

EPV1078

Cognitive function and cannabis use in first episodes of psychosis

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Introduction: The association between cannabis use and cognitive functioning in individuals experiencing a first episode of psychosis (FEP) is inconsistent, with some studies reporting deterioration (Bogaty et al. J Psychiatr Res 2018; 99:22-32), others indicating improved performance (Rodríguez-Sánchez et al. Schizophr Res 2010; 124:142-151), and some finding no difference.

Objectives: This study aims to evaluate the effect of cannabis use on cognitive functioning among individuals diagnosed with FEP.

Methods: A cross-sectional study was conducted on FEP patients enrolled in the ITPCan Program (Santander, Spain) between January 2020 and July 2024. A total of 207 participants (57 cannabis users and 145 non-users) with a FEP diagnosis were included. A descriptive-univariate analysis was performed on clinical, analytical, and cognitive variables. A Student's t-test compared baseline cognitive performance between cannabis users and non-users, while a multivariate general linear model (GLM) assessed differences related to cannabis consumption, adjusting for sex, age, and educational level. Statistical analyses were performed using SPSS.

Results: Out of the 207 FEP participants, 53.6% were women, with an average age of 36.8 years. Cannabis users comprised 28.1% of the group, and present at baseline a lower mean age at intake (27 years; $p < 0.001$). Cannabis users exhibited significantly higher scores in mania and positive psychotic symptoms. Cognitive assessments, completed by 148 patients (39 users and 108 non-users), revealed that cannabis users performed better than non-users on "processing speed" task; however, after adjusting for sex, age, and educational level, these differences were attributed to educational level and sex.

Conclusions: In consonance with some previous studies (Sánchez-Gutiérrez et al. Eur Psychiatry 2020; 63(1)), cannabis use does not appear to be a determining factor in cognitive performance in early psychosis.

Disclosure of Interest: None Declared

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Neuropsychiatric Effects of HIV and Toxoplasma Gondii Encephalitis: A Case Study

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Introduction: HIV infection can lead to neurological and psychiatric disorders by affecting the central nervous system. Toxoplasma

gondii can cause encephalitis in immunocompromised individuals, particularly damaging brain regions such as the frontal cortex and basal ganglia. These neuroanatomical disruptions can influence dopaminergic and serotonergic neurotransmitter pathways, resulting in severe neuropsychiatric symptoms like behavioral disinhibition, personality changes, and psychotic manifestations (Vidal JE. J Int Assoc Provid AIDS Care 2019; 18:2325958219867315).

Objectives: This case seeks to demonstrate the neuropsychiatric effects of HIV and Toxoplasma gondii infections on neuronal function and behavior, emphasizing the importance of continuous assessment and integrated treatment approaches.

Methods: A 49-year-old male patient, with no known medical history, developed Toxoplasma gondii encephalitis after being diagnosed with HIV. Following discharge from the infectious disease ward, the patient exhibited increased irritability, impulsivity, hypersexuality, visual hallucinations, disorganized speech, and risky behaviors, along with cognitive dysfunction and social norm violations. The patient's symptoms partially improved with a treatment regimen of 15 mg olanzapine and 1000 mg valproic acid. However, the patient's ongoing course, parkinsonism symptoms emerged, prompting the discontinuation of valproic acid and the consideration of quetiapine. The patient's follow-up treatment is ongoing.

Results: HIV and Toxoplasma gondii disrupt neuronal function through inflammatory responses and microglial activation in the central nervous system. HIV does not directly damage neurons, but inflammation from cytokines released by microglia can lead to neurodegenerative conditions. Dopaminergic dysfunction is linked to psychotic symptoms, while serotonergic disruptions contribute to depression and anxiety. Frontal lobe and basal ganglia damage impair executive functions (planning, decision-making, impulse control), causing increased impulsivity, risky behaviors, emotional dysregulation, irritability, lack of empathy, behavioral disinhibition, hypersexuality, and cognitive dysfunction. These changes are tied to structural damage in the frontal cortex, necessitating long-term follow-up and comprehensive treatment. Diagnostic imaging often shows white matter lesions, basal ganglia damage, and frontal cortex atrophy. Treatment involves antiretroviral therapy, low-dose antipsychotics, mood stabilizers for managing symptoms (Bartolomé Del Pino LE et al. Actas Esp Psiquiatr 2024; 52(2):149-60).

Conclusions: HIV and Toxoplasma gondii infections induce significant central nervous system impairment, leading to neuropsychiatric disorders. This case illustrates their impact on neurotransmitter systems, manifesting as psychosis, cognitive dysfunction, and behavioral disturbances. Continued monitoring and comprehensive treatment are essential.

Disclosure of Interest: None Declared

EPV1078

Relationships between parameters of EEG and glutamate-ergic system activity in patients with depressive-delusional disorders

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Introduction: Activity of the glutamate neurotransmitter system contribute to the development of many mental disorders, in particular of depression and schizophrenia. While the glutamate is the principal excitatory neurotransmitter in the brain there are poor data on its relationships with EEG.

Objectives: The aim of the study was to search for possible relationships between parameters of EEG and glutamate dehydrogenase (GDH) activity in patients with depressive-delusional disorders.

Methods: The study involved 28 female in-patients aged 16-35 years (mean age 22.0 ± 8.1 years) with depressive-delusional disorders in the frames of schizophrenia (F20.01, by ICD-10). Pre-treatment multichannel resting EEG recordings with spectral power analysis in narrow frequency sub-bands were performed in all patients. Patients were divided into two groups with relatively "normal" ($n=18$) and "slow" ($n=10$) EEGs. "Slow EEG" group statistically differed ($p<0.05$) from "normal EEG" group by greater spectral power values in theta2 (6-8 Hz) EEG sub-band in frontal-central-temporal regions of the left hemisphere. GDH enzymatic activity was measured in platelets' extracts from blood samples by spectrophotometric kinetic method and was assessed by the rate of NAD•H absorption decrease at 340 nm. The descriptive statistics and the rank correlation analysis (Spearman) were used for statistical processing of EEG and neurochemical data.

Results: "Slow EEG" and "normal EEG" groups did not statistically differ ($p>0.05$) in age, HDRS and PANSS scores, while the clinical severity was somewhat greater in "slow EEG" group. Nevertheless, "slow EEG" and "normal EEG" groups apparently differed in correlation structure between EEG and GDH activity parameters. Thus, in "slow EEG" group values of GDH activity correlated positively ($p<0.05\div 0.01$) with values of spectral power in delta (2-4 Hz), theta1 (4-6 Hz) and theta2 (6-8 Hz) EEG sub-bands. In "normal EEG" group values of GDH activity did not correlate with any studied EEG parameters.

Conclusions: Greater GDH activity in "slow EEG" group of patients with depressive-delusional disorders have to provoke the glutamate mediated excitation deficit reflected in EEG slowing. The predominance of this phenomenon in the left hemisphere may underlie some features of the clinical conditions in these patients and need further studying.

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EPV1079

Temporal-lobe functional connectivity in future converters to Alzheimer's disease

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Introduction: Preclinical stages of Alzheimer's disease (AD) are characterized by structural and neurochemical abnormalities in temporal lobe and temporoparietal junction (T-TPJ; Popuri *et al.* Hum Brain Mapp 2020; 41 4127-4147; Jagust. Nat Rev Neurosci 2018; 19 687-700). However, the role of functional characteristics of T-TPJ in conversion to AD is understudied.

Objectives: Our aim was to clarify whether any patterns of functional connectivity (FC) within T-TPJ differentiate patients with amnesic mild cognitive impairment (aMCI) and future conversion to AD from stable aMCI and healthy controls.

Methods: Patients with aMCI and future conversion to dementia due to AD (converters, $n = 15$; mean age 74.31 ± 7.86), patients with stable aMCI (non-converters, $n = 12$; mean age 66.77 ± 9.54), and healthy individuals without cognitive deficits ($n = 29$; mean age 64.17 ± 11.30) underwent resting-state fMRI (3T Philips Ingenia scanner). Eighteen T-TPJ cortical ROIs in each hemisphere were defined according to Harvard-Oxford atlas (Desikan *et al.* Neuroimage 2006; 31 968-980). FC between these ROIs was compared between groups separately for each hemisphere (one-way ANCOVA with connection threshold $p[FDR] < 0.05$ and *post hoc* between-group comparisons). Age, sex, and number of outlier scans due to motion were included into the models as covariates of no interest. The analyses were implemented via CONN (RRID: SCR_009550; www.nitrc.org/projects/conn).

Results: FC between the posterior parts of left middle and inferior temporal gyri was different between converters, non-converters, and healthy individuals ($p[FDR] = .0256$). Converters demonstrated an increased FC between these regions compared to other groups ($p = .0003$ for both tests). We also observed a trend for inverse correlation between this FC and delayed recall of words (MoCA) in the entire sample ($p = .055$).

Conclusions: An increased FC between the temporal brain regions may reflect either pathological processes that have already started, or compensatory mechanisms, or both in future converters to AD. The posterior part of left middle temporal gyrus is critical for auditory verbal memory, whereas the posterior inferior temporal cortex in the left hemisphere stores visual images associated with a word. The observed trend-like correlation might indicate that patients with worse auditory verbal memory rely on visual images associated with a word, however, this assumption should be supported in further studies.

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EPV1082

Central effects of soluble factors derived from human macrophages, polarized to M2 phenotype, in depression

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Introduction: In depressive disorders caused by chronic psychological stress, cognitive decline is produced by neuroinflammatory and neurodegenerative changes in the brain. M2-type macrophages possess high pronounced regenerative potential due to high production of neurotrophic, neuroprotective and angiogenic factors. We have previously shown that M2 macrophage-derived soluble factors (M2-SFs) edit stress-induced depressive-like behavior.