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CASE REPORT

Complex Long-Term Catatonia

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Catatonia comprises a neuropsychiatric syndrome characterized by altered psychomotor and behavioral symptoms, as well as autonomic dysfunction.[1,2] Catatonia is currently classified in the *DSM-5* as a possible specifier of a psychotic, affective, neurologic, or other medical condition or unknown cause (idiopathic).[3] Here, we present a case of an inpatient with catatonic schizophrenia with multiple systemic repercussions and complex etiology.

CASE REPORT

In February 2018, a 23-year-old male university student was admitted to the hospital to undergo electroconvulsive therapy (ECT). The patient presented with mutism, and the main sources for history taking were family members and his clinical reports. He had a 2-year progressive onset of diverse symptoms not consistently described until he experienced full-blown catatonia. The following remarks were included in his clinical report: "catatonic stupor, with psychomotor inhibition, selective mutism, negativism, active resistance to feeding with significant weight loss and insomnia . . . affective indifference and sitophobia." He had been fed with a permanent nasogastric tube for 8 months at the time of admission. During the previous admission, psychotropic medications with benzodiazepines, antidepressants, antipsychotics, and mood stabilizers were tried. The examinations at admission showed no important alterations. At admission to our unit, he looked emaciated and was uncooperative with avoidant contact, staying silent with negativism and *mitgehen* (an observation finding consisting of the examiner being able to move the patient's body with the slightest touch). He scored 12 + 3 on the Bush-Francis Catatonia Rating Scale.[4] The clinical examination showed a lesion in the corpus callosum splenium compatible with Marchiafava-Bignami syndrome (**Figure 1**) and malnutrition. No other relevant alterations were found on extensive blood, urine, and

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Figure 1.

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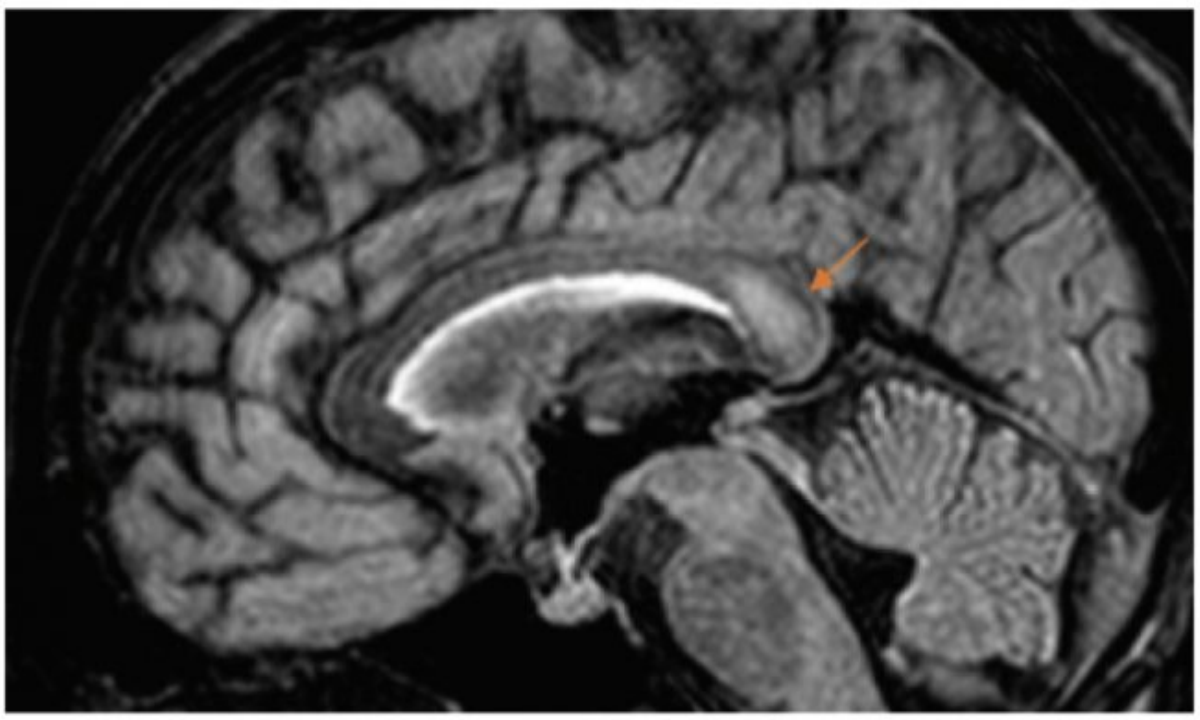
and Hypersignal in FLAIR in the Splenium of the Corpus Callosum (red arrow) With Mild Hyposignal on T1[a]



[a]The lesion measures approximately 15 mm in dimension in the anteroposterior direction, conditioning mild insufflation, with central isolation and sparing the peripheral fibers, aspects that are correlated with a transient lesion of the splenium of the corpus callosum (Marchiafava-Bignami).

Abbreviations: FLAIR = fluid-attenuated inversion recovery, MRI = magnetic resonance imaging.

Based on the history provided by the family, the medical team first considered an affective episode as the underlying disorder, and ECT (total of 18 sessions) and lorazepam titrated to 15 mg/d and venlafaxine titrated to 225 mg/d were started. The nasogastric tube feeding was optimized. Due to the sparse response and later clarification of symptomatology as having a clearer psychotic undertone (schizophrenia with catatonic features), olanzapine was initiated and titrated to 15 mg/d, with haloperidol also being tried at a later stage. In time, the patient began to communicate through gestures and written language, started feeding by his own hands, and became more socially interactive, with remission of most psychomotor symptoms.

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He needed a tracheostomy. Therefore, his medications were temporarily reduced and ECT was suspended as a precaution. After therapeutic revision, he was started on clozapine and haloperidol and was discharged in November 2018 with clozapine

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treatment and care. The findings of Marchiafava-Bignami syndrome were reversed on the discharge magnetic resonance imaging scan. He currently presents no active psychotic or affective symptoms, although some cognitive symptoms remain, and takes clozapine 200 mg/d and haloperidol 200 mg/month. With time, it was possible to remove the tracheostomy, and he now speaks with no restrictions. The patient is now well integrated socially and started a job after directed training.

DISCUSSION

Acute- and long-term catatonia share the initial treatment regimen of benzodiazepines and ECT, but chronic catatonia presents no clinical response and needs further treatment, with the management of nonpsychiatric causes prioritized.[5–7] Long-term catatonic presentation in psychotic illness is usually associated with a worse prognosis, incomplete recovery, and several years of neuropsychiatric symptoms,[2,6,8] but the treatment approach in this case was successful.

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Patient Consent: Consent was received from the patient to publish the case report, and information (including the dates) has been de-identified to protect anonymity.

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