Conclusions: The choice of molecules must take into account the associated somatic symptoms for a better tolerance. In the absence of a biological or iconographic examination with good sensitivity and specificity, the therapeutic test remains the only way to decide.

Disclosure of Interest: None Declared

EPV0382

Esketamine for resistant depression in older people with cognitive impairment: a case report

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Introduction: Depression represents a significant challenge in terms of disability among the elderly population and its responsiveness to conventional treatment approaches tends to diminish in this population group. Esketamine has shown both effectiveness and safety in addressing treatment-resistant depression in older patients.
Objectives: Currently, there is a lack of available literature regarding the use of esketamine in the treatment of patients experiencing both cognitive decline and treatment-resistant depression (TRD). We administered esketamine to a 79-year- old patient to evaluate the effectiveness and tolerance of the medication.
Methods: We recruited a 79 year old female referred to the outpatient clinics of Pavia suffering from TRD with current Severe Depressive Episode (scoring 42 on the MADRS) with cognitive impairment (MMSE 16/30). The patient was on a fourth-line treatment. First-line treatment was started with paroxetine 40 mg from september 2021 to May 2022, switched to sertraline 50 mg. Second-line treatment with quetiapine 150 mg from June 2022 to December 2022 failed, despite optimal compliance for both lines of treatment. Then third line treatment with fluoxetine 20 mg, olanzapine 10 mg was initiated from December 2022 to May 2023. Study duration was 12 weeks. Anamnestic data and psychometric (MADRS, HAMD-21, HAM-A) and cognitive (MMSE and MoCA TEST) assessment were collected from medical records at baseline (T0), one month (T1) and three month (T2) follow-ups.
Results: MADRS, HAM-A and HAM-D values decreased significantly at T1 and T2 follow-ups. T0: MADRS 42, HAM-D 33, HAM-A 54; T1: MADRS 18, HAM-D 12, HAM-A 15; T2: MADRS 4, HAM-D 5, HAM-A 10. We also observed an improvement in cognitive test: T0: MMSE 16/30, MoCA test 4/30; T1: MMSE 18/30, MoCA test 6/30; T2: MMSE 20/30, MoCA test 8/30. The patient reported one episode of hypertension treated with clonidine after two moth of treatment, and mild prolonged motor slowing lasting about two hours after esketamine in the first month.
Conclusions: This case documented a successful treatment using intranasal esketamine in combination with an SSRI (Fluoxetine) for an older individual with cognitive impairment and a persistent anxiety-depressive syndrome. This approach was employed as a therapeutic intervention after multiple unsuccessful attempts with other antidepressant medications. Our findings confirmed the safety and tolerability of esketamine in an elderly female with cognitive impairment. Although a minor improvement in cognitive abilities has been noted, secondary dysfunction attributable to vascular-based cognitive decline remained. In terms of cognitive tolerance, derivatives of ketamine could potentially serve as an alternative to electroconvulsive therapy in cases of treatment-resistant depression, potentially improving short-term cognitive outcomes.

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EPV0389

The use of memantine for depressive symptomatology

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Introduction: Depression is one of the most prevalent and incapacitating disease in current times and depressive symptoms have important global functioning implications.
The serotonergic and glutamatergic systems are involved in the pathophysiology and treatment of depression. Ketamine is an N-methyl-d-aspartic acid (NMDA) receptor antagonist that has demonstrated an important role on depressive symptoms, but its use is restricted due to its dissociative effects and other possible adverse effects.
Memantine is a noncompetitive antagonist of the NMDA receptor that modulates glutamate transmission. Memantine is used for the treatment of moderate to severe Alzheimer's disease.
Objectives: In this review, we aim to investigate, organize and synthetize the current data about the use of memantine for depressive symptoms.
Methods: Our literature research focused on some of the most significative articles published in the last decade, including meta-analysis and systematic reviews.
Results: Most of the relevant literature suggests that memantine may effectively reduce depressive symptoms in patients with mood disorders.
The literature also supports that memantine's glutamatergic mechanism of action could reduce apathy and treat depression comorbid with alcohol abuse.
Memantine affects brain-derived neurotrophic factor(BDNF) production suggesting that glutamate assumes an essential role in the pathology and etiology of depression. Also, the relationship between depression and the NMDA receptor is further supported by the fact that people with major depressive disorder demonstrate higher glutamate levels in the brain and blood.
Moreover, current studies demonstrate that treatment with memantine as adjunct to selective serotonin reuptake inhibitors (namely sertraline) manifested a favourable safety and efficacy profile in patients with major depressive disorder.
Conclusions: Memantine may have a wide therapeutic use beyond its utility in neurodegenerative diseases.
More studies should be performed, especially larger controlled studies of longer duration focusing on long-term safety and efficacy.

Disclosure of Interest: None Declared

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