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Periodic Catatonia after Thyroid Cancer

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Authors' contributions

This work was carried out in collaboration between all authors. Author DA wrote the case report and participated in every step of manuscript writing. Author PH designed the case report, managed part of literature searches and helped with revisions, author MH wrote parts of introduction and discussion section, helped with literature searches and help with manuscript editing. Author RGB designed the paper, wrote parts of introduction, case report and discussion, introduced the concept of periodic catatonia in this context and supervised the work to completion. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

We are presenting the case of a 52 year old female with three distinct episodes of clinical deterioration over a 20 year period after thyroid cancer treatment. The first decrease in functioning happened after the diagnosis of thyroid cancer, resulting in the patient not achieving her PhD thesis. The second deterioration happened ten years later when she presented with psychotic symptoms and the symptoms of anorexia. The last period of deterioration occurred one year before this hospitalization. During that time the patient worsened to the point where she became bedbound and dependent on a PEG tube for feeding. Once hospitalized, the patient had partial response to lorazepam (27 mg a day) and so dextroamphetamine was added with positive response. The addition of memantine helped with the residual symptoms. The PEG tube was finally able to be removed and the patient was discharged home in stable condition.

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1. INTRODUCTION

Thyroid conditions, including autoimmune, hypo, and hyperthyroidism can cause a myriad of psychiatric conditions, including catatonia [1-3]. The underpinning mechanisms of catatonia are unknown, but there are many theories that attempt to explain the development of catatonia in different medical conditions and in psychiatric conditions [4]. Several decades ago, a number of papers reported the concept of periodic catatonia [5,6] which is defined as a subtype of catatonia in which a person exhibits intermittent hyperkinetic and/or akinetic episodes that appear suddenly and fail to remit completely.

Lorazepam, corticosteroids, electroconvulsive therapy (ECT), and medical correction of the thyroid disorder are the most studied treatments for catatonia in the setting of thyroid dysfunction. In one case, surgical treatment of the thyroid disorder resolved the catatonia [7]. Both lorazepam alone and treatment of the thyroid disorder alone have been shown to improve catatonia [1,8]. Neuroleptics have been useful as adjunctive treatments in some studies, while they failed to relieve catatonia in other studies [2,3,7-9]. Interestingly, in one case report, the use of a neuroleptic resulted in catatonia in a patient with hyperthyroidism [10]. There is a theory that thyrotoxicities can cause sensitization to neuroleptics and induce a catatonic reaction [10].

Chronic illnesses that cause catatonia at one point are likely to cause catatonia again and notably, the response to an agent in the past does not necessarily predict the response in a future recurrence [3]. In this paper we present the case of a patient with a thyroidectomy that had three major deteriorative episodes, without a return to the baseline between episodes, leading the patient to be bedbound. Catatonia as result of medical conditions is often described in association with psychosis and other psychiatric conditions [11-13]. We propose that catatonia has played a role throughout this patient's illness and suggest that people with chronic thyroid conditions should be periodically evaluated for catatonia, especially when new psychiatric conditions emerge.

2. CASE REPORT

The patient is a 52 year old female with a prior diagnosis of schizophrenia and an eating disorder who was brought in by her family from an outside hospital for treatment of catatonia. According to the patient's family, the patient was a PhD student 20 years ago but quit school and moved home after having a total thyroidectomy and for stage II papillary thyroid cancer. Around ten years ago the patient started exhibiting symptoms of psychosis that eventually remitted but her functioning did not improve. Four months prior to admission the patient was sent to an outside hospital, as her symptoms had worsened significantly. She refused to eat, refused medications, and was selectively mute. A g-tube had to be placed in an outside hospital for feeding and the patient was transferred to UCI for further psychiatric care.

During the initial interview, the patient exhibited mutism, negativism, and stupor. The patient received an extensive medical workup to determine if there was an organic cause for her catatonia, as her diagnosis of schizophrenia was unclear. The following workup recommended by neurology and rheumatology revealed no significant findings: Electroencephalogram, magnetic resonance imaging (MRI) brain, MRI neck, Vitamin B12, thyroperoxidase antibodies, erythrocyte sedimentation rate, complete blood count, hepatitis panel, IgG subsets, immunoglobulins, anti-diuretic hormone, homocysteine, ammonia, c-peptide, insulin, West Nile antibody and Tetanus antibody, urinary porphobilinogen, and urine creatinine. Initially patient's liver function tests were elevated and they trended down, it was likely secondary to olanzapine that the patient was taking at the outside hospital. On admission, patient's thyroid-stimulating hormone (TSH) was elevated to 62 and her free-thyroxine (FT4) was 0.57. Her levothyroxine was managed by endocrinology and optimized. Patient had antinuclear antibody positive 1:320 but her subsets were negative (dsDNA, Smith, SSA, SSB, RNP) and inflammatory markers normal. Patient's C3 was initially low but returned to normal and C4 remained at 2. Patient's low complement level was attributed to malnutrition. Rheumatology concluded that patient did not have an underlying autoimmune disease. A pelvic ultrasound showed a complex mass, but an MRI pelvis showed intramural uterine fibroids and normal ovaries.

On her admission to psychiatry, the patient was started on lorazepam 2 mg QID which was titrated up to a dose of 7 mg QID. She was

started on amphetamine-dextroamphetamine 20 mg daily titrated up to a dose of 40 mg in the AM and 30 mg at 3 pm. After starting the stimulant, the patient complained of insomnia so she was started on trazodone 50 mg QHS titrated up to a dose of 150 mg QHS. Patient reported vivid nightmares with the trazodone and she was switched to mirtazapine 15 mg QHS which was titrated up to 45 mg QHS. Ramelteon 8 mg was added for adjunctive treatment of insomnia. Patient scored 14/30 on the Montreal Cognitive Assessment (MoCA) test. MoCA) is a 10 minute screening tool used to determine if a person has mild cognitive impairment by testing 8 different cognitive domains [14]. Memantine 10 mg was added for memory problems and it was titrated up to a dose of 20 mg daily. After being on memantine patient scored a 24 and then 30/30 on the MOCA test. Patient responded very well to the combination of the high doses of stimulants and benzodiazepines. She was not started on any antipsychotics, as she did not exhibit any symptoms of psychosis. She began interacting with staff, communicating her needs, and showing her emotions. She began to eat by mouth and tube feeds were tapered off. She gained around 6 lbs throughout her hospitalization and reported that she was content with her weight gain. The g-tube was removed during her hospitalization. Originally she was placed with a sitter due to being too frail and a fall risk. Physical therapy was done during her hospitalization and the sitter was able to be discontinued. Once patient appeared at baseline, her amphetamine-dextroamphetamine and lorazepam were decreased. She was discharged to a partial hospitalization with amphetamine-dextroamphetamine 20 mg QAM and 20 mg at 3pm, lorazepam 2 mg BID, memantine 20 mg daily, mirtazapine 45 mg QHS, and ramelteon 8 mg QHS.

3. DISCUSSION

Benzodiazepines are the first line of catatonia treatment [4]. The use of electroconvulsive treatment (ECT) as the second line of treatment is often difficult because of availability (due to problems with insurance coverage) or inability to obtain legal consent from the patient for the procedure due to catatonia. Therefore, often timely medication combinations are used to target presenting symptoms.

In this case, the patient tolerated lorazepam well, however she exhibited limited improvement and she started to develop mild sedation at the dose of 7 mg every six hours. A decrease in dose led to losing the noted improvement- increase in verbal output. After the first dose of dextroamphetamine, she reported an increase in anxiety. As we increased the dose of the stimulant, the anxiety symptoms did not increase and the patient began wanting to participate in the swallow test and start eating again after four months of PEG tube feeding.

As she started interacting more with staff, she started reporting memory problems and a Montreal Cognitive Assessment (MoCA) was done in which she scored a 14/30. There are reports of catatonia that state that the condition can cause cognitive impairments [15]. Also there are several reports in the literature that report that memantine is helpful for the treatment of catatonia in patients with limited response to benzodiazepines [16,17]. After starting memantine, the patient reported no change in her symptoms, but MoCA scores improved gradually to 24 and then to 30 over a couple of weeks.

Interestingly, the patient remembered many events while she was catatonic and in bed. She described memories in a relatively precise manner, however there were no emotional values attached to them. She reports that she was somewhat puzzled during the months of catatonia as she would not interact with people. She did not ask herself why she was in bed and why people were taking care of all her needs.

After two weeks of hospitalization she started eating and started working with physical therapists to walk again. Around that time she also started to have a fuller range of affects. Anxiety symptoms continued to be present but mirtazapine kept them relatively well controlled.

Treatment of catatonia as result of thyroid conditions is not well understood. A report of a patient with grave disease with hyperthyroidism developed subsequent episodes of catatonia. The first time around, the patient responded well to a combination of antithyroid and anxiolytic medications, but the second episode responded only to ECT [3]. This suggests the complexity of catatonia, as the treatment can vary from episode to episode, even if the cause is thought to be similar.

4. CONCLUSION

We propose that patients with thyroid conditions that develop new psychiatric manifestations should be evaluated for catatonia and treated

accordingly. It is difficult to say now if in this case the patient had psychosis and catatonia ten years earlier, however the patient regained the function she had 10 years earlier, according to the family.

We are also suggesting a revision of the concept of periodic catatonia in the context of chronic or recurrent conditions.

ETHICAL APPROVAL

It is not applicable.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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