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Management of antipsychotic-induced tardive dyskinesia with calcium channel blockers and botulinum toxin: a case report

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Introduction: Tardive dyskinesia (TD) is an iatrogenic disorder characterized by a range of movement abnormalities, described as irregular, stereotyped, and choreiform caused by exposure to antipsychotics. This condition can lead to significant or disabling symptoms that affect quality of life. However, the exact mechanism underlying TD remains unclear. Pharmacotherapy of TD includes cholinergic drugs, benzodiazepines, antioxidants, amantadine, propanolol, botulinum toxin, whereas the non-pharmacotherapy approach includes modified electroconvulsive therapy and deep brain stimulation. We successfully treated a chronic schizophrenia patient with comorbid TD using Aripiprazole and botulinum toxin after trying calcium channel blockers in association with Aripiprazole and vitamin E.
Objectives: To determine the effects of calcium channel blocker drugs (Amlodipine) for treatment of neuroleptic-induced tardive dyskinesia in people with schizophrenia, and the efficiency of botulinum toxin in treating induced tardive dyskinesia.
Methods: Through a case report and a literature review, our work aims to study antipsychotics induced tardive dyskinesia and its pharmacotherapy especially the use of calcium channel blocker and botulinum toxin in association with Aripiprazole.
Results: A 22 years male, diagnosed with schizophrenia since the age of 16 years, with one hospitalization. The evolution of his disease was marked by the development of a tardive dyskinesia, cervical and brachial movements disorders with rapid worsening in few months, the patient was treated with olanzapine oral administration. During his follows-up, his tardive dyskinesia disn't resolve despite switching the incriminated antipsychotic to Aripiprazole , lowering the dose since he was on complete remission. we added benzodiazepines and vitamin E to his treatment , before trying the calcium channel blocker for two months, with no improvement , on the contrary a worsening was noted using the Unified Dyskinesia Rating Scale . The aggravation of the movements was a reason for the patient attempted suicide , after this late incident we chose to try the botulinum toxin injections. His tardive dyskinesia was spectacularly improved within the fisrt injections.
Conclusions: Antipsychotic drugs are known to cause a variety of adverse effects, including tardive dyskinesia. Hence the importance of knowing the pharmacotherapy and non-pharmaco- therapy to manage this effect, through this case report where it tardive dyskinesia got treated after using botilinum toxin injections with a spectacular improvement in its Unified Dyskinesia Rating Scale. In our case , we had no response after adding calcium channel blocker .The effects of calcium channel blockers for antipsychotic-induced tardive dyskinesia are unknown, and it use is still limited.

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Treatment Strategies in Managing Negative Symptoms in Schizophrenia

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Introduction: Negative symptoms pose a significant challenge for the treatment and management of schizophrenia. They refer to the loss or diminishment of normal emotional and behavioural functions, which profoundly impact one's quality of life and socio-occupational outcomes. They are often persistent and difficult to treat.
Objectives: To explore and assess different treatment strategies for addressing negative symptoms of schizophrenia, including pharmacological, psychosocial, and non-invasive neurostimulation interventions. The goal is to provide an overview of current evidence and recommendations for enhancing the quality of life and functional outcomes in individuals with schizophrenia.
Methods: We conducted a review of the extant literature to determine treatment strategies for negative symptoms in schizophrenia. We incorporated findings from randomised controlled studies, meta-analyses and systematic reviews.
Results: We have identified several treatment strategies for negative symptoms in schizophrenia. The literature indicates that second generation antipsychotics such as Cariprazine and Amisulpride are associated with better functional outcomes with lower cognitive impairment. Adding on an anti-depressant, particularly to first-generation antipsychotics, has demonstrated positive effects. Psychosocial interventions including Cognitive Remediation (CR), Social Skills Training (SST) and exercise programs also alleviate negative symptoms. Additionally, non-invasive neurostimulation intervention such as rTMS applied to the left dorsolateral prefrontal cortex (DLPFC) has shown encouraging results in reducing negative symptoms.
Conclusions: The findings highlight the important of comprehensive and holistic treatment approach integrating both pharmacotherapy and non-pharmacotherapy strategies to address the heterogeneity of negative symptoms. There is a need for further research into personalised treatments that address individual symptom profiles.

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