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Catatonia and Positive Serum Antibodies against N-Type Calcium Channel

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Abstract

Catatonia occurs in a range of psychiatric, neurologic, medical, and toxic conditions. Several neurobiological abnormalities predispose individuals to catatonia. Here we present a case of a 57-year-old man with a major depressive disorder with psychotic features and catatonia, whose evaluation was notable for positive serum antibodies against N-type calcium channel, without evidence for malignancy.

Keywords: Catatonia; N-type voltage-gated calcium channel antibody

Introduction

Catatonia occurs in a range of psychiatric, neurologic, medical, and toxic conditions. Several neurobiological abnormalities predispose individuals to catatonia. Here we present a case of a 57-year-old man with a major depressive disorder with psychotic features and catatonia, whose evaluation was notable for positive serum antibodies against N-type calcium channel, without evidence for malignancy.

Case Report

A 57-year-old man with a history of post-traumatic stress disorder and major depressive disorder with psychotic features was admitted for depressed mood, paranoia and catatonia characterized by stereotypies, mutism, and involuntary resistance to passive movement (negativism). His past medical history included hypertension, hyperlipidemia, gastroesophageal reflux, and sleep apnea. The patient met Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5) criteria for major depressive disorder recurrent, severe with psychotic features, and began treatment with sertraline, risperidone, clonazepam, prazosin, and trazodone. These were titrated up to 200 mg, 6 mg, 5 mg, 8 mg, and 100 mg, respectively, without much clinical response. Because catatonia persisted, he was treated with high-dose of oral lorazepam up to 12 mg/day, which also proved ineffective. Subsequent extensive medical work-up, including lumbar puncture, cerebrospinal fluid analysis, and electroencephalogram, was largely unremarkable. The only positive findings were mildly elevated C-reactive protein at 2.0 mg/dL, mildly elevated erythrocyte sedimentation rate at 24 mm/hour, and positive serum antibodies to N-type calcium channel at 0.19 nmol/L. MRI of the brain with and without contrast showed diffuse nonspecific white matter signal abnormalities. Computerized tomography (CT) scans of the abdomen and pelvis with and without IV contrast revealed a 9 mm small adrenal adenoma, unchanged from prior CT scan 3 years ago. CT scan of the chest without IV contrast showed no lung masses. Given lack of response to psychopharmacological interventions, the patient began right unilateral electroconvulsive therapy (ECT), with the plan to pursue immune-modulatory therapy if he did not respond to ECT. All medications except lorazepam 1.5 mg/day were discontinued prior to ECT. The patient began to improve after ECT #7, after which he became more verbal and regained some of his normal mobility without stereotypies. By ECT #11, the patient was nearly back to his baseline with normal movement, speech, cognition (montreal cognitive assessment (MoCA) score 28/30), and euthymic mood (Hamilton Depression Rating Scale (HAM-D) score 4). The patient had 13 RUL ECT treatments and was maintained on a post-ECT psychopharmacological regimen of lithium 900mg per day (level 0.7 mEq/L) and nortriptyline 30 mg per day with a nortriptyline plasma level of 50 ng/ml.

Discussion

To our knowledge this is the first reported case of a patient with catatonia and elevated titers of N-type voltage-gated calcium channel antibody. There may be a genetic vulnerability for catatonia as evidenced by aggregation in families and linkage to chromosome [1,2]. GABA, dopamine, and glutamate are neurotransmitters of interest. NMDA antagonists have demonstrated some anti-catatonic effects, even in cases that have not responded to traditional treatment [3]. Voltage gated calcium channels mediate physiological processes in multiple organ systems, including the brain. Animal studies have localized clinical phenomena to calcium channel variants. For example, knockout of Cav2.1 (P/Q-type) channels give rise to ataxia and seizures [4]. L-type channels function in long-term potentiation, which has implications in animal models for depression. The Cav1.3 (L-type) subtypes have been used to explain the selective degeneration of dopaminergic neurons [4]. L-type calcium channel agonism is associated with a phenotype of dystonia in mice. Symptoms of the motor syndrome precipitated by calcium channel agonism were attenuated by calcium channel antagonism, suggesting that calcium channels are important mediators of dystonia [5].

This case describes a patient with a major depression with catatonic features, with positive serum antibodies to the N-type calcium channel, with a negative work up for malignancy. Given evidence that voltage gated calcium channels mediate movement disorders and specifically dystonia, as well as the positive serum antibodies to the N-type calcium channel in this patient, we hypothesize that calcium channelopathies may underlie particularly the motor dysfunctions seen in catatonia. Therefore, studying calcium channelopathies may help elucidate the biological mechanism of the various presentations of catatonia and possibly reveal novel pharmacological targets for catatonia.

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