—Case Reports—

Importance of Early Diagnosis and Treatment of Perioperative Catatonia: A Case Report

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Symptoms of catatonia include silence, motionlessness, and postural retention. Although it is important to detect and treat catatonia early, before it becomes severe, postoperative cases have inherent risks that hinder diagnosis and treatment. A 60-year-old man with schizophrenia underwent endoscopic/thoracoscopic esophagectomy and was extubated in the operating room. In the intensive care unit (ICU), he had stiffness in the neck, ankles, and knees, catalepsy during passive knee flexion, mild disturbance of consciousness, mild creatine kinase elevation, and respiratory depression. Intravenous diazepam was administered for diagnosis, and the patient's rapid improvement indicated catatonia. He was intubated and started on lorazepam; tapering produced no recurrence of symptoms. The patient was extubated and transferred to the general ward on postoperative Day 2. Because this patient was extubated in the operating room and was managed postoperatively in the ICU with a full-time doctor, his symptoms were easily recognized and early diagnosis was possible. Thus, we were able to administer drug therapy quickly and adequately and perform forward management that accounted for postoperative risks, thereby achieving a favorable outcome. (J Nippon Med Sch 2024; 91: 347–350)

Key words: catatonia, postoperative risks, benzodiazepine therapy

Introduction

Catatonia is a disorder characterized by silence, motionlessness, and postural retention. Left untreated, it leads to malignant catatonia accompanied by fever and autonomic imbalance, which progresses to myolysis, renal failure, electrolyte abnormalities, and arrhythmia, which can be fatal. Because benzodiazepine administration is an effective treatment, early diagnosis and initiation of treatment are important[1].

Although there have been reports of postoperative catatonia[2,3], the number of reports is small, and details regarding diagnostic pitfalls and treatment risks are unclear. We here present a series of procedures for diagnosis, treatment, and postoperative management of catatonia that we developed after experiencing a patient with long-term complications of surgery for schizophrenia.

Case Presentation

A 60-year-old man was scheduled for thoracoscopic esophagectomy and gastric tube reconstruction. He had been diagnosed at another hospital with schizophrenia, insomnia, and alcoholism and had been prescribed various antipsychotics, some of which he was taking at higher than the standard doses. As perioperative management, the psychiatric department of our hospital carried out a preoperative evaluation and drug adjustment. General anesthesia was administered alone, with 60 mg propofol, 0.1 mg fentanyl, and 50 mg rocuronium as induction drugs; 5% desflurane and 0.2-0.3 mcg/kg/min remifentanil as maintenance drugs; and fentanyl (intravenous patient-controlled analgesia; 20 μg/mL/hr, lock time 10 min; bolus dose of 1 mL) as a postoperative analgesic. The operating time and anesthesia time were 13 hours and 41 minutes, and the total intraoperative dose of fentanyl was 1.33 mg. After the inhalational anesthetic

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Table 1 Prescribed medicines

Drug	By previous doctor	Pre-operative	Day of surgery (morning)	At discharge	General usage and dosage in Japan	Type, mechanism of action
Olanzapine (®Zyprexa)	25 mg/day	25 mg/day	5 mg	15 mg/day	Administer 5-10 mg once a day Do not exceed the daily dose of 20 mg	Atypical antipsychotic: multi-receptor antipsychotic (MARTA)
Chlorpromazine (®Contomin)	150 mg/day	150 mg/day	interrupted*	Canceled	50-450 mg daily in divided doses	Phenothiazine, typical antipsychotic: primarily a dopamine receptor blocker
Flunitrazepam (®Silence)	3 mg/day	1 mg/day	interrupted*	1 mg/day	Oral administration of 0.5-2 mg once before going to bed or before surgery	Benzodiazepine (intermediate type)
Nitrazepam (®Benzarine)	10 mg/day	10 mg/day	interrupted*	10 mg/day	Oral administration of 5-10 mg once before bedtime	Benzodiazepine (intermediate type)

*due to surgery

was discontinued, neck stiffness was noted, but voluntary response was observed, and the patient's respiration and circulation were stable. When he was returned to the intensive care unit (ICU), he responded to speech by blinking, but had difficulty speaking, and no follow-up movements including limb movements were observed. Muscle rigidity was noted in the neck, ankle joints, and knee joints, as was catalepsy after passive knee flexion. The patient had a blood pressure of 157/98 mm Hg, a heart rate of 87 beats/min, a respiratory rate of 10 breaths/min, an oxygen saturation of 97%, and a bladder temperature of 36.8°C.

The patient's blood test results showed the following: white blood cells, 19.0 × 103 /μL; hemoglobin, 12.3 g/dL; platelets, 274 × 103/μL; aspartate aminotransferase, 146 U/L; alanine transaminase, 153 U/L; lactate dehydrogenase, 372 U/L; creatine kinase, 580 U/L; total bilirubin, 0.6 mg/dL; potassium, 4.4 mmol/L; blood urea nitrogen, 14.1 mg/dL; creatinine, 0.91 mg/dL; myoglobin, 319 ng/mL.

When diazepam 5 mg was administered by intravenous injection for diagnostic purposes, the above symptoms improved rapidly, indicating catatonia. As additional treatment, oral (intestinal fistula administration) lorazepam was started. The patient's respiratory rate at this time was 8 breaths/min. Given the risk of developing respiratory depression associated with interaction with fentanyl, tracheal intubation was performed before dosing. The dose was reduced to 3 mg/day on the 1st day after the operation and to 1.5 mg/day on the 2nd day, and no recurrence of symptoms was observed. After confirming that the patient's biomarkers were stable, he was extubated on the second day after surgery and was transferred to the general ward. Lorazepam was tapered to 1 mg/day on the 4th postoperative day, to 0.5 mg/day on the 8th postoperative day, and then stopped on the 11 th postoperative day. The patient was discharged home 38 days after surgery. **Table 1** shows the prescribed doses of medications taken by the patient.

Discussion

Before publication of the 4th edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-4), catatonia was considered a subtype of schizophrenia called catatonic schizophrenia, but after DSM-5 was published in 2013, it was considered a syndrome with specific psychosomatic symptoms brought about by various causative diseases, a history of use of antipsychotics and/or selective serotonin reuptake inhibitors, or organic brain disorders[1,4,5]. Although the mechanism of catatonia has been suggested to be related to γ-aminobutyric acid, dopamine, and *N*-methyl-D-aspartate receptors, this has not yet been elucidated[1]. In severe cases (malignant catatonia), fever, autonomic imbalance, and elevated creatine kinase appear and require intensive care management, but catatonia can be resolved with appropriate treatment. A diagnosis is made when 3 of the 12 characteristic symptoms are present[5]. Benzodiazepine therapy (BT) is the first choice for treatment, and electroconvulsive therapy is indicated in treatment-resistant cases[6]. Regardless of the underlying disease, the above treatment methods are effective, and early treatment increases the cure rate. In cases of malignant catatonia, it is essential to treat catatonia as a syndrome and start treatment promptly be-

cause the severity of the disease affects the mortality rate. Its differential diagnosis from neuroleptic malignant syndrome becomes problematic because the 2 syndromes are treated differently. Lang et al.[7] reported that differentiation is possible but is often difficult in clinical practice, as there are many overlapping symptoms. Furthermore, differentiation becomes more difficult when symptoms of catatonia progress and autonomic symptoms appear (malignant catatonia). A report that BT was remarkably effective in a patient diagnosed with malignant syndrome who showed resistance to dantrolene treatment highlighted the difficulty of differentiation as well as the importance of BT[8]. Therefore, it is important to detect the psychosomatic symptoms characteristic of catatonia in the early stages.

The present case highlights postoperative risks associated with the diagnosis and treatment of catatonia because it developed perioperatively. Postoperative delirium, residual anesthetic, use of opioids for postoperative analgesia, and use of sedation for systemic management may obscure the characteristic symptoms of catatonia, leading to delayed diagnosis and increased severity. Indeed, previous reports confirm that this diagnosis often takes time[2,3]. Even when symptoms are severe, it is necessary to distinguish catatonia not only from neuroleptic malignant syndrome, but also from late-onset malignant hyperthermia induced by inhalation anesthetics. These diseases have similarities in terms of the intensive care management required when they become severe, and even if definitive diagnosis is not possible, initial treatment can be started. However, considering the efficacy of benzodiazepine administration for catatonia, early diagnosis is critical[8]. In the present case, early diagnosis was possible because it was relatively easy to recognize the symptoms because the patient woke from general anesthesia and was extubated in the operating room. Because we were able to recognize the symptoms quickly, BT was administered promptly for diagnosis under the guidance of a psychiatrist, even though there were only 2 symptoms that clearly corresponded to the diagnostic criteria: stupor and catalepsy.

Furthermore, we administered benzodiazepine while taking into consideration the possibility of respiratory depression due to interactions with the opioids used for postoperative analgesia. The patient's respiratory rate was 10 breaths/min immediately before administration of diagnostic diazepam and <8 breaths/min before additional oral lorazepam (intestinal fistula administration). Prioritizing adequate BT increases the risk of respiratory depression and hypoxia due to metabolism, drug interactions and synergies, as well as other uncertainties, especially after surgery. Previous cases of emergency admission to the ICU for hypotension and hypoxia after BT have been reported[3]. However, use of inadequate BT, to avoid respiratory and circulatory depression, increases the risk of catatonia becoming malignant and decreases the survival rate[9]. Because the present patient had undergone surgery for a malignant esophageal tumor, esophageal intubation had a high risk of rupturing the reconstructed gastric suture, and reliable tracheal intubation was necessary. Emergency tracheal intubation should be avoided whenever possible. After comparing respiratory depression and airway management problems with the risk of progression to malignant catatonia from delayed treatment, we chose a safer method of tracheal intubation followed by adequate benzodiazepine administration to ensure the patient's safety while providing appropriate treatment.

In summary, we experienced a case of catatonia that developed after long-term surgery for schizophrenia. Despite the risks associated with the postoperative period, diagnosis and treatment were appropriate, worsening of catatonia was prevented, and a favorable outcome was achieved.

Conflict of Interest: The authors have no conflict of interest to declare.

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