Electroconvulsive Therapy in a Patient With Chronic Catatonia

Clinical Outcomes and Cerebral ^{18}[F]Fludeoxyglucose Positron Emission Tomography Findings

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Abstract: Catatonia is a psychomotor syndrome that can be associated with both psychiatric diseases (mainly mood disorders, but also psychotic disorders) and medical conditions. Lorazepam (6-21 mg/day, occasionally up to 30 md/day) is the first choice treatment and electroconvulsive therapy (ECT) is the second line, regardless of the underlying clinical condition. There are some evidences also for effectiveness of other medications. Patients treated acutely usually show rapid and full therapeutic response but even longstanding catatonia can improve. However, some authors suggested that chronic catatonia in the context of schizophrenia is phenomenologically different and less responsive to lorazepam and ECT, especially if associated with echophenomena. We present here the case of a patient with longstanding catatonic schizophrenia treated with antipsychotics who significantly improved after ECT. Improvement regarded mainly catatonia, but also negative symptoms, cognition and psychosocial functioning. A slight amelioration in prefrontal metabolism (Brain^{18}[F]FDG PET) one month following the ECT course was also noted.

Key Words: electroconvulsive therapy, chronic catatonia, neurocognition, cerebral 18F-FDG PET/CT

(*J ECT* 2016;00: 00–00)

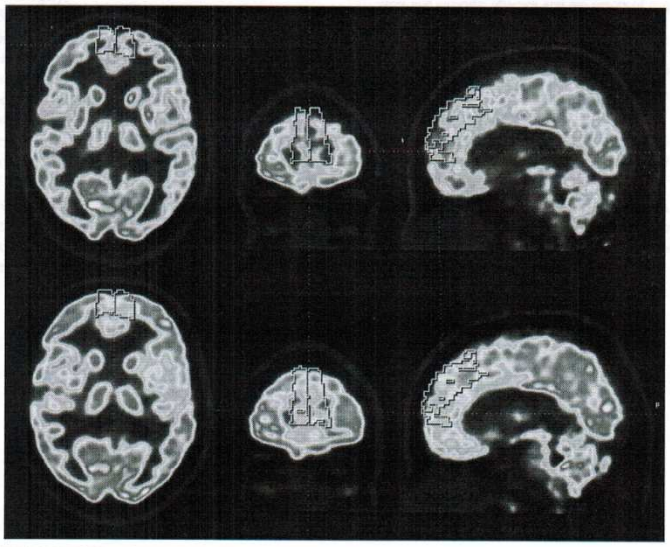

FIGURE 1. Cerebral ^{18}F-FDG PET/CT performed before (first raw) and 1 month after (second raw) the end of ECT showed qualitatively and semiquantitatively (SCENIUM included in the syngo via Siemens' health care software) a slight improvement in prefrontal cortex (white volumes of interest). From left to right: axial, coronal, and sagittal views.

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Received for publication January 21, 2016; accepted April 13, 2016.

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The authors have no conflicts of interest or financial disclosures to report.

Supplemental digital contents are available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's Web site (www.ectjournal.com).

DOI: 10.1097/YCT.0000000000000333

Electroconvulsive therapy (ECT) is known to be an effective and safe therapy for catatonia if benzodiazepine have failed, although some clinical features can predict a lower response.[1] The mechanisms underlying ECT are unknown. Few data are available on functional brain imaging in catatonic patient treated with ECT. As regard catatonia associated with schizophrenia, a single-photon emission-computed tomography study reported clinical response, but no changes in regional cerebral blood flow.[2] Another Cerebral ^{18}[F]fludeoxyglucose positron emission tomography (cerebral ^{18}F-FDG PET) study on a schizoaffective patient

showed increased metabolism more pronounced in left parietal-motor cortices.[3]

We present the case of a 48-year-old man with chronic catatonia associated with schizophrenia who showed a slight amelioration in prefrontal metabolism 1 month after the end of ECT and an improvement concerning catatonic symptoms, cognition, and social functioning.

Catatonic symptoms started insidiously at the age of 26 years as enduring psychomotor disturbances. Many years later, an acute catatonic episode (retarded subtype) occurred and relieved with lorazepam treatment (12 mg/d for 2 weeks). Nevertheless, some catatonic symptoms persisted and worsened over time. At the age of 47 years, we observed posturing, echoprassia, echolalia, automatic obedience, thermal hypoalgesia (see Video, Supplemental Digital Content 1, at http://links.lww.com/JECT/A46), staring, episodic hyperdiaphoresis, tachycardia, progressive weight loss.

Extensive clinical and biochemical examination including whole body computed tomography (CT) excluded underlying medical conditions.

Twelve bifrontotemporal ECT sessions were administered, with a frequency of three sessions per week.[4] Sertraline was added to the ongoing polypharmacy.

Rating scales (Bush-Francis Catatonia Rating Scale, Positive and Negative Symptoms Scale), neurocognitive tests (Matrics Consensus Cognitive Battery, Cognitive Estimation Test and Wisconsin Card Sorting Test) and a cerebral ^{18}F-FDG PET/CT[5] were performed before and 1 month after the end of ECT. We observed a decrease of both catatonic symptoms (Bush-Francis Catatonia Rating Scale reduction = 63%) and psychotic symptoms (reduction of Positive and Negative Symptoms Scale scores: total = 38%; 23%, 54%, 31% for positive, negative and general psychopathology, respectively). Cognitive tests showed an amelioration especially in attention/vigilance, cognitive estimation processes, abstract thinking, set shifting. Cerebral ^{18}F-FDG PET/CT showed diffused cerebral hypometabolism, especially in thalamus and temporal regions and slight improvement in prefrontal cortex (Fig. 1). These findings were highlighted both visually/qualitatively and semiquantitatively.

In the following 18 months, the patient remembered many episodes of his life, interacted appropriately in social contexts and was successfully engaged in a residential rehabilitation program.

This case report showed efficacy of ECT in a patient whose clinical features are usually associated with lower response (chronicity of catatonia, chronicity of psychosis, long-lasting antipsychotic exposure, echophenomena). Electroconvulsive therapy was also well tolerated because no cognitive side effects were observed.

Brain imaging findings are particularly interesting because we obtain improvement in specific catatonic signs (echophenomena, perseveration, posturing) and neurocognitive domains (executive functions, reasoning skills) for both of which prefrontal cortex plays a relevant role.

We present these images to add to the literature of brain areas and the effectiveness of ECT in chronic catatonia.

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