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EV0719

Case report Klinefelter syndrome and multiple sclerosis as the cause of psychosis

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Introduction and goals Forty-three-year-old male diagnosed with Klinefelter syndrome and showing radiological findings suggesting a demyelinating pathology who presents several psychiatric manifestations including megalomaniacal ideation, delusion, lack of impulse control and behavioral alterations.
Clinical case Forty-three-year-old male diagnosed with Klinefelter syndrome at the age of 31, presenting several psychiatric pathologies since adolescence: delusions, megalomania, mood fluctuation, and high impulsiveness. The patient had a poor therapeutic response to anti-psychotic drugs and ECT. He was hospitalized up to 9 times, but the full control of the symptomatology was not achieved. During his last hospitalization, a MRI revealed lesions compatible with a demyelinating pathology.
Discussion A higher prevalence of schizophrenia spectrum disorders has been described among patients suffering from Klinefelter syndrome, which might explain the role of the X chromosome in the susceptibility to psychiatric disorders, particularly to psychosis. Furthermore, the brain structure alterations presented by patients suffering from Klinefelter syndrome are similar to those described among schizophrenic patients: small brain volume, lateral cerebral ventricular enlargement and reduced temporal gyrus, amygdala, insula and cingulate cortex. Patients suffering from multiple sclerosis are more prone to psychiatric disorders, such as mood swing, aggressiveness or psychosis, which are not concurrent with the physical progression of the disease, sometimes being its first manifestation. Even when being patchy and multifocal, demyelination seems to be concentrated in the frontal lobes, related to the cognitive and affective functions and the personality.
Conclusions Both multiple sclerosis and Klinefelder syndrome may alter the brain structure, mainly in the frontal lobe, and predispose to psychiatric disorders.
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Neuroretinal dysfunctions in regular cannabis users: An impact of cannabis on retinal neurotransmission?

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Introduction Although cannabis is very widespread worldwide, its brain toxicity is poorly understood. The neuroretina is an accessible extension of the brain and could be a relevant site for investigating neurotransmission abnormalities in neuropsychiatric disorders. The retina has a functional endocannabinoid system involved in the regulation of retinal neurotransmission. In animals, the modulation of this system led to retinal dysfunctions measured with the electroretinogram (ERG).
Objectives To assess whether the regular cannabis use could affect the neuroretinal function.
Aims Assessments of the neuroretinal function in cannabis users compared with controls.
Methods Recordings of pattern, flash and on-off ERG were performed in 55 cannabis users and 29 controls. The amplitude and implicit time of the following waves were evaluated: N95 (pattern); a – and b – (flash); a –, b- and d1 – (on-off).
Results Cannabis users showed a significant increase in implicit time of the waves N95 ($P = 0.0001$), a- ($P = 0.029$) and b – ($P = 0.002$) for the flash ERG and b – ($P = 0.016$) and d1 – ($P = 0.027$) for the on-off ERG, compared with controls. No significant difference was found between groups in terms of wave's amplitudes.
Conclusions These results show a delay in the response of cones, bipolar and ganglion cells of the on and off pathways to constitute a delay of ≈ 6 ms in the transmission of information from the retina to the brain in cannabis users. Cannabis could disrupt the regulatory role of the cannabinoid system and impair retinal glutamatergic neurotransmission. The consequences on visual perception should be explored in future studies.
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In search of possible peripheral biomarkers for suicide: Similarities between platelet and cerebrospinal fluid proteome (preliminary results)

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Despite the fact that proteomic analysis is becoming widely used in various medical branches its use in psychiatry is still very limited. Majority of psychiatric proteomic research is still oriented mostly on Alzheimer's disease, schizophrenia and depression but very few studies focus on suicidality. We decided, based on the current knowledge, to study suicidal behaviour with the use of proteomics to compare cerebrospinal fluid and platelets. We hypothesized that the same protein group can be detected in pathways that are part of platelet degranulation process in the platelet proteome and cerebrospinal fluid proteome. Based on these findings we suppose, that with use of proteomic analysis a specific protein (group of proteins) can be identified in both, cerebrospinal fluid and platelet proteome in patients with suicidal behavior.
Group of proteins identified in our sample in the reactome pathway database (release of platelet secretary granule components and exocytosis of platelet granule contents) supports the idea of link between central nervous system and platelets ("the periphery"). Further research is needed to clarify whether the identified group of proteins taking part in platelet pathways can be used as peripheral biomarkers for suicidal behavior.
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