

Conclusions: It is much needed more than ever before that proper consideration to the diagnosis of iron deficiency anemia must be given with the assistance of predesigned guidelines. Misdiagnosis of iron deficiency anemia as a psychiatric disorder can be misleading toward the insidious usage of psychiatric medications. Proper attention must be provided to this neglected area so that management of iron deficiency is tailored in the right direction and it is diagnosed at less severe stages. It will be helpful for general physicians and practicing psychiatrists in the field.

Keywords: Iron deficiency, Psychiatric Disorders, Anxiety, depression.

Disclosure of Interest: None Declared

Psychoneuroimmunology

EPP0232

Assessment of serum high mobility group box 1 protein (HMGB1) levels and its change with treatment in patients with manic episode of bipolar disorder

A. Kara^{1*}, Ö. D. Balaban² and N. Karamustafalıoğlu³

¹Van Education and Research Hospital; ²Istanbul Bakirkoy Prof. Dr. Mazhar Osman Research and Training Hospital for Psychiatry, Neurology and Neurosurgery and ³Istanbul Bakirkoy Prof. Dr. Mazhar Osman Research and Training Hospital for Psychiatry, Neurology and Neurosurgery, İstanbul, Türkiye

*Corresponding author.

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Introduction: There has been an increasing evidence in recent years that inflammation plays a role in the pathophysiology of bipolar disorder (BD). In addition to central inflammation, systemic immune response is thought to be associated with disease stages and course. HMGB1 protein is a member of damage-associated molecular pattern which plays a role in the regulation of cytokine release and is important for innate immunity. It has been revealed that HMGB1 levels change in many autoimmune and inflammatory diseases

Objectives: Our study was designed to compare serum HMGB1 levels of inpatients with mania and healthy controls as well as analyzing its relationship with other inflammatory markers and disease severity. Another aim of our study is to determine the changes in serum HMGB1 levels before and after treatment in manic patients.

Methods: Our study included 35 patients who were hospitalized in our hospital between November 2020 and April 2021, diagnosed with bipolar disorder, manic episode according to DSM-5 criteria, and 35 healthy controls who matched to the patient group in terms of age and gender. The sociodemographic and clinical characteristics data forms of the participants were filled in, and the patients were evaluated with the Young-Mania Rating Scale (YMRS) and the Hamilton Depression Scale (HAM-D). Serum HMGB1, CRP levels and complete blood count values were measured in healthy controls and patients before and after the treatment.

Results: The main finding of our study is that HMGB1 levels did not show a statistically significant difference in the patient and healthy control groups ($p > 0.05$). In addition, there was no significant change in serum HMGB1 levels of the patients after treatment compared to the level before treatment ($p > 0.05$). Our study has also revealed that manic patients had a higher level of CRP than the ones in control group at a statistically significant level. The platelet/

lymphocyte and neutrophil/lymphocyte rates of the patients increased after the treatment compared to the pre-treatment period (respectively $p = 0.026$, $p = 0.003$).

Conclusions: The higher CRP levels in manic patients support the hypothesis of low-grade chronic inflammation, which is thought to be involved in the etiology of BD. The most important finding of this study is that there is no statistically significant difference of serum HMGB1 levels between the two groups. This can be explained by the fact that HMGB1 is one of the late mediators of inflammation and does not rise immediately during the acute period. However, since the inflammation process includes complex mechanisms involving many systems, it makes it difficult to comment on this issue.

Disclosure of Interest: None Declared

EPP0233

Choroid plexus volume as a proxy of neuroinflammation in depression

B. Bravi^{1,2*}, E. Melloni^{1,2}, L. Servidio¹, E. Agnoletto¹, M. Paolini^{1,2}, S. Poletti¹, C. Lorenzi¹, C. Colombo³ and F. Benedetti^{1,2}

¹Division of Neuroscience, Psychiatry and Clinical Psychobiology, IRCCS San Raffaele Hospital, Milan; ²Vita-Salute San Raffaele University, Milan and ³Mood disorder Unit, IRCCS San Raffaele Hospital, Milan

*Corresponding author.

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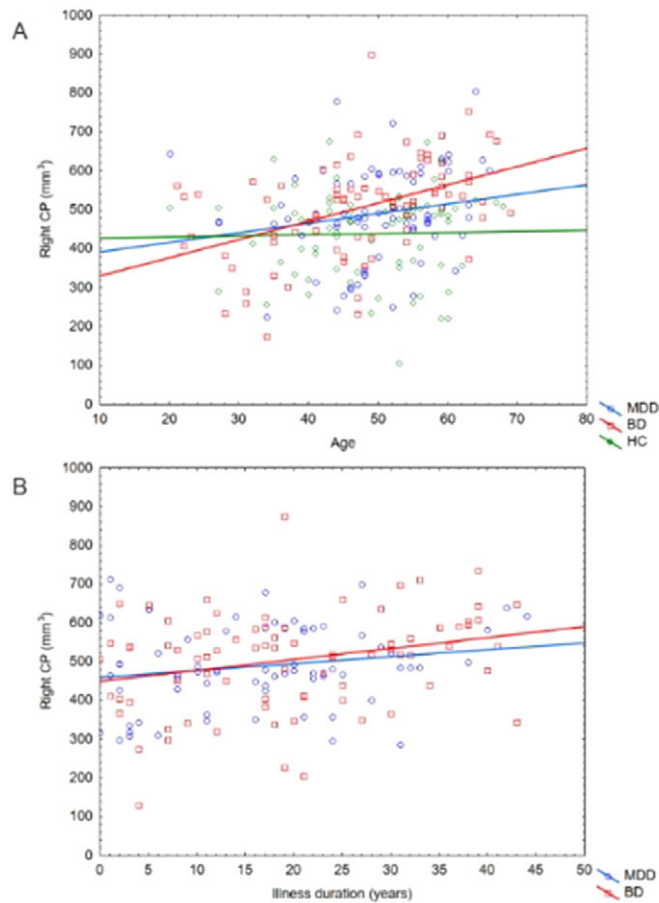
Introduction: Choroid plexus (CP) is a physiological barrier, producing cerebrospinal fluid (CSF), neurotrophic, and inflammatory factors. It's also involved in the neuro-immune axis, facilitating the interplay between central and peripheral inflammation, allowing trafficking of immune cells. Coherently, CP enlargement has been found in psychiatric diseases characterized by inflammatory signature. Although CP volume correlates with central microglia activation in major depressive disorder (MDD), it's never been directly associated with peripheral markers in mood disorders.

Objectives: Examine CP volume in mood disorders and healthy controls (HC) in relation to clinical features and peripheral inflammatory markers.

Methods: CP volume was extracted with FreeSurfer in 72 HC and 152 age- and sex-matched depressed patients: 79 BD and 73 MDD. Plasma analytes in patients were collected through immunoassay technology (Bioplex). We tested for the effect of age by group on CP volume. Then we focused on the interaction between illness duration and diagnosis in predicting CP volume. After testing the effect of specific analytes by diagnosis, we calculated moderated moderation models (SPSS, PROCESS) setting each analyte as independent variable, CP volume as predicted variable and illness duration and diagnosis as moderators. We get the effects' significance with the likelihood ratio statistic, always controlling for age, sex, and intracranial volume.

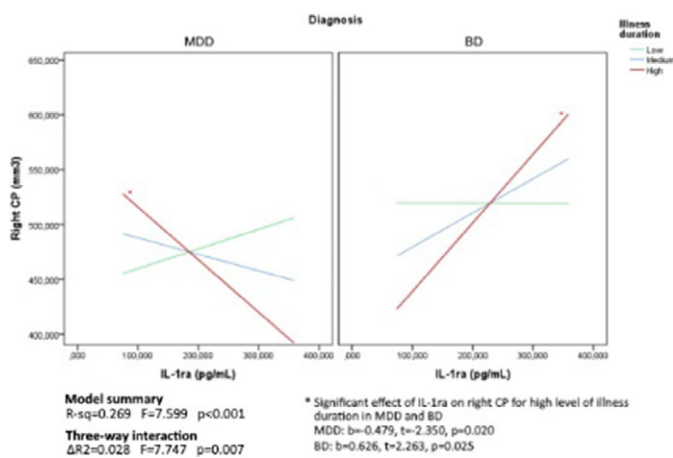
Results: Patients were comparable in illness duration and severity. CP volume is differentially distributed through groups (right: $p = 0.04$; left: $p < 0.01$), with higher volumes in the clinical groups. Age by group significantly predict right CP volume ($p = 0.01$). Also, duration of illness differently predicts right CP volume in MDD and BD ($p = 0.03$) (Figure1). Then, given the significant interaction effect of IL13 ($p = 0.02$) and IL1ra ($p = 0.01$) in predicting right CP, we run the moderated moderation model. Longer illness duration has an effect in strengthening the opposite predicting value of IL1ra ($\Delta R^2 = 0.03$, $p < 0.01$) on right CP volume in MDD and BD (Figure2).

Image:



In A) effect of age by group on right CP volume; in B) illness duration by diagnosis interaction in predicting right CP volume.

Image 2:



Conclusions: Our findings propose CP as a proxy of inflammation in depression, being significantly predicted by peripheral immune markers in MDD and BD. In particular, the signature of inflammation in depression, could represent the neurotoxic load of the disease over the illness, with a worse effect in BD, with possible

disruption of brain barriers permeability and an opposite effect of tightening and central segregation in MDD. Further analyses are needed to better elucidate this neurobiological mechanisms across mood disorders.

Disclosure of Interest: None Declared

EPP0236

Biological markers of impending psychosis :A systemaic literature review

M. Stambouli*, B. N. Saguem and J. Nakhli

psychiatry department, Farhat hached hospital, sousse, Tunisia

*Corresponding author.

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Introduction: Despite the encouraging research findings concerning the predictive validity of the psychosis risk criteria, they are still insufficient to justify treatment recommendations and attempts at preventive interventions for this early, non-specific illness phase. So far, diagnosis of the prodromal symptoms is largely based on patient reports of symptoms and/or collateral reporting, and yet remains relatively devoid of objective, brain-based biological markers. Adding specific predictors and biological markers to the clinical psychosis risk approach that could increase the predictive power of current psychosis risk criteria is a crucial step for early intervention efforts.

Objectives: We reviewed all studies examining the biological markers in subjects with an "At Risk Mental State" (ARMS) for psychosis and we discussed their predictive psychosis transition value.

Methods: A systematic search of the literature was performed using PubMed. The key words «Early psychosis / Prodromal symptoms / At risk mental state» in combination with «Biomarkers / Inflammatory markers / Stress» were used for the search.

Results: We selected 18 papers: 2 literature reviews, 7 cross-sectional studies and 9 prospective studies. Aiello et al. (2012) reviewed studies examining biological markers of the stress response in the relatives of psychotic patients and in individuals at Ultra-High Risk (UHR) for psychosis. Karanikas and Garyfallos (2015) systematically reviewed data concerning the role of cortisol in patients at risk for psychosis mental state and its associations with psychopathological correlates. There was no review available of all biomarkers.

The biological markers that have been reported are mainly stress markers, endocrine and inflammatory markers.

Serum or salivary cortisol was the most studied marker. Studies stated that UHR subjects had higher cortisol levels than controls and that there was a positive correlation between cortisol levels and the severity of symptoms. Results of prospective studies were controversial.

High levels of prolactin were reported by 3 studies, including a prospective one which concluded that prolactine may be considered as a biological marker of transition.

A prospective study found higher levels of interleukin-6 (IL-6) in UHR subjects than in controls. This study also reported significantly higher IL-6 levels in subjects who transitioned to psychosis compared to those who did not.

Conclusions: Despite advances in mental health research, there is still a lack of biological markers with clinical utility. The results of current studies should be interpreted with caution due to the small