temporarily closed at the outset of the COVID pandemic. Subjects completing the DB during this time were allowed delayed entry into the OLE upon requalification. Safety and efficacy measures were assessed relative to DB (or OLE re-entry) Baseline) at OLE Weeks 2, 4, and ~ every 8 weeks thereafter. The trial was planned for 3 years or until commercial availability of viloxazine ER.

Results. Subjects (N= 159; including 133 immediate- and 26 delayed-rollover) received viloxazine ER for 265 ± 254.9 days (mean ± SD). Nine subjects used adjunctive stimulant medication at some point after Week 12. Primary reasons for discontinuation included withdrawal of consent (25.6%), loss to follow up (17.7%), and adverse events (17.6%). Adverse events (experienced by 72.3%) were largely mild (26.4%) or moderate (40.3%) in severity and included (≥10%) insomnia (13.8%), nausea (13.8%), headache (10.7%), and fatigue (10.1%). Changes in clinical laboratory measures, vital signs, and ECG parameters were consistent with those observed in DB and product labeling. Suicidal ideation (wish to be dead) was reported by 3 subjects at a single visit each; no subject reported suicidal behavior. ADHD symptom (AISRS), executive function (BRIEF-A), global function (CGI), and quality of life (AAQOL) measures showed continued improvement in the OLE relative to that seen in DB. Baseline [mean ± SD)] AISRS Total, CGI-S, BRIEF-A GEC T-score and AAQOL ratings, respectively, were 37.9 ± 6.34), 4.6 ± 0.60, 70.4 ± 10.94 and 54.9 ± 14.96. All showed significant improvement (P<.0001 relative to Baseline) by the first OLE follow-up assessment (Week 2 for AISRS and CGI-S, Week 4 for BRIEF-A and AAQOL). Improvement continued with long-term use. Subjects maintained on viloxazine ER for at least 3 months (≥ Week 12) showed changes at Last OLE Visit of -20.0 ± 11.63 (n=106), -1.8 ± 1.34, -13.6 ± 13.64 (n=104), and 12.7 ± 17.90 (n=87) respectively.

Conclusions. Subjects maintained on viloxazine ER showed continued improvement in ADHD symptoms, global and executive function, and quality of life measures during long-term treatment.

Funding. Supernus Pharmaceuticals, Inc.

Cogwheel Rigidity in Subacute Combined Degeneration Unresponsive to Vitamin B12 Therapy

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Introduction. Parkinsonian symptoms seen with B12 deficiency have been described in five cases where B12 therapy has led to their elimination. Subacute combined degeneration (SCD) presenting with parkinsonian signs of cogwheel rigidity, unresponsive to B12 supplementation, has not heretofore been described.

Methods. Case Study: This 62-year-old right-handed woman with a past medical history of hypothyroidism presented with complaints of trouble with memory. Cogwheel rigidity and pernicious anemia with low intrinsic factor and B12 levels (165 pg/ml) were found. SCD was diagnosed and treated with monthly B12 injections over the past three years, providing symptomatic relief, yet the cogwheeling persisted. She described never developing trouble with gait, movement disorders, autonomic abnormalities, olfactory dysfunction, disorders of sleep, visual hallucinations, or other parkinsonian symptoms.

Results. Abnormalities in Neurologic Examination: Cranial Nerve (CN) Examination: CN I: Alcohol Sniff Test: 9 (hyposmia). CN III, IV, VI: Bilateral ptosis. Motor Examination: 1+ Cogwheel rigidity both upper extremities. Drift Testing: Right pronator drift with left abductor digiti minimi sign. Reflexes: 3+ throughout other than 4+ ankle jerks. Quadriceps femoris bilaterally pendular. Bilateral Hoffman and Babinski reflexes present. B12 Level: 394 pg/ml (normal).

Discussion. While predominantly affecting the posterior columns and the lateral corticospinal tract, the demyelination may further extend into adjacent fibers including the reticulospinal tract and the rubrospinal tract, the tracts which, in Parkinson's Disease, have been cited for their role in maintenance of tone and thus cogwheeling. Additionally, low B12 and elevated homocysteine levels have been noted as potential contributory factors in the pathogenesis of Parkinson's Disease. It is also possible that this is a violation of Occam's razor, that this individual has two separate distinct diseases — the prominent subacute combined degeneration as well as a subclinical parkinsonism which was revealed on neurologic examination. The parkinsonian signs may have been present prior to the B12 deficiency, and if not for the examination findings, could have remained undiscovered for decades. In those that present with Subacute Combined Degeneration, evaluation for parkinsonism is warranted.

Funding. No Funding

Pseudo-Wellens Syndrome: Demonstrating the Importance of Electrocardiogram in Emergency Psychiatric Patients

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Introduction. Pseudo-Wellens syndrome (PWS) is a rare but clinically significant condition characterized by electrocardiogram (ECG) abnormalities that mimic acute ST-segment elevation myocardial infarction (STEMI) in the absence of obstructive lesions on coronary angiography. The occurrence of PWS should be on the differential for any patient who visits the Emergency Room

https://doi.org/10.1017/S1092852924001287 Published online by Cambridge University Press

(ER) with known and/or suspected drug overdose. This will avoid the potential for inappropriate invasive diagnostic modalities in otherwise cardiac healthy individuals. This literature review aims to explore the ECG changes in patients diagnosed with PWS in the context of drug intoxication presenting to the ER.
Methods. Specific keywords such as "Pseudo-Wellens Syndrome," "ECG/EKG," and "substance abuse," were used to search PubMed, Google Scholar, and PsycInfo. Articles on PWS that were not on patients presenting with substance use or not in English were removed. We extracted substance use history, ECG parameters, and their clinical presentations to the ER for review.
Results. We found cases of PWS in patients presenting with cannabis, PCP, methamphetamine, opioid, and cocaine intoxication, either in combination or singularly. The most common ECG finding across the cases was biphasic T wave inversion in V2 and V3 with involvement in the anterior leads. The authors were unable to find any characteristic ECG changes associated with individual substances. This might be attributed by the small number of patients in the studies and due to the use of multiple drugs by patients at presentation to the ER, especially with drugs known to cause ECG abnormalities such as opioids. While PWS typically resolves spontaneously in most cases, this review revealed a concerning trend. Patients who consumed cocaine were at a higher risk of developing life-threatening cardiac conditions, including myocardial infarction. This finding underscores the importance of considering the pretest probability of acute coronary syndromes and avoiding misinterpretation of PWS as a less severe entity. For those patients presenting with a suspicious diagnosis and high pretest probability indicating an interruption of coronary blood flow, a comprehensive work up should be done to investigate any other possible life-threatening cardiac conditions.
Conclusions. PWS has ST segment and T wave abnormalities, which are ECG abnormalities that are already very common in psychiatric patients seen in-hospital. Recognizing the specific ECG findings in PWS is of utmost importance for clinicians to prevent unnecessary interventions and potential harm to patients. For further understanding, more comprehensive analysis of ECG findings with larger sample sizes while considering comorbid conditions and contributing factors to the patient presentation should be conducted.
Funding. No Funding

Ketamine Plus Propofol (Ketofol) Administration Before ECT for Depression: A Systematic Review for Hemodynamic Parameters and Depression Outcomes

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Introduction. Ketamine, in theory, as an anesthetic prior to electroconvulsive therapy (ECT) can potentiate anti-depressant effects given that both ketamine and ECT have demonstrated benefit. However, ketamine can cause significant hemodynamic instability, which can limit its use. Propofol can help mitigate these issues, resulting in ketofol, a combination of ketamine and propofol as an anesthetic agent. In this systematic review, we see if this anesthetic cocktail proves to be effective pre-ECT for patients with depression and evaluate the hemodynamic side effects and post-ECT depression outcomes as compared to ketamine or propofol alone.
Methods. Search strategies using keywords were created to explore two databases: PubMed and Google Scholar. After removing duplicates, 71 articles were found. All abstracts and full texts were examined by 3 independent reviewers to solve discrepancies in the screening decisions. We included 15 observational articles that are not reviews which had a direct comparison of ketofol with ketamine or propofol only in regards to hemodynamic or depression outcomes.
Results. The review included 12 studies looking at the anesthetic effects of ketofol and 8 studies looking at the depressive effects of ketofol. Based on the GRADE approach, the evidence level of primary and secondary outcomes regarding anesthetic effects ranged from low (1/12 observational study) to moderate-high (11/12 randomized control trials (RCTs)) while the evidence level for depressive effects were moderate to high (8/8 RCTs).

Anesthetic effects were mostly assessed using hemodynamic parameters such as systolic and diastolic blood pressure, mean arterial pressure, and heart rate. Out of the 12 studies, 4 RCTs concluded that ketofol was significantly more hemodynamic favorable compared to propofol, whereas 3 RCTs found the opposite. The remaining 4 RCTs and 1 observational study concluded that there was no statistically significant difference in hemodynamic parameters when administering ketofol.

Depressive effects were assessed using validated psychometric testing. Out of the 8 studies, 2 RCTs concluded that ketofol had a greater reduction in depressive symptoms compared to propofol. The remaining 6 RCTs concluded that there was no statistically significant difference in reduction of depressive symptoms with the use of ketofol.
Conclusion. Ketofol is an anesthetic combination that is being used for a variety of procedures, including ECT. While theorized to assist with the hemodynamic changes that ketamine or propofol alone cause in opposite directions, most studies have demonstrated no significant difference between ketofol and one medication. Furthermore, most studies also show no difference in depression outcomes after ECT between ketofol and ketamine or propofol monotherapy. If a statistically significant change is seen, it is usually attributable to the ketamine portion of ketofol rather than an effect from the combination.
Funding. No Funding

https://doi.org/10.1017/S1092852924001287 Published online by Cambridge University Press