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Title: Clinical utility of electroconvulsive therapy for the treatment of multidrug-resistant psychosis emerging in older adults: A case report

Running title: ECT for psychosis emerging in old age

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Introduction

'Late paraphrenia' and 'very-late-onset schizophrenia-like psychosis (VLOSLP)' describe primary psychotic disorders, not attributed to dementia and affective disorders, that emerge at age 60 or older (1, 2). Although such patients are classified as patients with schizophrenia or delusional disorders according to the Diagnostic and Statistical Manual of Mental Disorders Fifth Edition, characteristics such as female preponderance and lack of negative symptoms support the validity of differentiating VLOSLP from average-onset schizophrenia spectrum disorders. Antipsychotics are considered the first-line treatment for both average-onset schizophrenia spectrum disorders and VLOSLP. However, some patients with VLOSLP do not respond sufficiently to antipsychotics, although many reports support their effectiveness(3). Regarding average-onset schizophrenia, clozapine and electroconvulsive therapy (ECT) are often administered in multidrug-resistant cases. However, only a few reports have highlighted the effectiveness and safety of ECT for the treatment of primary psychotic disorders that emerge later in life, and the factors that contributing to long-term good response has not been revealed (1, 4, 5). Herein, we report a case of a patient with late-onset multidrug-resistant psychosis who achieved remission for over a year after a course of ECT and combined low-dose risperidone. Additionally, we discuss the potential of ECT for a good long-term prognosis of late paraphrenia and VLOSLP based on our experience and previous reports on this topic.

The Osaka University Clinical Research Review Committee approved this report, and the patient and her family provided consent for the publication of the report.

Case Presentation

The patient was a 74-year-old, ambidextrous, middle school graduate woman. She had a history of idiopathic subarachnoid haemorrhage (SAH) in the left frontal lobe, which occurred at the age of 57 years without subsequent complications, but no history of psychiatric disorders. The patient lived alone for over 20 years after her husband died. From the age of 70, the patient had fewer opportunities to visit her two daughters and her grandchildren due to the marriage and relocation of her daughters. At age 71, she underwent surgery for a severe lumbar fracture, which decreased her outings. At age 73, the patient complained of auditory hallucinations. She was diagnosed with late paraphrenia at a memory clinic and was placed on 1 mg/day of risperidone. Initially, the drug relieved her symptoms. However, she relapsed, and the hallucinations worsened after 1 month. She was then referred to our hospital and hospitalised.

On admission, the patient complained of auditory hallucinations, such as hearing threats, running commentary, and commanding voices. She did not exhibit any other hallucinations and apparent affective symptoms. Her formal thoughts were usually intact, and she recognised that her auditory hallucinations were strange and wanted treatment. Her neurological examination and blood test results were normal. Her Mini-Mental State Examination and Frontal Assessment Battery scores were 28 and 15, respectively. She did not show any apparent cognitive fluctuations. Other than strange behaviours associated with her psychosis, we did not observe any cognitive impairment in her daily behaviour in the ward. The Physical Self-Maintenance Scale score and Lawton Instrumental Activities of Daily Living score were 6/6 and 7/8, respectively. Her global Clinical Dementia Rating (CDR) score was 0.5 and CDR sum of box score was 0.5. Her electroencephalogram showed high-voltage slow waves in the left frontal area. Brain magnetic resonance imaging showed an old SAH lesion in the left frontal lobe and mild atrophy in the bilateral medial temporal lobes (Figure 1-A, B). [123I]-metaiodobenzylguanidine (MIBG) myocardial scintigraphy showed heart-to-

mediastinum ratios of 3.87 and 4.30 in the early and delayed phases, respectively (Figure 1-C). Brain single-photon emission computed tomography showed decreased perfusion in the old lesion and the bilateral parietal lobes (Figure 1-D). We considered that the contribution of the SAH to her psychosis was negligible because of the over 10-year gap between the occurrence of the SAH and the onset of her psychotic symptoms. Although the atrophy in the temporal lobes and hypoperfusion in the parietal lobes suggested neurodegenerative disease, we did not attribute her psychosis to Alzheimer's disease due to the lack of apparent memory impairment in her neuropsychological test results. The lack of core features of dementia with Lewy bodies and the normal accumulation on MIBG scintigraphy were unsupportive of Lewy body disease.

Therefore, we diagnosed the patient with schizophrenia spectrum disorder and initiated antipsychotic treatment. The patient was administered risperidone (up to 4 mg/day), quetiapine (up to 150 mg/day), aripiprazole (up to 30 mg/day), blonanserin (up to 16 mg/day), asenapine (up to 10 mg/day), and paliperidone (up to 6 mg/day). Sufficient doses of some of the drugs did not improve her symptoms, whereas the doses of some drugs could not be increased sufficiently due to side effects, such as muscle rigidity and dry mouth. Despite continuous antipsychotic treatment for over 3 months, her condition gradually deteriorated. Finally, the patient refused to eat and drink, affected by command hallucinations, which lead to the intravenous hydration. The patient displayed no catatonic symptoms. After assessing the risks and benefits, we initiated a course of ECT on the 92nd day, using Thymatron System IV with bifrontal electrode placement, a pulse width of 0.5 ms, and propofol as an anaesthetic agent. After three ECT sessions, her psychotic symptoms improved dramatically, and she began oral intake of food and fluids. Her apathy and social withdrawal improved as well. The patient did not show any side effects of ECT other than lower back pain, which was alleviated by increased usage of muscle relaxants. After ten ECT sessions, her psychosis disappeared, and she reported

her psychosis had been 'a strange experience'. Apart from tapering risperidone, we did not change any drug administered during the course of the ECT. The patient was discharged on the 163rd day with additional social support, such as visiting nurses and elderly day-care services, for better social communication. The patient lived by herself without any apparent psychotic symptoms for over a year on only 1 mg/day of risperidone. The clinical course of the hospitalisation is shown in Figure 2.

Discussion

In the present case, ECT dramatically improved late-onset psychosis after anti-psychotic drug therapy failed. It is noteworthy that remission was maintained for 1 year with only 1 mg/day of risperidone. Although this dose of risperidone was adequate for maintaining remission, the same dose did not alleviate the patient's active psychosis before ECT was initiated. Thus, ECT was essential for achieving remission. Our patient's auditory hallucinations, persecutory delusions, gender, social isolation, and lack of negative symptoms are compatible with late paraphrenia (1). On the other hand, the newer diagnostic term, VLOSLP, requires etiological differentiation from psychosis and brain abnormalities. Since our patient had an old SAH lesion in the frontal lobe, we did not describe her case as 'VLOSLP'. However, except for the imaging abnormalities, the clinical course and symptoms of the patient in the present case met the consensus criteria for VLOSLP (2). Therefore, we decided to discuss this case in the context of late-onset primary psychotic disorders, such as late paraphrenia and VLOSLP.

There are several reports of psychotic disorders in non-catatonic older patients treated with acute ECT (Table 1) (1,4,5). Kay and Roth reported that seven of 12 patients with late paraphrenia treated using ECT improved temporarily (1). Kumagaya reported that all seven patients with late-onset (41 - 60 years of age) or very-late-onset disorders (≥ 60 years of age) responded to ECT and achieved full remission (4). On the other hand, only

five of the 13 patients with early-onset (≤ 40 years of age) psychosis achieved full remission. Although there is little data regarding the clinical difference between groups divided according to onset age, patients with late-or very-late-onset psychosis may respond to ECT better than those with young- or average-onset psychosis.

The long-term prognosis of late-onset primary psychotic disorders following ECT has not yet been elucidated. In the study by Kay and Roth (1), five of seven patients who responded to ECT were readmitted within a year, and only one of the seven remained in good condition for a long time. The patient moved from isolated housing to her daughter's home, suggesting that environmental modification favoured the outcome. Kumagaya showed that a larger proportion of patients in partial remission transitioned to maintenance ECT than those in full remission (71% vs. 33%) (4). The good response of our patient to acute ECT, which may be linked to the late onset of psychosis, and the post-treatment social support provided may be factors associated with long-term remission after ECT for late paraphrenia and VLOSLP.

Figiel et al. reported that only one of six patients with late-onset psychosis treated with ECT responded to treatment (5). The other five patients showed structural changes in the brain, including lateral ventricular enlargement and/or large white matter hyperintensities. Structural brain changes were suggested to predict poor response to ECT. Although our patient had an old SAH lesion, she responded well to ECT. This may be explained by the lack of complications after the onset of SAH and the minimal effect of SAH on her psychosis.

In conclusion, ECT showed great efficacy for the treatment of late-onset multi-drug resistant psychosis in the present case. Although there is little evidence regarding the use of ECT for the treatment of late paraphrenia and VLOSLP, ECT may be a treatment

option for multidrug-resistant cases and social support may contribute to good outcome of ECT.

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Table 1. Comparison of previous reports on electroconvulsive therapy for the treatment of late-onset psychotic disorders

Author, year	Patients (n)	Age (mean, range)	Diagnoses	Responder (n)	Characteristics for good response	Adverse events	Limitations
Kay and Roth, 1961	12	NA (all patients were over 60 years old)	Late paraphrenia	7	Social isolation in clinical history	NA	No imaging and cognitive data; one patient showed catatonia
Figiel et al., 1992	6	74.8 (65-89)	Late-onset psychosis	1	Absence of structural brain changes	Five patients experienced interictal delirium	Most patients had moderate or severe ventricular enlargement
Kumagaya, 2019	7 (and 13 early-onset cases)	NA (all patients were over 60 years old)	Late-onset and very-late-onset schizophrenia or schizoaffective disorder	7	Shorter duration of psychotic illness; later onset of psychosis, later age at the time electroconvulsive therapy was administered	4 of 20 whole samples showed adverse effects. Two showed a post-ictal confusional state.	No imaging and cognitive data; several patients may have had catatonia
Present case	1	74	Late paraphrenia	1	NA	none	Old subarachnoid haemorrhage lesion

Figure legends

Figure 1. Clinical images

A:T1-weighted magnetic resonance imaging (MRI) shows mild atrophy in the bilateral medial temporal lobe. B:T2-weighted MRI shows the old lesion of subarachnoid hemorrhage in the left frontal lobe. C: Delayed image of [123I]-metaiodobenzylguanidine myocardial scintigraphy. Heart-to-mediastinum ratios were 3.87 and 4.30 in early and delayed phases, respectively. The washout ratio was 33.5%. D: N-isopropyl-p-[123I]iodoamphetamine single-photon emission computed tomography shows hypoperfusion in bilateral temporal, parietal lobes and the old lesion.

Figure 2. Clinical course during hospitalisation

RIS, risperidone; QUE, quetiapine; APZ, aripiprazole; BLN, blonanserin; ASP, asenapine; PAL, paliperidone; ECT, electroconvulsive therapy.