some neuropsychiatric states. There is evidence that patients with schizophrenia have altered corticocerebellar connectivity.
Objectives To evidence a case with early onset psychosis accompanied with brain structural abnormalities.
Method Case description.
Results The patient is 15 years old girl with an acute psychotic episode. For more than two months she had demonstrated odd behavior, getting around all the time purposelessly, abandoned school etc. She presented with disorders of perceptions, disorganized speech, insomnia and fluctuations in her mood and behavior. In her brain, MRI was found vermian atrophy, and CT was found hypocampal glyosis and dilatation of temporal corn.
Conclusions Although the structural mapping studies have been equivocal, the weight of evidence supports extending the study of cerebellar activity in schizophrenia. For example, the finding that unaffected first-degree relatives of probands with schizophrenia have reduced cerebellar volumes, along with the observation of reduced cerebellar volumes in neurolepticnaïve patients with schizophrenia, suggests that cerebellar atrophy may be a hereditary trait rather than a psychotropic associated epiphenomenon.
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EV1215

Traumatic brain injury as psychosis development factor

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Introduction The pathophysiology of psychosis is not fully discovered yet. However, during the last years many different risk factors are shown to prove to have a strong influence within the development of this pathology. Traumatic brain injury (TBI) is one of them.
Objectives Show TBI as a psychosis development risk factor.
Methods Case report. A clinical vignette is presented followed by the results obtained in a bibliographic review.
Results A young 19-year old immigrant man, who lives with his parents in a social exclusion situation is brought to the hospital after having been observed making estrange religious rituals within a local river. During the anamnese he declares that God is "getting in touch with him" while he shows to be changed, with suspicion about being pursued. He also reveals to have suffered a mild-severe TBI with 8 years, having right ear audition problems since then. During the hospitalization some medical test were done, such as MRI, showing the lack of the inner right ear, as well as white matter abnormalities in his right hemisphere, which could be consequence of the TBI. Those findings make us think that this pathology might have been influenced, within other factors, by the traumatic brain injury.
Conclusions This bibliographic review shows that traumatic brain injury may increase the risk of developing psychosis up to 65% from healthy controls, with a medium gap of 3.3 years between the TBI and the appearance of psychotic pathology.
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EV1216

Psychosis and creativity. Genetic and structural relation between them

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Introduction Madness and creativity are thought to be related from ancient ages. Nowadays, thanks to new scientific developments and researches we are able to identify common genetic and brain patterns between creativity and psychosis.
Objectives Taking the inspiration of a psychotic patient with some shocking drawings, we want to get deep into the actual knowledge about the relation between creativity and psychosis.
Methods Case report and bibliographic review.
Results A 19-year-old man was brought to the hospital after having been found making strange rituals in the public way. In the anamnese he showed to have experienced mystic delusions and hallucinations. He made some particularly creative drawings.
We made a review which showed that this patients may have a diminished latent inhibition, which could make them experiencing usual live irrelevant stimuli as something very exciting and creative at the same time. Genome wide association studies show also that people having creative jobs and psychotic patients share some genes, which could be linked to this abnormal latent inhibition.
Conclusions Latent inhibition abnormalities could be related with psychosis and creativity. There are differences within the course of people having this oversensibility, which could be explained due to the presence of protective and risk factors.
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EV1217

A fine line between schizophrenia and Hashimoto encephalopathy

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Introduction Hashimoto encephalopathy (HE) is an uncommon syndrome associated with Hashimoto thyroiditis. The relationship between these entities is unclear. Even being rare, it appears to be underrecognized.
Objectives Report a case of an atypical presentation of psychosis in a patient with elevated serum levels of antithyroid antibodies and review the literature about similar situations.
Methods Access clinical process, research PubMed, using the mesh terms "Hashimoto encephalopathy" and "psychosis".
Results A 21-year-old Portuguese female was conducted by authorities to our emergency department after she called for help and was spotted walking barefoot on the streets. Throughout clinical course she presented persecutory ideas, thought blocks, auditory hallucinations, soliloquies, perplexity, total insomnia, bizarre behaviors like coprophagia, trichotillomania and self-injured burns. After some tests, it was found that the patient had high serum levels of antithyroid peroxidase antibody (TPO) and antithyroglobulin antibody (TGO) and reduced folic acid, without other changes. Trials with corticosteroids showed clinical improvement for short periods, as with antipsychotics. No consistent remission was achieved with either approaches.
Conclusion HE is an uncommon syndrome presenting with high titers of antithyroid antibodies that may preconize an acute state of atypical psychosis. Usually, it responds to corticosteroids and so, has a generally good prognosis when treated accordingly. Evidence

suggests that HE is an autoimmune disorder instead of thyroid disease.

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EV1218

Brain metabolic abnormalities in schizophrenia patients

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Introduction Main schizophrenia symptoms result from abnormalities in brain function, such as hypofrontality and structural deficits on the prefrontal-thalamic-cerebellar circuit, as shown in brain imaging studies in first-episode SCZ patients. Whether metabolic alterations may be underlying these events is being studied thoroughly.

Objectives/aims To assess brain metabolic disturbances in first episode and/or drug-naïve SCZ patients.

Methods We conducted a literature review through Pubmed search for MeSH: schizophrenia, metabolism, glucose, insulin, brain. Controlled studies on first episode and/or drug-naïve SCZ patients were included.

Results Lower metabolic activity in the frontal regions of the brain is associated to an increase in norepinephrine transmission and decrease in dopaminergic transmission with reduced dopamine efflux in the frontal cortex. This seems to lead to cellular changes resulting in resulting lower blood flow and glucose demand. Molecular analysis of postmortem SCZ patients' brains has indicated alterations in glucose metabolism and insulin signalling pathways, showing evidence for prefrontal cortex decreased expression of glucose metabolism, namely glycolytic enzymes such as glyceraldehyde 3-phosphate dehydrogenase, hexokinase, phosphoglycerate mutase, enolase and pyruvate kinase and decreased levels and phosphorylation of the insulin receptor and insulin signalling proteins AKT1 and GSK3β. Significantly elevated glucose concentrations in cerebrospinal fluid were observed in SCZ patients, but with no serum levels differences. A SCZ brain specific increased glucose could be explained by preferential utilization of lactate, predominantly produced by astrocytes, over glucose as an energy substrate.

Conclusions Abnormalities in brain glucose metabolism and insulin signalling seem to appear in early stages of SCZ, suggesting a role in SCZ onset and pathophysiology.

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EV1219

Peripheric metabolic abnormalities in schizophrenia patients

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Introduction Schizophrenia (SCZ) is frequently associated with metabolic symptoms including dyslipidaemia, hyperinsulinemia, type 2 diabetes and obesity. In fact, SCZ patients have been reported to present higher prevalence of these conditions than general population, commonly associated to second generation antipsychotic therapy. Recent studies, however, have demonstrated that peripheral metabolic disturbances can appear at disease onset or drug-naïve patients.

Objectives/aims To assess metabolic disturbances in first episode and/or drug-naïve SCZ patients.

Methods We conducted a literature review through Pubmed search for MeSH: schizophrenia, metabolism, glucose, insulin. Controlled studies on first episode and/or drug-naïve SCZ patients were included.

Results Several studies showed no change in SCZ patients' fasting blood glucose, while others found increased glucose levels and impaired glucose tolerance in SCZ patients compared to healthy controls in several recent studies. Hyperinsulinemia and insulin resistance have also been identified in antipsychotic-naïve SCZ patients and it has been suggested that early onset patients are more likely to present insulin resistance. In addition, there's evidence of increased circulating levels of chromogranin A, pancreatic polypeptide, prolactin, cortisol, progesterone, thus emphasising that multiple components of the hypothalamic-pituitary-adrenal-gonadal axis may be affected in SCZ. These elevations were associated to normal glycaemia suggesting there may be insulin intolerance during early stages of SCZ, requiring an increased secretion from pancreatic Bcells to maintain normal glucose levels.

Conclusions Recent studies of first onset and/or drug-free schizophrenia patients have shown impaired fasting glucose tolerance, hyperinsulinemia and insulin intolerance, suggesting that metabolic abnormalities may play a role in SCZ onset and pathophysiology.

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EV1220

Systemic review: High dose olanzapine treatment for treatment resistant schizophrenia

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Objectives Schizophrenia is a major mental illness with a progressive course. Thirty percent of cases of patients with schizophrenia do not respond to adequate trials of at least 2 different groups of antipsychotics,are currently classified as having treatment resistant schizophrenia (TRS). Clozapine remains the gold standard, treatment of choice for TRS. However, clozapine does not come without its own challenges. Its risk profile, particularly agranulocytosis, reported in 1% of cases, has led to the necessity of weekly blood counts within the first 18 weeks of treatment and subsequently every month with slow dose titration. Clinically, sedation, weight gain and hypersalivation may further hamper the compliance of patients. Non-compliance has been reported to cause rebound psychosis. Recent studies have raised questions as to which antipsychotic is most efficacious for TRS. Thus, we conducted a systematic review of high dose olanzapine treatment for people with TRS.

Method A systematic review of prospective studies found through search of PubMed, Scopus and hand-searched key papers which included randomized controlled trials and open-label studies which looked at high dose of olanzapine treatment response for TRS.