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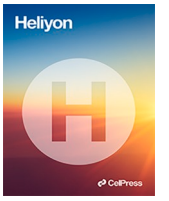

A case of comorbidity of schizophrenic catatonia and chronic intestinal pseudo-obstruction Successfully managed with lorazepam

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ABSTRACT

It is challenging to manage schizophrenic catatonia and comorbid chronic intestinal pseudo-obstruction (CIPO). The pathology of catatonia is unclear. There are few reports or research on this issue. In this case, we present a middle-aged woman diagnosed with schizophrenia with catatonic features and comorbid CIPO. In the treatment process, modified electroconvulsive therapy (mECT) improved her stupor and CIPO partially. Lorazepam alleviated her stupor and CIPO completely. It is the first report describing complete remission with lorazepam in patient suffering from comorbid schizophrenic catatonia and CIPO, which may benefit the exploration of pathophysiology and treatment of comorbidity of schizophrenia with catatonia and CIPO.

1. Introduction

Catatonia is defined as the presence of three or more of the following symptoms in the Diagnostic and Statistical Manual of Mental Disorders-Fifth Edition (DSM–5): catalepsy, waxy flexibility, stupor, agitation (not influenced by external stimuli), mutism, negativism, posturing, mannerisms, stereotypies, grimacing, echolalia, and echopraxia. Catatonia has not been a specifier or a subtype of schizophrenia. It has been included as a specifier for four other psychotic disorders (brief psychotic disorder, schizophreniform disorder, schizoaffective disorder, and substance-induced psychotic disorder), major depressive disorder and bipolar disorder [1]. Some experts hold that catatonia can be distinguished from other behavioral syndromes by a recognizable cluster of clinical features. They support the consideration of catatonia as an individual category in psychiatric diagnostic systems [2].

There are currently no broadly accepted treatment guidelines for catatonia. Drug therapies and electroconvulsive therapy (ECT) have been the mainstay of catatonia's treatment [3]. The most extensively researched treatment methods are benzodiazepines and ECT [4]. However, catatonia in schizophrenia may be somewhat less likely to respond to benzodiazepines alone, with a response in 40–50 % of cases [5]. A patient with catatonia and additional features of schizophrenia may require the judicious combination of benzodiazepine and antipsychotics, preferably second-generation antipsychotics (SGAs). Relapsing and chronic courses of catatonia are

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challenging. Long-term treatment with benzodiazepines, clozapine, or maintenance electroconvulsive therapy is often necessary [6].

One small, short-term trial suggested that risperidone may improve catatonic and positive symptoms scale scores amongst people with schizophrenia spectrum disorders and catatonic symptoms. Low-quality evidence indicated that ECT may result in greater improvement in the first three weeks of treatment [7].

For the treatment of schizophrenia spectrum disorders with catatonic symptoms, the Chinese Guidelines for the Prevention and Treatment of Schizophrenia (Second Edition) recommended sulpiride or atypical antipsychotic medications or mECT as the first line of treatment [8]. The Maudsley Prescribing Guidelines in Psychiatry 14th edition suggests 'consider SGA e.g. clozapine, olanzapine, some authorities recommend co-therapy with benzodiazepines' [9].

The pathology of catatonia has remained elusive [10]. There is evidence that catatonia is related to abnormal gamma amino butyric acid (GABAergic), dopaminergic, and glutamatergic systems, especially the gamma-aminobutyric ($GABA_A$) receptor [11,12].

Chronic intestinal pseudo-obstruction (CIPO) is characterized by impaired gastrointestinal (GI) motility which is similar to mechanical intestinal obstruction but without GI mechanical obstruction. Explicit diagnostic criteria for CIPO has not yet been established. The three main principles of investigating CIPO include: (i) to confirm the absence of GI mechanical obstruction; (ii) to identify underlying causes of secondary CIPO; and (iii) to identify the character of the disease and any complications present [13]. An integrated investigative approach is required to utilize radiology, endoscopy, laboratory, manometry, and histologic analysis. Small bowel manometry may provide pathophysiological relevant information on the mechanisms involved in CIPO patients. Its place as a diagnostic test is contested, as it has a low diagnostic specificity and as it will not influence treatment in most patients [14]. CIPO is underdiagnosed. Its treatment options are suboptimal and largely supportive [15].

CIPO is generally classified into primary or idiopathic, and secondary. Primary CIPO is usually seen in congenital or familial cases. Familial forms of CIPO have been described which can be autosomal dominant, autosomal recessive, and X-linked [15–17]. Secondary CIPO is usually caused by endocrine disorders (such as diabetes, hypothyroidism or hypoparathyroidism), autoimmune disorders (such as systemic sclerosis, paraneoplastic syndromes), neurological disorders, and certain infections. Worth noting, medications such as anticholinergic agents, or narcotics can also result in secondary CIPO [15,16,18]. Congenital and acquired CIPO both constitute three histological categories: disruptions in smooth muscle cells (myopathy), interstitial cells of Cajal (mesenchymopathy), intrinsic intestinal innervation and/or extrinsic autonomic innervations (neuropathy). Neuropathies, myopathies, and mesenchymopathies may act alone or together, to cause weakened or unsynchronized peristalsis [15,19]. The exact pathophysiology is unclear [15]. Gastrointestinal motility disorders can be caused by diseases of the entire neural axis, from the cerebral hemispheres to the peripheral autonomic nerves. Stroke, dementia, parkinsonism, multiple sclerosis, and diabetic neuropathy are the most prevalent neurologic conditions that impact gastrointestinal function [20]. Together with the area postrema, the nucleus of the solitary tract (NTS) and dorsal motor nucleus of the vagus (DMV) collectively form the dorsal vagal complex (DVC), the locus for central control of gastric functions, including motility [21].

2. Case presentation

A 56-year-old female was hospitalized because of stupor and abdominal distension at 15, December 2017.

2.1. History of present illness

In 1998, she presented with disorganized behavior, hallucination, and prominent delusion. Then she was sent to the Seventh People's Hospital of Hangzhou. The psychiatrist diagnosed her with schizophrenia and prescribed quetiapine (the maximum dose was 650mg daily) for her. Her symptoms improved. Gradually she was able to complete her work (as a universal lecturer), and take care of herself well. According to her doctor's advice, she took quetiapine 300–450mg daily for decades, and her illness was stable.

In 2016, she had another psychotic episodes without obvious inducement. Along with the previous symptoms, she presented a new one which was stupor. She was diagnosed with schizophrenia with catatonic features. When quetiapine was taken 600mg per day, her symptoms improved. After that, quetiapine 600mg per day was maintained.

In February 2017, her psychotic episodes occurred without obvious inducement. The symptoms were similar to previous episodes. She was diagnosed with schizophrenia with catatonic features once more. Her symptoms improved but were not completely relieved when she was given 650mg of quetiapine per day. Her hallucination and delusion were almost gone. However, her movements were a little slow, and her facial expression was somewhat stiff. Higher dosages of quetiapine, trihexyphenidyl (1–4mg per day), clonazepam (1–2mg per day), or lorazepam (0.25–1mg per day) had no effect. She was able to take care of herself and get along well with others. Quetiapine dosage was maintained at 650 mg per day.

On November 26, 2017, her situation deteriorated without obvious inducement. She lay in bed with her eyes open all day but did not speak or move. Meanwhile, her abdomen was distensible. Her symptoms persisted and were not accompanied by fever, diaphoresis, tachycardia, or unstable blood pressure. Her family had no way to give her food or medicine because she wouldn't swallow. Then she was sent to the community hospital. She was treated with intravenous fluids and gastrointestinal decompression there. However, her symptoms persisted without improvement. On December 15, 2017, she was admitted to our hospital.

2.2. Medical history

She had been diagnosed with diabetes mellitus (DM) for several years, and was taking metformin followed the endocrinologist's advice. The blood glucose indexes (fasting blood glucose and glycated hemoglobin) were within the normal range.

There is no substance abuse history. Her family members disclosed no mental illness.

2.3. Physical examination

A physical examination revealed abdominal distension and tympany. Bowel sounds were none. There was neither guarding nor rigidity on the abdomen. Neurological examination revealed bilateral rigidity. Apart from those, the other abnormal physical and neurological signs were not detected.

During the mental status examination, the patient laid still, spontaneously opened her eyes, stared vacantly into space, and made no eye contact with others. She remained unexpressive and did not respond when others spoke to her. She was well-groomed, alert, and mute. She also had systemic stiffness. She would maintain an unusual posture accompanied by external manipulations of the limbs. Her thought process, attention, mood and insight into her symptoms were unable to evaluate. Her Bush-Francis Catatonia Rating Scale (BFCRS) score was 23 (Table 1).

2.4. Laboratory testing

During the hospitalization, laboratory testing to exclude the possibility of organic diseases, blood examinations and electrophysical examinations, ultrasonic examinations, CT scan and magnetic resonance imaging (MRI) were ordered upon admission, including peripheral blood cells, coagulation function, hepatic function, renal function, blood electrolyte, thyroid function, infection markers, C-reactive protein, antistreptolysin-*O*-titer, fasting blood glucose, postprandial blood glucose and glycosylated hemoglobin tests， the quantitation of HBV markers, prolactin, sex hormone, alexin, antinuclear antibodies, antinuclear extract antibody spectrum determination, folic acid, vitamin B12, serum ferritin, and cortisol, urine sediment quantification, urine analysis, fecal analysis, and occult blood analysis; Electroencephalogram and electrocardiograph; a thyroid ultrasound, color doppler echocardiography, liver, gallbladder, pancreas and spleen ultrasound; a brain CT scan, a brain MRI, and a abdomen CT. These tests were conducted, but there were no remarkable findings, except for hypomagnesemia and hypokalemia. Her abdominal CT scan revealed apparent accumulated air and fecal, dilated intestine with air-fluid levels but without intestinal adhesions and specific digestive tract obstruction (Fig. 1). Cerebrospinal fluid (CSF) examinations, endoscopy and routine biopsies were not conducted because the parents' consent was not obtained.

2.5. Diagnosis and treatment (Table 2)

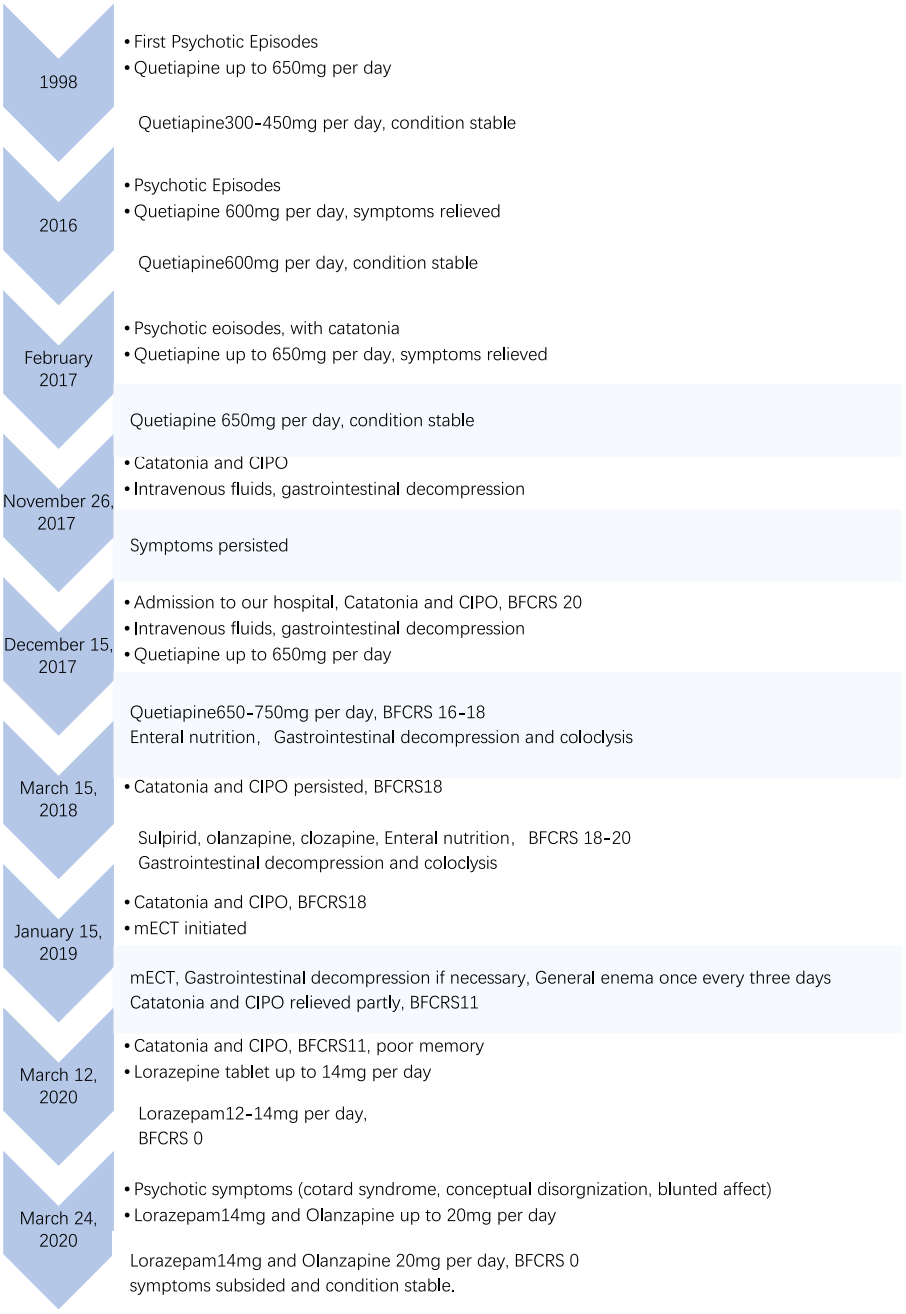
Multi-disciplinary consultation was held. According to DSM-5 criteria, a diagnosis of schizophrenia with catatonic features was made. Besides, CIPO was made, so were DM and electrolyte disturbance (Hypomagnesemia and hypokalemia).

The treatment plan was temporary fasting, gastrointestinal tube placement, gastrointestinal decompression, general enema if necessary, intravenous fluids to restore fluid and electrolyte balance and provide nutritional support. Quetiapine was prescribed. Her medication was fed nasally. Her symptoms appeared to slightly improve when quetiapine was taken 650mg per day. She could sit or walk alone. She had 0 to 1 bowel sound per minute. However, her movement was stiff. She didn't eat, drink or speak. The BFCRS score

Table 1
Bush francis catatonia rating scale.

Items	On Admission	Treatment of Quetiapine	Treatment of MECT	Treatment of Lorazepam
Excitement	0	0	0	0
Immobility/stupor	3	2	1	0
Mutism	3	3	0	0
Staring	3	0	0	0
Posturing/catalepsy	3	3	3	0
Grimacing	0	0	0	0
Echopraxia/echolalia	0	0	0	0
Stereotypy	0	0	1	0
Mannerisms	0	0	0	0
Verbigeration	0	0	0	0
Rigidity	0	0	0	0
Negativism	0	0	0	0
Waxy flexibility	1	1	1	0
Withdrawal	3	3	2	0
Impulsivity	0	0	0	0
Automatic obedience	0	0	0	0
Mitgehen	0	0	0	0
Gegenhalten	3	2	2	0
Ambitendency	0	0	0	0
Grasp	0	0	0	0
Reflex Perseveration	0	0	0	0
Combativeness	0	0	0	0
Autonomic abnormality	1	1	1	0
Total score	20	16	11	0

Table 2
Timeline with Relevant Treatment Data.



was 16 (Table 1.). Higher dosages of quetiapine had no further effect. When she was fed up with gastrointestinal nourishment, gastrointestinal decompression, and coloclysis were needed. Other pharmacologic treatments, including metformin tablet (500mgTID), mosapride tablet (5mgTID), simethicone (2mlTID), paraffin oil (10mlTID) potassium magnesium aspartate tablet and folium sennae, could relieve the abdominal distention and the decreased gastrointestinal hypomotility. Gastrointestinal decompression and coloclysis were still needed.

After that, the treatment strategy was changed. Sulpiride (the maximum dosage was 1200mg per day), olanzapine (the maximum dosage was 20mg per day), and clozapine (the maximum dosage was 500mg per day) have been prescribed separately. Her symptoms were not alleviated.

Then mECT was considered in January 2019. The treatment strategy was mECT (Thymatron System IV brief-pulse square-wave

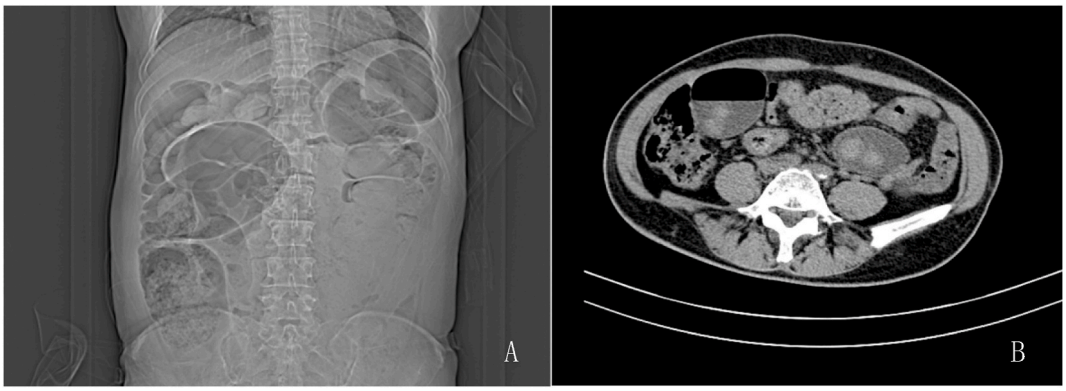


Fig. 1. CT image of the abdomen on admission (December 26, 2017). The scan revealed apparent accumulated air and fecal, dilated intestine with air-fluid levels but without intestinal adhesions and specific digestive tract obstruction. A was coronal, B was transverse.

apparatus, Somatics, LLC, Lake Bluff, Ill); bilateral electrode placement; maximum charge, 504 mC; mean seizure duration, 12 seconds) twice per week [22]. She was capable of eating food after three mECT sessions. After eight mECT sessions, she was able to follow the conversation but with less speech, spontaneous but few movements, and slowed movement responses. The abdominal distension was relieved but did not disappeared. Her bowel movements improved but remained less frequent. She was said to have 1 bowel movement every minute. The BFCRS score was 11 (Table 1). Her abdominal CT scan still showed apparent accumulated air and fecal (Fig. 2). Therefore, nasogastric tube feeding and coloclysis were still necessary. The patient received mECT for over a year, but her symptoms did not get any better. And her symptoms would relapse once stopped mECT therapy. She however lost her memories as a result of the therapy. Even some of the most important people from her recent past slipped her mind. The patient, her attendants, and her guardian all objected to the continuation of this therapeutic approach.

Thus, instead of continuing with mECT, we tried a high-dose lorazepam tablet (on the first day, 2mg, three times a day; on the second day, 4mg three times a day; on the third day 4mg twice a day and 6mg once per night, and then maintained the dosage). Other treatments remained the same. Significant clinical improvements were observed on the fourth day. Psychomotoric negativism and abdominal distension had significantly improved. She was able to eat and defecate independently. The abdominal distension subsided. Meanwhile, the patient was more communicative and paid more attention to the activities of her caregivers. She played mahjong with the other patients every day. All her symptoms of catatonia and CIPO disappeared. Nonetheless she displayed cotard syndrome (she believed her stomach had vanished), conceptual disorganization, impairment of immediate recall, affective flattening, and abulia. The BFCRS score showed 0 (Table 1). Her gastric tube was removed and enteral nutrition was discontinued. The mosapride tablet, simethicone, paraffin oil, and folium sennae were also discontinued. Besides, the delusion and conceptual disorganization were relieved after the prescription of olanzapine (20mg per day).

The patient and her guardian have consented for all clinical data and images included in the manuscript to be published.

3. Discussion

A synthesis of the psychiatric history, course of illness, and the psychiatric and medical evaluations suggested the diagnosis of schizophrenia with catatonic features, CIPO, DM, and electrolyte disturbance (Hypomagnesemia and hypokalemia).

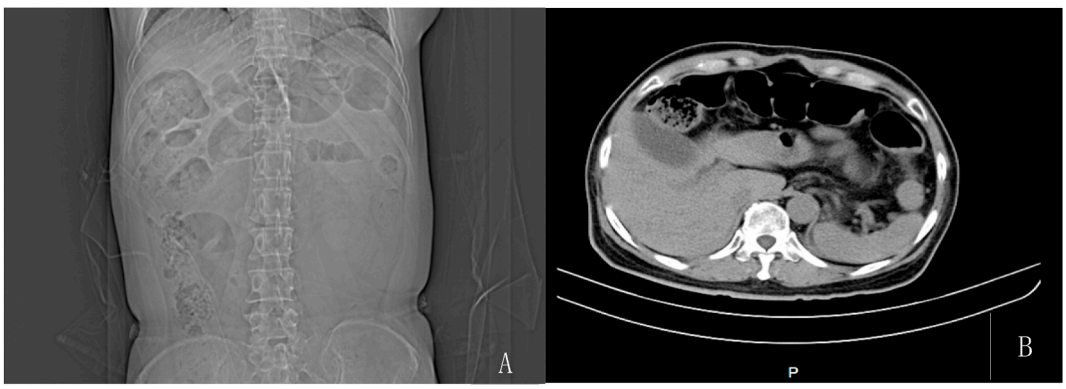


Fig. 2. CT image of the abdomen when the patient was treating with MECT (March 27, 2019). The abdominal CT scan still showed apparent accumulated air and fecal. A was coronal, B was transverse.

According to DSM-5, a diagnosis of schizophrenia with catatonic features was made. Because she had hallucinations and delusions prior to the stupor, and there were insufficient findings to suggest organic cerebral disease or drug-induced catatonia.

A diagnosis of CIPO was made because her physical examination revealed abdominal distension, her bowel sounds were none, and her abdominal CT scan revealed apparent accumulated air and fecal, dilated intestine with air-fluid levels but without intestinal adhesions and specific digestive tract obstruction.

Despite having received numerous antipsychotics, she had earlier displayed catatonia and CIPO symptoms when she was only taking quetiapine which she had been taking for many years. Her CIPO and catatonia symptoms did not improve when she ceased taking quetiapine. She then took quetiapine once more, which relieved her stupor and abdominal distension. Moreover, there were no indications of a fever, tachycardia, or unstable blood pressure. Therefore, malignant catatonia or neuroleptic malignant syndrome, and antipsychotics-induced CIPO were ruled out.

The patient has been diagnosed with DM and electrolyte disturbance. However, her blood glucose and glycosylated hemoglobin were within normal limits, which meant good glycemic control. There has been reported that gastric emptying and intestinal transit are unaltered in Type2 DM patients with good glycemic control [23]. Her electrolyte disturbance emerged after the CIPO. CIPO induced by DM or electrolyte disturbance was ruled out based on these.

In this case, quetiapine was effective for the symptoms of catatonia and CIPO initially. Then SGAs had no effect on the catatonia or CIPO.

Antipsychotics are extremely effective medications for schizophrenia, but there is insufficient research on their use in treating catatonic schizophrenia. SGAs may lessen acute catatonic and psychotic symptoms, according to data from naturalistic studies and randomized controlled trials that specifically targeted schizophrenia patients with catatonic symptoms [24]. A case series of 39 patients suggests that quetiapine is a promising agent for the treatment of schizophrenia with catatonic stupor during the acute phase [25].

A favorable response to antipsychotic treatment is contingent on dopaminergic dysfunction, whereas treatment resistance may be characterized by an abnormality of a non-dopaminergic mechanism-a glutamatergic mechanism would be a possible candidate [26]. This could explain why antipsychotic medications are no longer effective as a treatment in this case.

It has been reported that electroconvulsive therapy is effective and safe for serious catatonia-related ileus. That was a ‘cases report’ with two cases. The two patients didn’t response to the treatment of lorazepam [27]. In our case, mECT partially alleviated the symptoms of catatonia and CIPO, Lorazepam completely improved the symptoms of catatonia and CIPO. Besides, we observed that the catatonia and CIPO appeared and disappeared at the same time in the later stage of the patient’s disease. Therefore, we considered that the CIPO was related to catatonia.

Catatonia has been associated with a reduced density of $GABA_A$ receptors in the left sensorimotor cortex on iomazenil single-photon emission CT, suggesting that GABAergic hypofunction may be central to the disorder [28]. ECT and lorazepam, which are effective in catatonia, may exert their effect through GABA systems [12]. It is reported that benzodiazepines may act by increasing GABAergic transmission or reducing levels of brain-derived neurotrophic factor [29].

DMV neurons regulating gastric functions are tonically active and exhibit ‘pacemaker’ properties. It is the ongoing tonic GABAergic inhibitory synaptic input that plays the most significant role in regulating DMV neuronal activity under basal conditions [30–32]. Therefore, modulation of these GABAergic synaptic inputs has the potential to exert profound effects on DMV neuronal excitability, and vagal efferent control of gastric motility [21].

GABAergic NTS-DMV synaptic transmission is important in regulating vagal efferent outflow to the stomach, and this synapse is remarkably resistant to modulation. Browning KN and Travagli RA’s research has shown that the ‘state of activation’ of this critical inhibitory NTS-DMV synapse is determined by ongoing vagal afferent activity [21,33]. Vagal afferents make monosynaptic connections with inhibitory NTS terminals [34]; Removal of this tonic metabotropic glutamate receptor activation, either by decreasing vagal afferent input, blocking metabotropic glutamate receptors, or by activating adenylate cyclase directly, disinhibits the activity of these inhibitory terminals, allowing such previously resistant synapses to be modulated [35].

Lorazepam, whose main target is $GABA_A$, may impact the quantity or distribution of $GABA_A$ receptors [11], and further lead to the removal of the tonic metabotropic glutamate receptor activation, which may be related to the patient’s CIPO being cured. ECT is known to enhance dopaminergic, serotonergic, and adrenergic neurotransmission, GABA and glutamate have been implicated as well [36,37], which may explain why mECT partially improved the CIPO symptoms.

Limitation

Antipsychotics were used intermittently. The risk of uncommon adverse effects including antipsychotic-induced catatonic states or more seriously, precipitating neuroleptic malignant syndrome, remain a concern requiring careful monitoring [24]. It seems likely that gastrointestinal motility will remain reduced for a long time even after the drug has been stopped once drug-induced gastrointestinal hypomotility has chronicized [38]. However, in our case, the patient displayed catatonia and CIPO during the long-term use of quetiapine. The symptoms did not relieve after discontinuing the medication, but they did after the resumption of the antipsychotic. As a result, we do not take antipsychotic-induced catatonia and CIPO into account. High-quality trials continue to be necessary to differentiate treatments for people with symptoms of catatonia in schizophrenia spectrum disorders. The lack of consensus on the psychopathology of catatonia remains a barrier to defining treatments for people with schizophrenia. Better understanding of the efficacy and safety of antipsychotics may clarify treatment for this unique subtype of schizophrenia [7].

CSF examinations were not conducted because the consent was not obtained.

The symptoms scales, such as the Positive and Negative Syndrome Scale, Montreal Cognitive Assessment, and Electroconvulsive

Cognitive Assessment were not estimated because the patient was unable to cooperate. We do not have enough quantitative information about her situation, such as cognitive function, psychotic symptoms.

Further studies are needed to monitor and collect data on patients with schizophrenia with catatonic features comorbid with CIPO in large cohort studies with long-term follow-up.

4. Conclusion

To the best of our knowledge, it is the first report describing complete remission with lorazepam in patient suffering from comorbid schizophrenic catatonia and CIPO.

In the case report, we witnessed a patient diagnosed with schizophrenia with catatonic features comorbid with CIPO. During treatment, SGAs failed to alleviate the stupor and CIPO, mECT partially improved the stupor and CIPO, and lorazepam completely relieved her stupor and CIPO. Lorazepam might be an effective option in patients with comorbidity of schizophrenic catatonia and CIPO. The effective treatment of catatonia and CIPO may be related to the glutamate and gamma-aminobutyric acid systems.

Such a case report may provide some valuable information for schizophrenia with catatonic features and comorbid CIPO. Nevertheless, greater identification, more focused treatment, and further research directed at identifying the neurobiological substrate of the disorder are required.

Data availability statement

Data will be made available on request.

Additional information

No additional information is available for this paper.

CRediT authorship contribution statement

Yang Cao: Writing – original draft, Visualization, Data curation. **Weixin Wang:** Writing – original draft, Investigation, Data curation, Conceptualization. **Jiong Chen:** Writing – review & editing, Supervision, Conceptualization. **Bo Jiang:** Writing – review & editing, Validation. **Sugai Liang:** Writing – review & editing. **Xianyu Chen:** Writing – review & editing. **Hui Dong:** Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] R. Tandon, et al., Catatonia in DSM-5, Schizophr. Res. 150 (1) (2013) 26–30.
[2] M. Fink, E. Shorter, M.A. Taylor, Catatonia is not schizophrenia: kraepelin's error and the need to recognize catatonia as an independent syndrome in medical nomenclature, Schizophr. Bull. 36 (2) (2010) 314–320.
[3] H. Zaman, R.C. Gibson, G. Walcott, Benzodiazepines for Catatonia in People with Schizophrenia or Other Serious Mental Illnesses, Cochrane Database of Systematic Reviews, 2019.
[4] A. Pelzer, F. van der Heijden, E. den Boer, Systematic review of catatonia treatment, Neuropsychiatric Dis. Treat. 14 (2018) 317–326.
[5] M.M. Rosebush Pi, Catatonia: re-awakening to a forgotten disorder, Mov. Disord. 14 (3395–397) (1999) 395–397.
[6] S. Heckers, S. Walther, Caring for the patient with catatonia, JAMA Psychiatr. 78 (5) (2021) 560–561.
[7] M.W. Huang, et al., Antipsychotics for schizophrenia spectrum disorders with catatonic symptoms, Cochrane Database Syst. Rev. 7 (7) (2022), CD013100.
[8] J. Zhao, S. Shi, Chinese Guidelines for the Prevention and Treatment of Schizophrenia (Second Edition), first ed., China Medical Electronic Audio Visual Press, Beijing, 2015.
[9] D.M. Taylor, T.R.E. Barnes, A.H. Young, The Maudsley Prescribing Guidelines in Psychiatry, fourteenth ed., Wiley, Hoboken, NJ, 2021.
[10] S. Walther, et al., Structure and neural mechanisms of catatonia, Lancet Psychiatr. 6 (7) (2019) 610–619.
[11] K. Samra, et al., Catatonia with GABAA receptor antibodies, Practical Neurol. 20 (2) (2020) 139–143.
[12] A. Unal, et al., Effective treatment of catatonia by combination of benzodiazepine and electroconvulsive therapy, J. ECT 29 (3) (2013) 206–209.
[13] R. De Giorgio, et al., Chronic intestinal pseudo-obstruction: clinical features, diagnosis, and therapy, Gastroenterol. Clin. N. Am. 40 (4) (2011) 787–807.
[14] S. De Meulder, T. Vanuytsel, Chronic intestinal pseudo-obstruction: a case report with review of the literature and practical guidance for the clinician, Acta Gastroenterol Belg 85 (1) (2022) 85–93.
[15] T.J. Downes, et al., Pathophysiology, diagnosis, and management of chronic intestinal pseudo-obstruction, J. Clin. Gastroenterol. 52 (6) (2018) 477–489.
[16] X. Piao, et al., Chronic idiopathic intestinal pseudo-obstruction, Cureus 13 (7) (2021), e16563.
[17] A. Gargiulo, et al., Filamin A is mutated in X-linked chronic idiopathic intestinal pseudo-obstruction with central nervous system involvement, Am. J. Hum. Genet. 80 (4) (2007) 751–758.
[18] M. Selgrad, et al., JC virus infects the enteric glia of patients with chronic idiopathic intestinal pseudo-obstruction, Gut 58 (1) (2009) 25–32.
[19] M.T. Muñoz-Yagüe, P. S-M, I. Salces, C. Ballestín, F. Colina, C. Ibarrola, G. López-Alonso, P. Carreira, F. Cruz Vigo, J.A. Solís Herruzo, Chronic intestinal pseudo-obstruction: a diagnosis to be considered, Rev. Esp. Enferm. Dig. 101 (5) (2009) 336–342.
[20] M. Camilleri, Gastrointestinal motility disorders in neurologic disease, J. Clin. Invest. 131 (4) (2021).
[21] K.N. Browning, R.A. Travagli, Central control of gastrointestinal motility, Curr. Opin. Endocrinol. Diabetes Obes. 26 (1) (2019) 11–16.

[22] Chinese Association of Neurological Regulation Committee for Electroconvulsive Therapy and Nerve Stimulation, Chinese association of sleep committee for mental psychology, and C.A.o. Anesthesiology, Expert consensus on modified electroconvulsive therapy (2019) Translational Medicine Journal 8 (3) (2019) 129–134.
[23] E. Sena, et al., Gastrointestinal motility in elderly patients with well-controlled Type 2 diabetes mellitus, Arq. Gastroenterol. 59 (2) (2022) 231–237.
[24] S.N. Caroff, G.S. Ungvari, G. Gazdag, Treatment of Schizophrenia with Catatonic Symptoms: A Narrative Review, Schizophr Res, 2022.
[25] B. Yoshimura, et al., Is quetiapine suitable for treatment of acute schizophrenia with catatonic stupor? A case series of 39 patients, Neuropsychiatr Dis Treat 9 (2013) 1565–1571.
[26] L.D. Vanes, et al., Differential neural reward mechanisms in treatment-responsive and treatment-resistant schizophrenia, Psychol. Med. 48 (14) (2018) 2418–2427.
[27] Y. Yamada, et al., Electroconvulsive therapy is effective and safe for serious catatonia-related ileus: two case reports, J. ECT 36 (4) (2020) e49–e51.
[28] G. Northoff, R. Steinke, C. Czcervenka, R. Krause, S. Ulrich, P. Danos, D. Kropf, H. Otto, B. Bogerts, Decreased density of GABA-A receptors in the left sensorimotor cortex in akinetic catatonia: investigation of in vivo benzodiazepine receptor binding, J. Neurol. Neurosurg. Psychiatry 4 (67) (1999) 445–450.
[29] T.L. Huang, Y.Y. Hung, Lorazepam reduces the serum brain-derived neurotrophic factor level in schizophrenia patients with catatonia, Prog. Neuro-Psychopharmacol. Biol. Psychiatry 33 (1) (2009) 158–159.
[30] R.A. Travagli, R. G, C.D. Rossiter, S. Vicini, Glutamate and GABA-mediated synaptic currents in neurons of the rat dorsal motor nucleus of the vagus, Am. J. Physiol. 260 (10) (1991) 305–313.
[31] K.Z. Sivarao Dv, P.J. Hornby, Role of GABAA receptors in rat hindbrain nuclei controlling gastric motor function, Neuro Gastroenterol. Motil. 10 (1998) 305–313.
[32] T. Babic, K.N. Browning, R.A. Travagli, Differential organization of excitatory and inhibitory synapses within the rat dorsal vagal complex, Am. J. Physiol. Gastrointest. Liver Physiol. 300 (1) (2011) G21–G32.
[33] K.N. Browning, R.A. Travagli, Plasticity of vagal brainstem circuits in the control of gastrointestinal function, Auton. Neurosci. 161 (1–2) (2011) 6–13.
[34] L. Rinaman, J. C, J.S. Schwaber, R.R. Miselis, Ultrastructural demonstration of agastric monsynaptic vagal circuit in the nucleus of the solitarytract in rat, J. Neurosci. 9 (1989) 1985–1996.
[35] K.N. Browning, R.A. Travagli, Functional organization of presynaptic metabotropic glutamate receptors in vagal brainstem circuits, J. Neurosci. 27 (34) (2007) 8979–8988.
[36] E. Esel, K. K, Y. Hacimusalar, S. Ozsoy, M. Kula, Z. Candan, T. Turan, The effects of electroconvulsive therapy on GABAergic function in major depressive patients, J. ECT 24 (3) (2008) 224–228, 2008 Sep;24(3):224-228.
[37] Charles H. Kellner, R.M. G, James W. Murrough, Ethan O. Bryson, Mimi C. Briggs, Rosa M. Pasculli, ECT in treatment-resistant depression, Am J Psychiatry 169 (12) (2012) 1238–1244.
[38] S. Every-Palmer, et al., Clozapine-treated patients have marked gastrointestinal hypomotility, the probable basis of life-threatening gastrointestinal complications: a cross sectional study, EBioMedicine 5 (2016) 125–134.