34 points in the Montgomery-Asberg-Depression Rating Scale (MADRS). Randomised patients took 80 mg Silexan, 50 mg Sertraline, or placebo once daily over 8 weeks. Primary efficacy endpoint was the change of the MADRS total score between baseline and week 8. Response (a reduction of the MADRS total score ≥50%), remission (MADRS total score <10 at the end of the treatment), the Patient Health Questionnaire PHQ-9, the Beck Depression Inventory, the Clinical Global Impressions, and the Sheehan Disability scale served as secondary endpoints.
Results: The full analysis set consisted of 498 patients. Between the start and end of treatment, the MADRS total score decreased by 12.1 (13.3, 11.0) points (adjusted mean, 95% confidence interval) in patients treated with Silexan, by 12.6 (13.7, 11.5) points in patients treated with Sertraline, and by 9.95 (11.1, 8.77) points under placebo. The confirmatory analysis proved that Silexan was significantly superior to placebo ($p<0.01$, ANCOVA). Internal validity could be shown since the treatment effects of the active comparator Sertraline were also more pronounced compared to placebo ($p<0.01$). There were no relevant differences between Silexan and Sertraline. Response was achieved by 53.5% of the patients in the Silexan group, by 54.0% of the patients in the Sertraline group, and by 41.5% of the patients in the placebo group. 44.4% of the patients treated with Silexan were remitter, compared to 45.2% under Sertraline and 32.6% under placebo. In both active treatment groups responder and remission rates were higher than in the placebo group ($p < 0.05$). Results of the secondary endpoints were in line with the results of the primary endpoint.
Conclusions: In a large phase III clinical trial, Silexan was more effective than placebo and not different to Sertraline in patients with a major depressive episode. Treatment effects were clinically relevant.

Disclosure of Interest: S. Kasper Consultant of: In the past 3 years Dr Kasper served as a consultant or on advisory boards for Angelini, Biogen, Boehringer, Esai, Janssen, IQVIA, Mylan, Recordati, Rovi, Sage and Schwabe; and he has served on speakers bureaus for Angelini, Aspen Farmaceutica S.A., Biogen, Janssen, Recordati, Schwabe, Servier, Sothema, and Sun Pharma., Speakers bureau of: In the past 3 years Dr Kasper served as a consultant or on advisory boards for Angelini, Biogen, Boehringer, Esai, Janssen, IQVIA, Mylan, Recordati, Rovi, Sage and Schwabe; and he has served on speakers bureaus for Angelini, Aspen Farmaceutica S.A., Biogen, Janssen, Recordati, Schwabe, Servier, Sothema, and Sun Pharma., E. Seifritz Consultant of: Schwabe, Janssen, Speakers bureau of: Schwabe, Janssen, H.-P. Volz Consultant of: Schwabe, Janssen, Speakers bureau of: Schwabe, Janssen

EPP0301

Ketamine enhanced ECT in refractory recurrent depression.

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Introduction: Recurrent Depressive Disorder is a chronic condition that significantly impacts the quality of life. Despite various treatment options, some patients face severe and treatment-resistant relapses. This case is related to research on ketamine in Electroconvulsive Therapy (ECT) for RDD. One study highlighted the efficacy and safety of ketamine compared to other anaesthetic agents in ECT for major depression. Additionally, another study explored subanesthetic doses of ketamine before each ECT session to improve therapeutic outcomes and sleep quality in patients with major depressive disorder.
Objectives: To present a clinical case of a patient with Recurrent Depressive Disorder (RDD) who improved following a change in the Electroconvulsive Therapy (ECT) protocol using ketamine as an anaesthetic inducer.
Methods: We examined the patient's medical records, including her medical history, previous treatments, and therapeutic responses.
Results: A 65-year-old childless woman with a history of stroke, bilateral carotid atheromatosis, and hypothyroidism suffered from RDD. Despite multiple prior treatments and ECT, she experienced a severe depressive relapse. Eight intensive ECT sessions were administered, with observed memory lapses. Due to the lack of response, the anaesthetic inducer etomidate was replaced with ketamine, resulting in a positive response. The patient continued pharmacological treatment with improved mood, but recent and evident memory alterations persisted, possibly related to anterograde amnesia.
Conclusions: This case highlights the complexity of RDD in patients with comorbidities and treatment-resistant relapses. The change in the ECT protocol using ketamine was effective, emphasizing the importance of alternative therapeutic approaches in refractory cases. The successful treatment of RDD in this patient using ketamine in ECT underscores the need for personalized therapeutic options in treatment-resistant patients. These scientific resources reinforce the relevance of exploring therapeutic alternatives in contemporary clinical practice. We need more research to understand the underlying mechanisms and how this approach could be enhanced in similar cases.

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EPP0302

Revealing complexity: beyond the whole—segmentation of hippocampal subfields in adolescents with depression and its relationships with cognition

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Introduction: The occurrence of depression in adolescence, a critical period of brain development, linked with neuroanatomical and cognitive abnormalities. Neuroimaging studies have identified hippocampal abnormalities in those of adolescent patients. However, few studies have investigated the atypically developmental trends in hippocampal subfields in adolescents with depression and their relationships with cognitive dysfunctions.
Objectives: To explore the structural abnormalities of hippocampal subfields in patients with youth depression and examine how these abnormalities associated with cognitive deficits.
Methods: We included a sample of 79 first-episode depressive patients (17 males, age = 15.54±1.83) and 71 healthy controls