evolutionary survival advantages) can become maladaptive and destructive. This comprehensive, 'interventionist' model will hopefully lead to better clinical conceptualization of AAV, especially since most AAV presents more commonly in the context of interpersonal/situational stressors and to non-forensically trained clinicians.

Objectives:

1. To identify biological, cognitive, disorder-based, developmental and personality predispositions to AAV
2. To link neuroanatomical and functional brain circuitry with onset and maintainence of AAV
3. To identify *how* pharmacological and non pharmacological interventions work, and 'where' in the brain,
4. To review the evidence-base supporting various treatment interventions

Methods: Database review of PUBMED, PsychINFO, Google Scholar and treatment guidelines dating back 15 years (review articles, treatment guidelines, meta-analyses and randomized controlled trials).

Results: This will be visually depicted in an electronic slide (or >1 slide, if permitted) to identify:

1) The inter-relationship between contextal factors, the psychopathology of AAV and undelying brain 'circutry'
2) The specific nature of the impulsive anger, 'reactive' aggression and violence response 'cycle' and where in this cycle various interventions can be effectively utlized
3) Which patient populations respond best to which types of interventions

Conclusions:

1. AAV are hyperarousal states, with outcomes that can be successfully managed
2. The evidence-base for the role of medications is more limited
3. The evidence-base for the role of various psychotherapies is greater, with specific therapies being particularly useful across psychiatric disorders and populations
4. Psychodynamic approches remain curently underutilized and underappreciated
5. Both medications and therapy influence brain neuroplasticity separately and synergistically

Disclosure of Interest: None Declared

EPV1061

Title: Identifying Neuropsychiatric Symptoms in Lewy Body Dementia: A Revealing Case Report

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Introduction: Lewy body dementia (LBD) is characterized by a range of complex neuropsychiatric symptoms that can initially mimic other psychiatric disorders, particularly melancholic depression. Failure to recognize these symptoms can lead to delayed diagnosis and inappropriate management.

Objectives: This case report highlights the importance of a thorough evaluation of neuropsychiatric symptoms in the diagnosis of Lewy body dementia (LBD).

Methods: This case report was compiled through clinical observations, patient history from family members, and medical testing. Literature on LBD was reviewed to assess treatment strategies in light of this patient's condition.

Results: Mr. T.C., 75 years old, followed for hyperthyroidism and benign prostatic hyperplasia, was admitted for a severe depressive episode with melancholic features. Despite treatment with fluoxetine, mirtazapine, and olanzapine, his clinical condition did not improve. The patient exhibited food refusal, active suicidal ideation, and thoughts of incurability, along with a delusional syndrome. The Mini Mental State Examination (MMSE) revealed cognitive impairment with a score of 21/30. An initial brain MRI showed no abnormalities. The treatment was adjusted with the introduction of paroxetine alongside olanzapine and mirtazapine, but there was no significant improvement. A follow-up brain MRI revealed periventricular vascular leukoencephalopathy and moderate cortical atrophy, directing the diagnosis towards Lewy body dementia. The appearance of additional neuropsychiatric symptoms, including visual hallucinations, cognitive fluctuations, and mild parkinsonian signs, further supported the diagnosis of LBD. Treatment was then adjusted with the introduction of quetiapine, which is better tolerated in the context of LBD due to its favorable therapeutic profile.

Conclusions: This case emphasizes the importance of accurately diagnosing neuropsychiatric disorders in Lewy body dementia. Appropriate management, based on a thorough clinical evaluation, can prevent treatment errors and improve the patient's quality of life.

Disclosure of Interest: None Declared

EPV1062

Sex differences in the levels of key mitochondrial markers in subjects with mild cognitive impairments and Alzheimer's disease

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Introduction: The lack of accessible plasma biomarkers to identify target populations limits the promise of precision medicine for Alzheimer's Disease (AD). Amnestic mild cognitive impairment (aMCI) is an important risk for AD and often occurs years before the onset of AD.

Objectives: Based on an emerging mechanistic model of mitochondrial mechanisms of brain plasticity, we studied the role of