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Personality changes in patients with schizoaffective disorder (a review)

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The aim of the work is to study the world experience and views on the clinic and systematics of non-psychotic manifestations in schizoaffective disorder (SAD) by analyzing modern scientific literary sources.

The relevance of study on SAD is determined by the significant prevalence in the population (0.2–0.6 %) and negative socio-economic consequences of the disease. Significant problems in SAD are family and work maladjustment, suicidality and states of psychoactive substance use comorbidity.

The nosological independence of the disease was put into question and it was reflected in diagnostic approaches and classifications. In the clinical practice, SAD is considered independently and as a variant of schizophrenia or affective disorders. Diagnostics and prognostic criteria regarding personality changes in SAD are still at the stage of determining.

Conclusions. Understanding of definitions "prognosis", "outcome", and "maladjustment" in SAD is not unified. Signs of SAD in remission and intermission is the most recent aspect of the SAD clinic, which dictates the need to assess personality changes and the level of social adaptation and functioning.

Особистісні зміни у хворих на шизоафективний розлад (огляд літератури)

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Мета роботи – шляхом огляду й аналізу сучасних наукових літературних джерел дослідити світовий досвід та погляди на клініку і систематику змін особистості при шизоафективному розладі (ШАР).

Актуальність дослідження ШАР зумовлена істотною поширеністю в популяції (становить 0,2–0,6 %) та суттєвими негативними соціально-економічними наслідками захворювання. Важливою проблемою при ШАР є сімейна та трудова дезадаптація, суїцидальність і коморбідність станів вживання психоактивних речовин. Нозологічна самостійність захворювання сумнівна, і це можна підтвердити різноманітністю діагностичних підходів і класифікацій. У клінічній практиці ШАР визначають і самостійно, і як варіант шизофренії або афективного розладу, адже діагностичні та прогностичні критерії ШАР щодо особистісних змін перебувають на етапі визначення.

Висновки. Наповнення понять «прогноз», «наслідок», «дезадаптація» при ШАР залишається неуніфікованим. Ознаки ШАР у станах ремісії та інтермісії – найменш досліджений аспект клініки ШАР, а тому необхідним є оцінювання змін особистості та ступеня збереження / втрати соціальної адаптації / функціональності.

Schizoaffective disorder (SAD) is characterized by a prevalence in the population ranging between 0.2 % and 0.6 % and leads to significant negative socio-economic consequences [1–3]. The cost of treating a patient with SAD is higher than with schizophrenia due to long hospital stay, higher frequency of hospitalizations [4] and the need to actively involve more medical staff, while there are no differences in drug costs [5,6]. An even more serious problem with SAD is family and labor maladaptation, suicide and comorbidity of psychoactive substance use (PAS).

Since J. Kasanin (1933) introduced the concept of "schizoaffective psychosis", the nosological independence of SAD has been repeatedly questioned, which was reflected in diagnostic approaches and classifications [7,8]. In clinical practice, SAD is considered independently and as a variant of schizophrenia or affective disorders (AD) [9–14,21,22], because the diagnostic and prognostic criteria of SAD are still being determined.

However, scientists whose diagnostic and classification approaches are based on the symptoms of SAD exacerbation assessment, come to the need to assess the similarity / difference with AD and episodic schizophrenia. Thus, according to L. Rink, T. Pagel, J. Franklin, C. Baethge (2016), L. Tondo, G. H. Vázquez, C. Baethge and co-authors (2016) as well as other scientists – SAD occupies intermediate position between schizophrenia and AD in almost all clinical and prognostic features [14–16]. At the same time, clinical signs of exacerbation and features of productive (hallucinatory) symptoms are close to schizophrenia and prognostic – to AD [15,17].

Given the above problems of clinical definition, taxonomy and diagnosis, independent groups of researchers concluded the overall low validity of the SAD criteria in ICD-10 and low diagnostic congruence of classification systems used in practice today [10,18].

Aim

To review and analyze data on clinic and systematics of personality changes in SAD by reviewing and analyzing modern scientific literature sources.

Given the lack of clear clinical and diagnostic criteria in DSM-IV and ICD-10 in 2006, A. Vollmer-Larsen, T. B. Jacobsen, R. Hemmingsen and co-authors proposed the adoption

of a moratorium on the clinical use of the SAD diagnosis [19]. Soon, given that clear neuropsychological, neuroimaging, neurobiological, and genetic data on differences in episodic endogenous psychosis (EEP) are quite ambiguous, it has been suggested that SAD is a consequence schizophrenia and AD comorbidity and it was recommended to exclude SAD as a diagnostic category from future classifications of mental disorders [20].

However, SAD remained a nosological unit in DSM-V and ICD-11, although it retained its diagnostic uncertainty. A significant proportion of clinicians use their own principles and ideas about the signs of SAD, which do not fully comply with the guidelines of diagnostic classification developers [24].

It should be remembered that E. Kraepelin for diagnostics of psychosis recommended to take into account not one of the signs, but the aggregate characteristics of all semiotics and dynamics of the disease. In order to fully cover the clinical manifestations of EEP, more and more attention is paid to the study of personality changes – persistent symptoms of nonpsychotic level and their impact on social functioning, which, in turn, determines the quality of patients' life and their social environment.

The consequence of the formation of personality changes in SAD is the existing in periods of remission (as previously thought – intermissions) persistent labor and social maladaptation of varying degrees. The notion of "intact" personality of patients with SAD seems to be questionable. In recent decades, other separate components of personality changes and personal regression in SAD have been identified, namely: neuropsychological disorders, decreased "social" and "emotional" intelligence, deficient (negative) symptoms, the presence of residual cyclothymic symptom complex and postdepressive pathocharacterological disorders, consequences of psychopharmacotherapy, nosogenic and stigmatizing effects, etc. [25,26].

Research interest in studying the psychogenic and nosogenic effects on the clinical features of SAD and related mechanisms of adaptability / maladaptation is not high, despite the fact that it is of great clinical importance [27]. Undoubtedly, life circumstances and important life events affect the development, course and outcome of mental illness, but only some phenomena in SAD have been studied, there are data on greater vulnerability of women and greater susceptibility of SAD to post-traumatic stress disorder regardless of gender [28,29].

Uncertainty about the presence, structure, dynamics, and severity of personality changes in SAD led some authors to use the wording "almost not accompanied by a defect", "there are mild manifestations", "better prognosis for the impact on the personality than in schizophrenia" and others [23]. At the same time, the fact stated in the study by R. Kotov et al. (2017) is indisputable, that in most clinical cases, the disease led to social dysfunction, family and labor maladaptation even in the period of long-term remission in psychosis [30].

The vast majority of studies show an increase in the degree of social maladaptation and reduced workability in the direction of AD – SAD – schizophrenia, however, the main factor of maladaptation is the severity of psychopathological symptoms, rather than the presence of certain nosology [16,17,31,32]. Also, O. S. Serikova (2018) identified the following anamnestic prognostic criteria for low quality remission in SAD: gradual onset of the disorder, premorbid behavioral disorders, disease manifestation at the age of 16–21 years, use of PAS, schizophrenia or bipolar disorder in the family history [33].

Thus, researchers of SAD have a common opinion that this disease requires further research on the characteristics of epidemiology, clinical course, etiological factors and treatment [34,35]. A 20-year study has found a decrease in social functioning in all forms of endogenous psychosis, and this decrease was not associated with age or changes in antipsychotic therapy [30]. Thus, in the long-term prognosis, the lack of models of social support led to a loss of success achieved by a treatment at the beginning of the endogenous disease development [30]. These and other results of studies on the pathopersonalogical aspect of the clinic in SAD and its relationship with indicators of social adaptation need to be supplemented and generalized. The study on personality changes and the definition of their clinical nosospecific differences will make it possible to perform a differential diagnosis with a high level of reliability and determine the individual functional diagnosis. This will make it possible to optimize treatment and rehabilitation effects and increase the level of adaptation and quality of life of patients with SAD, as personality changes in schizophrenia are one of the fundamental manifestations of the disease and determine the functional outcome.

Maruta N. O. and Linska K. I. (2018) believe that the pathogenic impact of most mental illnesses, which include SAD, is multimodal and includes factors associated with the progression of clinical signs by endogenous mechanisms, adverse effects of psychopharmacological interventions and psychogenic (in particular, nosogenic) effects [36]. Each of the above vectors of the disease's impact on the personality is also a complex. Thus, the ideas about the clinical consequences of SAD formed during the twentieth century are now subject to revision, taking into account the multifactorial nature of their genesis and the phenomena of true and therapeutic pathomorphosis.

In schizophrenia and SAD, negative symptoms have a more pronounced transforming effect on the personality compared to positive symptoms and become a "character trait" – a component of the personality changes complex [37]. According to R. J. Gurrera, R. W. McCarley, D. Salisbury (2014), deficient symptoms, combined with neurocognitive disorders, form the core of personality disorders after the first hospitalization for psychosis of the schizophrenic spectrum [38]. The study on anxiety in SAD and AD performed by V. M. Goghari and M. Harrow (2019), which lasted 20 years, has found that the presence of anxiety at the beginning of the disease gave hope for a high quality remission, was a prognostic sign of declining global functioning in general [39]. İpçi K., Yildiz M., İncedere A. and co-authors (2020) have obtained data that the indicators of "subjective recovery" as a component of "clinical recovery" in patients with SAD in stable remission were directly correlated with indicators of general and social functioning, self-esteem and showed an inverse correlation with the severity of psychopathological symptoms, the level of internal stigma, depression [40].

In contrast to healthy individuals, the personal profile of patients with schizophrenia and SAD has higher rates

of neuroticism, decreased openness and extraversion [41]. According to C. Ridgewell, J. U. Blackford, M. McHugo, and co-authors (2017), the above personal characteristics can also predict indicators of quality of life and overall functioning, as high neuroticism is associated with low quality of life [41].

The presence of therapeutic pathomorphosis of SAD remission has proved that the use of atypical antipsychotics, modern antidepressants and normothymics leads to a decrease in the number of patients with depressive and subdepressive symptoms in remission. Significant difficulties arise in assessing and interpreting the existing semiotics of emotional and volitional disorders in EEP and differentiation of mild manifestations of emotional and volitional decline, subdepression (endogenous, psychogenic and neuroleptic) and "depressive behavior", which occurs in periods of remission / intermission. Grove T. B., Yao B. and Mueller S. A. (2018) have suggested that the above emotional disorders deepened and possibly caused neurocognitive deficits, the formation of which has been proven after the first episodes of SAD [42].

According to most authors, cognitive impairment is common to all EEP (in comparison with healthy individuals); however, a study by A. J. Lynham, L. Hubbard, K. E. Tansey and co-authors (2018) has shown a dependence on the clinical type of SAD. Thus, cognitive impairment in the mixed type is less pronounced and close to the indicators of AD, and in the depressive type – cognitive impairment is more pronounced and almost no different from the indicators in schizophrenia [43]. In addition, patients with SAD and schizophrenia show lower level of intellectual indicators in comparison with AD [23].

It was found that cognitive disorders are "family" in nature, confirming the continuum model – increasing severity of neurocognitive deficit in the direction of AD – SAD – schizophrenia and the relationship of neurocognitive disorders with the presence of personality characteristics of cluster A by DSM-V [44].

Complementary (or alternative) to psychometric assessment of clinical symptoms, there are "biobehavioral" methods for measuring negative symptoms using objective voice, speech, gesture, facial expressions, decision-making, electrophysiological, neurobiological characteristics [31].

The concept of "social cognition" was highlighted and considered a more important factor in successful social functioning than cognition in general. According to B. E. Buck, K. M. Healey and E. C. Gagen et al. (2020), the factor structure of social cognition disorders in schizophrenia and SAD consists of hostile style (caused by positive and general psychopathological symptoms) and social cognition skills disorders (caused by negative psychopathological symptoms) [45].

It is revealed that the ability to recognize emotions by facial expression is a key component of socio-emotional competence and impaired in SAD [46]. Studies confirm that facial emotion recognition disorders are specific to negative emotions and that there is a link between this deficiency and cognitively disorganized symptoms, regardless of the general cognitive level. In a study, S. J. Barkl, S. Lah, A. W. Harris, and L. M. Williams (2014) obtained the results of a lacking recognition of certain emotions in the examination of a face after the first psychotic episode of EEP, which allows us to consider these symptoms as premorbid [47]. The increase in the deficit of emotion recognition by facial expression is identified in the direction of AD – SAD – schizophrenia, in addition, the structure and features of recognition disorders are similar in patients and their relatives, which gives grounds to attribute these signs to the endophenotype of EEP [48].

Conclusions

1. Clinical manifestations of SAD in remission and intermission are the least studied aspect of clinic, which dictates the need to assess both residual psychopathological symptoms and the degree of preservation / loss of social adaptation / functionality.

2. Numerous studies have shown that the leading factors of maladaptation in EEP (including SAD) are a wide range of psychopathological symptoms of personality changes and closely related neurotic and cognitive symptoms arising from endogenous, exogenous (psychopharmacological) psychogenic (by the mechanisms of nosogenic influence and stigmatization) factors.

3. Each of the abovementioned concepts is again heterogeneous and there are no verified tools for their evaluation. Thus, the content of the concepts of "forecast", "outcome", "maladaptation" in SAD remains unified.

4. A comprehensive study of non-psychotic symptoms in patients with SAD will expand the theoretical understanding of this nosological unit and it will be used for differential diagnosis, identification of necessary psychopharmacological, psychotherapeutic and rehabilitation interventions to prevent social maladaptation of patients.

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