11th International Congress on Psychopharmacology & 7th International Symposium on Child and Adolescent Psychopharmacology Case Reports Addendum

[Abstract:0232] [Schizophrenia and other psychotic disorders]

Early adolescent-onset schizophrenia: a case of report

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ABSTRACT

It is defined as childhood onset or early onset schizophrenia when it starts at the age of 13. In this presentation, a male patient who was diagnosed with schizophrenia at age 10 was discussed.

Case presentation: A 10-year-old male patient presented to our outpatient clinic with complaints of nervousness, aggression, introversion, and self-talk, which started about 9 months ago. The parents reported observed disorganized behaviour in the form of self-talk and laughter, nightly fears, repetitive conversations and gestures, trusting people, and wanting to kill them, putting his hand in his mouth and butt, going under the table all the time in class. The patient had aphonia, which started three days before he arrived in our outpatient clinic. No organic pathology was found in the neurological examination and his brain MRI exam was unremarkable. No psychiatric history in the family were reported. Parents reported that he started walking in 15 months, and he started speaking when he was 5–6 years old. In his psychiatric examination, distraction of the patient during the interview, incoherence, the blocks of thought, self-talk and laughter, and disorganized behaviour were remarkable. Patient was diagnosed with schizophrenia and olanzapine treatment was started and he was followed regularly. Our case is important in terms of starting at an early age, quick onset of symptoms, and poor functionality. Due to the facts that the age of our patient was young and his parents were not cooperating in the treatment, these affected the clinical prognosis negatively. Treatment guidelines for early-onset schizophrenia are based on adult literature and clinical experience, and therefore further studies are needed in the child age group for effective treatment.

KEYWORDS

Adolescent; disorganized behaviour; schizophrenia

[Abstract:0434] [OCD]

Psychiatric manifestations of corpus callosum agenesis: an adolescent case with obsessive compulsive disorder and psychotic symptoms

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ABSTRACT

Corpus callosum connects the left side of the brain to the right side. Agenesis of corpus callosum causes many physical, cognitive, developmental, social, and behavioural disorders such as intellectual disability, seizures, feeding problems, developmental delay, cognitive problems, learning difficulty, mental retardation, hyperactivity, and psychotic disorders. Abnormalities in the corpus callosum have also been implicated in the pathogenesis of obsessive-compulsive disorder. Hereby, we present an adolescent with corpus callosum agenesis and obsessive compulsive disorder (OCD) with psychotic symptoms.

Case presentation: A 16-year-old boy was presented to our clinic with the self-talking, withdrawing into himself, spending long times in bathroom, and swinging when he sits or stands up. He always wants to be alone. He is reported to spend more than 1 h in bathroom. Normally, he does not like having a bath. He has a bath once in a week. When he is in the bathroom, he prefers washing his hair in sink. He never uses shower or tub. He usually leaves his hair with shampoo. He also spends long times in toilet. He puts his hands under the tap more than 5 min but he does not wash his hands.

KEYWORDS

Corpus callosum; obsessive compulsive disorder; psychotic symptoms

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He combs his hair for hours and spends most of the time in front of the mirror. He also has ritualistic eating behaviours. He eats six or seven millets in a day.

He was born via a normal vaginal delivery following an eventful pregnancy. His developmental history was reported to be normal. He walked and said his first word at the age of 12 months. His mother described him as a silent baby, rarely crying and easily comforted. Mother reported that the patient did not cry during getting injections or while being circumcision. He learnt reading and writing at elementary school first grade. Wechsler Intelligence Scale for Children-Revised score indicates that he has normal IQ. After his psychiatric evaluation, the patient was diagnosed with OCD and psychotic disorder not otherwise specified. His Yale-Brown Obsessive Compulsive Scale score was correlated with OCD. For the investigation of a possible organic aetiology, he was referred to paediatric neurology. His neurologic assessment included magnetic resonance imaging and revealed that he had agenesis of the posterior corpus callosum.

Patient was prescribed sertraline 25 mg/day and risperidone 0.5 mg/day. The dose of both medications was increased gradually within 2 months. His current medications include Sertraline 100 mg per day for his obsessive compulsive symptoms and risperidone 2 mg per day for swinging, self-talking, and augmentation. His OCD, swinging, and self-talking symptoms were reported to improve markedly. Previous case reports linked CC agenesis mainly with psychotic disorder. Our case shows that there may be a possible link between OCD and corpus callosal dysfunction. Patients with OCD possibly have microstructural abnormalities in the CC rostrum itself and fibre integrity abnormalities in the orbital prefrontal region that contains fibres extending into the rostrum. The orbital prefrontal region is involved in the pathophysiology of OCD. Clinicians should be aware of OCD and psychotic symptoms in adolescents with CC agenesis.

Schizophrenia and other psychotic disorders

Body dysmorphic disorders and prodromal psychosis: a case report

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ABSTRACT

Body dysmorphic disorder is a psychiatric disorder in an obsessive-compulsive spectrum disorder with a mean age of 16–18 years, characterized by extreme intolerance of the person with a non-reality defect [1,2]. In many psychiatric disorders, such as schizophrenia, there are extreme efforts related to the body [3,4]. The case of prodromal psychosis presenting with symptoms of body dysmorphic disorder is discussed with the literature.

Case presentation: The patient was brought to the psychiatry out-patient clinic by the family because of the thought that she was ugly. According to the anamnesis obtained, the age of 16 years, course success is good, the face of the region is ugly, these thoughts began 2 years ago, the process of preparing for the exam, the complaints have increased for the last 5 months, therefore, he could not look at the mirrors and people’s face, he didn’t go to his friends at school, he did not care for himself, he presented to a child psychiatry at the external centre, he started sertralin 50 mg/day and risperidone 1 mg/day, he was using the drugs for 5 months, his complaints did not change. Patient’s medical history was unremarkable. In his family history, it was learned that the patient’s uncle had psychotic disorder and received treatment. Her mental state examination showed her age and her dress was appropriate for the sociocultural level. He was conscious; his orientation was complete; his affect was inappropriate; his mood was depressive. Thought content is at the level of delusion. His judgment was impaired, he had insight and he had passive suicidal ideation. With preliminary diagnoses of body dysmorphic disorder and prodromal psychosis, the patient was treated with risperidone 2 mg/day, clomipramine 25 mg/day, and lorazepam 1 mg/day. The patient was called for follow-up and the patient’s complaints were regressed. In this case, prodromal psychosis, which starts with physical symptoms, was considered. In children and adolescents, psychotic disorder is rare (0.1%) and our case is important to start with body symptoms. Patients with body dysmorphic disorders have an over-valued view that their bodies appear to be defective. In the early stages of schizophrenia, some patients have somatic delusions related to the appearance of the body, and the disease may begin with symptoms of dysmorphic disorder in the body.

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Aripiprazole induced muscular and skeletal pains: a case report

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Aripiprazole is a third-generation antipsychotic with a dopamine receptor-binding profile distinct from other second-generation antipsychotics. The safety of aripiprazole in paediatric patients is influenced by age- and drug-specific features, but clinical information on specific risk of aripiprazole medicines in this population is limited.

Case presentation: A 15-year-old female patient with a diagnosis of other dissociative disorder and depression whose musculoskeletal pain developed after the onset of aripiprazole was presented. Aripiprazole was started at 5 mg/day. In parallel with the use of aripiprazole, the patient began to have muscular and skeletal pain which was more intense in the proximal parts of her arms and legs. The patient had difficulty in moving her limbs due to pain. The drug was discontinued. Two weeks after the discontinuation of medication, her pains completely disappeared. Aripiprazole was restarted at 5 mg/day and musculoskeletal pain reappeared similarly. Musculoskeletal pain emerged with the use of the drug and resolved with discontinuation of the drug. Muscular and skeletal pain associated with aripiprazole treatment is a rare side effect. In this case report, musculoskeletal pain occurs dramatically with the onset of aripiprazole and improves after discontinuation of the drug. In order to provide essential information that will help to prevent harm to children, pharmacovigilance reports should include data on the patient characteristics and circumstances involved in the reported adverse drug reaction. More controlled studies on aripiprazole should be performed in this area.

Symposium Abstracts Addendum

Signs and symptoms of ASD and destructive behaviours: novel treatment approaches

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Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by impairments in three developmental areas: social interaction, communication, and restricted and repetitive behaviours. The epidemiological research shows that about 1–1.5 per 100 children have ASD. Although the underlying etiology of ASD is unknown, it is suggested that complex genetic factors are largely responsible for ASD. To date, no medication has been proven to be effective in the treatment of core symptoms of ASD. The main target of available psychotropic medications is emotional and behavioural symptoms and repetitive behaviours. Although no medication other than risperidone and aripiprazole have been approved by the US Food and Drug Administration (FDA) in ASD currently, there is an increasing research interest on the candidate treatment options. Recent studies of oxytocin, arginine-vasopressin, glutamatergic, and gamma-aminobutyric acidergic (GABAergic) agents have reported mixed results. Among these candidate agents, oxytocin and arginine-vasopressin may deserve special attention. Although newer studies are needed in young children with ASD, oxytocin and arginine-vasopressin may be considered as promising agents at least for a subpopulation of individuals with ASD who have marked deficits on eye contact, certain genetic receptor defects, and lower plasma levels of these neurohormones. One of the new research directions in ASD is the gut microbiota, number of studies have
confirmed alterations in gut microbiota composition in children with ASD. However, the available data does not indicate a characteristic and unique gut microbiota profile in ASD. Probiotics may be defined as living non-pathogenic microorganisms that can provide health benefits in a variety of clinical conditions regarding the potential role of probiotics in ASD treatment, the research is limited and available results are conflicting. One of the main drawbacks of ASD medication trials is the extremely high placebo effect on parental ratings. In addition, most of the present studies recruited patients on a wide age range and different clinical features using varying doses of the candidate agents. Taken together, although some promising results have been published, the evidence is still limited for pharmacotherapy of core symptoms in ASD. New randomized placebo-controlled studies with large sample size are needed to reach conclusive results.

**Neurocognitive symptoms in children and adolescents with anxiety disorders**

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**ABSTRACT**

Studies show a mutual relationship between anxiety and cognitive performance. It is thought that the cognitive structuring that started in early development periods and shaped during the transition to adulthood paved the way for the development of anxiety disorders. Attention, learning, and memory-related problems predispose to anxiety disorders and determine treatment response. Implicit memory and episodic memory capacity are effective in the development and chronicity of fear and anxiety. Cognitive processes and specific somatic symptoms occur with the interaction of cortical and subcortical networks. Amygdala, an important brain area related to emotions and memory, shows increased activity. Autonomic and neuroendocrine responses in anxiety disorders lead to impairment of neurocognitive functions; high cortisol level affects the hippocampus and memory performance negatively. Loss of visual–spatial memory areas in verbal memory, visual memory, and visual learning, verbal learning and remembering were reported in panic disorders. According to other anxiety disorders, visual–spatial problems were more common in social anxiety disorder. And generalized anxiety disorder (GAD) patients were shown to have more neurocognitive deficiencies in planning ability/efficiency, cognitive flexibility, and visual processing. Short-term and long-term memory and cognitive flexibility are impaired in GAD. In a study conducted on people who were followed from birth to thirties, the relationship between cognitive performance and lifelong GAD diagnosis risk was investigated. After socioeconomic status and parental mental health control; a 15-point (1 SD) advantage in childhood cognitive performance was associated with a 50% reduction in lifelong GAD risk. The risk of GAD in childhood and adolescence was significantly associated with 89% and 57%, respectively [1]. And likely, children who had earlier maternal stimulation and exhibited a less anxious behaviour pattern in 1–36 months had a better cognitive performance at the age of 15 years [2]. A normal social cognition as a cognitive domain seems to be necessary for a healthy social-emotional development. The low level of social cognition in early childhood may result in less adaptation to social situations, more negative social experiences (neglect, rejection), less social self-confidence, and possibly more avoidance and social anxiety. It can be seen that children and adolescents with high social anxiety level or social anxiety disorder can hardly recognize emotions, which is a basic aspect of social cognition. In a study of 48-month-old children, socially anxious children showed lower social cognition performance and more negative shy expressions [3]. As a result, memory-related problems seem to be a common feature of anxiety disorders. Implicit and episodic memory bias may be associated with anxiety disorders. Although comorbidities are common, the relationship between these diagnoses and cognitive functions has not been studied sufficiently. Disorders of these neural networks may be seen because chronic fear and anxiety occupy and consume neurocognitive sources such as attention and memory. In anxiety disorders, the orientation of attention to the object or condition may affect performance in neurocognitive tasks. As with executive functions, it is possible to talk about a mutual relationship between social cognition and anxiety and fear from early childhood.

**KEYWORDS**

Anxiety; neurocognitive symptoms; cognitive performance; social cognition; children; adolescent

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Neurocognitive symptoms in children and adolescents with depressive disorders

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ABSTRACT
There may be important differences between early-late onset depression with early onset depression being more severe and with higher levels of recurrence. The long-term consequences of early-onset depressive symptoms on the aforementioned outcomes underline the need for a thorough examination of the neuropsychological correlates of depressive symptoms among adolescents. Early research has suggested that depression is characterized by cognitive impairment. Adults with depression suffer from significant cognitive deficits, but the current neuropsychological data on the cognitive performance of children and adolescents with major depressive disorder suggest mixed results. With various brain regions undergoing different patterns of maturation, neural effects of stress-related conditions such as depression depend on the developmental level at which the stress and depression started [1]. So, stress and stress-related depression may have a greater effect on cognitive and emotional function in childhood and adolescence as the brain experiences important alterations compared to adulthood. Based on these facts of natural brain development, it can be concluded that a pathological brain condition like depression impact on neuropsychological function during development. Depression in adolescents is usually associated with cognitive impairment [2]. It is still unclear whether adolescents with depressive symptomology are prone to global or domain-specific cognitive deficits. Recent review and meta-analysis suggest that children with depression have impairment in inhibition, verbal fluency, sustained attention, planning, and verbal memory [3]. In this session, we review the cognitive impairments and possible interventions about depression in children and adolescence with current evidences.

KEYWORDS
Adolescent depression; cognitive impairment; inhibition; neurocognition

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Exercise and cognitive functions
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ABSTRACT
The term “physical activity (PA)” should not be confused with “exercise,” which is a subcategory of PA that is planned, structured, repetitive, and aims to improve or maintain one or more components of physical fitness. Beyond exercise, any other PA that is done during leisure time, for transport to get to and from places, or as part of a person’s work, has a health benefit. Further, both moderate- and vigorous-intensity PA improve health. Should do at least 150 min of moderate-intensity PA throughout the week, or do at least 75 min of vigorous-intensity PA throughout the week, or an equivalent combination of moderate- and vigorous-intensity activity. In animal studies, the structural changes analysed concern the cellular (neurogenesis, gliogenesis, synaptogenesis, and angiogenesis) and molecular (alteration in neurotransmission systems and increasing in some neurotrophic factors) level,

KEYWORDS
Exercise; neurocognitive symptoms; cognitive performance; physical activity; children; adolescent
while the functional activity has been measured using the levels of performance in behavioural tasks, such as spatial tasks that allow to analyse the different facets of spatial cognitive functions. In humans, indicators of structural changes correspond, for example, to brain volumes, measures of white matter integrity, or modulation in neurotrophins levels (by correlation with trophic factors plasma levels). Such metrics can be correlated to cognitive performances, defining the functional neural efficiency. To this regard, it should be emphasized that any morphological change results in a modification of the functional properties of a neural circuit and vice versa any change in neuronal efficiency and functionality is based on morphological modifications. A large amount of evidence has demonstrated the power of exercise to support cognitive function, the effects of which can last for considerable time. An emerging line of scientific evidence indicates that the effects of exercise are longer lasting than previously thought up to the point to affect future generations. The action of exercise on epigenetic regulation of gene expression seem central to building an “epigenetic memory” to influence long-term brain function and behaviour. Recent research has shown that PA interventions that jointly involve physical effort and emotional and social engagement challenge core cognitive functions, as well as cognitive life skills, such as goal setting, problem-solving, and self-regulation, a life skill relying on the efficiency of a core executive function such as inhibition. Recent research has summarized evidence regarding the effectiveness of PA interventions on children’s and adolescents’ non-executive cognitive functions, core executive functions, and metacognition. Late systematic review and meta-analysis support that PA interventions are useful strategies to foster the development of children’s cognition (non-executive cognitive functions, core executive functions, and higher-level executive functions). Moreover, it has been shown that curricular exercise and programmes aimed at increasing the time dedicated to PA are more likely to produce an effect on children’s and adolescents’ cognition. This information should be jointly considered with previous research that has highlighted the positive effects of PA on children’s health. It also might be relevant for policy and decision-makers in order to design new strategies not only for promoting children’s cognitive development, and as a consequence better academic performance, but also for improving mental health (reducing anxiety/depressive disorders, improving self-esteem) and preventing cardiovascular disease. Including this evidence into good practice guidelines in education and public health might lead to counteracting the current school trend of PA reduction. For this purpose, qualified professionals are essential in designing PA strategies tailored to enhance children’s and adolescents’ executive functions, and therefore motor and cognitive development promotion.

The effects of chronic diseases on neurocognitive functions in children
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ABSTRACT
Over the last century, the primary burden of disease in children and adolescents has shifted from infectious diseases to chronic diseases. It is thought that the frequency of chronic diseases is increasing in the world and it affects 13–27% of the children. Chronic diseases affect many aspects of children’s lives and their results extend to adulthood. The most important of these results is the deterioration of brain development and neurocognitive functions. Chronic diseases may affect brain development by mechanisms such as lesions that cause tissue damage to the brain (e.g. brain tumours and stroke), impaired oxygen conduction to the brain (e.g. hypoxia in sickle cell anemia), chronic inflammation, and disruption of glucose delivery to the brain (e.g. diabetes). The side effects of cranial radiotherapy and drugs (e.g. insulin, antiepileptics) are also among the reasons. The development of white matter structures is mostly in childhood and adolescence. Therefore, there is evidence that children with chronic diseases may have a decrease in white matter. In addition, the stress experienced in childhood was associated with smaller prefrontal cortex volume and weaker executive function.

There are studies on the neurocognitive effects of various chronic diseases, such as cancer, epilepsy, and diabetes. Children with cancer are at risk of deterioration in processing speed, attention, working memory, and executive function areas. This risk is even greater in radiotherapy and central nervous system tumours. In children with epilepsy, working memory and processing speed often deteriorate and may negatively affect learning and problem-solving. It has been shown that epilepsy may impair mental development and lead to permanent behaviour problems in children aged 10 years and especially with recurrent seizures. Children with type 1 diabetes mellitus have fewer gray and white matter volumes than healthy controls. Changes in blood glucose levels are associated with these volume differences. It is assumed that reduced white matter leads to lower IQ and academic success, leading to deficiencies in executive functions. It was observed that children with chronic
kidney disease experienced more cognitive problems compared to their sibling controls and healthy controls. Poorer kidney function and longer duration of renal failure are associated with increased intellectual, academic, and memory problems. The effects of congenital heart diseases on cognitive functioning in children and adolescents have been shown in a meta-analysis study. Studies with children with sickle cell anemia have shown that the magnitude of cognitive impairment is associated with the severity of brain injury. However, there are cognitive problems in patients with sickle cell anemia without evidence of cerebrovascular accident. These effects may in part be explained by cerebral oxygen deprivation and inflammatory cytokine levels. In conclusion, there are findings about the neurocognitive effects of various chronic diseases in children and adolescents. These neurocognitive effects may adversely affect life and should be considered in disease management.

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Symposium Abstract Addendum

Presented at the 10th International Congress on Psychopharmacology & 6th International Symposium on Child and Adolescent Psychopharmacology

GDNF Gene rs2910702, rs3096140, and rs3812047 polymorphisms in early and late-onset patients with obsessive-compulsive disorder

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ABSTRACT

OBJECTIVES: Obsessive-compulsive disorder (OCD) is psychiatric disorder in which people have unwanted, intrusive, and repeated thoughts, feelings, ideas, sensations (obsessions), or behaviours that make them feel driven to do something (compulsions). The neurobiology of OCD is evidenced by a strong demonstration of impairments in the serotonergic and dopaminergic system. Recently, GDNF (glial cell derived neurotrophic factor) gene polymorphisms have been emphasized in psychiatric disorders and treatment strategies that have been tried to be developed in this regard. In the literature, there are several studies examining the relationship between GDNF gene polymorphisms and psychiatric disorders excluding OCD. Therefore, in this present study, we aimed to examine the symptomatology and GDNF gene polymorphisms in early and late-onset OCD patients.

METHODS: For this purpose, patients diagnosed with OCD according to DSM-5 diagnostic criteria in structured clinical interviews were grouped as early and late-onset based on their age of initiation. Sociodemographic information were collected, and the Yale-Brown Obsessive-Compulsion Scale (Y-BOCS), the Hamilton Depression Rating Scale (HDRS), the Hamilton Anxiety Rating Scale (HARS), the DSM-III-R-Personality Inventory (SCID-II), and Structured Clinical Interview (SCID-I) for DSM-IV Axis I Disorders were given to all subjects. The DNAs were isolated from blood samples collected from all 98 (69 OCD and 29 healthy) subjects in ethylenediaminetetraacetic acid tubes, and rs2910702, rs3096140, and rs3812047 polymorphisms in GDNF gene were examined by Quantitative Real-Time PCR method combined with melting curve analysis.

RESULTS: No significant correlations were detected between GDNF rs2910702, rs3096140, rs3812047 polymorphisms and OCD subjects (P = 0.115, P = 0.511, and P = 0.436, respectively).

CONCLUSIONS: Failure to detect correlations between OCD and GDNF gene polymorphisms might be due to the variable expression pattern of the GDNF gene in different tissues and pathologies. Therefore, future studies were required by including a larger group of patients and examining a wider range of tissues for the expression pattern of GDNF.

KEYWORDS
Obsessive-compulsive disorder; GDNF; single nucleotide polymorphism; gene