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Is the Current Diagnosis of Schizophrenia Useful or Harmful?

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ABSTRACT

In recent decades, evidence has appeared in various scientific fields—genetic, psychopharmacological, neuro-psychological, etc.—which makes it difficult to maintain the positive and negative syndrome of schizophrenia under one and the same diagnosis. On the other hand, there are social and legal reasons recommending the conception of these two syndromes as different entities. In this paper, we conduct appropriate bibliographical researches to reveal these evidences. We discuss these findings and conclude proposing the split of positive and negative syndromes of schizophrenia in two different disorders.

KEYWORDS

Antisocial Personality Disorder; Drug Abusers; Genetic Markers; Neurocognitive Deficits; Negative Syndrome; Positive Symptoms

1. Introduction

According to the current concept of schizophrenia, this psychiatric disorder includes positive and negative symptoms, but not necessary for both be present to make such diagnosis. The inclusion of negative and positive syndromes under the same diagnosis has been questioned since years ago [1] and until present [2]. Many authors defend that these two syndromes are of different nature, not only because of different symptoms, but also because of its different response to antipsychotic drugs, and even its different pathogenesis.

In addition, many neuro-psychological, neuro-physiological, and neuro-psychopharmacological hypotheses have appeared during the last decades, which provide evidence about the different nature of these two syndromes.

The paranoid schizophrenias constitute almost half of the total schizophrenic diagnoses [3]. They are often erroneously diagnosed, being necessary to change such diagnosis in a second instance [4]. Now then, schizophrenic diagnosis carries a strong social stigma [5] resulting in a great damage for these wrong diagnosed patients.

In light of these facts, we think it is not advisable to maintain negative or positive syndromes under the only and same diagnosis of schizophrenia.

We will carry out a bibliographical review of different areas in which these two syndromes appear to have different profiles. This research includes the following sections:

- Distortion of Bleuler's concept of schizophrenia;
- Different performance of negative and positive schizophrenic syndromes regarding genetic and neurocognitive markers;
- Analysis of both syndromes in light of the glutamate hypothesis of schizophrenia.

2. Distortion of Bleuler's Concept of Schizophrenia

When Eugen Bleuler created the neologism schizophrenia a hundred years ago, to replace Kraepeilinian term dementia praecox, he referred to a group of processes of

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constitutional nature characterized by fragmentation of the psyche as a whole. From that change, psychical functions (cognition, affectivity, motivation, execution, etc.) start to act in a not coordinated way. In fact, the term schizophrenos, coming from Greek, means fragmented psyche [6].

This uncoordinated and fragmented functioning generates symptoms that are the direct expression of the division suffered by the psyche. These symptoms consist in a drop or even a suspension of some psychic functions, and that is why they are termed negative symptoms:

- Alogia: denotes the cognitive function deficits. Thought is disorganized, the words are not linke by logical reasoning, and speech is disjointed and often incomprehensible.
- Flat affectivity: the subject seems to have lost the ability to feel or express emotions appropriately, and speaks with coldness and indifference, regardless of the emotional charge of the events described.
- Ambivalence: emotions and thoughts opposites coexist in the patient's mind at the same time. This inability to separate opposing psychic productions leads to a paralysis of will, called amotivational syndrome.
- Autism: due to the three symptoms above, the patient moves away of common reality, taking refuge in an imaginary world where he neglects his obligations, including basic tasks necessary to survive.

Bleuler considered these negative symptoms as the primary and characteristic expression of psyche fragmentation. Alongside them other symptoms can appear: hallucinations, delusions, anguish, psychomotor agitation, etc. These symptoms, which can be the most outstanding in some cases, are always conceived by Bleuler as secondary to the splitting of the mind.

This conception Bleuler suffered successive and progressive changes over time, until it lost its initial meaning. First, it appears the concept of reactive schizophrenia [7], which is a schizophrenic-like syndrome reactive to different psycho-social stressing situations. It usually ends in a fast and non-defective way. Generally negative symptoms don't appear in this benign form of schizophrenia.

A second step was the appearance of DSM-II in 1968, which already contemplated the possibility of schizophrenic diagnosis, e.g. paranoid schizophrenia, without any negative symptom. Since then, following editions of DSM and ICD allow the diagnosis of schizophrenia without the concurrence of any of Bleuler's primary symptoms [8].

3. Different Performance of Negative and Positive Syndromes in the Light of Genetic and Neurocognitive Markers

All manuals of psychiatry attributed a high hereditary component to schizophrenic disorder. For example, Kaplan *et al.* [9] report 47% risk of developing schizophrenia in monozygotic twins compared to a risk of 1% in the general population. In turn, Bray *et al.* [10] and Harrison and Weinberger [11] argue that schizophrenia has a heritability of approximately 80%.

In recent years there have been a number of publications of potential genetic markers of schizophrenia. Allen *et al.* [12], after conducting a meta-analysis of over a thousand studies on the genetics of schizophrenia, they conclude that there is no genetic variant that serves to determine genetic susceptibility to schizophrenia.

Meanwhile, Gallander [13], in a monograph devoted entirely to this subject, notes that while wide-genome studies have linked certain dopamine-related genes with the onset of schizophrenia, however it has not been possible to identify any specific biomarker of the disease.

Finally, Cross-Disorder Group of Psychiatric Genomics Consortium [14], in a recent single-nucleotide polymorphism analysis, found common genetic factors shared by five psychiatric disorders: autism spectrum disorder, attention deficit-hyperactivity disorder, bipolar disorder, major depressive disorder, and schizophrenia.

This lack of specific biomarkers for a disease that, in theory, involves a high inherited risk, is very surprising. We believe that the limited information provided by these studies is due in part to the erroneous inclusion of positive and negative syndromes in a single diagnosis. In fact, when these studies focus only on negative/disorganized schizophrenias the results seem more promising [15].

On the other hand, there are numerous neuropsychological researches about neurocognitive deficits as markers for schizophrenia. These neurocognitive deficits appear in children and young people who later develop schizophrenia. These shortfalls have been postulated as potential neurobiological markers of this disorder. They concern many areas of development: neuromotor, language, cognitive, emotional and interpersonal development [16,17]. Well, the researches carried out link these neurocognitive deficits more clearly with negative symptoms [18].

4. Different Performance of Negative and Positive Syndromes Regarding Glutamatergic Hypothesis of Schizophrenia

A new pharmacological hypothesis of schizophrenia came to light years ago: the glutamate hypothesis. This new pharmacological model of schizophrenia has been gaining more authority in the last two decades, and it is a complementary explanation to the former dopaminergic hypothesis [19-20]. According glutamatergic hypothesis, an altered glutamate activity can underlie the symptoms

of schizophrenia, with two opposite proposals about it.

Several authors defend an increased glutamatergic activity as pathogenic mechanism of certain schizophrenic symptoms [21,22]. Some of these investigations show evidences indicating that this increase of glutamate neurotransmission correlates more clearly with the positive symptoms of schizophrenia [20].

On the other hand, there are also investigations defending the opposite pathogenic mechanism, that is, a decreased glutamate activity as pathogenic mechanism of schizophrenia [23,24]. That seems to be particularly valid for negative symptoms and these authors postulate a rising of glutamate activity as treatment of these negative symptoms of schizophrenia [25].

So, negative and positive syndromes seem to have a different profile regarding glutamate hypothesis of schizophrenia.

5. Discussion

So far we have referred to neuro-cognitive and neurophysiological pieces of information pointing that negative and positive syndromes of schizophrenia have a different, even, opposed nature. But, at this moment we consider necessary to add other kind of information that reinforces our proposal about the necessity of individuate these two syndromes. We are referring to the fact that current diagnosis of schizophrenia, which is possible with the presence of two positive symptoms, increases the probabilities of a misdiagnosis [4]. Many patients with hallucinations and delusions, without apparent cause, and having time criteria of schizophrenia, receive such diagnosis. After some time it is possible to evidence the existence of a neurological or psycho-toxic cause that explains better the origin of such syndrome. At that moment it is necessary to change the first diagnosis and substitute it, for example, by a hallucinatory-delusional organic disorder due to a cerebral lupus until this moment neurologically asymptomatic, event well established after many years ago [26]. Now, during this time the patient has been burdened with a strong stigmatic diagnosis [5].

Very often this wrong diagnosis occurs in patients with a borderline personality disorder (BPD). In fact, sometimes it is difficult the differential diagnosis between schizophrenia and BPD [27]. This is especially true when the borderline patient is also a drug abuser, a very often association [28]. It is well known that many psycho-toxic substances produce a like-schizophrenic syndrome, often very difficult to differentiate of a true paranoid schizophrenia [29].

However, these two features—use of recreational drugs and such personality disorder—in the same person are indicative of potential aggressiveness. Indeed, patients diagnosed in the first instance of paranoid schizophrenia and later of personality disorder with abuse and/or psycho-toxic substances dependence frequently commit aggressive and/or criminal acts [30,31].

This confusion of paranoid schizophrenia with paranoid reaction in recreational drug abusers with personality disorder is contributing to exacerbate the consideration of schizophrenic people as dangerous and, so, increasing the harmful stigma of this nosological entity. This is one more reason to separate the positive syndrome of the schizophrenic diagnosis.

Therefore, in addition to the previously mentioned scientific reasons, there are also important social and legal arguments that advise to put end to the current situation in which the only presence of two positive symptoms are enough to diagnose schizophrenia.

6. Conclusion

There are scientific and social reasons that recommend the individuation of the negative and positive syndromes of schizophrenia in two different nosological entities.

Conflict of Interest Statement

None.

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