Fighting the fire: anti-inflammatory effects of aripiprazole as mechanism for improvement in medical catatonia?

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Introduction

Disorders of central nervous system inflammation have a strong association with catatonia.. We present a case of a patient with multiple sclerosis who presented with new onset psychosis and catatonia secondary to an MS exacerbation. Her psychiatric symptoms quickly improved after addition of aripiprazole to her benzodiazepine regimen.

Case Presentation

Ms. M was a 49-year-old female. Her medical history was significant for MS, multinodular goiter, and marijuana use. She presented due to mania and psychosis with sudden onset. Brain imaging demonstrated lesions consistent with demyelination.

On psychiatric evaluation, she was diagnosed with severe major depressive disorder with psychotic features and catatonia. Catatonia was felt to most likely be secondary to her MS exacerbation. The patient was started on lorazepam, 2 mg every 4 hours. After completion of steroids , she was transferred to an inpatient psychiatric facility. She was re-admitted to a neurologic service after sustaining a fall.

Her presentation was noticeable for psychotic symptoms, specifically persecutory and nihilistic delusions. Quetiapine was briefly introduced but discontinued due to worsening of catatonic symptoms. We decided on a trial of aripiprazole 2.5 mg nightly. The next day, the patient showed a marked improvement in her spontaneity, and was less focused on her delusions. The patient was then transferred to an inpatient psychiatric facility, where she began electroconvulsive treatments. Her aripiprazole was increased to target residual psychotic symptoms. She was discharged on 15 mg daily.

Brain MRI

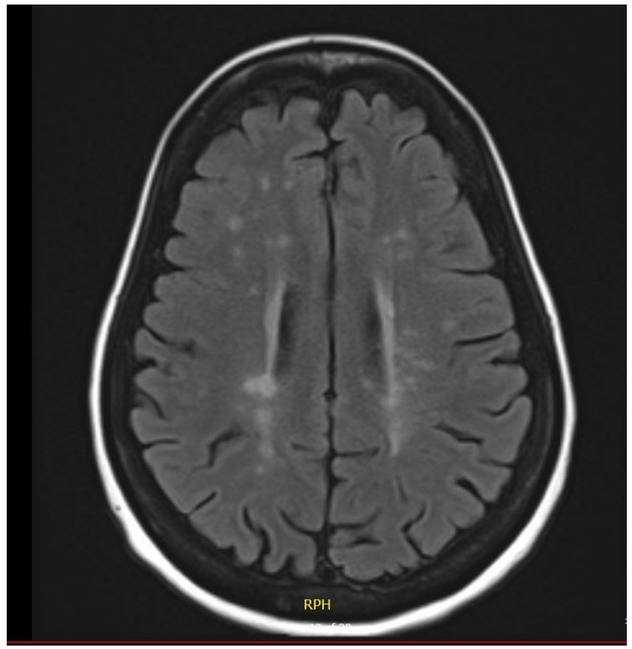

Figure 1: Axial flair imaging of patient showing new demyelinating lesions

References

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Discussion

The pathophysiology of catatonia is still not completely understood. However, there are increased theories that hypo-dopaminergia in the striatum is instrumental. Additionally, there are notable theories regarding catatonia and inflammation, including one systemic review that found increased acute phase reactants, such as activation of macrophages and monocytes. As these cells are activated, there is release of pro-inflammatory cytokines including interleukin- 1, Interleukin-6, and TNF-alpha. Interestingly, the utilization of lorazepam in catatonia could reduce cytokine expression by stimulating the GABA-A receptor on leukocytes.

However, Ms. M did not respond to only lorazepam. Our utilization of aripiprazole was both to target the imbalance of dopamine (Ms. M was both having paranoia and catatonia), but also to approach the hypothesized inflammation. There is reasonable evidence that aripiprazole (in addition to other antipsychotics) may reduce expression of inflammatory cytokines. It was due to these mechanisms of action that we felt aripiprazole an appropriate choice for concurrent delusions and catatonic symptoms.

This case adds to the growing body of literature discussing catatonia in the neurologically ill population. It is becoming clear that there is a growing association between encephalitis, multiple sclerosis, epilepsy, and overall neuronal inflammation and catatonic symptoms. Treatments need to act not just as means of re-regulating dopamine but also reducing neuronal inflammation.