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Childhood and Adolescent Schizophrenia and Other Early-Onset Psychoses

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1. Introduction

Children and adolescents can have a variety of psychotic disorders from acute and transitory psychoses to chronic ones, such as schizophrenia. Their psychotic symptomatology, however, can also be the consequence of an organic cause or drug-inducted. Comparing to adolescence and adulthood, psychotic disorders in childhood seem to occur more rarely, which can be explained by lower incidence and diagnostic problems - hallucinations and delusions in children are quite difficult to recognise (Turk et al., 2007).

While schizophrenia is a very rare disorder in childhood, it becomes increasingly common during adolescence. It is actually the most frequent psychotic disorder in the age group of more than 12 years. This is the reason why this chapter will discuss schizophrenia in greater detail, highlighting possible difficulties a therapist can face during a diagnostic process. Some of these diagnostic difficulties will also be emphasized by the case report describing a metabolic disease which can mimic schizophrenia symptoms.

Even though childhood and adolescent schizophrenia lie on a continuum with schizophrenia in adults and the same diagnostic criteria are valid no matter the age group, there are special difficulties in applying the adult criteria to children. Symptoms at an early stage are less specific and in addition show remarkable overlap with a number of developmental disorders. So, diagnosing schizophrenia in children or adolescents could be a much harder task than diagnosing adults.

In childhood and adolescence, schizophrenia most often has a slow and insidious onset with many precursors in the shape of developmental, cognitive and emotional symptoms or retardation. None of these precursors is schizophrenia-specific, therefore it is striking to see the number of children and adolescents with schizophrenia that seem to be multidimensionally impaired.

The fact is that schizophrenic psychoses are a very heterogeneous group of disorders. Consequently, it is difficult to find a unique etiology. Studies in recent years have focused on the neurobiological and neurodevelopmental approaches, including genetics, and have produced interesting results which have not been yet integrated into conclusive and convincing theory of schizophrenia (Remschmidt, 2001).

The prognosis of early-onset schizophrenia is worse than schizophrenia that starts in adulthood, hence early recognition and treatment is crucial. Children and adolescents with any psychotic disorder, especially with schizophrenia, thus require a broad,

multidimensional treatment approach, very often including hospital treatment, with the focus on the socialization and rehabilitation. Pharmacotherapy does not differ a lot from adult patients.
As it will be seen in a case report and afterwards discussion the importance of excluding possible organic causes, whose clinical manifestation can mimic a psychotic disorder, can not be overestimated. Thus careful clinical, laboratory and imaging diagnostic evaluation must always be performed.

2. History of psychotic disorders in childhood and adolescence

The term psychosis was used very broadly in children and adolescents. It also covered the children with behavioural and autistic spectrum symptoms. The clinical distinction between autism and other psychotic disorders was first established by Kolvin in 1971 (Kolvin, 1971), but there is still confusion and misdiagnosis between the two disorders. The presence of fleeting hallucinations and delusions in nonpsychotic children can also be often misleading in a variety of other diagnoses, such as acute or reactive psychosis. But the onset of schizophrenia during adolescence and childhood has been accepted already in the twentieth century, and it is even more today. The child, adolescent and adult forms of schizophrenia could be regarded as a qualitatively similar and continuous, while allowing for developmental variation (Remschmidt, 2001).

3. Classification of psychotic disorders

Psychotic disorders can be classified into (modified after Turk et al., Turk, 2007):
- acute and transitory psychotic disorders
- schizophrenia
- organic psychotic disorders or psychotic disorders due to general medical condition
- substance-induced psychotic disorders
- schizoaffective disorders.

In schizoaffective disorders, symptoms of schizophrenia and affective symptoms are present at the same time. Since they are very rare in children and adolescents, they will not be considered in detail here. Psychotic symptoms, on the other hand, can be found in 58% of patients with bipolar disorder (see the subchapter 5.6 Differential Diagnosis of schizophrenia for more detail), which is classified under affective disorders.

4. Developmental aspects of psychotic disorders

Main characteristic for any psychotic disorder is loosing the reality control. Understanding the reality and sharing the same view on reality with other members of the same culture has a strong developmental basis. Young children cannot distinguish between fantasy and reality (Remschmidt, 2001). Therefore, it is extremely difficult to demonstrate psychotic processes until children develop reasonably cognitive and linguistic abilities. Beliefs in fantasy figures, imaginary friends are common in preschool children. Transient psychotic-like symptoms, such as hallucinations, can be thus observed in preschool children in relation to stress and anxiety (Rothstein, 1981). Children under 4 years cannot develop typical psychotic symptoms (delusions of persecution or influence) because they lack a fully formed perception of social relations (Remschmidt, 2004).

In school children, psychotic phenomena are not common, but if they are present, they show the tendency to persist and are frequently associated with schizophrenia (Carlson & Kashani, 1988; Del Beccaro et al., 1988; Russel et al., 1989; Volkmar et al., 1988). In adolescents, the levels of psychotic disorders increase markedly and clinical pictures are similar to those seen in the adulthood. Also, the differential diagnosis becomes difficult in the adolescence because of frequent substance abuse in this age range and also because of increased frequency of brief psychotic episodes associated with other conditions such as borderline personality and others (McKenna, 1994).

5. Childhood and adolescent schizophrenia

5.1 Epidemiology

Early-onset schizophrenia can be divided into a very early-onset or childhood-onset schizophrenia (major symptoms of schizophrenia are present at or under the age of 12) and adolescent-onset schizophrenia (with major symptoms in the 13-19 year age range) (Martin & Volkmar, 2007; Remschmidt, 2001).

The lifetime prevalence of schizophrenia is around 1%. It is very rare before the age of 12 and it peaks between the age of 13 and 17 (Remschmidt, 1994). The prevalence of early-onset schizophrenia (childhood and adolescent schizophrenia) is 0.23%. Only 0.1-1% of schizophrenic disorders start before the age of 10, 4% start before the age of 15 and 10% start between the age of 16 and 20. According to one population study, adolescent onset schizophrenia affects 0.23% of the general population and according to another population study, it affects 1.34% of the general population of teenagers with mental retardation (Remschmidt, 2001). Before the age of 15, there is a higher proportion of boys (male:female ratio is 3:1), but soon after the age of 15 the male:female ratio reaches 1:1 (Remschmidt, 2004).

In Europe, childhood-onset schizophrenia is only occasionally diagnosed. An outcome study in Germany covering a 30-year period reported only 40 cases, many of which had onset after the age of 10, but before the age of 13 (Eggers,1978). The NIHM childhood-onset schizophrenia study, which has been ongoing since 1990 in USA has found 89 cases of childhood schizophrenia (Martin &Volkmar, 2007).

In general, mean IQ in children with schizophrenia seems to be lower than in the general population. About 10-20% score about 70 or under. This fact is considered a premorbid feature and not a consequence of schizophrenia (Aylward, 1984). The same seems to be true for adolescents with schizophrenia. Altogether, 1.34% of them have an IQ under 70, a percentage that is much higher than in the general population where it is just 0.23% (Remschmidt, 2001).

5.2 Etiology

Schizophrenia is a complex multifactor disorder, where genetics is an important vulnerability factor. The actual occurrence of the disease and its form, however, depend upon many other familiar and unfamiliar internal and external – environment factors (Sadock & Sadock, 2007). Schizophrenia can thus be seen as a syndrome at the end of dynamic processes, which can be explained with neurodevelopment and neurodegenerative models.

Neurodevelopmental model proposes that the biological origins of schizophrenia lie in the fetal neurodevelopment, and this early developmental lesion can be traced in premorbid

developmental, behavioural and cognitive impairments. This neuropathology is finally expressed as classical psychotic symptoms (Purves &Lichtmen, 1980). There is still a lively debate about the value of neurodevelopmental model, since not all patients with schizophrenia show premorbid abnormalietis.

In the last 50 years, neurotransmitter (dopaminergic, serotonergic and glutaminergic) hypotheses have prevailed in an etiology of schizophrenia. However, neurobiological in-vivo and post-mortem studies of neurotransmitter system have yielded inconsistent and contradictory results rather than providing more precise knowledge of schizophrenia etiology. But on the other hand, they were crucial in pharmacotherapy improvement for schizophrenia (Haroutunian, 2007).

Also, in the last decades, it is the genetic studies that have come to the forefront of schizophrenia research. Linkage analysis of microsatellite regions in schizophrenic families has shown that regions associated with schizophrenia can be found on many chromosomes. The impact of one single gene on the disease is very small, probably less than 1% (Gill, 1996; Riley, 2006). The second frequently used genetic method is searching for candidate genes for schizophrenia (Riley, 2006). Among those that are researched in relation to schizophrenia are also the genes that encode dopamine and serotonin receptors and promoters.

As evidenced by the research, there are no genes that would significantly impact the onset of schizophrenia. Instead, schizophrenia seems to be a product of various risk factors in people that are genetically sensitive. This sensitivity is complex and it is most likely represented by a changing combination of various genes with small impact (Sadock & Sadock, 2007). Genetic sensitivity alone, however, is not sufficient for the development of the clinical picture of schizophrenia. Other risk factors must be present too (Riley, 2006; Prathikanti & Weinberger, 2005).

It is possible that childhood schizophrenia has a higher genetic loading, since the outcome of childhood schizophrenia is very poor and probably much worse than for adolescent- and adult-onset schizophrenia (Remschmidt, 2001).

It seems that multiple gene influences result in continuous dimensions rather than in categorical disorders. Thus the dimensional model for schizophrenia seems to be more appropriate than the categorical one (dimensional model will be explained in the subchapter on Diagnosis).

Unfortunately, genetic studies too have failed to fully explain schizophrenia etiology. It remains vaguely and phenotypically broadly defined disorder that represents a complex disease due to the incomplete penetrance and heterogenic genetics (Kendler, 1993; Prathikanti & Weinberger, 2005).

There is, however, a new concept emerging in schizophrenia research, namely endophenotypes. Endophenotypes are particularly useful for understanding the etiology of complex disorders – such as schizophrenia – in which several genes and environmental factors influence the phenotype. Synonyms for endophenotypes are biological markers, biological phenotypes, latent phenotypes or intermediate phenotypes (Gottesman, 2003; Gould, 2006; Holden, 2003; Preston & Weinberger, 2005; Weinberger, 2002). The endophenotype is less genetically complex than the disorder it underlies (Castellanos & Tannock, 2002). It is assumed that it is more closely related to one or more pathophysiological genes for the nosological category, compared with the entire spectrum of disorders included in the nosological category. It is not necessary for an endophenotype to belong to a specific nosological category because nosological categories usually do not have biological background. It is, however, necessary for it to be heritable and to segregate with illness within families (Berrettini, 2005). Cognitive and

biochemical features of psychiatric patients are more associated with genetic factors than the behavioural phenotypes. The idea of endophenotypes is basically to relate some features of schizophrenic patients to the genes (Gottesman & Gould, 2003; Berrettini, 2005). Endophenotypes are stable, state-independent characteristics in contras to behavioural symptoms (Berrettini, 2005). The most studied endophenotypes today are cognitive deficits, an abnormality of the P50 auditory evoked potential and imagining phenotypes (Berrettini, 2005; Burdick et al., 2006; Turner et al., 2006).

Schizophrenia peaks at the age of 15 and this is one of the reasons why many authors see the puberty as a risk factor for schizophrenia (Remschmidt, 2004; Hyde, 1992). Neurobiological changes that occur during the puberty and adolescence can influence more frequent occurrence of schizophrenia during this period of life. These neurobiological changes are: myelinisation of the associative cortex and hippocampus, maturation of the prefrontal cortex, diminishing of the cerebral plasticity, effect of sex hormones on synapses development and changes in dopaminergic innervations (the last two proved only in animals) and changes in neurotransmission (Remschmidt, 2004).

5.3 Diagnosis

No symptom or sign is pathognomonic for schizophrenia. Moreover, patient symptoms change with time. During the last decade, schizophrenic symptoms have been linked to the underlying neurocognitive processes and further to the genetic substrate (for detail please see subchapter on Eetiology – Endophenotypes). It seems that multiple gene influences result in continuous dimensions rather than in categorical disorders. For schizophrenia, five symptom dimensions can be described (positive, negative, disorganized, cognitive and affective symptoms).

Positive symptoms

- Hallucinations, which can be auditory, visual, somatic-tactile, and olfactory. Typical for schizophrenia are voices commenting and/or conversing patient's behaviour or commending him or her what to do (imperative hallucinations).
- Delusions, which can be persecutory, jealousy, guilt and sin, grandiose, religious, and somatic. Typical for patients with schizophrenia are delusions of reference, of being controlled, of mind reading, though broadcasting and thought withdrawal.
- Positive formal thought disorder like distractible speech, pressure of speech, illogicality, and derailment. Some authors classified some of these symptoms among disorganized symptoms.

Negative symptoms

- Affective flattening, which can be seen as unchanged facial expression no matter the circumstances, decreased spontaneous movements, paucity of expressive gestures, and poor eye contact etc.
- Alogia (poverty of speech, poverty of content of speech, blocking, and increased response latency).
- Avolition-apathy (grooming and hygiene, impersistence at work or school, physical anergia).
- Attention (social inattentiveness and inattentiveness during testing).

Disorganized symptoms

- Bizarre behaviour, including bizarre clothing and appearance, social and sexual behaviour, giggling for no reason, aggressive or agitated behaviour and repetitive or stereotyped behaviour.

- Disorganized thinking with illogical, nonsensical thought patterns jumping from one unrelated idea to another, so that it is impossible to understand what the person is trying to say. Making up words is common.

Cognitive symptoms refer to the difficulties with concentration and memory. Thy may include:

- slow thinking
- difficulty understanding
- poor concentration
- poor memory
- difficulty expressing thoughts
- difficulty integrating thoughts, feelings and behaviour.

5.3.1 DSM-IV diagnostic criteria (Diagnostic and Statistical Manual of mental disorders)

DSM-IV (American Psychiatric Association, 1994) contains the American Psychiatric Association's official diagnostic criteria for schizophrenia, which describes several types of schizophrenia. The presence of hallucinations or delusions is not necessary for the diagnosis of schizophrenia. A patient's disorder is diagnosed as schizophrenia also when the two of the symptoms listed as symptoms 3-5 in criterion A are present.

Criterion A characteristic symptoms: two or more of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated):

1. delusions
2. hallucinations
3. disorganized speech
4. grossly disorganized or catatonic behaviour
5. negative symptoms.

For a significant portion of the time since the onset of the disturbance, one or more major areas of functioning such as school or work must be markedly below the level achieved prior to the onset of the disturbance to fulfil the diagnostic criteria for schizophrenia. Continuous symptoms of the disturbance have to persist for at least 6 months. Prodromal symptoms are included in this 6-month period. At least 1 month of acute symptoms (or less if successfully treated) has to be present for the diagnosis of schizophrenia. Schizoaffective disorder, mood disorder with psychotic features, pervasive developmental disorder, direct physiological effects of a substance (a drug abuse, a medication) or a general medical condition have to be ruled out.

DSM-IV advocates the application of the same diagnostic criteria in early-onset schizophrenia as for adult-type disorders with some allowance for different manifestations. It recognises several different types of schizophrenia.

5.3.1.1. *Paranoid type* is the most common type of schizophrenia, typically beginning in adult life. It is the prototype of positive symptoms. The course of disease usually does not lead to personality and cognitive changes (Sadock & Sadock, 2007; Remschmidt, 2005).

5.3.1.2 *Disorganized type* (also cold hebephrenic type) begins most often in puberty with an insidious, slow and unspecific onset. It can often be mistaken for an adolescent crisis. This type is characterized by a marked regression to a primitive, disinhibited and disorganized behaviour. Depersonalization phenomena can be present. The contact with reality is poor, the emotional responses are inappropriate. They can burst into laughter without any

apparent reason. Incongruous grinning and grimacing is also common. The behaviour can be described as silly or fatuous (Sadock & Sadock, 2007; Remschmidt, 2005). These patients are usually intelligent with the premorbid personality characteristics such as shyness, passiveness, introverted, with few/no friends. This type can be diagnosed after a few months of observations. Patients with disorganized type show negative symptomatics early in the course of disease. This is also the reason why this type is a prototype for negative symptoms (Remschmidt, 2005).

5.3.1.3 *Catatonic type* was common several decades ago. In Europe and North America, it is quite rare today, most likely because of the antipsychotic treatment. The classic feature is a marked disturbance in motor function which can involve stupor, negativism, rigidity, excitement or posturing. Rapid alternations from one extreme (stupor) to another (excitement) are also possible (Sandock & Sandock, 2007).

5.3.1.4 *Residual type* is a chronic state characterized by the presence of continuing evidence of the schizophrenic disturbance in the absence of a complete set of active symptoms or sufficient symptoms to meet the diagnosis of another type of schizophrenia. Emotional blunting, social withdrawal, eccentric behaviour, illogical thinking and mild loosing of associations commonly appear. Hallucinations or delusions can be present from time to time but are neither prominent nor accompanied with a strong affect (Sandock & Sandock, 2007).

*5.3.1.*5 *Undifferentiated type* is a type characterized by the presence of symptoms for schizophrenia, but does not meet criteria for any other type.

5.3.1.6 *Simple schizophrenia* is a type of schizophrenia according to ICD-10 classification (International Statistical Classification of Diseases and Related Health Problems, 1993) with particular insidious onset of withdrawal and social deterioration associated with very poor outcome. It usually starts slowly and untypically in adolescence. In the absence of prominent delusions, hallucinations or disorganized speech, it leads to a profound personal and social destruction. Patients are depressed, anhedonic, without energy and/or motivation. They can leave a school or a job and many of them can be found in marginal populations (homeless etc.). The diagnosing of this type can be quite difficult, since it is not clearly demarcated neither from the schizoid nor from the schizotypal personality disorder (Remschmidt, 2005).

In early-onset schizophrenia there are typically fewer well-formed systematized types when compared to adult schizophrenia. It appears that there is a relative predominance of disorganised and undifferentiated cases and fewer paranoid cases in early-onset schizophrenia, when compared with adult schizophrenia (Remschmidt, 2001).

The definition of schizophrenia is symptom based, so the diagnostic criteria using the adult manifestation of the disorder could miss some of the developmental variability in symptoms seen in children and adolescents. This could result in missed diagnoses, especially in presentation with a slow onset and negative symptoms, which are more commonly seen in children and adolescents (Remschmidt, 2001).

Another factor that has to be taken into consideration is the patient's age, which has a strong impact on the schizophrenic symptom types. Positive symptoms increase linearly with age, while the negative ones occur most frequently in the early childhood and late adolescence (Remschmidt, 2004). Bettes and Walker found a correlation between symptoms and IQ: children with high IQ showed more positive and fewer negative symptoms than low-IQ children (Bettes & Walker, 1987). In the course of schizophrenic symptomatology, there is a clear shift from positive and mixed symptoms to negative ones, which correlates with disease chronicity (Remschmidt, 2005).

Slow and insidious onset of childhood and adolescent schizophrenia is a rule rather than an exception. This is the reason why cases of early-onset schizophrenia are diagnosed with up to two-year delay (Schefer & Ross, 2002). Such delays worsen the course and prognosis of the disease, so early diagnosis and treatment is crucial. Early-onset schizophrenia with acute onset, on the other hand, poses less problems, mostly due to easily recognisable positive symptoms.

5.3.2 Other possible diagnostic aspects

An interesting feature of early-onset schizophrenia compared to adult one seems to be the higher rates of early language, social and motor developmental abnormalities, which are possibly reflecting greater impairment in early brain development (Martin & Volkmar, 2007). This is the reason why Werry suggests at least two clinical phenotypes of schizophrenia. The first one is associated with a long-standing developmental abnormality where the acute psychotic episode develops after some years of pre-existing abnormality. The second phenotype develops in children and adolescents who have had previously normal development (Werry, 1996).

Schizophrenic symptomatic in children and adolescents as well as in adults can also be described using another distinction which differentiates between two type of schizophrenia differing in psychopathological symptoms and premorbid functioning of the patient (Remschmidt, 2005). Type I is characterized by positive symptoms – hallucinations and illusions. Patients are aggressive, restless and excited, they can talk a lot and can make up new words (neologisms). The CAT scan usually reveals a normal brain structure and the response to the pharmacological treatment is usually good. Type II, on the other hand, is characterized by negative symptoms – affective flattening, social withdrawal, loss of interests and cognitive slowness. Here, the CAT scan shows brain structural changes and the response to pharmacotherapy is worse than in type I (Kaplan & Sadock, 1998).

5.4 Preclinical symptoms and associated features

5.4.1 Prodromal states

Before full symptoms of schizophrenia are manifested in a clinical picture, disease pre-stage – prodromal state can be present in 75% of patients with schizophrenia. Prodromal state can last a few days or some years (Remschmidt, 2005). The symptoms are unspecific, ranging from problems with concentration, changes in motivation and sleep disturbances, unspecific fears, irritability and suspiciousness. Family and friends may notice that the person has changed and is no longer functioning well in social, school and/or occupational activities. A patient may develop an interest in abstract ideas, philosophy and in the occult and religious questions. Peculiar behaviour, abnormal affects, unusual speech, bizarre ideas and strange perceptual experiences can be present too. Somatic complains such as headache, back and muscle pain, weakness and digestive problems can also be seen in prodromal state (Sadock & Sadock, 2007). Unspecific prodromal state could be misdiagnosed as adolescent crises or same other psychiatric diagnosis.

Negative symptoms are usually the first schizophrenic symptoms present in a prodromal state, while positive ones usually appear later during the acute state of schizophrenia (Remschmidt, 2005).

5.4.2 Premorbid personality characteristics

Typical, but not invariable, for some children and adolescents with schizophrenia are their premorbid personality characteristics. They are usually quite passive, introverted with few or no friends. They may avoid sport and social activities such as dating and watch TV or listen to the music instead. As children they are often described as special, perpetually unsatisfied and sensitive. Described characteristics may be detected in half of the children and adolescents with schizophrenia (Remschmidt, 2005).

5.4.3 Unspecific developmental abnormalities

In addition to prodromal state and premorbid personality characteristics, patients with early-onset schizophrenia can also show many unspecific developmental abnormalities such as soft neurological signs, unspecific sensory and motoric neurological abnormalities, aggravated autonomic reactions, different perinatal complications and symptoms of hyperkinetic syndrome (Remschmidt, 2004). Prevalent findings in a personal history of children and adolescents with schizophrenia are also cognitive impairments, lower IQ, transient symptoms of pervasive disorder and social withdrawal. A study of Alaghband-Rad et al. found that 36% of patients with childhood schizophrenia had a premorbid history of at least one pervasive developmental disorder feature and 13% had full autism (Alaghband-Rad et al., 1995). In all, 60-70% of children with childhood onset schizophrenia had language and/or motor impairments in infancy (Jacobsen & Rapoport, 1998; Watkins et al., 1988). All described features and impairments are not specific for early-onset schizophrenia, they can also be found in other psychiatric disorders, so they have no diagnostic value.

There is a vivid debate on primary prevention for subjects at high risk for schizophrenia. In short, our knowledge today is not sufficient enough to reliably diagnose pre-schizophrenic states, which is why preventive pharmacological treatment cannot be recommended as yet.

5.5 Course and prognosis

Early-onset schizophrenia has worse prognosis compared to schizophrenia occurring in adulthood. Only 23% of patients with early-onset schizophrenia reach full remission (compared to 25% of adult patients), while 25% of them reach partial remission (compared to 50% of adult patients). Chronic course will have 52% of patients with early-onset schizophrenia (compared to 25% of adult patients) (Remschmidt, 2005). After the hospitalization, 40% of patients with early-onset schizophrenia cannot go back to the same educational/professional level as before the hospitalization. Besides the chronicification of the disease, disfunctional family environment may also be the reason (Remschmidt, 2005).

Moreover, a total of 10% of people with adult schizophrenia die by suicide (Kaplan & Sadock, 1998). There are not many studies on this topic in children and adolescents, but one provides interesting results. Eggers points to the risk of suicide in the prodromal state and growing suicidal risk with onset of schizophrenia; where 65% of adolescents were preoccupied by death thoughts, 20% attempted suicide and 5% committed suicide. Compared to adults, suicide was relatively late phenomena, with the average time of 8.5 years between the onset of schizophrenia and the attempted suicide, and the average time between 6 and 14 years for the committed suicide (Eggers, 1978).

In general, the prognosis for the early-onset schizophrenia is as follows: the earlier the onset, the greater the disability (Remschmidt, 2001). Regarding the gender, girls seem to have somewhat better prognosis, both because they seem to be less vulnerable to the early-onset

than boys, and because they seem to be less affected by the illness. This, however, does not mean that their chance of full recovery is any better than in boys.
Studies show that children with prodromal state that lasted over one year have worse prognosis for clinical improvement during hospitalization (Amminger et al., 1997).

5.6 Differential diagnosis

5.6.1 Conduct and emotional disorders

Hallucination can be present in conduct and emotional disorders, particularly at the times of stress. When focusing on persecutory ideas and ideas of reference, which are often present in conduct and emotional disorders, the misdiagnosis can be the problem (Remschmidt, 2001).

5.6.2 Affective psychosis

In a follow-up study of Werry, over 50% of the bipolar disorder cases had initial diagnosis of schizophrenia (Werry, 1991), which makes a valid distinction between schizophrenia and bipolar disorder in children and adolescents questionable. In affective psychosis, however, the onset of psychotic symptoms is often rapid with relatively good premorbid social and school functioning.

5.6.3 Autistic spectrum disorders

Positive symptoms may develop in autistic spectrum disorders in adolescence, however, they are usually transitory. Children and adolescents with autistic spectrum disorders have more long-standing and progressive deterioration in social and cognitive fields prior to the onset of psychotic symptoms.

5.6.4 Organic brain conditions

At first episode of schizophrenia, one must exclude all other possible organic causes for psychotic symptomatology (drug-inducted psychosis, epilepsy, neuro-degenerative disorders etc.) described in special subchapters.

5.7 Treatment of schizophrenia

5.7.1 Pharmacological treatment

Antipsychotics are the first choice drugs in a pharmacological treatment of schizophrenia. They act suppressively on the symptomatology of the disease, but do not cure the disease. They calm, reduce and eliminate psychotic symptoms in an acute state of schizophrenia. When the acute state has settled down, they act prophylactic against psychotic recidives. Pharmacological treatment in schizophrenia can be divided into three phases:

- Acute phase, in which antipsychotics act suppressively on major schizophrenic symptoms.
- Maintenance phase, which starts in full remission and should last at least six months with the same doses of antiphsychotics as in acute phase. The purpose is to prevent possible relapse, to improve patient's social skills and further relief of symptoms.
- Prophylaxis phase prevents recidive and helps maintaining and/or exceeding the achieved level of patient's functioning and quality of life. This phase should last at least one year after the first episode of schizophrenia and several years after the second episode of schizophrenia. As schizophrenia is a chronic disease, the pharmacological

treatment can be lifelong. The doses of antipsychotics should be lower than in the first two phases (Sandock & Sandock, 2007).

5.7.2 Psychotherapeutic measures

It is crucial that schizophrenic patients and their families are educated about the disease, its etiology, its course and the treatment. Patients and their families have to have the knowledge about the disorder in order to recognise a relapse on time, to prevent preterm drug reduction or cessation, and to strengthen the compliance. Among psychotherapeutic measures, only the most used/applied will be mentioned, i.e. cognitive and other behavioural approaches, emotion management therapy, group programs, family-oriented measures, supportive and structural family therapy and extended development-oriented family therapy.

5.7.3 Rehabilitation

Since nearly half of the patients suffering from schizophrenia cannot return immediately after the hospitalization to their school/occupational and family environment, they are usually directed to rehabilitation centres. On average, the rehabilitation lasts two years and is crucial to the treatment of schizophrenia.

The rehabilitation can be divided into two phases, aiming at growing stepwise independence. During the first phase, much effort should be assigned to the areas of schooling, working skills, social skills training and interpersonal problem solving. It usually lasts one year. The patient is living together with other patients in a rehabilitation centre in a family-like structured program. The second rehabilitation phase, however, should bring major change to the patient in order to gain more independence, self-sufficiency, psychosocial reintegration and the continuation of school and professional work without the significant support from the rehabilitation centre (Remschmidt, 2001).

6. Acute and transient psychotic disorders

There are no reliable data on incidence and prevalence, gender ratio and the mean age at the onset for this psychotic disorder. It seems, however, that it is more frequent in a younger population. The disorder is characterized by a sudden onset (less than in a two-week time) of psychotic symptoms, which may include delusions, hallucinations, disorganized speech or behavior, or catatonic behavior. Also, the symptoms can change frequently during an episode. The duration of an acute and transient psychosis is generally short, non re-occurring, and not better accounted for by another condition. Symptoms generally do not last long, usually just a few days or weeks, but 2-3 months at most. There is a possibility of an eventual return to full baseline functioning (Kaplan & Sadock, 1998). The prognosis is good – 50-80% of patients do not have any psychiatric problems after the acute psychosis has ended. Occasionally, a relation to a stressor can be found. In such cases, the diagnosis of a reactive disorder is appropriate (Sandock & Sandock, 2007).

6.1 Reactive psychoses

Reactive psychosis occurs shortly after (maximum 2 weeks after) and in response to a significant stressor in a person's life (death in the family, war, divorce, abuse, etc.). Psychotic symptoms can be thematically closely related to the trauma. The reactive psychoses are short. Their duration is usually not longer than 2-3 months.

7. Substance-induced psychotic disorder

There are 3 facts that are important when dealing with psychoactive substances associated with psychotic disorders (Kaplan & Sadock, 1998; Remschmidt, 2004).

- Psychoactive substances can cause psychotic symptoms in anyone (typical hallucinogens are LSD, phencyclidine –"*angel dust*", cocaine and amphetamine – "*speed*" and "*ecstasy*").
- In a person who is genetically predisposed to developing a psychosis, psychoactive substances can accelerate the onset of the psychosis and worsen its clinical picture, course and treatment.
- Patients with psychotic disorder (most often schizophrenia) can use psychoactive substances as self-medication because of their effect on symptomatic recovery. Heroin, for instance, can reduce hallucinations, while cannabis, on the other hand, calms the psychotic excitement.

Psychotic symptoms in substance-induced psychotic disorders develop during the intake of a substance or not later than 48 hours after it. Partial remission occurs within one month, while full remission is achieved in 6 months (Remschmidt, 2004). Prominent hallucinations (they typically appear on more than one sensorial level, even though acoustic hallucinations are the most frequent ones), delusions, psychomotor abnormalities (excitment or stupor) and a variety of affective symptoms are typical for a substance-induced psychotic disorder. It is worth mentioning that canabis can cause chronic psychotic state too (Sandock & Sandock, 2007).

In an acute psychotic state, it is difficult to make a distinction between substance-induced psychotic disorders, brief and acute psychotic disorders and schizophrenia. The main facts that militate against the substance-induced psychotic disorders are (Remschmidt, 2005):

- The presence of psychotic symptoms prior to the drug consummation.
- The presence of psychotic episodes prior to the drug consummation.
- Drug-induced psychotic symptoms persist longer than expected.

8. Psychotic disorders due to general medical condition

The evaluation of a psychotic patient requires also a consideration of the possibility that the psychotic symptoms are the result of a general medical condition (brain tumour, head injury, poisoning, infection, metabolic diseases, epilepsy, etc.). These conditions can represent a real diagnostic problem if the psychotic symptoms are the only symptoms in the clinical picture. In such a case, there is a danger to overlook the organic/somatic cause of the symptomatology and to treat the patient incorrectly. When dealing with a case of first psychotic/schizophrenic episode there is a need to preform laboratory tests and imaging investigations in order to exclude the most possible causes of psychotic symptomatology (Sandock & Sandock, 2007).

8.1 Case report

An 18-year-old girl was admitted to the Child and Adolescent Psychiatry Unit of the Maribor University Clinical Centre in Slovenia for suspected disorganized (formerly called hebephrenic) schizophrenia. A year before that, at the age of 17, she had begun to laugh without reason, and her behaviour had become silly and disorganized. Her emotional

responses were inappropriate. She started grimacing and became paramimic **a**nd parathymic. She withdrew socially and began to shut herself in her room. She had auditory hallucinations, consisting of a running commentary on her behaviour. At that time, there were no changes in her school performance, which was already consistently below average; however, her school performance deteriorated 3 months before admission to hospital, when she became unable to do any schoolwork. She also became disoriented as to her whereabouts and got lost several times.

There had been no aberrations in her developmental milestones. Until recently, she had not had any diseases or needed any medical attention.

Her family history was interesting. She had a 24-year-old brother with moderate mental retardation according to the DSM-IV criteria. He had delayed developmental milestones and difficulties in walking and speaking. She also had a 25-year-old sister who, 4 years before, had had an episode of postpartum depression (according to DSM-IV criteria) that started a few days after she had delivered a baby. She had depressed mood and no interest in her baby or in any other activities. She was motor retarded, complained about loss of energy and diminished ability to think and concentrate, and she reported feelings of worthlessness and guilt about being ill and not being able to take care of her baby. She cried constantly, had no appetite and suffered from insomnia. She recovered in a month without any antidepressants. Her school performance had been below average throughout all 10 years of her education, and she had been unable to find a job either before or after the episode of postpartum depression. She attended night school to become a cook and had achieved average success at the time of writing. There was no history of psychiatric disturbance before or after the episode of postpartum depression. The patient's mother had 7 half-siblings. Two had died, probably of pneumonia, at the age of 6 months. The third sibling was mentally retarded and probably had poliomyelitis at the age of 2 years. Otherwise, there was no family history of psychiatric disorders.

At admission, our patient was disoriented in terms of time, place and person. Her thought process was disorganized, and her behaviour was disorganized and uninhibited. She was paramimic and parathymic with blunted affect. Her rapport with others was poor and her speech monotonous, and her understanding of questions was poor. She had a poor attention span and reported auditory hallucinations, consisting of a running commentary on her behaviour, which she found pleasant. She described the hallucinations as a single young male voice, which was talking to her using short sentences, mostly encouraging her to do everyday tasks and commenting on her behaviour. She revealed a lack of initiative, had poor contact with reality and had no insight into her illness. She was incontinent of urine and had dyspraxia. Psychological tests (Rorschach projective test and Bender Visual–Motor Gestalt test) revealed psychotic disturbance. The results of routine laboratory tests including thyroid-stimulating hormone, vitamin B12 and folic acid were normal. The findings of syphilis and HIV-1 serology were negative, as was serology for Borrelia burgdorferi. Levels of proteins in the cerebrospinal fluid were elevated (0.77 g/L). Electroencephalography (EEG) showed abnormal bilateral synchronous, mainly theta activity, which was most prominent temporally.

According to DSM-IV diagnostic criteria, the patient's disease presented like disorganized schizophrenia (disorganized thought process and behaviour, flattening of the affect, auditory hallucinations and social dysfunction). These symptoms persisted for about 9

months without any marked signs of cognitive impairment, apart from poor attention and slowing in thought process. Therefore, treatment with small doses of an atypical antipsychotic, risperidone (1 mL twice a day) was initiated.

After 1 week of treatment, the patient stopped reporting auditory hallucinations. Although her behaviour was still disorganized, she was more willing to cooperate during the required medical examinations. Neurologic examination revealed positive pyramidal signs in the upper extremities. Tendon-stretch reflexes in the lower extremities were absent distally. Computed tomography revealed signs of advanced cortical atrophy, symmetrical ventricular enlargement and periventricular white-matter hypodensity. Magnetic resonance imaging of the brain showed diffuse signal hyperintensity of the white matter, especially in the periventricular area, as well as in the corpus callosum, cerebral atrophic changes and symmetrical ventricular enlargement. Electromyography showed a slowing of the nerve conduction velocities (NCV) (NCV of the peroneal nerve was 23 m/s [reference range 44–57 m/s]) and marked prolongation of the F-wave latency. Visual evoked potentials showed a normal retinogram, but cortical responses had prolonged latencies with a normal response distribution. Somatosensory and acoustic evoked potentials were within normal limits. Abdominal ultrasonography revealed polyposis of the gall bladder.

Four weeks later, the disorganized and psychotic clinical picture diminished and the patient's cognitive impairment became increasingly obvious. Apart from poor attention and slowing of the thought process, memory (recollection and recent past) was disturbed the most. Therefore neuropsychological tests (Wechsler Intelligence Scale, Wechsler Memory Scale, Trail-making Test, Stroop Test, Hooper Visual Organization Test, Rivermead Behavioural Memory Test and the Controlled Oral Word Association Test) were carried out. The patients' full-scale IQ fell to within the range of severe mental retardation (according to DSM-IV criteria). There was a minor difference between the patient's verbal and performance IQ, favouring the former. The impairments were severe over the whole range of mental functioning. Attention processes, perception, executive functions, communication and motor skills were impaired. Her processing speed was very slow. Memory for verbally presented information and visual memory were found to be severely impaired. She could not independently perform any simple or routine operations. Therefore, an acetylcholinesterase (AChE) inhibitor was added to the therapy (galantamine, 4 mg, twice a day for 1 month, then galantamine, 8 mg, twice a day).

Taking into account the diagnostic findings of neurologic examinations, neuropsychological tests, urinary incontinence and imaging, it was clear that there was most probably an organic disorder underlying the disorganized schizophrenia–like symptoms, which also later caused symptoms of dementia. Therefore, further tests were performed, focusing especially on inherited metabolic disorders.

The results of urine screening tests and screening for very long chain fatty acids in the serum were normal, excluding adrenoleukodystrophy and several disorders of peroxisomal function. Arylsulfatase A activity in leukocytes was markedly reduced (0.047 nmol/h per milligram; normal values in controls 2.82 ± 1.24 nmol/h per milligram). Measurement of urinary sulfatides by electrospray ionization–tandem mass spectrometry showed a 10-fold elevation of 609 nmol/L (values in healthy controls 51.5 ± 33.45 nmol/L), thus confirming the diagnosis of metachrometic leukodystrophia (MLD).

In the patient's sister, who remained clinically asymptomatic apart from the single episode described earlier, biochemical tests also showed clear arylsulfatase, a deficiency in leukocytes (0.155 nmol/h per milligram) and markedly elevated sulfatides in urine (436 nmol/L), thus proving the presence of a metabolic disease metachrometic leukodystrophy (MLD). In accordance with their obligate heterozygosity, intermediate arylsulfatase A activity was found in both of our patients' parents. Our patients' mentally retarded older brother had normal arylsulfatase A activity and normal values of sulfatides in the urine.

8.2 Discussion

Metachromatic leukodystrophy is one of the most serious genetic demyelination disorders (Tylki-Szymanska et al., 1996). It is an autosomal recessive lysosomal disease characterized by demyelination of the white matter in the central nervous system and the peripheral nerves. The relevant gene is located on chromosome 22q13. The disease is caused by a deficiency of the enzyme arylsulfatase A, which hydrolyzes various sulfatides, including the major sulfate-containing lipids of the nervous system. Sulfatide accumulation can be found in the brain and peripheral nerves and nonneural organs (kidney and gallbladder) (Hageman et al., 1995). The incidence of MLD is estimated between 1 and 5 cases per 100 000 newborns (Rentrop et al., 1999)

There are 3 types of MLD: late infantile, juvenile and adult. The late-infantile form, which has its onset at the age of 1–2 years, is characterized by gait and behavioural disturbances. The course of the disease is rapid and the outcome fatal. The juvenile form, which has its onset between the ages of 3 and 15 years, displays a less distinct phenotype, varying from peripheral nerve involvement in younger children to learning problems and behavioural difficulties in older children (Kaye, 2001). It has a more protracted course. The symptoms of adult MLD include dementia, psychosis, behavioural abnormalities, ataxia, polyneuropathy and epileptic seizures. Other psychiatric disorders can present with the following MLD symptoms: personality changes, depressive disorders, alcohol addiction, and worsening of school and/or work performance. Adult MLD has a slowly progressive course. Compared with the late-infantile and juvenile forms, the adult variant of MLD appears to be quite rare. The mean survival for adult MLD is at least 12 years, which is longer than the survival in late-infantile MLD (3–4 years) and juvenile MLD (7–9 years) (Kaye, 2001).

The diagnosis of MLD is based on arylsulfatase A activity in leukocytes or fibroblasts and on sulfatide excretion in the urine.

There is much disagreement in the literature regarding the incidence of psychosis in adult MLD. Hyde et al. (Hyde et al., 1992) suggested that in 53% of patients with adult MLD psychosis is present and is often the initial manifestation. Cengiz et al. (Cengiz et al., 2002) reported the cases of 3 sisters with adult type MLD, 2 of whom were initially diagnosed as having schizophrenia. Hageman et al state that psychosis is a less common symptom than previously suggested. In their group of 13 patients with confirmed adult MLD, the most common symptoms were ataxia and behavioural abnormalities with only 1 patient suffering from psychosis. The findings were similar in the group of 24 patients with confirmed MLD described in the literature, among whom only 4 were psychotic (Hageman et al., 1995).

Disorganized schizophrenia-like symptoms were the initial manifestation of MLD in our patient, and they persisted without any marked signs of cognitive impairment for at least 9 months. Then she got lost several times, and her school performance deteriorated. It is open to discussion whether her school performance deteriorated because of the disorganized schizophrenia-like symptoms or whether this was the first symptom of dementia. The differential diagnosis of dementia became problematic when disorganized and psychotic symptoms diminished after treatment with antipsychotics. We wondered whether disorganized schizophrenia-like symptoms just masked dementia? The first symptoms of disease were disorganized and silly behaviour, disorganized thought process, inappropriate affect, social withdrawal, incongruous grimacing, outbursts of laughter without any apparent reason, paramimia, parathymia, auditory hallucinations, poor contact with reality and no prominent signs of cognitive impairment. All these symptoms can also be found with dementia; however, they usually occur later, have a gradual onset and are rarely all present at the same time. At some point, as MLD progressed, disorganized schizophrenia–like symptoms most probably masked dementia, but dementia was not the initial manifestation of MLD in our patient.

The patient's older sister, in whom the diagnosis of MLD was confirmed biochemically, had had an episode of postpartum depression 4 years before. The literature describes some cases of adult MLD manifesting as major depression, but to our knowledge none of postpartum depression (Ricketts et al., 1996; Vella et al., 1998).

Little is known about the symptomatic treatment of psychotic symptoms and dementia in MLD. Our patient responded well to treatment with small doses of an atypical antipsychotic, showing no side effects. More questionable is the treatment with AChE inhibitors, which is only indicated in Alzheimer's disease.

This report underlines the importance of metabolic or any other somatic disease as a cause of what appears to be a psychosis or schizophrenia. It is crucial to always bear in mind that a full clinical, biochemical and imaging diagnostic evaluations must be performed in any patient with psychotic symptoms (Seidl et al., 1981).

9. Treatment of psychotic disorders

In diagnosing psychosis or any other psychiatric disorder it is important to know how to talk to a child or an adolescent. The communication has to be age appropriate, clear and without any questions that could be misunderstood or confusing. To create a positive transfer it can be helpful to know the topics, films, toys and plays that are popular in a specific age group. Children and adolescents usually like to talk openly without any reservations. They are frank in their answers and compared to adults they minimalize or deny their problems and fears less frequently.

It is equally important that the therapist is calm and relaxed, using short and understandable questions, talking slowly and understandably. Unclear question formulations and irony, must be avoided because they can additionally confuse and excite the patient. Furthermore, the therapist should also clearly explain the patient that he or she is here to help and that in order to do so some questions need to be answered. Discussion about the (un)reality of patient's symptoms and feelings when dealing with an acute psychotic patient should be postponed until the remission phase. It is possible that

patient's state of disease will not allow interviewing the patient what have to be recognised soon enough in order not to excite the patients further. But general are psychotic children and adolescents willing to talk about themselves and their symptomatic.

When meeting an acute psychotic patient for a first time, it is difficult to know if the psychotic symptomatics is substance-induced, if it is a result of a general medical condition or whether it is a case of schizophrenia. If the patient's condition allows taking auto- and hetero- anamnestic data, these could be very helpful in further differential diagnostic considerations. For instance, any information on the febrile state or a head injury is very probably associated with a possible organic background. Information on drug consumption is needed to exclude or include a substance-induced psychotic disorders in the diagnosis. Also, schizophrenia positive family history, a symptomatic that is typical for prodromal state or premorbid personal characteristic can be frequently found in the first schizophrenic episode.

Recommended investigations in any first psychotic episode are (Sandock & Sandock, 2007):

- blood biochemical tests (including liver functional tests), thyroid hormones
- drug and alcohol tests
- EEG, ECG, brain imagining investigations (CAT scan or MRI).

If metabolic diseases are suspicious further diagnostic testing should be performed: lactate, pyruvate, ammonia test, aminoacids in serum and urine, organic acids in urine; copper, ceruloplasmine (Mb Wilson); encime arylsulphatase A activity in leukocytes and sulphatides in urine (metachrometic leukodystrophia) (Gregoric Kumperscak, 2005).

Roughly speaking, psychotic patients can clinically present as two different types. Firstly, as acute psychotic, excited and/or physically aggressive patients with vivid hallucinations and illusions without any reality or impulse control and without disease insight. Secondly, as socially withdrawn adolescents with peculiar ideas and interests, diminished school achievements, in whom psychotic symptoms are present but hardly fulfil the needed diagnostic criteria for any psychotic disorder. The first type of patients should be hospitalized and treated with antipsychotics and other medicaments described in following chapter. The second type with a slowly developing clinical picture of psychosis just needs to be carefully followed in order to establish a proper diagnosis. Special care should be taken as prodromal state can be easily misdiagnosed or overlooked. Therefore, it is of outmost importance for the therapist to be familiar with all variations of development and growing up. Sometimes the search of adolescents for their identity can be mistakenly considered as a prodromal state. Only when there is a big difference between premorbid functioning and actual functioning (lower school achievements and social interests), there is a strong likelihood of prodromal state. Adolescents in general feel and describe psychotic symptoms before they can actually be seen in their behaviour and be objectivised. Thus, children and adolescents in a pre-psychotic state often talk about their inner changing, about a strong feeling that there is something happening to them, often stating that they are losing control and their mind. They are very upset and tense without any genuine reason.

9.1 Medication in an acute psychotic and/or excited patient

Acute psychotic and/or excited patients need medication prior to their admission to the hospital. Atypical antipsychotics (AA) and/or benzodiazepines (BD) can be used. The

recommendations for acute psychotic and excited child or adolescent treatment follow the recommendations for adult patients. All doses have to be age-adjusted.
AAs and BDs are broadly equally effective in sedation, however the effect of BDs can be quicker. Sometimes the combination of AAs and BDs is necessary and also more effective (Tayler et al., 2009). If the patient is already on AA, then BDs are recommended. If substance-induced psychosis is suspicious, then AAs are appropriate (Tayler et al., 2009).
Before resorting to intramuscular medication (im.), oral medications are always first choice. Clinicians have a good experience with risperidone in suspension, which can be given with water or tea. Oral risperidone suspension 0.5 to 2ml can be first administrated and if no effect is achieved after 2 hours, another dose of risperidone can be given. Olanzepine 5-10mg or haloperidol 0.05-0.15mg per kg per day can also be administrated. Among BDs, lorazepan 1-2 mg or diazepam 5-10mg can be used and the same dose can be repeatedly given after 1-2 hours if no effect is noted. The combination of AAs and BDs could also be administrated.
If there is no possibility for oral medication, im. medication must be used. Among AAs, olazepine 2.5-10mg im. can be administrated or a first generation antipsychotic haloperidol 0.025-0.075mg im. per kg per dose. Among BDs, lorazepam 0.05-0.1 mg im. per kg per dose or diazepam 0.1mg per kg per dose by slow iv. injection can be administrated.

10. Conclusion

To summarize, there are several traps when dealing with children and adolescents who present with symptoms suspicious of being psychosis. The above-described case report clearly shows that it is possible for an organic disease to present with psychotic symptoms only. This, of course, causes problems in accurately diagnosing the disease. Therefore, in order not to overlook a possible organic cause in a psychotic clinical picture, a biochemical, neurological and imagining tests and procedures must always be performed first.
Developmental period with its own special characteristics is another factor that needs to be considered when establishing the diagnosis. Each developmental state can colour the clinical picture differently. It can either overlap or aggravate the psychotic symptoms. For instance, it is completely normal for a 5-years old child to have an imaginary friend, but at age of 15 such friend may indicate pathology.
Also, adolescence is associated with many developmental tasks such as identity formation, independence and autonomy, and development of one's own view of life. All this, of course, brings along at a certain stage of development a detachment from parents, a retreat into their own world, as well as changes in the way of thinking and one's own philosophy, which however should not be confused with the schizophrenia prodromes. On the other hand, due to their key neurodevelopmental changes, puberty and adolescence represent an important risk factor for the development of schizophrenia. Therefore, this should be taken into consideration when establishing the correct diagnosis.
To conclude, though infrequent in childhood, schizophrenia is a major psychiatric disorder of adolescence (Remschmidt, 2001). Bearing in mind that early-onset schizophrenia has worse prognosis than the adult-onset one, early diagnosis followed by treatment is essential. However, in order to diagnose schizophrenia properly, a thorough knowledge of the negative and positive symptoms, prodromal state symptom varieties as well of the full

range of normal developmental changes is indispensable. Early-onset schizophrenia frequently starts with negative symptoms, but schizophrenia is usually recognised only when positive symptoms appear in the clinical picture. It also is noteworthy to mention that children and adolescents feel the symptoms and describe them much earlier than their effect on their behaviour can be objectivised and diagnosed.

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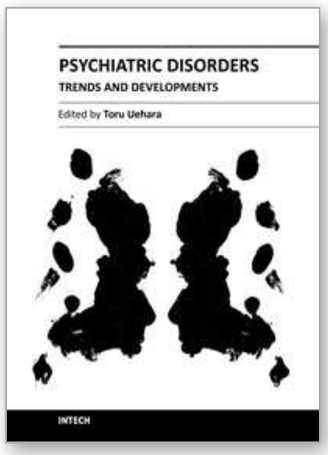

Psychiatric Disorders - Trends and Developments
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Due to their prevalence, pervasiveness and burden inflicted on men and women of today, psychiatric disorders are considered as one of the most important, sever and painful illnesses. This impairment of cognitive, emotional, or behavioural functioning is in some cases tragic. Aside from knowing the physical organic factors, such as infections, endocrinal illnesses or head injuries, the aetiology of psychiatric disorders has remained a mystery. However, recent advances in psychiatry and neuroscience have been successful in discovering subsequent pathophysiology and reaching associated bio-psycho-social factors. This book consists of recent trends and developments in psychiatry from all over the world, presented in the form of multifarious and comprehensive articles. The first two sections of the book are reserved for articles on schizophrenia and depression, two major illnesses present in this field. The third section of the book is reserved for addiction psychiatry, related not only to socio-cultural but also biological alterations. The last section of the book, titled Biological Neuropsychiatry, consists of three topics - updated molecular biology, fundamental neuroscience and clinical neuropsychiatric conditions. Doubtlessly, this book will be fruitful for future developments and collaboration in world psychiatry.

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