Lithium-Induced Rash

Sir: Lithium has been used as a mood stabilizer for decades and has been approved by the U.S. Food and Drug Administration for acute and maintenance therapy of mania in bipolar disorder. The *Physicians' Desk Reference* lists generalized pruritus with or without rash as one of the dermatologic side effects of lithium.[1] We present an interesting case of maculopapular rash that we believe was induced with lithium.

Case report. Ms. A, a 60-year-old woman with a past psychiatric history of bipolar disorder, was brought to the emergency room in 2005 by her family for some odd behaviors. The family described a 2-week history of spending sprees, staying up all night, talking excessively, and making inappropriate phone calls. She called her former employers and was verbally abusive to them, which resulted in a restraining order being issued against her. A thorough medical work-up in the emergency room did not reveal any significant findings. Her past medical history was significant for coronary bypass graft, end stage renal disease requiring dialysis, and gastritis. Her current medications were captopril, omeprazole, atrovastatin, aspirin, spironolactone, amiodarone, and cetrizine. She was found to have pressured speech with a circumstantial thought process without any psychosis or cognitive impairment. She was later transferred to the psychiatric floor for stabilization. The patient had a history of bipolar disorder with 4 past psychiatric hospitalizations, the last being 6 years ago. She had been on divalproex and lithium in the past with no adverse effects, but noncompliance has been an ongoing issue in her case. There had been no psychiatric follow-up for the last 5 years.

A diagnosis of bipolar disorder, most recent episode manic, was made using the DSM-IV diagnostic criteria, and she was started on lithium 300 mg at bedtime and quetiapine 25 mg at bedtime on an as-needed basis for sleep. Within a few days a significant symptomatic improvement was noticed. On the sixth day after initiation of lithium, she was found to have a red maculopapular rash about 5 cm × 3 cm in size over the left pretibial area. There were no complaints of itching or any signs of systemic infection. Her lithium level a day before was 0.5 mEq/L. Her lithium was withheld, resulting in a fading of her rash over the next 2 days. Unfortunately, she left the hospital against medical advice and went to the home of her daughter, who convinced us that the patient would follow up with outpatient psychiatry within the next few days.

It has been reported that women are at increased risk of cutaneous lesions when treated with lithium, usually within the first year of its initiation.[2] In our case, the temporal association of the onset of the rash within a week of the initiation of lithium and its fading within 2 days of its discontinuation points to a causal relationship. Although the exact mechanism is unclear, it has been proposed that inhibition of adenylate cyclase/cyclic AMP systems induced by lithium could be responsible for the cutaneous conditions.[2]

Other lithium-induced skin conditions include psoriasis, acne, folliculitis, exfoliative dermatitis, seborrheic dermatitis, and herpetiform dermatitis. The prevalence rate of lithium-induced cutaneous reaction has been reported to be 34% in one study[2] and 45% in another.[3]

Further investigation regarding cutaneous lesions associated with lithium therapy is warranted as this distressing side effect could adversely affect the medication compliance.

Drs. Sharma and Padala report no financial affiliations or other relationships relevant to the subject of this letter.

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Effectiveness of Olanzapine for Systemic Lupus Erythematosus–Related Psychosis

Sir: We report a case of olanzapine treatment of psychotic symptoms related to systemic lupus erythematosus (SLE). There is evidence that SLE can affect the central nervous system, among other parts of the body. Reports of psychotic presentation in SLE are rare, but such presentations are often dramatic, causing impairment in quality of life and difficulties for treatment.[1,2] To our knowledge, there are no reports of effectiveness of olanzapine, an atypical antipsychotic, for SLE-related psychosis.

Case report. Ms. A, a 14-year-old adolescent girl, was diagnosed with SLE in 2002 at age 10, when she presented with high fever, polyarthritis, purpura, systemic arterial hypertension, and laboratory abnormalities (lymphopenia and positive antinuclear and native DNA antibodies). She fulfilled the American College of Rheumatology diagnostic criteria[3] for SLE. At the time she was originally diagnosed, she received therapy with methylprednisolone (pulse therapy) for 6 months, followed by azathioprine (50 mg/day) for 2 years.

Later, in 2004, she was followed in a pediatric outpatient clinic and treated with prednisone (20 mg/day) and hydroxychloroquine (400 mg/day), which she continued throughout follow-up. At age 14, she was admitted to an inpatient unit presenting with progressive agitation, persecutory and auto-reference delusions, visual and auditory hallucinations with command voices that encouraged her to commit suicide (2 attempts to self-harm), soliloquies, and insomnia. Symptom onset was rapid and became apparent 10 days prior to admission. Her rheumatologist concluded that she had not had a new SLE crisis, since SLE markers were not different from previous exams, and that the psychotic symptoms were not caused by medication adjustment.

The findings of her brain computed tomography (CT) and serologic exams (HIV and syphilis) and biochemical examination results were all within normal limits. She had no family history of psychiatric disorders. She was administered haloperidol 5 mg/day with minimum improvement and significant extrapyramidal side effects. That medication was stopped, and olanzapine 5 mg/day was initiated and was increased to 7.5 mg/day within 15 days. She became symptom free within 15 days of olanzapine initiation and remained so for 4 months. The dose was then decreased to 5 mg/day due to weight gain (5 kg), and the patient remains symptom free to date.

This patient had a 4-year history of SLE with multiple complications, including her psychotic outbreak. She had been taking corticosteroids since her diagnosis and had no dose alteration in the last 2 years before her psychiatric manifestation.

Since she had no family history of psychosis or changes in her SLE treatment, this case is not likely to be a first episode or a medication-induced psychosis. Moreover, diffuse brain disease in SLE can be difficult to assess because of its sparse biological expression, usually resulting in normal neuroimaging and laboratory findings.[4] Thus, it is likely that psychiatric presentation in SLE patients should be considered as SLE induced.

The usual treatment of SLE-related psychosis involves immunosuppressant and/or antipsychotic therapy.[5] Regarding atypical antipsychotics, as far as we know, only risperidone, alone or in combination with valproic acid, has been used.[6–8] On the other hand, there are reports of antipsychotic-induced lupus with traditional (chlorpromazine[9,10]) and atypical (clozapine[11,12] and ziprasidone[13]) agents.

Successful treatment of SLE-related psychosis with olanzapine brings another therapeutic option to those patients, although controlled studies would be necessary to further confirm this observation.

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Aripiprazole-Induced Tardive Dystonia

Sir: Tardive dystonia is a persistent syndrome of sustained muscle contraction that produces twisting and repetitive movements or abnormal postures. It usually involves the head and neck, producing torticollis, retrocollis, or anterocollis, but sometimes it involves back muscles, giving rise to opisthotonus and gait disturbances.[1] The most common causes for development of tardive dystonia are exposure to neuroleptics or other medications like metaclorpromide, prochloroperazine, or amoxepine.[1] Among the new generation of antipsychotics, aripiprazole has a unique receptor binding profile that combines partial agonistic activity at D_2 and 5-HT_{2A} receptors.[2,3] The risk of movement disorder in people taking aripiprazole has to date been considered insignificant as there is no published report to our knowledge on association of tardive dystonia with aripiprazole.

Case report. Ms. A, a 25-year-old woman, presented to the hospital in 2000 with schizoaffective disorder that included a past history of 2 psychotic episodes and 3 manic episodes. During the course of treatment, she developed skin rashes with the use of carbamazepine, experienced significant weight gain with the use of olanzapine, developed galactorrhea and severe extrapyramidal symptoms with the use of risperidone, and experienced severe tremors and excessive daytime drowsiness with the use of valproate. These side effects led to frequent changes in medications. While treated with quetiapine, she developed recurrence of mania. She was also given a trial of lithium with which she complained of memory loss and vomiting. The lithium dosage was reduced to 450 mg/day, and she remained on treatment with that dosage for the 8 months preceding the current episode.

For the current manic episode, in 2005, aripiprazole 10 mg/day was added to her lithium treatment, which she had been taking for the last 8 months without any significant problem. After 4 weeks, aripiprazole was increased to 15 mg/day. After 2 months of administration of aripiprazole, she developed backward arching of her body with spasm over the origin of la-

tissimus dorsi muscle near vertebrae L1 and L2. These effects worsened while she was standing and lifting her arms. The arching increased when she was asked to engage in physical activity. These symptoms were absent during sleep, caused significant dysfunction in her occupation and embarrassment in public, and gradually worsened. She did not experience perioral tongue movements, facial grimacing, or difficulty in chewing or breathing.

Ms. A had no history of head injury or loss of consciousness or a family history of movement disorders. A thorough neurologic examination, including neuroimaging, revealed no pathology. She was rated with the Extrapyramidal Symptom Rating Scale (ESRS)[4] and was found to suffer from moderate to severe levels of extrapyramidal symptoms. Aripiprazole was stopped and tablet trihexyphenidyl 6 mg/day was started with continued lithium treatment (450 mg/day) in view of worsening dystonic symptoms. She showed improvement in dystonia, and after 2 weeks, her ESRS score was zero. Four weeks after the start of trihexyphenidyl treatment, in view of her mood and psychotic symptoms, she was started on treatment with clozapine, which was increased to 150 mg/day. Currently, 1 year after discontinuing aripiprazole, she is symptom free.

This patient had several features suggestive of tardive dystonia following exposure to aripiprazole. The symptoms started after about 2 months of exposure to aripiprazole and worsened subsequently. They improved only after the stoppage of aripiprazole and addition of trihexyphenidyl. The neurologic examinations revealed no other causes of dystonia, and previous antipsychotic exposure was at least a year earlier. Association of lithium with tardive dystonia has been reported in literature,[5] but in this case she took lithium for 8 months with no dystonic symptoms.

Studies have reported no increase in incidence of extrapyramidal symptoms in patients treated with aripiprazole compared with those treated with placebo.[6,7] Moreover, treatment with aripiprazole has shown remarkable improvement in tardive dyskinesia.[8] But, as seen in this case, the possibility of developing dystonia, though remote, is present even with a safe drug like aripiprazole. It is even more likely to occur in a patient who is vulnerable to side effects with other neuroleptics.

The authors report no financial or other relationship relevant to the subject of this letter.

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Methylphenidate-Induced Akathisia in a Patient With Multiple Sclerosis

Sir: Methylphenidate is a short-acting psychostimulant that is structurally and pharmacologically similar to dextroamphetamine and is primarily used in the treatment of attention-deficit/hyperactivity disorder (ADHD).[1,2] Its use has also been explored for the treatment of apathy and for augmentation of effects of antidepressants.[3] It acts as a dopaminergic and noradrenergic reuptake inhibitor. Its dopaminergic activity has been implicated in causation of tics, choreiform disorder, and dyskinesias. Several reports[1,2] have highlighted increased incidence of tics and dyskinesia in particularly vulnerable groups of patients such as children with ADHD. In previous reports,[1,2] association of extrapyramidal symptoms (EPS) has been noted with chronic use of stimulants at high doses. This is the first report of EPS induced by methylphenidate in a patient with multiple sclerosis.

Case report. Ms. A, a 46-year-old white woman with a history of DSM-IV-TR major depressive disorder recurrent type and alcohol dependence in full sustained remission, presented to our clinic with apathy. Her psychiatric history included dysthymia and depression since age 18 years and an inpatient hospitalization for severe depression at age 25 years. The patient ever since had been treated in an outpatient setting and was currently being managed on a complex medication regimen including venlafaxine, mirtazapine, buspirone, and quetiapine that she felt worked best for her. Her medical history included nicotine dependence, chronic obstructive pulmonary disease, multiple sclerosis, and multiple pulmonary eosinophilic granulomas. The patient was taking several medications including tizanidine, oxybutynin, interferon-beta, hydrochlorothiazide, baclofen, diazepam, and clonazepam.

Ms. A also complained of memory problems. Initial cognitive testing yielded the following results: Mini-Mental State Examination[4] score, 29/30; long-term memory score, 8/12; abstraction score, 3/4; judgment score, 2/2. Detailed neuropsychological testing (word fluency, some attentional tasks, response inhibition, and executive functioning) revealed evidence of impairment in frontal lobe functioning. Her apathy was assessed using the Apathy Evaluation Scale.[5] She scored 47 on a scale of 18 to 72, with 72 being the worst apathy.

She was started on methylphenidate treatment at 10 mg twice a day for the treatment of apathy after discussing various treatment options. She reported feeling restless after the third dose of methylphenidate but continued to take the medication. By the fifth day of treatment, she described feeling as if wanting to crawl out of her skin and was restless and pacing. She took clonazepam and valium, which did not provide relief of her symptoms. Her left leg stiffened and tremors started in her left

arm by day 6. She was taken to the emergency room and was given an intramuscular injection of benztropine, which resulted in immediate relief of her symptoms. She was instructed to discontinue methylphenidate and was given oral benztropine, which she took for a week. No recurrence of the above symptoms was noted.

Akathisia is a subjective feeling of motor restlessness with anxiety. The objective signs of restlessness include pacing, rocking back and forth, marching-like movements of the feet, and other repetitive, purposeless actions. Its etiology is poorly understood, although it is thought to be due to adrenergic excess. Treatment includes reduction in the dose or discontinuation of the medication causing akathisia. The typical agents used to treat EPS are not very effective in reducing symptoms of akathisia. β-Blockers, benzodiazepines, and less commonly, anticholinergics and antihistaminergics are used to treat akathisia.[6]

Stimulants are implicated in the production of tics and dyskinesias with an estimated incidence ranging from 1.3% to 60% in children treated with stimulants.[7] Methylphenidate is similar to dextroamphetamine in pharmacologic properties, and both drugs have been shown to produce stereotyped behavior. In patients with alteration of postsynaptic dopamine receptor sites, an increase of dopaminergic activity together with altered receptor responsiveness exacerbated existing chorea.[2]

The patient in this report presented with symptoms suggestive of akathisia following the introduction of methylphenidate. These symptoms did not respond to benzodiazepines, which are usually the treatment of choice for akathisia. Symptom relief was obtained with the addition of benztropine, an anticholinergic agent. Our patient was also taking quetiapine, and although quetiapine has a low potential to cause EPS, her presentation could be due to the unmasking of latent EPS by the addition of methylphenidate.

The authors report no financial affiliations or other relationships relevant to the subject of this letter.

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Aripiprazole Worsens Psychosis: A Case Report

Sir: Aripiprazole is a second-generation antipsychotic medication with a unique mechanism of action. It acts as a functional antagonist or functional agonist at dopamine and serotonin receptors, depending upon the level of the relevant neurotransmitter in the immediate environment, and for this reason, it is known as a dopamine-serotonin system stabilizer.[1] Due to the partial-agonist D_2 receptor activity of aripiprazole, it has been suggested to worsen psychosis.[2]

We report a case in which the unique mechanism of action of aripiprazole probably contributed to relapse of psychotic symptoms.

Case report. Ms. A, a 24-year-old graduate student, presented in December 2000 with an insidious-onset illness of 5 years' duration characterized by delusions of reference, control, and persecution; thought broadcast; and auditory hallucinations of commenting and discussing type. The symptoms led to marked dysfunction (Global Assessment of Functioning[3] score = 30). She was diagnosed with paranoid schizophrenia (as per ICD-10 and DSM-IV) and was initially treated with risperidone (maximum dose = 6 mg/day, total duration of 24 months), trifluoperazine (maximum dose = 25 mg/day, total duration of 15 months), olanzapine (maximum dose = 25 mg/day, total duration of 4 months), and quetiapine (maximum dose = 600 mg/day, total duration of 4 months).

With each of these medications, Ms. A showed partial improvement initially (about 1 Clinical Global Impressions scale [CGI][4] point), with subsequent worsening to the previous level within a few weeks. As she (and her family) continued to refuse clozapine, she was switched to haloperidol (maximum dose = 30 mg/day, total duration of 2 months). She showed minimal initial improvement (1 CGI point) with haloperidol, so aripiprazole (maximum dose = 20 mg/day, total duration of 2 months) was added. With the combination of the 2 drugs, her symptoms worsened (2 CGI points) despite an increase in haloperidol dose (maximum = 60 mg/day). Ms. A started showing improvement in symptoms when aripiprazole was tapered off.

She maintained improvement on treatment with 60 mg/day of haloperidol over the next 5 months (about 3 CGI points beyond the level seen with the aripiprazole and haloperidol combination). According to her family, it was the best state achieved by the patient in the last 5 years. Objectively, her Positive and Negative Syndrome Scale[5] positive syndrome score at this time was 0.

Aripiprazole acts as a D_2 receptor antagonist in a hyperdopaminergic environment and as an agonist in a hypodopaminergic environment. In the index case, the presence of haloperidol, which is a potent D_2 antagonist, led to a hypodopaminergic state. In the hypodopaminergic milieu, aripiprazole acted as an agonist and led to worsening of symptoms. This hypothesis can be more strongly accepted for the present case in particular, because withdrawal of aripiprazole led to improvement in psychotic symptoms. Our case is an addition to the small number of cases in which this phenomenon has been reported[2,4,6,7] and suggests the need for caution while adding aripiprazole to augment the action of antipsychotics.

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