We report here the case of a woman comorbid with monosomy X karyotype Turner syndrome and schizophrenia. She suffered from multiple relapses of psychosis with prominent depressed and anxiety mood. Treatment with risperidone failed at the beginning, but later injections of paliperidone palmitate became successful. Informed consent for this report was obtained from the patient and her family.

A 48-year-old woman had been diagnosed with Turner syndrome at age 17 years. At age 20 years, she had become paranoid with auditory hallucination, and reference and persecutory delusions. She was referred to our psychiatric outpatient department where schizophrenia was diagnosed and she was treated with thirizine 25 mg four times a day. She remained in a relatively stable condition for the next 20 years. At age 45 years, she developed a depressed mood, with suspicious attitude, auditory hallucinations, and reference and persecutory delusions. Her treatment was then switched to amisulpride 400 mg once at night, and then to risperidone Consta 37.5 mg every 2 weeks for better adherence. A stable condition was maintained for the following 2 years.

Due to the discontinuation of medications by the patient, auditory hallucination, delusion, negative symptoms, and depressed mood recurred and lasted for 3 months. At age 48 years she was again admitted to our psychiatric ward. On admission, her scores on the Positive and Negative Syndrome Scale (PANSS) measured Positive 33, and Negative 38. Her psychotic symptoms improved after a 3-week treatment with risperidone 5 mg per day. Nevertheless, she still showed depressed mood and negative symptoms. After risperidone treatment, her PANSS scores improved to Positive 18 and Negative 25. We switched her antipsychotics from risperidone to paliperidone 12 mg once daily in the morning. After a 2-week medication with oral paliperidone (12 mg), her mood incontinence, negative symptoms, and depressed mood improved without psychotic relapses. Her PANSS scores at the time of discharge were Positive 13 and Negative 15. Because of the history of her poor drug compliance, we changed her oral medication of paliperidone to paliperidone palmitate. After discharge, she returned to the psychiatric outpatient department for regular injections with paliperidone palmitate 150 mg every 4 weeks. Her mental condition remained stable thereafter without any discomfort of adverse effects for at least 1 year. The patient was subsequently able to return to work (at a government agency). Both the patient and her family have been very satisfied with the outcome.

This is the first case reported of a patient with monosomy 45 X karyotype Turner syndrome who developed schizophrenia at a later stage, and is also the first case to show that the prominent negative symptoms and depressed mood were treated successfully with paliperidone palmitate injections. Several cases of Turner syndrome with schizophrenia have been treated successfully with first- or second-generation antipsychotics.[2,3] According to Kilic *et al.*, girls with Turner syndrome have lower self-esteem, and more serious symptoms of anxiety and depression.[4] Patients comorbid with Turner syndrome and schizophrenia may have depressive or anxiety symptoms more than those with schizophrenia alone. For paliperidone, its higher 5-HT_{2A}/D_2 binding ratio, higher receptor affinity for α_2-receptors and 5-HT_7 receptors, and its mood-stabilizer-like effects might well improve the depressed mood and negative symptoms.[5] Paliperidone may be an effective and tolerable treatment for individuals with comorbid Turner syndrome and schizophrenia, especially those with higher levels of anxiety and depression.

DISCLOSURE STATEMENT

There is no potential conflict of interest.

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Cotard's syndrome in anti-*N*-methyl-D-aspartate receptor encephalitis

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ALTHOUGH COTARD'S SYNDROME is most commonly associated with psychiatric diseases, it has also been associated with medical conditions. A 31-year-old nurse complained of difficulties in performing her work. At 4 days post-onset, she showed depersonalization, saying, 'I feel myself detached from my own body.' At 7 days post-onset, she began to throw herself against walls in an attempt to commit suicide and was admitted to a psychiatric hospital. The following day, however, she developed status epilepticus and was transferred to our hospital. Her neurological examination showed disorientation and an indication of Kernig's sign. Although no ovarian teratoma was found and magnetic resonance imaging of her head revealed no abnormalities, she was diagnosed with anti-*N*-methyl-D-aspartate receptor

(NMDAR) encephalitis as she had lymphocytic pleocytosis with 62 cells/μL and elevated antibodies against NMDAR in her cerebrospinal fluid. She received two courses of methylprednisolone pulse therapy followed by 4 weeks of prednisolone tapering and intravenous administration of immunoglobulin.

Her neurological conditions gradually improved, but her prominent psychiatric symptoms remained. She developed Cotard's syndrome along with cenesthetic hallucinations. She complained, 'My body goes round and round, and I am always upside down. I am pulled to the wall as if by a magnet and then sucked into it. Do I have my own body?' Another day she said, 'My upper body feels dead. The world seems to have ended.' She even asked that we check her heart, saying, 'I am dead. I have no heart. All of my blood has been drained. My body has been chopped into pieces.' However, other than those symptoms, her behavior was organized and she was not in a depressive state. These abnormal experiences gradually subsided and she returned to her previous work.

Our patient with anti-NMDAR encephalitis presented with depersonalization, Cotard's syndrome, and cenesthetic hallucinations, which could be summarized as extreme abnormal somatic sensations. Drugs that block NMDAR function (e.g., phencyclidine and ketamine) can produce depersonalization and abnormal sensations.[1–3] Considering the synaptically related functions of NMDAR and symptoms related to anti-NMDAR encephalitis, we hypothesized that by blocking the NMDAR, similar symptoms to those seen in subjects with a ketamine addiction would emerge. Depersonalization is also reportedly a potential mechanism underlying the development of Cotard's syndrome.[4] Given the close relation between Cotard's syndrome and depersonalization, anti-NMDAR encephalitis may produce Cotard's syndrome.

Ethical aspects of this study were approved by Ashikaga Red Cross Hospital Human Research Ethics Committee. This report was written after obtaining informed consent from the patient.

DISCLOSURE STATEMENT

The authors have no conflict of interest to declare.

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Anxious distress in monopolar and bipolar depression: Clinical characteristics and relation with mixed depression in Japan

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BASED ON THE effects of anxiety accompanying major depressive disorder (MDD) and bipolar disorder (BD), the DSM-5 introduced the 'anxious distress' (ANXD) specifier.[1] While several studies have reported the clinical characteristics of major depressive episode (MDE) with ANXD due to MDD,[2,3] to the author's knowledge, those of MDE with ANXD due to BD remain unexplored.

The author examined the clinical features of MDE with ANXD due to not only MDD but also BD and compared the two groups. The relation between ANXD and mixed depression was also examined. The presence of ANXD was prospectively evaluated in 70 MDD and 99 BD outpatients with MDE via clinical interviews. Mixed depressions were defined exclusively (≥3 non-overlapping manic/hypomanic symptoms [NOMS], i.e., DSM-5 mixed features[1]) or inclusively (≥3 NOMS or overlapping manic/hypomanic symptoms [OMS, i.e., irritability, psychomotor agitation, and distractibility]). The patients provided written informed consent, and the study was conducted in accordance with the 1989 Declaration of Helsinki and approved by the institutional ethics committee.

The proportion of ANXD in BD patients was comparable to that in MDD patients (26.3% vs 30.0%, $P = 0.593$). The median age of patients with ANXD was higher than that of patients without ANXD in both groups, the difference being particularly pronounced in MDD patients (MDD, 50 vs 31 years, $P = 0.005$; BD, 43 vs 35 years, $P = 0.067$). A significantly larger proportion of the elderly (≥50 years) MDE patients with ANXD was diagnosed as having MDD rather than BD (64.7% vs 35.3%, $P = 0.038$). Significantly higher median Hamilton Depression Rating Scale-17 total scores were observed in MDE patients with ANXD than in those without ANXD in both groups (MDD, 20 vs 16, $P = 0.002$; BD, 19 vs 16, $P = 0.006$). The prevalence of ≥3 NOMS was low (4 [2.4%] in the entire sample), while ≥3 NOMS/OMS was more frequent in MDE patients with ANXD than in non-anxious patients (MDD, 66.7% vs 26.5%, $P = 0.002$; BD, 65.4% vs 46.6%, $P = 0.099$). Both MDD- and BD-related ANXD was closely associated with greater depressive symptom severity and inclusively defined mixed depression. However, the age distributions were different between patients with MDD- and BD-related ANXD, suggesting that the nature of comorbid anxiety may be different in monopolar and bipolar depression.