

Introduction: ADHD (Attention-Deficit/Hyperactivity Disorder) is a common treatable disorder that impairs daily functioning along the life span. Pharmacotherapy plays a central role in managing ADHD, but adherence rates can be low, impacting treatment effectiveness.

Objectives: To compare the adherence to the specific medication used to treat ADHD on specific patient populations.

Methods: In this study, we used "Clalit Medical Services" anonymized data base and focused on the first year of treatment with the four available first-line pharmacotherapy products: Methylphenidate tablets, Methylphenidate Slow Release tablets, Methylphenidate Long Acting capsules, and Oros Methylphenidate tablets. Analyzing data from 214,035 patients of all ages diagnosed with ADHD who initiated pharmacotherapy between 2000 and 2022, we used a Negative Binomial Regression to develop a model to predict the number of prescriptions purchased in the first year of treatment, serving as a proxy for adherence. Our main focus was on identifying medications that enable better adherence.

Results: Oros Methylphenidate had the highest number of predicted purchases (RR CI 95%: 5.85-5.96). After adjusting for calendar year effects, our results identified gender, age group, and socioeconomic status (SES) as significant predictors of adherence. A significant interaction effect revealed that the predicted number of purchases for a specific medication is influenced by the patient's SES level, i.e., for the lower SES levels adherence with Methylphenidate was better than adherence with Oros Methylphenidate.

Conclusions: The choice of the specific medication available as first-line treatment for ADHD, has a significant effect on adherence. Oros Methylphenidate has better adherence than the other MPH formulas. This would guide physicians to prefer the use of Oros Methylphenidate as first line therapy. This is not true for the lower SES. Strengthening our assumptions that knowledge about medication adherence and patient characteristics are potential indicators for improving the treatment of ADHD.

Disclosure of Interest: None Declared

EPP599

Plasma microRNAs reveal a schizophrenia patient subgroup with high inflammation and severe symptoms

T. Miyano^{1*}, T. Mikkaichi¹, K. Nakamura¹, Y. Yoshigae², K. Abernathy³, Y. Ogura² and N. Kiyosawa¹

¹Translational Science Department II; ²Translational Research Laboratories, Daiichi Sankyo Co., Ltd., Tokyo, Japan and ³Clinical Research Department, Sirtsei Pharmaceuticals, Inc., North Carolina, United States

*Corresponding author.

doi: 10.1192/j.eurpsy.2025.821

Introduction: Schizophrenia is a complex and highly heterogeneous psychiatric disorder, and it is crucial to understand the different pathophysiology among patients to realize precision psychiatry.

Objectives: This study aims to evaluate the potential of plasma microRNAs (miRNAs) as clinical biomarkers to stratify schizophrenia patients based on molecular profiles and understand the heterogeneous pathophysiology.

Methods: We measured the Positive and Negative Syndrome Scale (PANSS) scores, which are severity scores of clinical symptoms of schizophrenia, along with levels of 179 plasma miRNAs in 26 schizophrenia patients experiencing acute psychosis. We applied hierarchical clustering analysis for the plasma samples on miRNA levels to explore patient subgroups. We then conducted miRNA set

enrichment analysis, literature-based text mining and manual literature survey on characteristic miRNAs for each patient subgroup to interpret the heterogeneous pathophysiology. This study has been approved by the Ethical Research Practice Committee of Daiichi Sankyo Co., Ltd.

Results: The schizophrenia patients were stratified into three subgroups based on the plasma miRNA profiles. One of these patient subgroups showed a tendency to have relatively high PANSS scores. This patient subgroup was characterized by distinctively low levels of four miRNAs. The enrichment analysis revealed an enrichment of 'Immune Response' pathways associated with these four miRNAs. Consistent with the enrichment results, literature-based text mining confirmed that these four miRNAs were frequently associated with 'inflammation' and IL-1 β , IL-6, and TNF α in the literature. We also identified literature-based experimental evidence demonstrating that these four miRNAs reduce IL-1 β , IL-6 and TNF α . These results suggest that the patient subgroup with high PANSS scores has relatively high inflammation.

Conclusions: miRNAs may potentially be clinical biomarkers that reflect both the symptoms and molecular pathology of schizophrenia, and they may be able to identify patient subgroups with relatively high inflammation. Such patient stratification based on molecular profiles is expected to be a key tool to realize precision psychiatry, e.g., prescribing right drugs for right patients.

Disclosure of Interest: T. Miyano Employee of: Daiichi Sankyo Co. Ltd., T. Mikkaichi Employee of: Daiichi Sankyo Co. Ltd., K. Nakamura Employee of: Daiichi Sankyo Co., Ltd., Y. Yoshigae Employee of: Daiichi Sankyo Co., Ltd., K. Abernathy Employee of: Sirtsei Pharmaceuticals, Inc., Y. Ogura Employee of: Daiichi Sankyo Co., Ltd., N. Kiyosawa Employee of: Daiichi Sankyo Co., Ltd.

Psychoneuroimmunology

EPP600

Hashimoto's encephalopathy in a patient with hypothyroidism and bipolar disorder: a case report

C. Bey^{1,2*}, T. Ach^{3,4}, A. Ben Abdelkarim^{3,4} and J. Mannai^{2,3}

¹Laboratory of Physiology and Pathophysiology of physical exercise; L.R.19ES09, University of Sousse, Faculty of Medicine of Sousse, 4000, Sousse; ²Psychiatry department, Ibn El Jazzar University Hospital of Kairouan, Kairouan; ³University of Sousse, Faculty of Medicine of Sousse, 4000 and ⁴Endocrinology department, Farhat Hached University Hospital of Sousse, Sousse, Tunisia

*Corresponding author.

doi: 10.1192/j.eurpsy.2025.822

Introduction: Hashimoto's encephalopathy (HE) is a rare, steroid-responsive neuropsychiatric disorder associated with Hashimoto's thyroiditis. The pathophysiology of HE remains unclear, but it is hypothesized to involve an autoimmune mechanism, distinct from thyroid hormone levels. The condition often presents with a variety of neurological and psychiatric symptoms, including cognitive decline, seizures, mood disorders, and movement abnormalities. Timely diagnosis and treatment are crucial to prevent further neurological impairment.

Objectives: This report highlights a case of HE in a patient with bipolar disorder and hypothyroidism.

Methods: A 42-year-old male patient, followed in psychiatry for bipolar disorder type I and in endocrinology for hypothyroidism secondary to Hashimoto's thyroiditis, was admitted to the

endocrinology department of Farhat Hached hospital, Sousse, due to fatigue, psychomotor retardation, and an enlarged goiter, in the context of discontinuation of his replacement therapy. Laboratory tests revealed a significantly elevated TSH level of 17.5mUI/L, indicating profound hypothyroidism. Hospitalization was therefore prompted by this endocrine decompensation to reinstitute treatment and to monitor him to prevent complications.

During the hospital stay, thyroid hormone replacement therapy was resumed. However, despite adequate treatment, the patient quickly became unstable, exhibiting vague persecutory delusions, marked irritability, changes in behavior, distractibility, attention problems, insomnia and confusion. This clinical picture raised the possibility of either a manic relapse with psychotic features, potentially triggered by the resumption of thyroid treatment, or Hashimoto's encephalopathy.

Further investigations, including brain imaging and anti-thyroid peroxidase antibodies (ATPO) measurement, were performed. Brain imaging was normal, and ATPO were elevated. Given the clinical history and elevated thyroid antibodies, the diagnosis of Hashimoto's encephalopathy was considered. The patient was started on corticosteroid therapy (prednisone), leading to a significant improvement in both psychiatric and cognitive symptoms within weeks.

Results: This case illustrates the importance of considering HE in patients with neuropsychiatric symptoms and underlying thyroid disease. The combination of elevated ATPO levels and progressive psychiatric deterioration, with normal neuroimaging, and significant improvement with immunomodulatory treatment supports the diagnosis of HE. It is a rare condition with a reported prevalence of 2.1/100000. It presents with a wide range of neurological and psychiatric symptoms and the presentation varies among patients.

Conclusions: This case underscores the need for increased awareness of HE as a differential diagnosis in patients with thyroid disorders and neuropsychiatric manifestations.

Disclosure of Interest: None Declared

EPP602

The hidden wolf – Case report of valproic acid drug-induced cerebral lupus erythematosus

S. C. Steininger^{1,*}, R. Mansky¹, A. Reichert¹, K. Brändle¹, S. Bacanovic², W. Kawohl^{3,4} and F. Xepapadakis^{3,4}

¹Old Age Psychiatry, Clénia Schloessli, Private Psychiatric Hospital and Academic Teaching Hospital of the University of Zurich and of the University of Nicosia Medical School, Oetwil am See/Zurich;

²Radiology, Männedorf Hospital, Männedorf; ³Clénia Schloessli, Private Psychiatric Hospital and Academic Teaching Hospital of the University of Zurich and of the University of Nicosia Medical School, Oetwil am See/Zurich, Switzerland and ⁴Department of Basic and Clinical Sciences at the University of Nicosia Medical School, Nicosia, Cyprus

*Corresponding author.

doi: 10.1192/j.eurpsy.2025.823

Introduction: Neuropsychiatric symptoms associated with valproic acid were already described in the 1980s. Valproic acid is known to cause valproate encephalopathy and hyperammonemia^(1,2). Rarely it can cause drug induced lupus erythematosus⁽⁵⁾. Previous studies and case reports have documented manifestations such as rash, pulmonary involvement, pleuritis and carditis due to valproate-

induced lupus erythematosus. However, little is known about other manifestations^(3,4,6). To date, there are no descriptions of cerebral drug-induced lupus erythematosus due to valproic acid.

Objectives: We report the case of a female 63-year-old patient who was initially treated in our inpatient clinic for prolonged delirium and major neurocognitive disorder following severe traumatic brain injury with bilateral traumatic intracerebral temporal bleeding eight months prior. Symptoms from the severe traumatic brain injury included aphasia, impulse control disorder, reduced frustration tolerance and depression, which were treated with valproic acid and quetiapine. The patient experienced a strongly fluctuating, progressively deteriorating neuropsychiatric syndrome that began six months before hospitalization. Additionally, a fluctuating neurological syndrome including temporary complete hemiparesis, hyperreflexia and loss of consciousness, which lasted from 30min to hours, and hallucinations was observed. The patient also developed epileptic seizures, which could not be managed by a combined antiepileptic therapy with valproic acid, brivaracetam and clobazam. Previous examinations (brain-MRI and CT, CFS, extended lab testing) excluded acute cerebrovascular insult, encephalitis and hyperammonemia.

Methods: The diagnosis of cerebral vasculitis was considered after excluding infection or cerebrovascular insult. Anti-nuclear antibodies and anti-histone Antibodies were detected in the blood sample. Anti-NMDA receptor antibodies as well as antibodies for paraneoplastic syndromes and Bickerstaff encephalitis were not detected. MRI scans over eight months showed an increase of white matter lesions periventricular and in the brain stem.

Results: Based on these results and the medical history, we considered a drug-induced vascular lupus erythematosus due to valproic acid. We initiated immunosuppressive therapy with high-dose prednisone while tapering valproate acid. Within days, the neurological symptoms declined and epileptic seizures ceased.

Image 1:

