Sayed Shahabuddin Hoseini
Faculty of Medicine, Medical Science/Tehran University, Tehran, Iran
Somayeh Mahdavi
Faculty of Science, Modares University, Tehran, Iran

References

1. Jansson ET: Alzheimer disease is substantially preventable in the United States—review of risk factors, therapy, and the prospects for an expert software system Med Hypotheses 2005; 64:960–967
2. Touyz RM: Reactive oxygen species, vascular oxidative stress, and redox signaling in hypertension: what is the clinical significance? Hypertension 2004; 44:248–252
3. Harman D: Alzheimer's disease pathogenesis: role of aging. Ann N Y Acad Sci 2006;1067:454–460
4. Heijer T, Skoog I, Oudkerk M, et al: Association between blood pressure levels over time and brain atrophy in the elderly. Neurobiol Aging 2002; 24:307–313
5. Poulet R, Gentile MT, Vecchione C, et al: Acute hypertension induces oxidative stress in brain tissues. J Cereb Blood Flow Metab 2006; 26:253–262

$GABA_A$ Versus $GABA_B$ in Catatonia

SIR: The role of GABA in catatonia has been identified. However, while hypoactivity at $GABA_A$ receptor may cause catatonia, increased activity at $GABA_B$ may worsen catatonia.[1] We report a patient who suffered from benzodiazepine withdrawal catatonia that could have been worsened by baclofen.

A 61-year-old man was treated for anxiety disorder without depressive features with diazepam 5 mg po TID. The patient was also on baclofen 20 mg po TID for cervicalgia.

The patient complained of chest pain and was admitted to the telemetry unit for observation. His stay was complicated secondary to refusal of medications, and he consequently suffered benzodiazepine withdrawal delirium accompanied by delusions of being poisoned. Laboratory studies including CBC and chemistry were within normal limits, but vital signs indicated mild autonomic elevations (pulse 110, normal sinus rhythm, blood pressure 165/116) consistent with benzodiazepine withdrawal delirium.

A psychiatric consultant noted the presence of catatonic signs including posturing, rigidity, waxy flexibility, negativism, and withdrawal as defined by the refusal of food and oral medications. The Bush-Francis Catatonia Rating Scale score was 20 indicative of moderately severe catatonia.[2]

The patient was given an initial dose of lorazepam at 1 mg IV with resolution of his immobility after approximately 1 hour at which time he appeared more agitated with verbal yelling. The patient received haloperidol and ziprasidone during the initial 3 day period to control verbal and physical agitation with mild improvement in overall catatonic signs, but with frequent switching from a psychomotor retarded state to an excited catatonic state. Lorazepam dosed from 3 to 6 mg IV daily was continued during this period to address his benzodiazepine withdrawal delirium as well. Catatonia improved gradually (BFCRS = 4), and by the seventh day, the patient showed improvements in orientation, but amnesia to recent events since his illness onset.[2]

He was seen 1 month later as an outpatient. The patient been discharged on diazepam 5 mg TID but baclofen was not restarted. Four months after this episode he was readmitted for an exacerbation of congestive heart failure. Diazepam was tapered from 5 mg TID to 2.5 mg TID without the return of catatonia or psychosis.

This patient's catatonic presentation appeared mostly likely secondary to benzodiazepine withdrawal delirium and a precipitous decline in $GABA_A$ activity after his refusal of po medications.[3] However, the presence of catatonia in this case is interesting because baclofen increases the activity of the $GABA_B$ receptor. In this case we found that the addition of baclofen may have upset the balance between $GABA_A$ and $GABA_B$ activity leading to catatonia that required prolonged treatment with lorazepam, a $GABA_A$ promoter for resolution.[4] We feel that this case may illustrate a potentially minor but clinically significant procatatonic role for $GABA_B$ activity.[1,5]

Brendan T. Carroll, M.D.
University of Cincinnati, and Chillicothe VA Medical Center, Chillicothe, Ohio
Christopher Thomas, Pharm.D.
University of Cincinnati, Ohio
Karen C. Tugrul, R.N.
University of Cincinnati, Ohio
Cristonel Coconcea, M.D.
Case Western Reserve University, Cleveland, Ohio
Harold W. Goforth, M.D.
Duke University Medical Center, Durham, N.C.

References

1. Lauterbach E C: Catatonia-like events after valproic acid with risperidone and sertraline. NNBN 1998; 11:157–163
2. Bush G, Fink M, Petrides, et al. Catatonia I: Rating scale and standardized examination. Acta Psychiatr Scand 1996; 93:129–136
3. Rosebush PI, Mazurek MF: Catatonia after benzodiazepine withdrawal. J Clin Psychopharmacol 1996; 16:315–319
4. Northoff G: What catatonia can tell us about "top-down modulation": a neuropsychiatric hypothesis. Behav Brain Sci 2002; 25:555–604
5. Carroll BT: The GABAa versus GABAb hypothesis of catatonia (letter). Movement Disord 1999; 14:702

Olanzapine-Induced Tardive Dystonia Successfully Treated by Tetrabenazine

SIR: Tardive dystonia (TD) is one of the tardive syndromes, along with