Results: A total of 72 patients were included. The main characteristics of the sample are shown in table 1. Among these patients, 65.28% exhibited NPS; notably, 49.3% had depression, 23.61% behavioral disturbances, 19.44% sleep disorders, 16.67% anxiety, 4.17% psychosis, and 2.82% suicidal ideation. In patients with a positive APscan, 29.79% had NPS, including 34.29% with depression and 66.67% with psychosis. Patients with abnormal FDG-PET scans showed higher NPS prevalence (65.96%), particularly behavioral disturbances (64.71%), sleep disorders (57.14%), and depression (62.86%).

Image:

Mean of age	61,75 years	
Gender	Male	45,83%
	Female	54,17%
Global deterioration scale	3	75,71%
	4	12,86%
Cognitive tests	MOCA	25,41%
	MMSE	26,07%
Neurological Diagnosis post PET	Alzheimer Disease (AD)	25,00%
	Lewy Body Dementia	5,00%
	Frontotemporal Dementia	10.00%
FDG- PET Scan Pattern	Alzheimer-like	16,67%
	Altered no AD Pattern	51,39%
	Non Conclusive	15,28%
Amyloid PET Scan Pattern	Positive	36,11%
	Negative	63,89%

Table 1. Main characteristics of the sample

Conclusions: This study underscores the high incidence of NPS in MCI patients, noting that NPS may exacerbate patient distress, contribute to autonomy loss, and increase institutionalization risk. Furthermore, molecular imaging patterns can help predict MCI progression to dementia and highlight NPS as potential predictors and outcomes of these biological changes.

Disclosure of Interest: None Declared

EPV1045

Apathy, Beta-amyloid Burden and Cognitive Decline in Parkinson's Disease Patients

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Introduction: Apathy is a common non-motor symptom in Parkinson's disease (PD), and its presence constitutes a risk factor for the development of cognitive impairment in this population (Burchill et al. Lancet Reg Health Eur 2024; 39:100870). β-amyloidopathy has been associated to shorter interval to dementia in PD and may also be a determinant of apathy.

Objectives: We aimed to investigate β-amyloid burden in non-demented PD patients based on the presence or absence of apathy, and how both factors influence the rate of progression to mild cognitive impairment or dementia over a 3-year period.

Methods: Forty-eight PD patients underwent clinical and comprehensive neuropsychological evaluation, as well as [18F]-flutemetamol positron emission tomography. They were classified as apathetic (n=22) or non-apathetic (n=26) based on their score on the Starkstein Apathy Scale. Brain imaging analysis was conducted using the SPM software package.

Results: We found statistically significant differences in disease duration when comparing clinical and demographic variables. Upon neuropsychological evaluation, apathetic patients performed significantly worse in attention domain (Digit Span and Trail Making Test A), executive function (Stroop Word-Colour Test and Trail Making Test B) and verbal memory (CERAD Total Word Recall). At follow up, 47.4% of apathetic patients progressed to dementia or MCI, compared to 12% of non-apathetic patients (χ^2 = 6.81, p <0.05). Brain imaging analysis revealed higher β-amyloid deposition in several cortical areas in apathetic PD patients (adjusted for disease duration and global composite cognitive z-scores).

Conclusions: The presence of apathy in PD patients is associated with greater cortical β-amyloidopathy and indicates a higher conversion rate to a worse cognitive diagnosis within a 3-year period.

Disclosure of Interest: None Declared

EPV1046

Gray matter macrostructure and brain aging in unhealthy alcohol users

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Introduction: In view of recent global trends in alcohol use, it becomes increasingly relevant to characterize health outcomes related to unhealthy alcohol use. Previous studies found that self-reported alcohol use was related to poor brain health. However, these studies remain inconclusive since they limited their analyses to very narrow demographic strata, considered only a subset of cortical regions, or didn't validate self-reported alcohol use with biomarkers such as gamma-glutamyltransferase (gamma-GT).
Objectives: This study aimed to comprehensively examine several aspects of brain health (cortical thickness, gray matter volume, and brain age gaps) in participants regularly exceeding the recommended limits of moderate alcohol use versus those who don't, and to validate self-reported alcohol intake by comparing gamma-GT levels across groups.
Methods: This analysis was based on cross-sectional data from the population-based cohort of the BiDirect Study conducted in Münster (Germany). Individuals aged between 35 and 65 years were randomly selected from the local population register and invited to participate in the assessment that included a 3 Tesla magnetic resonance imaging (MRI) of the brain and a blood collection. Unhealthy alcohol use was defined as the regular consumption of at least three units of alcohol (one unit = 0.2L beer or 0.1L wine or 2cl spirits) per occasion at least twice a week. Regional cortical thickness and subcortical gray matter volumes were extracted from T1-weighted images in participants who underwent MRI. In addition, brain age gaps were estimated using an elastic net algorithm based on the imaging-derived phenotypes. Associations between unhealthy alcohol use, cortical thickness, subcortical gray matter volumes, and brain age gaps were analyzed using multiple regression models adjusted for age, sex, lifetime smoking status, education, and childhood trauma.
Results: Participants engaging in unhealthy alcohol use had significantly higher gamma-GT levels. In addition, unhealthy alcohol use was associated lower regional cortical thickness across all four lobes of the brain. No differences in subcortical gray matter volumes were detected. In addition, we observed a significantly higher brain age gap (+ 1.11 years) in unhealthy alcohol users.
Conclusions: The results of this study indicate that the regular exceedance of the recommended levels of alcohol use is associated with poorer brain health as reflected by lower regional cortical thickness and advanced brain aging. The findings underscore the potentially adverse effects of alcohol on brain health, which are increasingly relevant in view of recent global trends in alcohol use.

Disclosure of Interest: None Declared

EPV1047

How Follow-Up Neuroimaging Techniques Enhance Care for a Veteran with Combat-Related TBI and PTSD

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Introduction: As traumatic brain injury (TBI) is a significant concern among military veterans, ongoing neuroimaging is a beneficial tool for monitoring functional brain changes and evaluating the progression of symptoms.
Objectives: Highlighting the importance of follow-up neuroimaging assessments in guiding treatment adjustments and understanding the evolving relationship between TBI, post-traumatic stress disorder (PTSD), and neurocognitive dysfunction.
Methods: A 27-year-old male veteran injured by an IED experienced trauma to the right side of his body, resulting in 80% vision loss in the right eye and 20% in the left. He reported memory gaps and sleep disturbances. After inpatient and outpatient rehabilitation, he was prescribed Olanzapine (5 mg/day), Quetiapine (150 mg/day), and Venlafaxine (75 mg/day). On his second admission for increased sleep disturbances and anxiety, Quetiapine was increased to 200 mg/day. One year later, the patient developed new cognitive impairments and reported memory deficits and anterograde amnesia, concurrently PET scans revealed hypometabolism in the frontal lobe.
Results: Neuropsychological Evaluation: The Raven Standard Progressive Matrices Test indicated potential issues in reasoning and problem-solving, while the Verbal Fluency Test suggested difficulties with cognitive flexibility and memory, and the Trail Making Test revealed problems with attention and sequencing. Imaging Findings: The initial CT scan demonstrated displaced linear fractures in the right temporal bone and two brain contusions shortly after the incident. On his visit nine months later, SPECT imaging showed relative hypoperfusion in the right posterior parietal cortex and bilateral temporal lobes. An EEG revealed slow wave anomalies in the right temporooccipital area and sharp spasms in the left temporal region. One year later, a follow-up PET scan revealed diffuse hypometabolism in the left frontal lobe, parietal lobes, and cerebellum.

Image:

