Tardive Dystonia–Associated Prescription of Aripiprazole

Article *in* Journal of Neuropsychiatry · February 2006

Impact Factor: 2.82 · DOI: 10.1176/appi.neuropsych.18.3.426-a · Source: PubMed

3 authors:

Narsimha R Pinninti
Rowan University
34 PUBLICATIONS **399** CITATIONS
SEE PROFILE

Rajnish Mago
Thomas Jefferson University
39 PUBLICATIONS **380** CITATIONS
SEE PROFILE

A. Adityanjee
Ohio Department of Mental Health and Addic…
93 PUBLICATIONS **1,161** CITATIONS
SEE PROFILE

post-exposure immunization. Although rabies was subsequently confirmed, the patient's history and presenting symptoms provided no straightforward explanation for the fatal outcome. Hence a diagnosis of rabies was not considered in the emergency room.

Clinicians in countries where rabies is still reported should consider its diagnosis while probing history in patients who mention death or sickness of their pet.

Vijay R. Prabhakar, M.D.
R.N. Monsy Abraham
Department of Emergency, Apollo Specialty Hospital, Chennai, India

Reference

1. Dutta JK: Rabies presenting with priapism. J Assoc Physicians India 1994; 42:430

Neuroacanthocytosis: Presenting With Depression

"Mr. A," a 38-year-old divorcee, presented to the department of neurology with complaints of alteration of the gait for the past 14 years. Since the age of 24, he had walked with a peculiar gait characterized by exaggerated lurching interspersed with sudden flexion movements at the hip and the knee joints bilaterally. He also had three episodes of generalized tonic clonic seizures when he was 26 years old; since then, he has been stabilized on 300 mg of phenytoin. For the past 3 years he has experienced difficulty with his speech and has found it difficult to articulate words. He also has experienced difficulty degluting solids. Following the onset of the illness, Mr. A lost his job, wife, and 6-year-old daughter. For the 8 months prior to presentation, Mr. A reported sadness and a loss of interest in those TV programs he usually watched. His interaction with his family members also diminshed and now he preferred to spend most of his time alone in his room. At times he experienced thoughts of helplessness and hopelessness. He found no purpose left in his life and thought of ending it. Neurological examination revealed dysarthric speech and chorieform movement of the bilateral upper limbs. There was no evidence of any sensory deficit. His magnetic resonance imaging (MRI) scans did not reveal any significant findings and his routine investigations, apart from the peripheral blood film (PBF), were within normal range. PBF showed accanthocytes that constitute 10% of the peripheral blood film. A detailed psychiatric evaluation produced evidence in favor of major depressive disorder following which he was put on a regimen of sertraline, 200 mg.

Neuroacanthocytosis is a progressive neurological disease first described by Levine in 1960 and characterized by movement disorders, personality changes, cognitive deterioration, axonal neuropathy, and seizures. Acanthocytes (>3%) are seen in peripheral blood smears. Neurochemical studies have revealed altered levels of catecholamines in various brain regions. Dopamine has been found to be decreased in almost the entire brain, and norepinephrine levels elevated in the putamen and globus pallidus. Studies have also reported decreased serotonin levels in the caudate nucleus and substantia nigra. The mean age of presentation ranges from 8 to 62 years with a possible male preponderance. Sporadic cases, as well as autosomal recessive, autosomal dominant, and X-linked inheritance patterns, have been reported. This disorder is linked to chromosome 9q21. Imaging studies have revealed caudate atrophy and increased signal in caudate and lentiform nuclei and hypometabolism in the neostriatum and the frontal cerebral cortical areas. The psychiatric manifestations of the disorder reported include personality changes, impulsivity, distractibility, anxiety, depression, apathy, loss of introspection, and compulsivity.[1,2] The treatment basically aims at supportive therapy and the management of associated behavioral symptoms.

Yatan Pal Singh Balhara, M.D.
Sunny T. Varghese, M.B.B.S.
Mridula Kayal, M.B.B.S.
Department of Psychiatry, All India Institute of Medical Sciences (AIMMS), New Delhi, India

References

1. Rafalowska J, Drac H, Jamrozik Z: Neuroacanthocytosis: review of literature and case report. Folia Neuropathol 1996; 34:178–183
2. Rinne JO, Daniel SE, Scaravilli F: The neuropathological features of Neuroacanthocytosis. Mov Disord 1994; 9: 297–304

Tardive Dystonia-Associated Prescription of Aripiprazole

SIR: Tardive dystonia is a delayed side effect of antipsychotic exposure that occurs at a frequency of 1% to 4.0%.[1] Most reports of tardive dystonia are from first-generation antipsychotics with recent case reports involving risperidone,[2] olanzapine,[3] and possibly clozapine. A MEDLINE/PubMed/Psylit search did not reveal any published reports of tardive dystonia associated with second-generation antipsychotics available in the United States, such as ziprasidone, quetiapine, or aripiprazole. We report a case of tardive

dystonia following the addition of aripiprazole to a patient's medication regimen.

A 35-year-old Caucasian woman diagnosed with schizoaffective disorder—bipolar subtype, panic disorder, and alcohol abuse was stable for a year on ziprasidone (160 mg), topiramate (300 mg), trazodone (100 mg hs) and temazepam (15 mg prn) for insomnia. She was hospitalized because of a relapse of psychotic, depressive, and panic symptoms. She was treated with clonazepam for panic and venlafaxine for depression. The dose of ziprasidone was increased to 200 mg/day with minimal improvement in psychotic symptoms. Aripiprazole was started and the dose titrated to 30 mg before the psychotic symptoms resolved completely. Discharge medications were ziprasidone (200 mg), aripiprazole (30 mg), clonazepam (3 mg), benztropine (2 mg), venlafaxine XR (225 mg), topiramate (300 mg), temazepam (30 mg), and trazodone (100 mg) a day.

Eight weeks after discharge she presented with 1 week of an inability to stand upright, being bent to the right, a mild degree of pain in the thoracic area, and significant emotional distress. Examination showed dystonic scoliosis of the thoracic spine with convexity to the left. No other muscle group was involved. There was no rigidity or involuntary movement in other parts of the body. She had a mild degree of akathisia. She had a history of normal birth and no associated medical problems. Aripiprazole was stopped while continuing all other medications. Within a week, akathisia subsided and in a month there was substantial improvement in dystonic symptoms. Three months later she remained free of dystonic symptoms.

The above clinical presentation satisfies the diagnostic criteria for tardive dystonia by Burke et al.[4] This case reminds us that despite its name, tardive dystonia is characterized more by the chronic nature of the dystonia than by the duration of antipsychotic exposure and is known to occur weeks or even days after use of antipsychotics.[4] Medications that could be implicated in tardive dystonia in this patient are ziprasidone, aripiprazole, venlafaxine, and trazodone. A drug-interaction software did not reveal any drug interaction relevant to this case. Trazodone and ziprasidone[5] have been associated with acute but not tardive dystonias. There are no reports of dystonia with venlafaxine. The dystonia, occurring 8 weeks after adding aripiprazole, showing dramatic improvement after stopping aripiprazole despite continuing ziprasidone and trazodone, makes aripiprazole the possible etiological agent. We cannot be sure if monotherapy with aripiprazole could have caused dystonia and prior treatment with antipsychotics may have predisposed the patient to developing tardive dystonia with addition of aripiprazole. Clinicians should realize that although monotherapy has a much lower incidence of extrapyramidal symptoms, when combined with second generation antipsychotics, like aripiprazole, they are not devoid of them. This includes symptoms of tardive dystonia.

Narsimha R. Pinninti, M.B.B.S., D.P.M., M.D.
Department of Psychiatry, UMDNJ School of Osteopathic Medicine, Cherry Hill, N.J.
Rajnish Mago, M.D.
Thomas Jefferson University, Philadelphia, Pa.
Adityanjee, M.D.
Schizophrenia and Psychotic Disorders Program, VA Medical Center, Minneapolis, Minn.

References

1. Marsalek M: Tardive drug-induced extra pyramidal syndromes. Pharmacopsychiatry 2000; 33(suppl):14–33
2. MVercueil L, Foucher J: Risperidone-induced tardive dystonia and psychosis. Lancet 1999; 353(9157):981
3. Dunayevich E, Strakowski SM: Olanzapine-induced tardive dystonia. Am J Psychiatry 1999; 156:662
4. Burke RE, Fahn S, Jankovic J, et al: Tardive dystonia: late-onset and persistent dystonia caused by antipsychotic drugs. Neurology 1982; 32:1335–1346
5. Dew RE, Hughes D: Acute dystonic reaction with moderate-dose ziprasidone. J Clin Psychopharmacol 2004; 24:563–564

Myasthenia Gravis Disclosed by Lithium Carbonate

SIR: A patient given lithium for bipolar disorder developed severe fatigue syndrome involving only the proximal limb muscles. The clinical and laboratory assessments revealed myasthenia gravis and thymic hyperplasia. Lithium was withdrawn and the patient underwent a thymectomy. A few months later the patient's symptoms followed a more classical myasthenic symptomatology pattern involving ocular and pharyngeal muscles. This change of the clinical manifestations may suggest that lithium modified the onset and pattern of myasthenic symptomatology.

Treatment with lithium may produce muscle weakness as a transient side effect, early in the course of therapy. A severe neuromuscular junction disorder during lithium treatment has been reported in five cases.[1–4] Lithium caused a myasthenic syndrome in four of them, which remitted fully after lithium withdrawal.[1–4] One case of myasthenia gravis was unmasked by lithium and followed the typical course of the disease after lithium discontinuation.[4] We report here a similar case.