Mrs. M is a 30-year-old, married, African-American woman with no prior history of mental illness who was initially admitted to a general hospital due to changes in behavior. Mrs. M’s husband reported that during the 2 days prior to her admission, she seemed to have difficulty paying attention and registering information; she would sometimes talk excessively and at other times would stare blankly and not engage in any conversation with her husband. She also experienced insomnia and was intermittently restless. When she initially arrived at the general hospital, she was agitated and was given a one-time dose of ziprasidone 20 mg orally to calm her down. While at the general hospital, the patient was also selectively mute.

The initial medical work-up included a complete blood count, comprehensive metabolic panel, thyroid panel, urine drug screen, urinalysis, and serum creatine kinase (CK); the results were within normal limits except for a white blood cell count of 13,000/µl, 1+ bacteria in her urine and CK of 2,252. The elevated CK was thought to be secondary to agitation and the fact that she needed physical restraint. She was started on ciprofloxacin 500 mg orally for a suspected urinary tract infection. Quetiapine 25 mg orally at bedtime was started for agitation and to target insomnia. She was seen by a neurologist who felt there was a low likelihood that Mrs. M.’s presentation was secondary to encephalitis or meningitis. Nevertheless, she was treated empirically with ceftriaxone, vancomycin, and acyclovir while the results of cerebrospinal fluid (CSF) analyses were awaited. Computed tomography (CT) scan of the head, magnetic resonance imaging (MRI) of the brain (with and without contrast), and magnetic resonance angiogram (MRA) of the head and neck (without contrast) were all negative for any acute findings. Electroencephalogram (EEG) was negative for any abnormal activity, and the CSF analyses showed no growth on culture after 72 hours. The CSF analyses were also negative for Neisseria meningitides CW135, Escherichia coli K1, Haemophilus influenzae, Streptococcus pneumoniae, and Group B streptococcus. She was medically cleared by neurology and internal medicine, and the psychiatry consult-liaison team recommended inpatient psychiatric evaluation and treatment. She was subsequently transferred to our acute inpatient psychiatric unit by the fourth day of admission at the general hospital.

When she arrived in our unit, Mrs. M was uncooperative, selectively mute, and had significant psychomotor retardation. She appeared internally preoccupied, would sit and stare blankly at times with some inappropriate smiling. She also intermittently exhibited stereotypic movement and facial grimacing. She could not answer questions, and the treatment team was unable to obtain

Nilesh S. Tannu, MD; and Olaoluwa O. Okusaga, MD, MScPHR

Address correspondence to: Nilesh S. Tannu, MD, The University of Texas Medical School at Houston, Department of Psychiatry and Behavioral Sciences, 1941 East Road, Houston, TX 77054; email: Nilesh.S.Tannu@uth.tmc.edu.

Disclosure: The authors have no relevant financial relationships to disclose. doi: 10.3928/00485713-20140403-02
any information from her. She required nursing assistance with feeding and other activities of daily living. Her husband confirmed that she had no past psychiatric history. She had been her normal self before the current episode, which started about 7 days prior to her presentation to our inpatient psychiatric facility. He was not aware of any hospitalizations, medications, illicit drug use, or suicide ideations/attempts. He was also unaware of any family history of mental illness in the patient. Mrs. M had a normal developmental history and did not have any delay in her milestones. She completed her General Education Development and later went on to graduate with a diploma in medical billing and coding. She was employed as a biller and coder at a doctor’s office. She had been married for 6 years, had a 6-year-old child, and did not have any legal history. There were no significant findings on physical examination.

The patient was started on ziprasidone 20 mg twice a day on the day of admission to our inpatient unit and by day 2, this was increased to 40 mg twice a day and lorazepam 1 mg three times per day was also started. Ziprasidone was discontinued on day 3 of admission and risperidone was started at a dose of 2 mg in the morning and 1 mg at night. Mrs. M’s Bush Francis Catatonia Rating Scale (BFCRS) score was 25 on the day of admission to our unit, and during the next 4 days, she remained selectively mute and intermittently wandered aimlessly around the unit. She seemed to also have minimal choreoathetoid-like movement, and on day 5 serum ceruloplasmin and copper were ordered to rule out Wilson’s disease. The CK level was also reevaluated. Ceruloplasmin, copper and CK were within the normal range. She started showing some treatment response by day 6, and by the eighth day, she had improved significantly.

**She started showing some treatment response by day 6 and by the eighth day, she had improved significantly.**

Mrs. M. was no longer having purposeless movements and was able to engage in an intelligible conversation. She did not have any mood or psychotic symptoms at the time of discharge on day 10, and her BFCRS was then zero. We started tapering down the dose of lorazepam by day 8, and she was discharged on risperidone 3 mg/day and advised to follow up with an outpatient psychiatrist within a week. Mrs. M’s discharge diagnosis was brief psychotic disorder. We recommended that risperidone be discontinued in the outpatient if she remained well during the follow-up appointment.

**DIAGNOSIS:**

**De Novo Catatonia / Brief Psychotic Disorder (298.8)**

De novo catatonia and idiopathic catatonia are synonymous terms used when catatonia is the first neuropsychiatric manifestation in an individual with no previous history of psychiatric, neurologic, or medical disorder.\(^3\)\(^4\) De novo catatonia is not a separate disease entity in the *Diagnostic and Statistical Manual for Mental Disorders*, fourth edition (DSM-IV) or *The International Statistical Classification of Diseases and Related Health Problems*, 10th edition (ICD-10), and both systems of classification assume catatonia to be a subtype of schizophrenia, secondary to a general medical condition, or a specifier for a mood disorder. Currently, the literature on de novo catatonia is very sparse and only a few case reports have been published.\(^3\)\(^5\) We now report a case of de novo catatonia that was successfully treated with a combination of risperidone and lorazepam. We decided to give Mrs. M a final diagnosis of brief psychotic disorder (298.8) because she had catatonic behavior that lasted less than 1 month and eventual full return to premorbid level of functioning, and her condition could not be better accounted for by another psychiatric condition, substance use, or general medical condition. The creation of the new category, “unspecified catatonia (codes 781.99 and 293.89),” will be very helpful in diagnosing cases such as this. DSM-V had not yet been published when we initially saw this patient.

**DISCUSSION**

This case illustrates the utility of the risperidone-lorazepam combination for the treatment of catatonia, a finding that has been documented in a number of case reports;\(^6\) two of the previous reports were in individuals with mood or psychotic disorders, whereas one was in an individual with disulfiram-induced catatonia.\(^7\) Ours is the first report on the effec-
tiveness of the risperidone-lorazepam combination in an individual without any apparent psychiatric, medical, or medication-based cause of catatonia. We initially started the patient on ziprasidone but decided to switch to risperidone based on the case reports that suggested that risperidone in combination with lorazepam was an effective pharmacologic option in the treatment of catatonia. Though there are no controlled clinical trials, an increasing body of evidence points to the clinical effectiveness of second-generation anti-psychotics in non-malignant catatonia. Some authors have proposed that the plausible mechanism of the beneficial effect of second-generation antipsychotics in catatonia could be the antagonism of 5HT2A, thereby increasing the dopaminergic activity in the prefrontal cortex. The few studies of de novo catatonia seem to suggest that the condition comprises a significant percentage of all cases of catatonia. For example, 46.15% of the 65 cases of catatonia studied by Benegal et al. were classified as idiopathic (de novo catatonia), and in the sample studied by Barnes et al., 40% were classified as idiopathic catatonia. De novo catatonia appears to share identical clinical features with catatonia that develops in the context of schizophrenia or mood disorder. For example in the study by Benegal et al. the distribution of catatonic symptoms (eg, stupor, posturing, negativism, etc.) did not differ between subjects with de novo catatonia and those with catatonia on the background of other illnesses. Furthermore, both groups had a good response to electroconvulsive therapy. In addition, de novo catatonia was more prevalent among women, and the illness was of a shorter duration compared to catatonia associated with either schizophrenia or depression but the difference was not statistically significant. Similarly, nine of the 10 cases of de novo catatonia in the study by Barnes et al. were female, and the de novo catatonia group also seemed to have higher rates of first-degree relatives with a history of catatonia.

CONCLUSION
We have presented the case of a 30-year-old woman with de novo catatonia who was effectively treated with a combination of risperidone and lorazepam, with the patient having complete recovery and return to premorbid level of functioning. The outcome of this case is consistent with the few previous reports of the effectiveness of risperidone-lorazepam combination in treating catatonic symptoms. Controlled clinical trials of the effectiveness of the risperidone-lorazepam combination for de novo catatonia (as well as catatonia in the context of other psychiatric and medical conditions) are needed.

REFERENCES