 2 cases (a man and his mother) indicating similarities consistent with the literature, including heredity pattern, comorbidity, and treatment response between BDD and OCD.

Case 1. Mr. S, a 46-year-old male civil servant, presented to our clinic with the fear of being murdered. His complaint had started 3 months ago. While he was assessing the applications for a free health insurance card in his office, he was confronted with a written application that was sent from a farmer whose application had previously been rejected. The farmer wrote that his property should be evaluated again. After reading it, Mr. S thought that the farmer would kill him if he had signed the application. He was sure that it was illogical to think like that, but he could not erase the idea of being murdered from his mind. After a few days, he was not able to think about anything except being murdered. Although he had known that he had not signed the application and it was impossible for him to be held responsible for anything, he continued to ruminate about the idea. He even visited the farmer and heard from him that he would certainly not kill him. However, his feeling of reassurance lasted only a few minutes, and his anxiety grew day by day. He was extremely frightened when first seen in our clinic. According to DSM-IV criteria, he received a diagnosis of OCD because of his obsession (an intrusive and recurrent persistent thought that caused marked anxiety and affected his occupational functioning significantly). After the treatment with clomipramine 75 mg/daily for the first 12 days and then 150 mg/daily for 2 months, his complaints ceased gradually.

Mr. S's psychiatric history was as follows: he was a brilliant student until, while he was in high school at age 15, he started to think that his nose was large and ugly. Although his friends and family struggled to convince him that his nose was not so big and was, in fact, in harmony with his face, he was so preoccupied with his nose that he would not listen to anybody. Finally, he dropped out of school for fear of being scrutinized and talked about by his classmates. Fortunately, he was convinced by the plastic surgeon to whom he applied for the reconstruction of his nose to visit a psychiatrist. He received a diagnosis of BDD according to DSM-IV criteria from the psychiatrist, and after 4 months of receiving clomipramine 75 mg/day, a marked improvement in his condition was noted. His thoughts about having an ugly nose disappeared completely.

Case 2. Mr. S's mother, Mrs. A, a homemaker aged 66 years, had a similar history. When she was 14 years old, she was convinced that her face was extremely asymmetric and that everybody around her felt that she was disgusting. Until she was forced to see a psychiatrist, she did not leave the house for 4 months. She received a diagnosis of BDD and was prescribed clomipramine 75 mg daily and was courageous enough to leave the house 2 months after the beginning of treatment. As interestingly similar to Mr. S's history, when she was 47 years old, she thought, after buying a TV, that the invoice paper she had signed made her a debtor to the TV seller not only for the TV but also for everything she had. She was sure that she did not sign anything else and the seller tore the invoice to pieces, but nothing made her feel reassured. She was afterwards suspicious about signing papers that made her a debtor to anyone she talked with, and she could not make conversation without asking, "Have I signed any paper?" although she was sure that her thought was illogical. Every conversation she had ended with this question. She admitted a need for psychiatric help and received a diagnosis of OCD according to DSM-IV criteria, as she had an obsession (about being a debtor) that was accompanied by a verbal compulsion. She was prescribed clomipramine 150 mg/daily for 2 months before she voiced an acceptable improvement. She has been taking clomipramine in variable doses for 19 years. Her compulsion has disappeared completely, while her intrusive thoughts occasionally arise.

OCD and BDD might frequently coexist in clinical samples and have several similarities,[16] including prominent obsessions and compulsions and similarities in treatment response. However, some differences also exist. For example, studies have found that BDD subjects have a significantly poorer insight than OCD subjects, and a significantly greater percentage of BDD subjects than OCD subjects were classified as delusional.[17] The poor insight generally characteristic of BDD may make it difficult to persuade patients to accept and remain in psychiatric treatment. Of interest, studies of both BDD and OCD have found that patients with poorer insight or even delusional thinking are as likely to respond to serotonin reuptake inhibitors (SRIs) as patients with better insight.[18] Regarding response to cognitive-behavioral therapy (CBT), several studies have found that OCD with poor insight responds as well to behavior therapy as OCD with good insight.[19] Descriptive studies have found that a higher proportion of BDD patients are unmarried, unemployed, and less educated[16] and that BDD patients have a higher prevalence of major depression, social phobia, and suicidal ideation and suicide attempts attributed primarily to their disorder (i.e., BDD or OCD).[20]

In our report, Mr. S and his mother, Mrs. A, had striking similarities. The beginning of BDD in both cases was in the early to middle teenage years. They both successfully responded to clomipramine in a few months. The age at onset of OCD was in the mid-40s for both the mother and the son. Clomipramine was again the excellent therapy choice. Mr. S's obsession—different from his mother's—was not associated with any compulsion and differentiated from delusion with the presence of insight. These findings lead us to emphasize the importance of hereditary relationship in and similarities, including treatment response and comorbidity, between BDD and OCD.

Studies from clinical settings tend to find higher comorbidity rates than studies from nonclinical settings because having more than one disorder may increase the probability of seeking treatment.[21] However, comorbidity is the rule rather than the exception, as is the case for many psychiatric disorders. Clinicians need to be mindful of the possible clinical impact of comorbid conditions—for example, their association with greater morbidity and the need to consider comorbidity in treatment planning.

A small magnetic resonance imaging study (N = 16) found that BDD subjects had a leftward shift in caudate asymmetry and greater white matter volume than healthy controls, whereas some OCD studies have found the opposite (i.e., a rightward shift in caudate asymmetry and reduced white matter volume).[22] BDD and OCD both appear to respond preferentially to SRIs,[23,24] but preliminary data suggest that, unlike OCD, BDD may not respond to SRI augmentation with antipsychotics.[25,26] However, additional research is needed to further examine the nature of BDD's relationship to OCD.

Taken together, these findings give some support to the hypothesis that BDD may be related to OCD and is an "OCD-spectrum disorder," but that BDD and OCD are not identical. There is also a need for future studies to evaluate evidence from other sources, such as neurobiological, family, and treatment studies, to further our understanding of the concept of obsessive-compulsive spectrum disorders.

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Risperidone and Dysphagia in a Developmentally Disabled Woman

Sir: Dysphagia represents an infrequently reported adverse effect secondary to use of neuroleptic agents.[1] The presentation of dysphagia is often associated with parkinsonian symptoms.[2] Autonomic dysfunction is due to changes in the hypothalamus, locus ceruleus, dorsal vagal nucleus, and sympathetic ganglia.[3] Neuroleptic medications may induce swallowing difficulties by similar mechanisms.[1] Atypical antipsychotic agents are believed to have a more advantageous side effect profile with fewer extrapyramidal effects.[1] This medication-mediated reversible effect may be underdiagnosed in populations with additional neurologic impairments.

Case report. Ms. A, a 60-kg, 46-year-old white woman diagnosed with dementia due to lithium toxicity and profound mental retardation (IQ = 20), resides in a state-run facility. Her verbal ability is limited to single words. The patient has a long-standing history of aggression and aberrant behaviors addressed with behavioral interventions and medication use. She presented to the psychiatric clinic in September 2006 with a

significant increase in drooling, difficulty swallowing, and gurgling sounds. The total daily doses for the psychoactive medication regimen at the time dysphagia was diagnosed included risperidone 2 mg daily for psychosis and mood lability, clozapine 500 mg for mood lability, clonidine 0.3 mg for aggression and dyskinesia, and benztropine 1 mg for drooling and extrapyramidal symptoms.

The most recent medication change, which occurred the previous month, was an increase in risperidone from 1.5 mg to 2 mg that followed increases in behavioral dyscontrol without an identifiable antecedent. In clinic, Ms. A was not appreciably calmer. The dose was subsequently decreased to the previous dose of 1.5 mg. On follow-up 3 days later, drooling and swallowing difficulties were resolving.

Case reports related to dysphagia secondary to atypical antipsychotic use have been reported previously. Two reports involved risperidone use; one followed an increase to a total daily dose of 1.5 mg and resolved with risperidone discontinuation and substitution of olanzapine 2.5 mg.[1] The other occurred following a single 4-mg risperidone dose and resolved after administration of benztropine.[4] Dysphagia was also reported 5 days after initiation of olanzapine 20 mg.[5] Olanzapine-associated dysphagia resolved with medication discontinuation.

Concurrent use of clozapine and risperidone may have predisposed our patient to adverse effects. Clozapine atypicality is thought to be associated with its greater affinity for serotonin 5-HT_{2A} receptors than dopamine D_2 receptors and rapid dissociation from the basal ganglia D_2 receptors. In addition, its anticholinergic effects may act to moderate the risk of extrapyramidal effects.[6] Risperidone has pharmacologic properties resembling those of clozapine, with antagonistic activity primarily at serotonin 5-HT_{2A} and dopamine D_2 receptors.[7] Concurrent therapy may negatively impact the rapid D_2 dissociation times that have been reported[8] and increase the potential for adverse effects.

We believe this is the first report of dysphagia identified secondary to atypical antipsychotic use in a person with developmental disabilities. The presence of mental retardation is an indication of neurologic impairment, and dysphagia may be overlooked.[9] Limited verbal abilities may impede self-reporting. Clinicians should be aware of the potential for this infrequently reported adverse effect in both the general population and those with developmental disabilities, particularly with concurrent therapy regimens.

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Chlorpromazine-Induced Skin Pigmentation With Short-Term Use in a Patient With Bipolar Disorder: A Case Report

Sir: Chlorpromazine is known, in rare instances, to induce skin pigmentation in areas exposed to sunlight.[1] The prevalence in chronic, hospitalized patients is reported as 1.0% to 2.9%.[2] Some authors report chlorpromazine-induced skin pigmentation as irreversible,[3] while some findings[4] indicate that it is completely reversible and that a variety of neuroleptics, including other phenothiazines, are used to replace chlorpromazine without risk of reemergence of pigmentation.[4] Some authors[3] suggest that chronic therapy with 500 mg/day or more of chlorpromazine is necessary to cause pigmentation, while others[5,6] report pigmentation at much lower doses. To our knowledge, there has been no report of skin changes in bipolar patients with short-term use of chlorpromazine.

Case report. Mr. A, a 30-year-old man diagnosed with bipolar affective disorder, current episode of mania with psychotic symptoms (per ICD-10 criteria), was treated with lithium carbonate tablets (1200 mg/day), chlorpromazine tablets (600 mg/day), and trihexyphenidyl tablets (2 mg/day). At the time of drug institution, there was no evidence of skin pigmentation or history of dermatologic problems. There was no history of prior exposure to chlorpromazine or of medical illnesses, and biochemical investigations revealed no abnormalities. He was still mildly euphoric and had difficulties initiating sleep, for which chlorpromazine was continued at the same dosage.

Three months later, in March 2004, he developed areas of diffuse hyperpigmentation over the sun-exposed parts of his face, neck, and shoulders compared to the rest of his body (Figure 1). There was no history of concomitant use of any other drugs except lithium and trihexyphenidyl. A possibility of chlorpromazine-induced skin hyperpigmentation was suspected. A slit-lamp examination of the eyes at this point revealed no corneal or lenticular deposits. Olanzapine tablets were started at 5 mg/day and raised to 20 mg/day, replacing chlorpromazine. There was only a slight decrease in pigmentation during follow-up in the next 2.5 years (Figure 2). A follow-up slit-lamp eye examination after 2.5 years revealed no corneal or lenticular deposits.

Figure 1. Photograph of Patient in March 2004 Showing Hyperpigmentation Over Face, Neck, and Sun-Exposed Area of Chest

Figure 2. Same Patient 2.5 Years Later

Our finding is different from past reports of skin changes following chronic exposure to chlorpromazine.[1,3] In contrast, our patient developed hyperpigmentation during short-term chlorpromazine exposure. Most of the cases described in the literature were chronically hospitalized patients diagnosed with schizophrenia,[1] whereas our patient was suffering from bipolar affective disorder. Previous reports show resolution of the skin changes occurring slowly over a period of 6 months to 5 years following substitution of chlorpromazine with other phenothiazines,[4] loxapine,[6] flupenthixol,[7] and the atypical antipsychotic clozapine.[8] The hyperpigmentation has remained unresolved in our patient in spite of replacing chlorpromazine with olanzapine, highlighting the need for a longer period of observation without chlorpromazine.[4]

This case underlines the need for clinicians and primary health care providers to be aware that chlorpromazine-induced skin pigmentation is a well-known side effect of this drug, regardless of the disease for which it is used, and that the time needed for developing the pigmentation seems to vary widely among patients.

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Topiramate-Induced Psychosis in an Individual With Alcohol Dependence: A Case Report

Sir: Topiramate, an antiepileptic drug, has been shown to reduce alcohol craving and heavy drinking and to improve abstinence among alcohol-dependent individuals.[1] By potentiating the inhibitory effects of γ-aminobutyric acid (GABA)[2] and depressing the excitatory action of kainate on α-amino-3-hydroxy-5-methylisoxazole-4-propionic acid (AMPA) glutamate receptors,[3] topiramate reduces mesocorticolimbic dopamine activity, which is a crucial pathway by which alcohol exerts its rewarding effects.[4–6] Doses of up to 300 mg per day have been effective in reducing craving in alcohol-dependent patients.

The drug is started at a low dose and increased slowly to avoid adverse effects. Psychiatric side effects occur in up to 23.9% of patients with epilepsy[7] and are related to a personal or family history of psychiatric illness and rapid dose escalation. These psychiatric side effects include depression, anxiety, aggression, cognitive slowing, and psychosis. Topiramate has also been reported to precipitate psychosis in patients with schizophrenia[8] and mood disorder.[9]

Case report. Mr. A, a 42-year-old man, was admitted to our Deaddiction Centre in January 2005 with a diagnosis of alcohol dependence syndrome (according to ICD-10 criteria) and presented with delirium tremens. The patient had a history of alcohol consumption for the last 15 years and had developed features of salience, tolerance, craving, and loss of control over drinking over the last 8 years. He presented with tremors; irritability; insomnia; loss of appetite; auditory, visual, and tactile hallucinations; disorientation; and clouding of consciousness, all of which began after 24 hours of abstinence. He had a history of possible withdrawal seizures in the past. There was no history of psychosis.

There was a family history of alcohol dependence in his father and 2 male siblings. There was no family history of psychosis or mood disorder. Laboratory findings were within normal limits.

The patient was started on a detoxification regimen with lorazepam, 16 mg per day PO in tapering doses, and vitamin supplementation. His withdrawal symptoms subsided within 48 hours, with hallucinations subsiding within 24 hours.

After detoxification for 10 days, the patient was started on topiramate 25 mg per day as long-term medication for alcohol dependence. On the third day after starting topiramate, the patient suddenly awoke at around 1:00 a.m., looked anxious, and reported that he could hear "voices calling out to him from hell" and discussing his death. He continued to hear these voices even after he was fully awake. He remained anxious, apprehensive, and distressed for the next 30 minutes and was sedated with intravenous haloperidol 10 mg and lorazepam 4 mg, both of which had to be repeated after 2 hours. During this episode, there was no disorientation, clouding of consciousness, or tremulousness. After topiramate was stopped, these psychotic symptoms remitted completely within 48 hours and did not recur.

Topiramate has been hypothesized to induce psychosis as a result of its antiglutamatergic properties in the nucleus accumbens and prefrontal cortex.[8] Brief psychosis was the most common presentation in a series of patients with epilepsy.[7] As patients with alcohol dependence syndrome show changes in glutamate receptors, especially during withdrawal,[6] they may be more sensitive to the effects of topiramate. To the best of our knowledge, this is the first report of topiramate precipitating psychosis in a patient with alcohol dependence. It is possible that patients presenting with alcohol withdrawal are more susceptible to this particular adverse effect.

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A Case of Hashimoto's Encephalopathy Manifesting as Psychosis

Sir: A high rate of autoimmune thyroiditis not associated with lithium treatment has been reported in patients with psychiatric disorders.[1,2] Hashimoto's encephalopathy is a rare subacute condition associated with high levels of thyroid antibodies that usually presents with neurologic symptoms. A few cases of this illness manifesting with predominant psychiatric symptoms have been reported.[3–5]

Case report. Mr. A, a 31-year-old man with no significant medical history, initially presented for treatment in January 2006 and had suffered from delusions, disorganized speech, talkativeness, racing thoughts, acute episodes of memory loss, reduced need for sleep, and irritability for 4 months. No alterations were found with physical examination or magnetic resonance imaging. Electroencephalography showed intermittent slow-wave activity. The results of general blood chemistries and a comprehensive drug screen were within normal limits. Thyroid function test revealed a free triiodothyronine (T_3) level of 0.33 ng/dL, a free thyroxine (T_4) level of 1.0 ng/dL, a total T_4 level of 6.2 μg/dL, and a thyroid-stimulating hormone level of 5.60 μIU/mL. The patient was unsuccessfully treated with numerous psychotropic medications during this 4-month period.

A new exhaustive physical examination showed myoclonic jerks and thyromegaly. Mr. A's thyroid peroxidase antibody titer was measured, and the result was positive at 7500 U/L. A diagnosis of Hashimoto's encephalopathy was considered, and steroid therapy was started, with significant improvement in the patient's mental status within 1 week. The steroid therapy was gradually tapered over 2 months, and the patient remained symptom-free and had thyroid peroxidase antibody titers within normal limits after 3 months.

Hashimoto's encephalopathy diagnosis should be considered in patients with potential autoimmune thyroiditis and neuropsychiatric manifestations not responding to conventional therapy.

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A Unique Case of Delirium Resulting From Electrical Accident–Induced Spinal Trauma

Sir: We would like to report a unique case of onset of delirium associated with electrical injury and spinal trauma in an adult. Reviewing the literature on psychiatric complications of electrical injury, we found no report of a relationship between adult-onset delirium and electrical injury.

Case report. Mr. A, a 25-year-old man, was admitted to our hospital in 2006 after having suffered an electrical injury with loss of consciousness and falling on his back from a height of 6 m. He received an electrical shock (25,000 V) for 12 to 15 seconds.

On admission, the patient was extremely agitated and his consciousness was very confused. No motor activity deficits were present. His cardiac and biochemical examinations revealed no abnormalities. Chronic drug use history was absent. A burn of approximately 1 × 1 cm was identified on the right thumb, and an 8 × 10 cm burn was exhibited on the left leg below knee. The head computed tomography also exhibited no abnormalities. His lumbar vertebral tomography revealed third lumbar vertebra burst fracture.

On day 2 after electrical injury, when sedation was discontinued, he began experiencing severe acute confusion, in which he screamed and manifested difficulty staying asleep. He felt extremely irritable. His thought process was not logical and fluctuated during the course of the day.

The patient underwent psychiatric consultation and was diagnosed as having delirium (based on his history and clinical findings). His acute confusion was not controlled with heavy-sedation–inducing administration of haloperidol 2 mg/mL (10 drops orally) twice daily for 7 weeks. Owing to resistant irrita-bility, quetiapine tablets 25 mg twice daily were administered additionally. After 3 weeks on treatment with haloperidol and quetiapine, the patient has remained better.

Although Baqain et al.[1] stated that an electrical current can produce nerve lesions, the mechanisms involved in the pathogenesis of neurologic damage after electrical trauma are unknown. Many patients with high-voltage electrical trauma suffer a bony displacement due to electrostatic repulsion, and a vertebral fracture may be evaluated. In our patient, however, we thought that the fracture was due to the fall. Bergman and Coletti[2] defined delirium (acute confusion) as diffuse impairment of higher cortical function characterized by a sudden onset of disordered cognition, dysfunction of the reticular activation system, and disturbed psychomotor behavior. Disturbance develops during a short period (usually hours to days) and tends to fluctuate during the course of the day, as it did in our patient. Bergman and Coletti[2] stated that decline in cholinergic activity may play a major role in the development of delirium. Treatment with anticholinergic drugs, opioids, antiparkinsonism agents, histamine-2 receptor blockers, cardiovascular agents, antibiotics, anticonvulsants, anti-inflammatory agents, or oral hypoglycemic agents may reveal acute confusion, especially in elderly people. However, our patient was young and exhibited the clinical symptoms preoperatively; thus, drug or surgical etiology cannot explain his condition. The patient was diagnosed as having delirium according to clinical findings.

We thought that the pathway followed by the electrical current might have caused an acute decline in cholinergic system function and was responsible for the neurologic damage. Finally, the diagnosis of delirium after electrical injuries is based on the patient's history and clinical findings. Ghaemi et al.[3] reported parasomnias, Pliskin et al.[4] reported narcolepsy and posttraumatic depression, and Khanna et al.[5] reported mania associated with electrical injury, but none of these reported delirium. The most interesting part of this case was that although the high-voltage electricity entered his body from his right thumb and came out from his left leg under his knee, he showed a unique case of delirium associated with his head.

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