lent in RD than other autistic disorders.[3] In addition, regression in speech, communication skills and social interaction is suggestive of a pervasive developmental disorder, which, taken together with the above observations and occurrence in a female are reminiscent of RD. It has already been argued that the prevailing diagnostic criteria for RD are too restrictive, which may exclude atypical or borderline variants of this condition.[2] In addition, while autistic disorder and childhood disintegrative disorder are associated with acquired neurological disorders, the criteria and description of RD in DSM-IV TR are silent on this aspect. A recent update on diagnostic criteria for RD omitted the exclusion criterion of acquired neurological disorders in the revised criteria for variant phenotypes of this condition.[2]

The other pertinent facet of discussion is the use of lamotrigine in RD. Kumandas et al. used lamotrigine in two girls with RD and found that apart from convulsions, lamotrigine also improved stereotypical hand movements and autistic behaviors as happened with our patient.[4] Another study showed that lamotrigine enhanced social behavior, temper tantrums and emotional problems in some patients with RD.[5]. Besides RD, lamotrigine has been used, albeit with mixed results, in either autistic disorder or patients with intractable epilepsy showing autistic symptoms. One such open-label study showed a positive effect of lamotrigine on some autistic symptoms,[6] while a placebo-controlled trial failed to show any such effect.[7] Whether patients with RD present with motoric symptoms such as stereotypic hand movements, ataxic gait, and dystonic posturing which are differentially responsive to lamotrigine as compared to other autistic spectrum disorders, remains to be ascertained. Moreover, as lamotrigine is now known to be effective in certain affective disorders, whether RD patients with emotional dysregulation respond better to it, is worth exploring. This is suggested by decreased irritability and improved emotional stability in patients displaying autistic symptoms treated with this drug.[6] More systematic studies are needed to address these issues. At a neurotransmitter level, there is some evidence to support the role of lamotrigine in RD. Glutamate is elevated in CSF of patients with RD and lamotrigine, by way of inhibiting release of glutamate, may be effective in ameliorating some of the symptoms of this rare condition.[8]

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Neuroanatomical Changes After Eye Movement Desensitization and Reprocessing (EMDR) Treatment in Posttraumatic Stress Disorder

Several authors have found smaller hippocampal volumes in patients with PTSD and some have suggested that psychotropic drugs may promote hippocampus neurogenesis and reverse the decrease in hippocampus volume.[1] However, the only study that has investigated the effects of psychotherapy on hippocampus volume failed to show a volumetric increase after effective psychotherapy.[2]

EMDR is a standardized psychotherapy for amelioration of traumatic sequelae.

We evaluated the hippocampus volumetric changes after successful EMDR treatment of a 27-year-old man with a chronic PTSD related to the suicide of his mother. Written informed consent was obtained after the study procedures had been fully explained. The patient did not receive any medication during the 8 weeks of EMDR treatment. Current and lifetime PTSD diagnoses and severity were established by the Clinician Administered PTSD Scale (CAPS DX)[3] and the severity by the Davidson Trauma Scale (DTS).[4]

Morphovolumetric evaluation through high resolution MRI scanning (Philips 1.5T MRI) consisted of coronal T1 Fast Field Echo (matrix 512×512, 1 mm thick) images lying on the plane perpendicular to major hippocampal axis. Hippocampal volume was calculated using dedicated software (Analyze VW 1.16, BIR, Mayo Clinic, MN, U.S.) by manual delimitation of hippocampal shape according to Watson Laboratories, Inc. anatomical criteria on each slice where detected, by an operator

blind to subject treatment status. The sum of each area provided right and left hippocampal volumes.

A first bilateral MRI-based measurement of hippocampal volume was obtained at baseline, when the patient met the CAPS criteria for a PTSD diagnosis and the total score on the DTS was 51. The baseline hippocampus volumes were 2,838.91 mm^3 for the left hippocampus and 3,259.00 mm^3 for the right hippocampus. After 8 weeks of EMDR treatment (one 90 minute session/week), the patient no longer met the CAPS criteria for PTSD and the DTS total score had decreased from 51 to 8. The second MRI showed that the left and right hippocampus volumes were 3,196.24 and 3,599.40 mm^3 respectively. Therefore, the increase in hippocampus volume was 357.33 mm^3 (left) and 340.40 mm^3 (right) respectively.

Clearly, our observation in a single case cannot challenge the findings of Lindauer and colleagues,[2] who conducted a randomized clinical trial. Also, the relatively short period of time in which the volumetric increase happened poses the question of whether the increase was due to neurogenesis or may simply be attributable to an increased water and electrolyte content in the hippocampus. However, the magnitude of the volumetric change that we observed after a documented improvement in the PTSD symptomatology suggests the opportunity to not dismiss the question on whether psychotherapy can increase hippocampus volume and possibly have a beneficial effect on neurogenesis, which would be consistent with the hypotheses of several authors, such as Kandel,[5] who have suggested that both psychotherapy and pharmacotherapy may induce alterations in gene expression and structural changes in the brain.

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A Case Report With Ziprasidone-Induced Catatonic Symptoms

Introduction

SIR: Double-blind placebo-controlled studies in schizophrenic patients without major comorbidities and not taking other antipsychotics suggest that some atypical antipsychotics such as ziprasidone may have extrapyramidal symptom profiles not different than placebo and definitively better that typical antipsychotics.[1] The use of atypicals in the general population of psychiatric patients with multiple comorbidities and polypharmacy has provided a less optimistic picture[1] and the different types of extrapyramidal symptoms have started to be described for the atypical antipsychotics.

Few cases of neuroleptic malignant syndrome (NMS) in patients taking atypical antipsychotics[2] have been described but there are almost no cases of uncomplicated catatonic syndromes associated with atypical antipsychotics. No prior cases have been published of ziprasidone-induced uncomplicated catatonia but we have found three cases of NMS associated with ziprasidone treatment.[3–5] This case appears to be the first case of ziprasidone-induced uncomplicated catatonic picture.

Case Report

The 21-year-old Caucasian man had a 3-year history of DSM-IV schizophrenia and no prior catatonic episodes on risperidone (up to 10 mg/day), olanzapine (up to 30 mg/day), quetiapine (up to 400 mg/day), clozapine, (up to 500 mg/day), aripiprazole (up to 30 mg/day), and haloperidol decanoate (50 mg/3 weeks).

In April 2003, the patient was taking 30 mg of aripiprazole and agreed to be switched to ziprasidone. A dose of 80 mg of ziprasidone was added on the first day, and the next day he received 160 mg of ziprasidone divided in two doses (added to the 30 mg/day of aripiprazole). The staff noted that the patient was mute, staring into space and exhibiting bizarre behavior. On day 3 after the fourth 80 mg ziprasidone dose (and no aripiprazole), the patient was examined by a psychiatrist who using the Bush-Francis Catatonia scale found the patient had posturing, mutism, bilateral arm cogwheeling and a parkisonian generalized resting tremor. He did not have other catatonic symptoms such as autonomic instability. Ziprasidone was discontinued and the patient was treated with a stat 1 mg dose of oral loraze-