Results: We present the case of a 64 years old woman, divorced and retired, who lives with her son since the aggravation of the depressive symptomatology, with no medical nor surgical background and no history in Mental Health before her first psychiatric internment in 2020. Between February 2020 and June 2023, 5 different treatments options with supervise intake were tried, including increment of the dose, antidepressant rotation, the combination of Desvenlafaxine + Mirtazapine and adding Topiramate and Lithium, with no improvement. Among this years, 3 psychiatric internments were needed because of the depressive symptoms and 1 more hospitalization in Internal Medicine was required because of the patients severe, malnutritional state. In June 2023 and after two complete analysis, a MR and a score of 28 points in the Hamilton Depression Rating Scale treatment with Esketamine was started with no incidences. She described one dissociative episode during which she assures "she was surrounded by soft, rubbery, yellow bubbles". After 4 months of treatment the patient has recovered her previous functional rate and has an 8 points score in the HDRS.
Conclusions: In conclusion, we can affirm that Esketamine is an effective and secure option for Resistant Depresion Dissorder. Nevertheless, Before considering a Depressive Episode as "resistant to treatment", treatment adherence and other medical, surgical and psychiatric comorbidities must be studied.

Disclosure of Interest: None Declared

EPV0377

Tardive Dysphoria: can antidepressants cause depression?

C. P. Desport*, D. O. Martins, J. R. Freitas, M. F. Santos and L. C. de Castro

Psychiatry, Hospital de Magalhães Lemos, Centro Hospitalar e Universitário do Porto, Porto, Portugal
*Corresponding author.
doi: 10.1192/j.eurpsy.2024.1101

Introduction: tardive dysphoria is a relatively new term used to describe the phenomenon of clinical worsening of depression after long-term antidepressant use. Most of the theories proposed to explain this talk about antidepressants tachyphylaxis that implies the loss of efficacy with its prolonged use, or even a pro-depressant effect of antidepressants when used for long periods of time.
Objectives: to explore the concept of tardive dysphoria, potential causes and clinical implications, by making a literature review on the topic. Moreover we pretend to understand the challenges in its diagnosis and treatment.
Methods: bibliographical search in PubMed database, using the key-words "long-term antidepressant", "tardive dysphoria" and "antidepressant tachyphylaxis", limited to works published in the last twenty years.
Results: from our search resulted 53 articles, 26 were chosen for further analysis.
Conclusions: the concept of tardive dysphoria is controversial, namely doubt persists if it constitutes a clinical entity by itself caused by long-term antidepressant use or if it simply relates to cases of treatment-resistant depression. We conclude that it is necessary further investigation in this area given the significant implications on clinical practice specifically in the psychopharmacological treatment with antidepressants, which is very common in psychiatric and general practices, with antidepressants being used to treat many mental health conditions.

Disclosure of Interest: None Declared

EPV0380

Depression in the elderly and dementia with Lewy bodies: A case report of a challenging diagnosis

E. Smaoui[1]*, D. Mnif[2], N. Reguaieg[2], F. Guermazi[2], S. Sakka[1], I. Baati[2] and J. Masmoudi[2]

[1]Neurology department, CHU Habib Bourguiba and [2]Psychiatry department, CHU Hédi Chaker, Sfax, Tunisia
*Corresponding author.
doi: 10.1192/j.eurpsy.2024.1102

Introduction: Depression and dementia with Lewy bodies (DLB) are two fairly common pathologies in the elderly which can have similar presentations or be associated and therefore pose a diagnostic challenge.
Objectives: We propose to illustrate, through our case, the diagnostic and therapeutic challenge of these two pathologies.
Methods: We present the case of Ms. S. BA aged 67, without organic or psychiatric history, admitted to the psychiatry department for massive anxiety and insomnia. The troubles date back to nineteen months when the patient isolated herself, remained bedridden, lost her appetite and no longer slept. The evolution quickly led to the appearance of an excessive agitation. The patient became distracted, talking and laughing to herself, and ran away from the house. She consulted several free-lance psychiatrists and received several antipsychotic medications without improvement. The admission interview revealed a very anxious patient with a difficult contact. Her speech was centred on well-detailed visual hallucinations with themes of death. The neurological examination was difficult at first. She was started on haloperidol and clonazepam. After 2 days, neurological examination showed a parkinsonian syndrome and a temporal disorientation. Other cognitive functions were difficult to assess. The two diagnoses evoked were DLB and a characterized depressive episode with psychotic features. Standard workup showed mild anaemia and thrombocytopenia. Brain MRI and electroencephalogram and immune tests were normal. However, PET imaging was not available in our hospital. Haloperidol was immediately stopped and the patient was treated with an anticholinergic corrector in combination with quetiapine at 200 mg. The evolution was characterized by a significant reduction in anxiety and visual hallucinations with a marked improvement of the parkinsonian syndrome. Depressive symptoms took the forefront of the clinical presentation; hence we associated sertraline with quetiapine. The subsequent evolution showed a clear improvement in the depressive symptoms with total resolution of the parkinsonian symptoms and a normal cognitive evaluation.
Results: In our case, the clinical evolution constituted a key element in the diagnostic orientation. So far, it is unlikely that our patient has DLB and the diagnosis retained was a characterized depressive episode with psychotic and melancholic features. Depression in the elderly can have atypical presentations, and raise the possibly of other differential diagnoses. Diagnostic uncertainty should not delay the implementation of treatment.

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

F. Mazzoni*, V. Arienti, S. C. Civardi, G. Carnevale Miacca, P. Leali, F. Santilli, A. Guffanti, N. Brondino and M. Olivola

Department of Brain and Behavioral Sciences, University of Pavia, Pavia, Italy
*Corresponding author.
doi: 10.1192/j.eurpsy.2024.1103

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.

Disclosure of Interest: None Declared

EPV0389

The use of memantine for depressive symptomatology

J. T. Coelho*, S. Timóteo and A. S. Machado

Department of Psychiatry and Mental Health, University Hospital Center of São João, Porto, Portugal
*Corresponding author.
doi: 10.1192/j.eurpsy.2024.1104

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