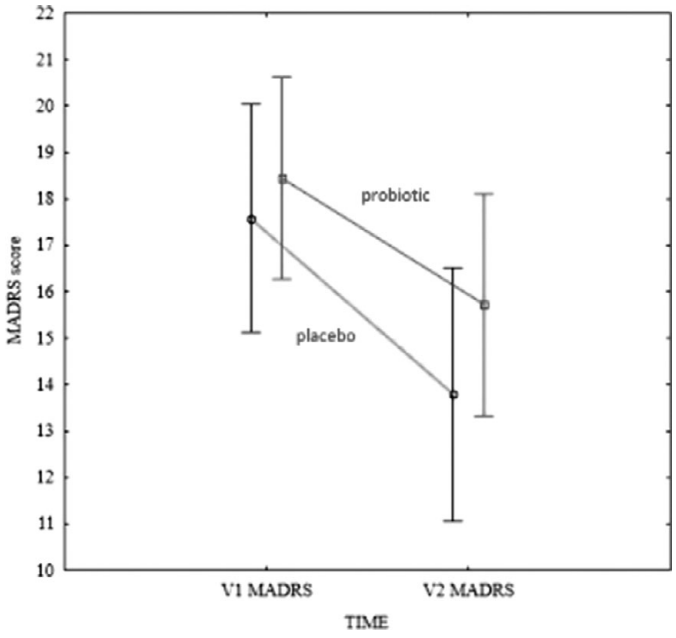
Image:



Conclusions: Whilst probiotics may benefit some individuals who do not fully respond to antidepressant medications, our study did not show the superiority of probiotics over placebo in managing depressive and anxiety symptoms. However, the target clinical sample, as well as the intervention period and dosage of preparation for this intervention is not fully recognized. Moreover, larger clinical sample may be needed to detect differences between placebo and probiotics.

Disclosure of Interest: None Declared

EPV0449

A Review of the Metabolism and Relevance to Form and Formulation of Ketamine

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Introduction: Ketamine is a phenylcyclohexylamine derivative comprising a racemic mixture of S- and R-ketamine that possesses anesthetic, analgesic, anti-inflammatory, and antidepressant activity. Oral (including extended release [PO]), intravenous (IV) sublingual (SL), transmucosal (TM), intranasal (IN), intramuscular (IM), rectal (PR), and subcutaneous (SC) formulations have been developed since its commercialization in 1970.
Objectives: To review and understand the impact of different forms and formulations on the pharmacokinetics of ketamine.
Methods: The extant literature on ketamine metabolism and formulations was reviewed and discussed.
Results: IV (racemic) ketamine (KET) has been shown to improve depressed mood within 4 hours with maximal effect at 24 hours. KET is a chiral molecule with two optimal isomers, R- and S-KET. KET is stereoselectively metabolized by CYP2B6 and CYP3A4 initially via nitrogen demethylation to active metabolite, norketamine (NK); there is no interconversion between R- and S-KET. NK is further metabolized to hydroxynorketamine (HNK) by CYP3A4 and CYP3A5; and dehydronorketamine (DHNK) by CYP2B6. Additional metabolic pathways exist including a direct enantioselective hydroxylation of KET to 6-hydroxyketamine (HK). Bioavailablity is greatest (100%) with the IV racemic KET formulation, but as low as 8% for oral S-KET due to extensive first-pass metabolism; the KET: NK ratio is a measure of first pass metabolism. NK plasma levels are higher with oral S-KET than KET as a result of local intestinal metabolism effects. Additionally, greater plasma concentrations are noted with IV bolus doses of S-KET vs. racemic KET or R-KET. S-KET possesses a longer elimination half-life than racemic KET due to inhibition by R-KET. KET is primarily renally eliminated and twice as fast in children vs. adults.
Conclusions: Complex interactions are reported between ketamine form (racemic/enantiomer), formulation, dose, and route of administration that impact on clinical variables and thus, outcome.

Disclosure of Interest: None Declared

EPV0450

Catatonia in depressive disorder, more usual than it is supposed to be

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Introduction: Catatonia is a psychomotor syndrome characterized by various motor, affective and behavioral symptoms. It can occur as a cause of various underlying organic and psychiatric disorders. In Psychiatric nosology is used to specify a subtype of the disorder underlying.Unlike what was assumed in the past, today it is accepted that catatonia is more frequent in affective disorders than in schizophrenia. But despite this, diagnosis and treatment are still late in affective cases on many occasions.
Objectives: -A case of catatonia is presented to review the diagnostic difficulties that can sometimes entail.-Review treatment algorithm.
Methods: We present the case of a 62-year-old woman, initially diagnosed of major depressive symptoms with psychotic symptoms, showing no response to different treatments, evolving to catatonia, which is diagnosed after screening for neurological and medical diseases.
Results: The patient had an adequate evolution after the withdrawal of antipsychotics and the application of ECT (Electroconvulsive therapy).
Conclusions: - It is important to carry out an adequate screening, because many times the symptoms are caused by medical or neurological diseases.
-Catatonia has a good prognosis with an early treatment, but it may increase the risk of mortality after 5 days from the onset of symptoms.
-It is important to avoid the use of antipsychotics or other dopamine blockers. The use of benzodiazepines and ECT is indicated.

Disclosure of Interest: None Declared