Objectives Reversion of this nucleotide to the ancestral type, –220A, co-occurs with severe deficit in higher brain cognitive functions.
Aims In the current study, we compare the pattern of protein binding between –220C and –220A.
Methods Antibodies reactive against transcription factors CREB, USF, and c-Myc were used to identify the specific proteins involved in complexes with DNA using electrophoretic mobility shift assay (EMSA).
Results Significant increase was observed in the overall protein complexes binding to the –220 C allele vs. –220A. The transcription factors, CREB, USF, and c-Myc, were differentially bound to –220C, represented by supershifts.
Conclusions We propose that differential binding of CREB, USF, and c-Myc to CALR nucleotide –220C may be linked with the evolution of higher brain functions in human.
Disclosure of interest The author has not supplied his/her declaration of competing interest.

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EV712

Neurofarmagen® testing and drug side effects: An evaluation of its use among a real-world case series

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Introduction Various pharmacokinetic and pharmacodynamics features have proven to be involved in the development of drug-induced side effects in psychiatry and thus pharmacogenetic profiling should be considered during drug selection to avoid the onset of side effects.
Aim To explore the usefulness of Neurofarmagen® testing in clinical practice by evaluating whether the genetic profile given by the tool could properly explain the onset of side effects during antipsychotic treatment.
Methods The pharmacogenetic profile of ten patients having a history of side effect appeared during to specific a psychopharmacologic treatment was determined by Neurofarmagen® testing tool. The relationship between genetic profile and side effects was evaluated and classified.
Results Sixty percent of the sample showed a genomic alteration related to a increased likelihood of having any side effects, one half of which presented pharmacokinetic alteration (slow or intermediate phenotype for the implicated cytochrome) whereas the other half had a pharmacodynamic gene variant (related to dopamine or serotonin pathway).
Conclusion the Neurofarmagen® testing tool may be useful in the clinical practice in order to avoid drug-induced side effects.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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EV713

Psychiatric manifestations of Niemann-Pick type C disease – two case reports

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Introduction Niemann-Pick type C disease (NPCD) is a rare metabolic illness, with autosomal recessive inheritance. NPCD has a heterogeneous presentation, with non-specific psychiatric symptoms, mostly affective and psychotic features and also cognitive deficits.
Objectives and methods We present the case reports of two brothers with an adolescent-adult onset and discuss the evolution of their neuropsychiatric manifestations.
Results The patients have now 35 and 31 years old and the youngest was the first to develop clinical manifestations of the disease. From 16 years old, he developed unspecified neurological impairment with gait imbalance. In the next years, the neurologic manifestations exacerbated, with dysarthria, ataxic gait, and his academic performance declined. With 24 years old, he presented acute psychosis, with unstructured delusion and auditory hallucinations. The acute psychotic symptomatology remitted with olanzapine but he revealed social withdrawal, apathy and progressive cognitive decline that persist until now. His brother, whose diagnosis was made in the course of the family genetic study, developed the first signs of the NPCD with 19 years old. He presented neuropsychiatric compromise, with impaired learning, social isolation and insomnia. They are receiving specific treatment with miglustat and symptomatic treatment for the psychiatric manifestations.
Conclusions NPCD is a rare metabolic disease, with neuropsychiatric compromise. No general psychopathological profile has been associated to NPCD. Sometimes psychiatric symptoms dominate the initial clinical presentation, with neuro-visceral signs appearing later. An atypical psychiatric symptomatology should be extensively investigated in order to exclude organic causes, including metabolic diseases like NPCD.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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EV714

Psychiatric disturbances in a patient with melas syndrome: A case report

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Introduction Mitochondrial disorders of energetic metabolism (MD) represent a heterogeneous group of diseases manifesting at any age and its one of a number of mitochondria syndromes that share the common characteristics of encephalopathy and myopathy. The clinical expression of MELAS (Mitochondrial Myopathy, Encephalopathy, Lactic Acidosis and Stroke-like episodes) is highly variable and ppsychiatric symptoms are rarely reported in literature even if are more common in MELAS syndrome than in the general population.
Objective The first aim of the study is describing the clinically observed primary psychiatric symptoms in a patient affected by MELAS syndrome admitted to the Psychiatric ward. The second aim is to go back over the diagnostic process, which led, from the uncommon psychiatric symptoms and signs to the final genetic diagnosis of MD.
Methods and results We report the case of a 44-year-old male with MELAS in whom psychiatric symptoms preceded the establishment of the clinical diagnosis for several months. Diagnosis was initially based on the neuroimaging and metabolic findings and subsequently confirmed with genetic analysis.
Conclusions In case of aggressive and paranoid behaviour with delusions of persecution and disorganised behaviour mmitochon-