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different pharmacokinetics minimize withdrawal symptoms, drug interactions, and fluctuations in plasma concentration, enhancing tolerability. No second-generation LAIA was found to be clearly superior in terms of efficacy. Various guidelines recommend offering this treatment option early, favoring an informed and collaborative decision-making process. However, despite documented benefits in robust studies, they do not consider LAIAs as a first-line treatment.

Conclusions: Discussion/Conclusions: Significant variations in the proportion of patients on LAIAs across countries suggest that factors other than efficacy may influence its use. Greater understanding of these factors could help identify potential barriers to optimal implementation. New evidence may prompt a review of the guidelines.

Disclosure of Interest: None Declared

EPV1593

The Effects of St. John's Wort and its Interactions with SSRI's

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Introduction: Hypericum perforatum, commonly known as St. John's Wort, is a widely used herbal remedy for mild to moderate depression. It significantly affects drug metabolism by inducing the Cytochrome P450 enzyme system, particularly the CYP3A4 enzyme. This interaction can alter the metabolism of various medications, including oral contraceptives, cancer drugs, HIV antiretrovirals, and antidepressants. St. John's Wort increases levels of serotonin, dopamine, and norepinephrine via reuptake inhibition, similar to the action of selective serotonin reuptake inhibitors (SSRIs). However, combining St. John's Wort with SSRIs can dangerously elevate serotonin levels, potentially leading to serotonin syndrome, a serious and potentially life-threatening condition. This review explores the interactions between St. John's Wort and SSRIs, focusing on metabolic effects and the risk of serotonin syndrome.

Objectives: To determine the impact St. John's Wort has on the metabolism of anti-depressants and to discover the differences in severity of serotonin syndrome between various SSRI's.

Methods: A comprehensive literature review was conducted using PubMed, Google Scholar, and Medline. The review focused on documented drug-drug interactions between St. John's Wort and SSRIs, particularly their effects on the CYP enzyme system and the incidence of serotonin syndrome in patients taking both therapies. **Results:** The review identified that St. John's Wort affects the metabolism of several antidepressants, primarily through the CYP3A4, CYP2C9, and CYP2C19 enzymes. SSRIs such as citalogram, escitalopram, and sertraline, metabolized by CYP2C19, are more likely to interact with St. John's Wort than those metabolized by CYP2D6, such as paroxetine, fluoxetine, and fluvoxamine. The most significant adverse effect observed was serotonin syndrome, with case studies highlighting sertraline and paroxetine as the most commonly involved SSRIs. Dosages of SSRIs ranged from 20 mg to 75 mg, with St. John's Wort dosages typically between 600 mg to 900 mg per day. All reported cases of serotonin syndrome involved both sertraline and paroxetine, suggesting that these SSRIs may have a higher risk when combined with St. John's Wort, though a larger sample size is needed for statistical validation.

Conclusions: The literature underscores the critical need to screen for patient's who may have added St.John's Wort into their treatment regimen, especially when taking SSRIs. St. John's Wort can significantly alter the metabolism of SSRIs and increase the risk of serotonin syndrome. While interactions with sertraline and paroxetine are well-documented, further research is necessary to determine the risk profile of other SSRIs in combination with St. John's Wort.

Disclosure of Interest: None Declared

EPV1594

Xerostomia Induced by Psychiatric Medications: Prevalence, Impact, and Management

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Introduction: Xerostomia, or dry mouth caused by reduced salivary flow, is a frequently reported adverse effect of various psychiatric medications, particularly tricyclic antidepressants (TCAs), antipsychotics, SSRIs, SNRIs, and anticholinergics. This condition can lead to severe oral health problems causing many patients to discontinue their medication. Anticholinergics and psychotropic medications cause xerostomia by blocking acetylcholine from binding to muscarinic receptors in the salivary glands. Determining rates of xerostomia among psychotropic medications could be useful for those who are at higher risk of xerostomia.

Objectives: To investigate the prevalence and clinical impact of xerostomia caused by psychiatric medications and to identify effective management strategies.

Methods: A narrative literature review of clinical trials, observational studies, and case reports was performed to gather data on the prevalence, severity, and management of xerostomia in patients on psychiatric medications. The review included TCAs (amitriptyline, nortriptyline), antipsychotics (clozapine, olanzapine, chlorpromazine), SSRIs (paroxetine, fluoxetine, sertraline, citalopram), SNRIs (venlafaxine, duloxetine), and anticholinergics (benztropine, trihexyphenidyl). Patient-reported outcomes and interventions were analyzed.

Results: Table 1: Prevalence of Xerostomia by Medication

Medication	Prevalence (%)	Severity (Mild/ Moderate/ Severe)	Management Strategies
Amitriptyline	30-50	Moderate to Severe	Saliva Substitutes, Pilocarpine
Clozapine	10-30	Mild to Moderate	Dose Reduction, Hydration
Paroxetine	20-40	Mild to Moderate	Sugar-free Chewing Gum, Hydration
Anticholinergics*	20-65	Moderate to Severe	Dose Adjustment, Saliva Substitutes

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*Anticholinergics include benztropine and trihexyphenidyl, commonly used to manage extrapyramidal symptoms.

The prevalence of xerostomia was highest with amitriptyline (30-50%), followed by paroxetine (20-40%) and clozapine (10-30%). Anticholinergics contributed to xerostomia in 20-65% of cases. Amitriptyline and anticholinergics often caused moderate to severe cases of xerostomia. Management strategies included the use of saliva substitutes and pilocarpine for TCAs and anticholinergics, dose reduction and increased hydration for antipsychotics, and sugar-free chewing gum for SSRIs. Other medications not listed that are notable for significantly inducing xerostomia among their medication class include citalopram for SSRIs, venlafaxine for SNRIs, and chlorpromazine for antipsychotics.

Conclusions: TCAs and anticholinergics pose the highest risk for the side effect of xerostomia among psychiatric medications. Effective management requires a multifaceted approach, including pharmacologic and non-pharmacologic interventions. Future research should aim to explore alternative medications with lower xerostomia risk.

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EPV1595

A meta-analytic approach on Vortioxetine and acute depression

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Introduction: Major Depressive Disorder (MDD) is a chronic, recurrent illness characterized by various combinations of signs and symptoms, severity levels, and loss of functions. Among the available pharmacological treatments for MDD, Vortioxetine, a serotonin transporter inhibitor (SERT), has been widely used for its multimodal action on serotonin neurotransmission, which produces important changes also on glutamate, GABA, norepinephrine, acetylcholine, and dopamine.

Objectives: The aim of this systematic review and meta-analysis is to evaluate the acute efficacy of Vortioxetine across multiple dosing to evaluate if there is a dose response effect and as well is there a dose response issue with respect to side effects.

Methods: According to PRISMA guidelines we systematically searched 3 major electronic databases (PubMed/MEDLINE, PsycINFO, and Cochrane Central Register of Controlled Trials) for Randomized Controlled Trial (RCT) studies published between January 2013 and April 2024. The RCTs evaluate the efficacy of Vortioxetine on acute depression, compared with placebo or other antidepressants, through improvements in depressive symptoms based on MADRS or HAM-D scores. Twenty-four studies were included in the analysis.

Results: In general, Vortioxetine significantly improved depression severity, anxiety symptoms, cognitive function, with high response and remission rates. It was also well tolerated with a relatively low occurrence of severe or serious treatment emergent adverse events (TEAEs), with nausea as the most frequent adverse event, and/or discontinuation due to intolerability. Observing the results of the meta-analysis, the effect was significant for both Vortioxetine 10 and 20 mg with a greater effect-size for vortioxetine 20 mg. These different results could explain a better clinical efficacy of Vortioxetine 20 mg than Vortioxetine 10 mg for acute symptoms of depression.

Conclusions: In conclusion, the results of the present study underlined the efficacy and safety of Vortioxetine and pointed out that the optimal dosage of Vortioxetine for the treatment of acute symptoms of depression varies between 10 and 20 mg with 20 mg for a better management of acute depression. When choosing initial therapy for MDD in clinical practice, it is important to consider not only drug efficacy but also patient preferences such as AEs and possible adherence. Thus, Vortioxetine should be consider efficacious as a first, and second line therapy.

Disclosure of Interest: None Declared

EPV1596

Clozapine response in patients with treatment-resistant schizophrenia: a preliminary report

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Introduction: Clozapine, the first atypical antipsychotic agent, serves as the gold standard treatment for treatment-resistant schizophrenia (TRS), being the most effective choice. Despite the fact that early commencement of clozapine is related to a higher response rate of patients, many psychiatrists remain reluctant towards its initiation. Identifying sociodemographic and clinical features correlating to clozapine response could facilitate the prompt clozapine initiation and ameliorate clinical outcomes.

Objectives: Our department presents the preliminary results of a prospective cohort study on patients diagnosed with TRS, as defined by the Treatment Response and Resistance in Psychosis (TRRIP) Working Group criteria, before clozapine initiation prospectively for 6 months. The study aims to evaluate the potential association of several sociodemographic and clinical factors with clozapine response.

Methods: The patients included in our study were required to have a history of treatment-resistant schizophrenia, as specified by the TRRIP criteria and no history of treatment with clozapine. The TRS patients were submitted to clinical assessments in baseline and 6 months after clozapine initiation included the following: the Positive and Negative Syndrome Scale (PANSS), the Perrsonal and Social Performance Scale (PSP), past medical history, sociodemographic data, blood tests, and monitoring of clozapine blood levels.

Results: 30 patients have been screened until now, of which, 26 met the inclusion criteria. 4 patients withdrew from the study before the