

Schizophrenia: A Systematic Review

Preksha Saparia*, Akshat Patel, Heer Shah, Kirtan Solanki, Aashal Patel and Maulin Sahayata

Second Professional Year Medical Students of Gujarat Cancer Society Medical College, Hospital & Research Centre, (GCSMCH & RC) -Asarwa, Ahmedabad, Gujarat, India

Corresponding Author*

Preksha Saparia
GCS medical college, Hospital & Research Centre,
Asarwa, Ahmedabad,
Gujarat, India.
Email: ppsapariya@gmail.com

Copyright: ©2022 Saparia, P. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Date of Submission: 12-June-2022, Manuscript No.: cep-22-69121; **Editor assigned:** 15-June 2022, PreQC No.: cep-22-69121 (PQ); **Reviewed:** 19-June-2022, QC No. cep-22-69121(Q); **Revised:** 22-June-2022, Manuscript No. CEP-22-6122 (R); **Published:** 28-June-2022, doi: 10.1111/j.1600-0447.2005.00687. 8(8).320

Abstract

Schizophrenia is a mental disorder characterized by disruptions in thought processes, perceptions, emotional responsiveness, and social interactions. Schizophrenia affects approx. 24 million people or 1 in 300 people (0.32%) worldwide. This rate is 1 in 222 people (0.45%) among adults. Here, a person's ability to think, feel and behave is affected. Difficulty with concentration and memory may also be present.

According to neurobiological theory, schizophrenia is the result of neurotransmitter dysregulation such as:

An Excess of Dopamine, Reduced Glutamate In The Cerebrospinal Fluid, Levels of Gamma-Aminobutyric Acid (Gaba) Expression In Prefrontal Cortex, Or Reduced Levels Of Serotonin Hence, one can say, Schizophrenia is the major mental illness of our time and causes serious disturbances for those with the condition as well as using up significant proportions of scarce health resources. This paper reviews the recent literature on advances in introduction, etiology, symptoms, and treatments. Methodological problems encountered in researching this condition are discussed. Advances in treatments offered for this condition have improved outcomes but whether the patient receives these treatments may depend on what local services are prepared to offer.

Keywords: Schizophrenia • Distruptions • Perceptions • Emotional responsiveness • Social interactions • Neurobiological theory • Neurotransmitter • Dysregulation • Dopamine • Glutamate • Cerebrospinal fluid • GABA • Prefrontal cortex • Serotonin

Introduction

What is schizophrenia?

The concept of schizophrenia is dying. Harried for decades by psychology, it now appears to have been fatally wounded by psychiatry, the very profession that once sustained it (Figure 1). Its passing will not be mourned. Today, having a diagnosis of schizophrenia is associated with a life-expectancy reduction of nearly two decades. By some criteria, only one in seven people recover. Despite heralded advances in treatments, staggeringly, the proportion of people who recover hasn't increased over time. Something is profoundly wrong. Part of the problem turns out to be the concept of schizophrenia itself. Arguments that schizophrenia is a distinct disease have been "fatally undermined". Just as we now have the concept of autism spectrum disorder, psychosis (typically characterized by distressing hallucinations, delusions, and confused thoughts) is also argued to exist along a continuum and in degrees. Schizophrenia is the severe end of a spectrum or continuum of experiences. Jim van Os, a professor of psychiatry at Maastricht University, has argued that we cannot shift to this new way of thinking without changing our language. As such, he proposes the term schizophrenia "should be abolished". In its place, he suggests the concept of a psychosis spectrum disorder. Another problem

is that schizophrenia is portrayed as a "hopeless chronic brain disease". As a result, some people given this diagnosis, and some parents, have been told cancer would have been preferable, as it would be easier to cure. Yet this view of schizophrenia is only possible by excluding people who do have positive outcomes. For example, some who recover are effectively told that "it mustn't have been schizophrenia after all". Schizophrenia, when understood as a discrete, hopeless, and deteriorating brain disease, argues van Os, "does not exist" [1].



Figure 1. Schizophrenia.

Breaking down breakdowns

Schizophrenia may instead turn out to be many different things. The eminent psychiatrist Sir Robin Murray describes how: I expect to see the end of the concept of schizophrenia soon ... the syndrome is already beginning to break down, for example, into those cases caused by copy number (genetic) variations, drug abuse, social adversity, etc. Presumably, this process will accelerate, and the term schizophrenia will be confined to history, like "drosy". Research is now exploring the different ways people may end up with many of the experiences deemed characteristic of schizophrenia: hallucinations, delusions, disorganized thinking and behavior, apathy, and flat emotion. Indeed, one past error has been to mistake a path for the path or, more commonly, to mistake a back road for a motorway. For example, based on their work on the parasite *Toxoplasma gondii*, which is transmitted to humans via cats, Researchers E. Fuller Torrey and Robert Yolken have argued that "the most important etiological agent (cause of schizophrenia) may turn out to be a contagious cat". It will not the proportion of people who recover hasn't increased over time. Something is profoundly wrong. Part of the problem turns out to be the concept of schizophrenia itself. Arguments that schizophrenia is a distinct disease have been "fatally undermined". Just as we now have the concept of autism spectrum disorder, psychosis (typically characterized by distressing hallucinations, delusions, and confused thoughts) is also argued to exist along a continuum and in degrees. Schizophrenia is the severe end of a spectrum or continuum of experiences. Jim van Os, a professor of psychiatry at Maastricht University, has argued that we cannot shift to this new way of thinking without changing our language. As such, he proposes the term schizophrenia "should be abolished". In its place, he suggests the concept of a psychosis spectrum disorder. Another problem is that schizophrenia is portrayed as a "hopeless chronic brain disease". As a result, some people given this diagnosis, and some parents, have been told cancer would have been preferable, as it would be easier to cure. Yet this view of schizophrenia is only possible by excluding people who do have positive outcomes (Figure 2) For example, some who recover are effectively told that "it mustn't have

been schizophrenia after all". Schizophrenia, when understood as a discrete, hopeless and deteriorating brain disease, argues van Os, "does not exist" [1].



Figure 2. Breaking down of schizophrenia.

Their Evidence does suggest that exposure to T. Gondii when young can increase the odds of someone being diagnosed with schizophrenia. However, the size of this effect involves less than a twofold increase in the odds of someone being diagnosed with schizophrenia. This is, at best, comparable to other risk factors, and probably much lower. For example, suffering childhood adversity, using cannabis, and having childhood viral infections of the central nervous system, all increase the odds of someone being diagnosed with a psychotic disorder (such as schizophrenia) by around two to threefold. More nuanced analyses reveal much higher numbers. Compared with non-cannabis users, the daily use of high-potency, skunk-like cannabis is associated with a fivefold increase in the odds of someone developing psychosis. Compared with someone who has not suffered trauma, those who have suffered five different types of trauma (including sexual and physical abuse) see their odds of developing psychosis increase more than fiftyfold. Other routes to "schizophrenia" are also being identified. Around 1% of cases appear to stem from the deletion of a small stretch of DNA on chromosome 22, referred to as 22q11.2 deletion syndrome. It is also possible that a low single-digit percentage of people with a schizophrenia diagnosis may have their experiences grounded in inflammation of the brain caused by autoimmune disorders, such as anti-NMDA receptor encephalitis, although this remains controversial. All the factors above could lead to similar experiences, which we in our infancy have put into a bucket called schizophrenia. One person's experiences may result from a brain disorder with a strong genetic basis, potentially driven by an exaggeration of the normal process of pruning connections between brain a cell that happens during adolescence. Another person's experiences may be due to a complex post-traumatic reaction. Such internal and external factors could also work in combination.

Either way, it turns out that the two extreme camps in the schizophrenia wars—those who view it as a genetically-based neurodevelopmental disorder and those who view it as a response to psychosocial factors, such as adversity—both had parts of the puzzle. The idea that schizophrenia was a single thing, reached by a single route, contributed to this conflict [2].

Symptoms

When the disease is active, it can be characterized by episodes in which the person is unable to distinguish between real and unreal experiences. As with any illness, the severity, duration, and frequency of symptoms can vary; however, in persons with schizophrenia, the incidence of severe psychotic symptoms often decreases as the person becomes older. Not taking medications as prescribed, the use of alcohol or illicit drugs, and stressful situations tend to increase symptoms. Symptoms fall into three major categories: Positive symptoms: (Those abnormally present) Hallucinations, such as hearing voices or seeing things that do not exist, paranoia, and exaggerated or distorted perceptions, beliefs, and behaviors.

Negative symptoms: (those abnormally absent) A loss or a decrease in the ability to initiate plans, speak, express emotion, or find pleasure. Disorganized symptoms: Confused and disordered thinking and speech, trouble with logical thinking, and sometimes bizarre behavior or abnormal movements. Cognition is another area of functioning that is affected in schizophrenia leading to problems with attention, concentration, and memory, and declining educational performance. Symptoms of schizophrenia usually first appear in early adulthood and must persist for at least six months for a diagnosis to be made. Men often experience initial symptoms in their late teens or early 20s while women tend to show first signs of the illness in their 20s and early 30s. More subtle signs may be present earlier, including troubled relationships,

poor school performance, and reduced motivation. Before a diagnosis can be made, however, a psychiatrist should conduct a thorough medical examination to rule out substance misuse or other neurological or medical illnesses whose symptoms mimic schizophrenia [3].

Social skills, stigma, and rejection

People with schizophrenia are often stigmatized and avoided by others. Societal misinformation about schizophrenia undoubtedly plays a role in contributing to this. However, it is also reasonable to expect that some of the social deficits that characterize people with this disorder create difficulties for them and for the people with whom they interact. Over time, this may lead to increased negativity, social distance, and rejection by others [4]. In an empirical demonstration of this, Nisenon, Berenbaum, and Good (2001) asked student research assistants (all of whom had been selected because they had pleasant dispositions) to form brief friendships with patients who suffered from schizophrenia [5]. Throughout the study, which lasted 2 weeks the behavior of the research assistants changed [6]. Most notable was that the amount of negativity that the students showed toward the patients increased considerably [7]. But do poor social skills explain why people tend not to want to marry, befriend, or employ someone who has schizophrenia? At least in part, the answer appears to be yes. Penn, Kohlmaier, and Corrigan (2000) videotaped clinically stable outpatients with schizophrenia during a 3-minute role-play conversation with a confederate. Trained research assistants then rated the patients' social skills, noting such things as how well they made eye contact, how they spoke, and whether their conversation was interrupted by pauses or stutters [8]. The videotaped role-plays were then shown to 41 undergraduates, who were asked how much social distance they would want to have from each of the patients they had seen [9]. The best predictor of the students expressing a desire to avoid interacting with the patient in the videotape was how "strange" the patient was rated as being [10].

This, in turn, was predicted by the patient's overall social skills. In short, what this study demonstrates is that people who have poor social skills seem strange to us; and when we regard people as strange we tend to want to avoid them [11].

Etiology

Several studies postulate that the development of schizophrenia results from abnormalities in multiple neurotransmitters, such as dopaminergic, serotonergic, and alpha-adrenergic hyperactivity or glutaminergic and GABA hypoactivity. Genetics also plays a fundamental role - there is a 46% concordance rate in monozygotic twins and a 40% risk of developing schizophrenia if both parents are affected. The gene Neuregulin (NGR1), which is involved in glutamate signaling and brain development, has been implicated, alongside Dysbindin (DTNBP1), which helps glutamate release, and Catecholamine O-Methyl Transferase (COMT) polymorphism, which regulates dopamine function. As aforementioned, there are also several environmental factors associated with an enhanced risk of developing the disease:

- Abnormal fetal development and low birth weight.
- Gestational diabetes.
- Preeclampsia.
- Emergency cesarean section and other birthing complications.
- Maternal malnutrition and vitamin D deficiency.
- Winter births - associated with a 10% higher relative risk.
- Urban residence increases the risk of developing schizophrenia by 2% to 4%.

The incidence is also up to ten times greater in children of African and Caribbean migrants compared to whites, according to a study conducted in Britain [12]. The association between cannabis use and psychosis has been widely studied, with recent longitudinal studies suggesting a 40% increased risk, while also suggesting a dose-effect relationship between the use of the drug and the risk of developing schizophrenia [13].

Pathophysiology

There are three main hypotheses regarding the development of schizophrenia. The neurochemical abnormality hypothesis argues that an imbalance of dopamine, serotonin, glutamate, and GABA results in the psychiatric manifestations of the disease. It postulates that four main dopaminergic pathways are involved in the development of schizophrenia. This dopamine hypothesis attributes the positive symptoms of the illness

to excessive activation of D2 receptors via the mesolimbic pathway, while low levels of dopamine in the nigrostriatal pathway are theorized to cause motor symptoms through their effect on the extrapyramidal system. Low mesocortical dopamine levels resulting from the mesocortical pathway are thought to elicit the negative symptoms of the disease. Other symptoms such as amenorrhea and decreased libido may be caused by elevated prolactin levels due to decreased availability of tuberoinfundibular dopamine as a result of blockage of the tuberoinfundibular pathway. Evidence showing exacerbation of positive and negative symptoms in schizophrenia by NMDA receptor antagonists insinuates the potential role of glutamatergic hypo activity while serotonergic hyperactivity has also been shown to play a role in schizophrenia development [14]. There are also arguments that schizophrenia is a neurodevelopmental disorder based on abnormalities present in the cerebral structure, an absence of gliosis suggesting in utero changes, and the observation that motor and cognitive impairments in patients precede the illness onset. Conversely, the disconnect hypothesis focuses on the neuroanatomical changes seen in PET and fMRI scans (Figure 3). There is a reduction in grey matter volume in schizophrenia, present not only in the temporal lobe but in the parietal lobes as well. Differences in the frontal lobes and hippocampus are also seen, potentially contributing to a range of cognitive and memory impairments associated with the disease.

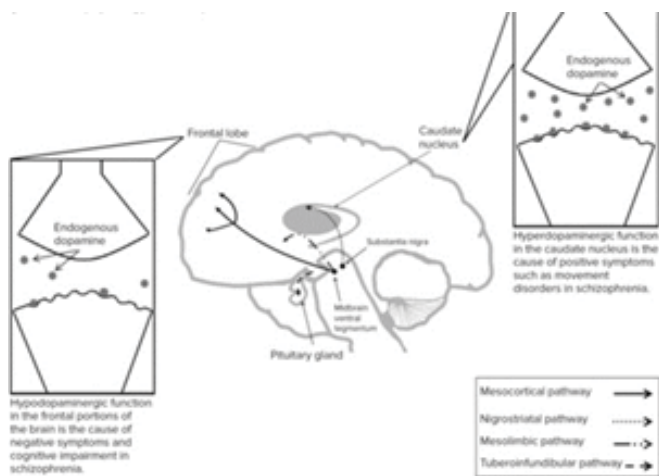


Figure 3. Pathophysiology.

Web differential diagnosis

Differential diagnoses that need to be considered are as follows:

- Bipolar I Disorder with psychotic features.
- Delusional disorders.
- Schizoaffective disorder.
- Brief psychotic disorder.
- Psychosis NOS.
- Certain personality disorders.
- Drug and medication-induced psychosis.

Online is a higher priority than simple web access in the connection among on the web and disconnected activism [15]. All in all, the present status of the writing lays out a fascinating picture of how web-based entertainment is used for the aggregate activity. The web is generally utilized for emancipatory activities to bring issues to light, rally individuals, set extremist plans, discuss and assess activities, yet additionally unfairly (by gatherings and specialists) to enrapture, misguide, and subdue undesirable activities. Indisputably, minority gatherings can all the more effectively connect and make themselves understood through online entertainment [16]. This gives online entertainment an extraordinary dynamic quality and pluralism, however, it might likewise isolate and spellbind social orders. Progressively, on the web and disconnected activism are indivisible and integral social-mental instruments for politicization, discussion, activation, and struggle [17].

Interventions for schizophrenia

In this section, we will discuss the various existing therapies used for treating schizophrenia symptoms as well as problems the patients face, such as unemployment, lack of education, and lack of social relationships [18].

Pharmacological intervention

It has been observed that full recovery from the symptoms of schizophrenia

occurs in 6% of individuals with schizophrenia after a single episode of psychosis [19]. In 39% of the patients, deterioration of symptoms has been reported [19]. Approximately, about one in seven individuals with schizophrenia achieve total recovery [20]. Table 1 identifies the issues related to the holistic management of schizophrenia and associated intervention options. The initial treatment of schizophrenia often includes various antipsychotic medications. The targets of antipsychotic medications are generally the symptoms of schizophrenia but not the root causes of it, such as stress and substance abuse (see above). As mentioned in Table 1, most antipsychotic drugs ameliorate hallucinations and delusions, while some attempt to also address the negative symptoms of schizophrenia. Antipsychotic medications are usually the only option for the treatment of schizophrenia. Most antipsychotic treatments work by reducing the positive symptoms of schizophrenia by blocking dopamine receptors [21].

In one research study by Girgis et al. [22], 160 individuals with schizophrenia were randomized to clozapine or chlorpromazine treatment for up to 2 years. The adherence to clozapine was found to be higher than that of chlorpromazine. In another study conducted on 34 individuals with schizophrenia, it was found that there was no beneficial effect of clozapine over conventional antipsychotics [23]. McEvoy et al. found that a large percentage of individuals with schizophrenia discontinued treatment due to the inadequate efficacy of some antipsychotic drugs [24]. An average daily dose of 523 mg/day and 600 mg/day of clozapine is effective in the treatment of positive and negative symptoms in individuals with schizophrenia [24]. Sanz-Fuentenebro et al. found that individuals with schizophrenia on clozapine continued their original treatment for a much longer period than patients on risperidone [25]. Specifically, the retention rate for clozapine was 93 point 4% whereas the retention rate for risperidone was 82 point 8%. However, patients in the clozapine group normally have more significant weight gain than those on risperidone [26]. In one study by Sahni et al. [27], a total of 63 patients were selected and randomly allocated to either clozapine or risperidone. The two groups were similar on sociodemographic variables including age, sex, education level, occupation, income, family type, and marital status. The mean duration of illness was 19 points 39 months, in the clozapine group, and 18 points 63 months in the risperidone group. There was a significant reduction of positive symptoms in both drugs. It was found that both clozapine and risperidone equally reduced positive symptoms whereas clozapine was much superior compared to risperidone in reducing negative symptoms. Clozapine has been found to reduce suicidal ideation in individuals with schizophrenia; Along these lines reported that the administration of clozapine in chronically psychotic patients has led to reduced suicidal ideation [28]. It was concluded that long-term treatment with clozapine resulted in a three-fold reduction in the risk of suicidal behaviors. Further, patients on clozapine are often administered metformin (500 mg twice daily) to lose weight. Aripiprazole is sometimes given along with clozapine to manage weight and improve metabolic parameters [29]. One study found that the administration of both aripiprazole and clozapine has led to a beneficial effect on the positive and general symptoms of individuals with schizophrenia, compared to clozapine alone. Antipsychotic drugs also help ameliorate disoriented behavior in day-to-day life. They are also used to improve cognitive impairment, which in turn improves the relationship and contributes to the attainment of education and employment. Antipsychotic drugs help improve disoriented behavior in day-to-day life. They are also used to improve relationships and enhance education [30, 31] and employment [32]. Table 1 summarizes the role of pharmacological intervention in the holistic management of schizophrenia [33].

TABLE 1. Abbreviations & Acronyms

OCD	Obsessive Compulsive Disorder
<i>T. Gondii</i>	<i>Toxoplasma Gondii</i>
DNA	Deoxyribose Nucleic Acid
Anti-NMDA Receptors	N-methyl-D-aspartate Receptors
GABA	Gamma-aminobutyric Acid
<i>NGR1</i>	<i>Negative Growth Regulatory Protein 1</i>
<i>DTNBP1</i>	<i>Dystrobrevin Binding Protein 1</i>
COMT	Catecholamine O-Methyl Transferase
PET	Positron Emmision Tomography
fMRI	Functional Magnetic Resonance Imaging
NOS	Not Specified Otherwise
CBT	Cognitive Behaviour Therapy
UK	United Kingdoms
NHS	National Health Sevices
PORT	Patient Outcomes Research Teams

Complementary intervention and diet

Found that the diets of schizophrenia patients contained more total fat and less fiber than the diets of a control group matched for age, gender, and education, although the intake of unsaturated fat was found to be similar in both groups. In another study studied the dietary intake of 30 individuals with schizophrenia living in assisted-living facilities in Scotland as well as a control group matched for sex, age, smoking, and employment status [34]. The majority of individuals with schizophrenia were overweight or obese, and saturated fat intake was higher than recommended in the diets for individuals with schizophrenia [35]. It was found that individuals with schizophrenia consumed less total fiber, retinol, carotene, vitamin C, vitamin E, fruit, and vegetables than the control group studied dietary habits of 102 individuals with schizophrenia with special emphasis on fruit and vegetable intake and smoking behavior [36, 37]. The study concluded that the patients (especially male patients) had poor dietary choices. Graham et al [38]. suggested that administering vitamin D to individuals with schizophrenia ameliorates their negative symptoms. In another study by [39], the dietary habits of a total of 146 adult community-dwelling individuals with schizophrenia were studied. It was observed that the patients consumed a higher quantity of food that includes protein, carbohydrate, and fat than that of a control group. Such habits can lead to cardiovascular diseases, type II diabetes, and systemic inflammation in individuals with schizophrenia [40]. These diseases are related to a short lifespan in individuals with schizophrenia [41]. In a research study by Joseph et al [42], it has been suggested that high-fiber diets can improve the immune and cardiovascular system, thereby, preventing premature mortality in schizophrenia.

Cognitive behavior therapy

Cognitive Behavior Therapy (CBT) is a therapeutic technique that helps modify undesirable mode of thinking, feeling and behavior. CBT involves practical self-help strategies, which are found to ameliorate positive symptoms in schizophrenia. CBT combines two kinds of therapies: "cognitive therapy" and "behavioral therapy." The combination of these two techniques often enables the patient to have healthy thoughts and behaviors. Morrison summarizes the use of CBT in individuals with schizophrenia to address the primary symptoms of illness as well as social impairments [43]. Morrison mentioned that many schizophrenia symptoms are resistant to pharmacological treatment and suggested CBT as an add-on to antipsychotics can be more effective than the administration of drugs alone. For example, several studies found that cognitive rehabilitation and CBT can ameliorate cognitive deficits and in turn positive symptoms [44, 45]. There are many techniques to alter thoughts and behavior using CBT. One research study described the key elements of CBT for schizophrenia [46], and concluded that various CBT techniques can be used effectively in schizophrenia. One of the techniques, known as cognitive restructuring, includes challenging the patient to come up with an evidence to prove that their beliefs are real. This technique assists the client to realize that they have delusions. This technique assists the patient to learn to identify and challenge negative thoughts, and modify the faulty thoughts with more realistic and positive ones. CBT was also found to be effective for managing homelessness. As CBT ameliorates cognitive impairment, it helps improve relationship and contributes positively to entertainment. Behavioral therapy aims to assist the patient to learn to modify their behavior. For example, they may rehearse conversational skills so that they can use these newly learned skills in social situations. CBT assists the patients in engaging in social circles which affects friendship and relationship as indicated in Table 1. There have been validation studies of CBT in schizophrenia over the last 15 years. In schizophrenia, CBT is one of the most commonly used therapy in the UK (generally in addition to medications). In fact CBT has been recommended as first-line treatment by the UK National Health Service (NHS) for individuals with schizophrenia. Similarly, the American Psychiatric Association recommended CBT for individuals with schizophrenia [47]. Recently the US Schizophrenia Patient Outcomes Research Team (PORT) has recommended CBT for patients who have persistent psychotic symptoms.

Yoga therapy

Yoga therapy can also manage schizophrenia symptoms, often in combination with pharmacological medications [48]. Pharmacological intervention alone might not produce all the desirable effects in managing schizophrenia symptoms, especially negative symptoms [49]. Yoga, as an add-on to antipsychotic medications, helps treat both positive and negative symptoms, more than medications alone. Furthermore, pharmacological interventions often produce obesity in schizophrenia [49]. Yoga therapy has been found to help reduce weight gain due to the administration of antipsychotic medications. Pharmacological interventions might cause endocrinological and menstrual dysfunction which may be positively treated by yoga therapy [49]. In a research study by Gangadhar et al. two groups of patients on antipsychotic medications were examined [49]. In one

group, yoga therapy was administered. In the other group, a set of physical exercises was applied. Both groups were trained for 1 month (at least 12 sessions). The yoga group showed better negative symptoms scores than the other group. Similarly, yoga therapy resulted in better effects on social dysfunction than the other group. Along these lines, Vancampfort et al. found that practicing yoga reduces psychiatric symptoms and improves the mental and physical quality of life, and also reduces metabolic risk [50]. The most probable explanation of the effectiveness of yoga therapy is the production of oxytocin in the body. Oxytocin is a hormone which contributes to wellbeing. In one research study, 40 patients were administered oxytocin along with antipsychotic medications [51]. It was found that both negative and positive symptoms improved in those patients. The results of yoga therapy are manifold. Yoga therapy can lead to a reduction in psychotic symptoms and depression, improvement in cognition, and an increase in quality of life. Table 1 identifies the issues related to holistic management of schizophrenia and associated yoga intervention options [51].

Comorbidities and their management in schizophrenia

The commoner co morbidities and their management are as follows:

Schizophrenia with substance use disorders: The most commonly abused drugs include alcohol, cannabis, and cocaine, and the use of these substances markedly worsens the course of illness. In addition, between 50% and 90% of schizophrenic patients smoke cigarettes, contributing to increased mortality from medical illness. Smoking also decreases the effectiveness of some antipsychotics. Co-morbid substance use disorder in schizophrenia is associated with greater deterioration of function, higher rates of psychotic relapse, and increased social dysfunction. Furthermore, the dual diagnosis is associated with increased suicidal ideation and victimization.

The uses of longer-acting oral medications and depot injections have also been shown to help, owing to poor treatment adherence in patients with dual diagnoses. Clozapine treatment seems to be most effective in reducing alcohol and substance abuse in schizophrenia. The increased potential for adverse effects from mixing prescribed medications with abused substances should also be considered in dual-diagnosis patients. Sibutramine, an anti-obesity medication is often used by patients to lose weight [52].

Depression in schizophrenia: The prevalence of depression in schizophrenia is 25%-81%. The presence of depressive symptoms in schizophrenic patients worsens quality of life and increases the risk for danger to self and others (including suicide), psychotic relapse, substance-related problems, and psychiatric hospitalization. In conclusion, concurrent depressive symptoms in schizophrenia are common and are associated with significantly poorer long-term functional outcome. Active treatment of depression targeting specific symptoms should be a standard of care.

OCD in schizophrenia

The common themes are of contamination, sexual, somatic, religious, aggressive, and somatic, with or without accompanying compulsions. These manifestations overlap with the underlying psychosis, demonstrating overvalued ideations and delusional manifestation. Recent evidence suggests a poorer clinical course and long-term outcome, as well as greater neuropsychological dysfunction.

The syndrome may manifest during the prodromal phase or during active psychotic illness, as obsessive ruminations during recovery or the remission phase, as a de novo OC syndrome associated with treatment with Atypical Antipsychotics, or as a concurrent independent OC disorder. Treatment is use of adjunctive anti-OC pharmacotherapy with antipsychotics like haloperidol. Cognitive Behavior Therapy could also be used [53].

Eating disorder in Schizophrenia

It is important to manage aggressive behavior in schizophrenia. Epidemiology revealed that co-occurring substance abuse and intoxication increase the risk of violence in patients with schizophrenia. Some studies have reported that ten percent of patients attack others within 24 hours after their admission in hospitals. Transient violence is associated with environmental factors and positive symptoms of psychosis. Several medication strategies are considered for treatment of persistently aggressive psychotic patients, including conventional neuroleptics, atypical neuroleptics, and mood stabilizers like sodium Valproate and occasionally lithium carbonate. A recent study revealed the effectiveness of clozapine on violence in patients with schizophrenia [54].

Schizophrenia and persistent aggressive behavior

It is important to manage aggressive behavior in schizophrenia. Epidemiology revealed that co-occurring substance abuse and intoxication increase the risk of violence in patients with schizophrenia. Some studies have reported

that ten percent of patients attack others within 24 hours after their admission in hospitals. Transient violence is associated with environmental factors and positive symptoms of psychosis. Several medication strategies are considered for treatment of persistently aggressive psychotic patients, including conventional neuroleptics, atypical neuroleptics, and mood stabilizers like sodium Valproate and occasionally lithium carbonate [55]. A recent study revealed the effectiveness of clozapine on violence in patients with schizophrenia

Conclusion

The conclusion about the whole picture of Schizophrenia could be drawn to point out what actually it is how it is prognosed, diagnosed, treated, and related. This clinical condition affects a person mentally which indirectly is an indication or whether to say a precautious warning to start living a "mentally healthy" life along with being socially & physically competent.

Presented below are the words of John Forbes Nash Jr. (noble prize winning mathematician and subject of the award winning book and movie: A Beautiful Mind)

He shares a piece of him while recalling his battle against schizophrenia: "I thought of the voices as...something a little different from aliens. i thought of them more like angels...It's really my subconscious talking, it was really that...i know that now."

References

1. Hooley JM. Social factors in schizophrenia. Current directions in psychological science. 2010 Aug;19(4):238-42.
2. Hooker C et al. Emotion processing and its relationship to social functioning in schizophrenia patients. *Psychiatry research*. 2002 Sep 15;112(1):41-50.
3. Mueser, et al. "Social skills and social functioning." (1998)
4. Mueser, et al. "Prevalence and stability of social skill deficits in schizophrenia." *Schizophrenia research* 5.2 (1991): 167-176
5. Mueser, et al. "Gender, social competence, and symptomatology in schizophrenia: a longitudinal analysis." *Journal of Abnormal Psychology* 99.2 (1990): 138.
6. Nisenson, et al. "The development of interpersonal relationships in individuals with schizophrenia." *Psychiatry* 64.2 (2001): 111-125.
7. Penn, et al. "Interpersonal factors contributing to the stigma of schizophrenia: social skills, perceived attractiveness, and symptoms." *Schizophrenia research* 45.1-2 (2000): 37-45
8. Pinkham, et al. "Neurocognitive and social cognitive predictors of interpersonal skill in schizophrenia." *Psychiatry research* 143.2-3 (2006): 167-178.
9. Zhu, et al. Impairments of social cues recognition and social functioning in Chinese people with schizophrenia. *Psychiatry and Clinical Neurosciences*, 61, 149-158
10. Messias, E.L., et al. Epidemiology of schizophrenia: review of findings and myths. *Psychiatric Clinics of North America*. 2007 Sep 1;30(3):323-338.
11. Davis, et al. "A review of vulnerability and risks for schizophrenia: Beyond the two hit hypothesis." *Neuroscience & Biobehavioral Reviews* 65 (2016): 185-194.
12. Patel, et al. "Schizophrenia: overview and treatment options." *Pharmacy and Therapeutics* 39.9 (2014): 638.
13. Carter, Marcia, Jean. "Diagnostic and statistical manual of mental disorders." *Therapeutic recreation journal* 48.3 (2014): 275.
14. Case, Jane, K. "Ferri's Differential Diagnosis: A Practical Guide to the Differential Diagnosis of Symptoms, Signs, and Clinical Disorders." *Mayo Clinic Proceedings*. Vol. 81. No. 10. Elsevier, 2006.
15. Paris, Joel. "Differential diagnosis of borderline personality disorder." *Psychiatric Clinics* 41.4 (2018): 575-582.
16. Jablensky, Assen. "The diagnostic concept of schizophrenia: its history, evolution, and future prospects." *Dialogues in clinical neuroscience* (2022).
17. Morgan, V. A., et al. "Psychosis prevalence and physical, metabolic and cognitive co-morbidity: data from the second Australian national survey of psychosis." *Psychological medicine* 44.10 (2014): 2163-2176.
18. Jääskeläinen, E., et al. "Una revisión sistemática y metaanálisis de la recuperación en la esquizofrenia." *Schizophrenia Bulletin* 39.6 (2013): 1296-1306.
19. Figgins, et al. "Self-reported physical activity levels of the 2017 Royal Australian and New Zealand College of Psychiatrists (RANZCP) conference delegates and their exercise referral practices." *Journal of Mental Health* 29.5 (2020): 565-572.
20. Girgis, et al. "Clozapine v. chlorpromazine in treatment-naive, first-episode schizophrenia: 9-year outcomes of a randomised clinical trial." *The British Journal of Psychiatry* 199.4 (2011): 281-288.
21. Woerner, et al. "Clozapine as a first treatment for schizophrenia." *American Journal of Psychiatry* 160.8 (2003): 1514-1516.
22. McEvoy, Joseph, P., et al. "Effectiveness of clozapine versus olanzapine, quetiapine, and risperidone in patients with chronic schizophrenia who did not respond to prior atypical antipsychotic treatment." *American Journal of Psychiatry* 163.4 (2006): 600-610.
23. Sanz-Fuentenebro, Javier, et al. "Randomized trial of clozapine vs. risperidone in treatment-naïve first-episode schizophrenia: results after one year." *Schizophrenia research* 149.1-3 (2013): 156-161.
24. Taylor, D. M., and R. McAskill. "Atypical antipsychotics and weightgain a systematic review." *Acta Psychiatrica Scandinavica* 101.6 (2000): 416-432.
25. Sahni S, et al. Comparative study of clozapine versus risperidone in treatment-naive, first-episode schizophrenia: A pilot study. *The Indian Journal of Medical Research*. 2016 Nov;144(5):697.
26. Hennen, John, and Ross J. Baldessarini. "Suicidal risk during treatment with clozapine: a meta-analysis." *Schizophrenia research* 73.2-3 (2005): 139-145.
27. Muscatello, Maria Rosaria A., et al. "Effect of aripiprazole augmentation of clozapine in schizophrenia: a double-blind, placebo-controlled study." *Schizophrenia research* 127.1-3 (2011): 93-99.
28. Lysaker, Paul H., et al. "Effects of cognitive behavioral therapy on work outcomes in vocational rehabilitation for participants with schizophrenia spectrum disorders." *Schizophrenia research* 107.2-3 (2009): 186-191.
29. Drake, et al. *Individual placement and support: an evidence-based approach to supported employment*. Oxford University Press, 2012.
30. Kinoshita, et al. "Supported employment for adults with severe mental illness." *Cochrane Database of Systematic Reviews* 9 (2013).
31. Brown, Steve, et al. "The unhealthy lifestyle of people with schizophrenia." *Psychological medicine* 29.3 (1999): 697-701.
32. McCreadie, Robin, et al. "Dietary intake of schizophrenic patients in Nithsdale, Scotland: case-control study." *Bmj* 317.7161 (1998): 784-785.
33. Gothelf, et al. "Weight gain associated with increased food intake and low habitual activity levels in male adolescent schizophrenic inpatients treated with olanzapine." *American Journal of Psychiatry* 159.6 (2002): 1055-1057.
34. Kalaydjian, A. E., et al. "The gluten connection: the association between schizophrenia and celiac disease." *Acta Psychiatrica Scandinavica* 113.2 (2006): 82-90.
35. McNamara, et al. "Deficits in docosahexaenoic acid and associated elevations in the metabolism of arachidonic acid and saturated fatty acids in the postmortem orbitofrontal cortex of patients with bipolar disorder." *Psychiatry research* 160.3 (2008): 285-299.
36. Graham, K. A., et al. "Relationship of low vitamin D status with positive, negative and cognitive symptom domains in people with first-episode schizophrenia." *Early intervention in psychiatry* 9.5 (2015): 397-405.
37. Strassnig, Dietary intake of patients with schizophrenia." *Psychiatry (Edgmont)* 2.2 (2005): 31.
38. Kraft. "Schizophrenia, gluten, and low-carbohydrate, ketogenic diets: a case report and review of the literature." *Nutrition & Metabolism* 6.1 (2009): 1-3.
39. Mao-Sheng, Ran, et al. "Mortality in people with schizophrenia in rural China." *The British Journal of Psychiatry* 190.3 (2007): 237-242.
40. Joseph, Jamie, et al. "Modified Mediterranean diet for enrichment of short chain fatty acids: potential adjunctive therapeutic to target immune and metabolic dysfunction in schizophrenia?." *Frontiers in*

- Neuroscience* 11 (2017): 155.
41. Ganguly, et al. "Holistic management of schizophrenia symptoms using pharmacological and non pharmacological treatment." *Frontiers in public health* 6 (2018): 166.
 42. Eack, et al. "Effects of cognitive enhancement therapy on employment outcomes in early schizophrenia: results from a 2-year randomized trial." *Research on social work practice* 21.1 (2011): 32-42.
 43. Subramaniam, et al. "Computerized cognitive training restores neural activity within the reality monitoring network in schizophrenia." *Neuron* 73.4 (2012): 842-885.
 44. Gumley, A., et al. "QUIET NARCISSISM CONTINUED FROM PG. 9." *IACP PRESIDENT'S MESSAGE STEFAN G. HOFMANN, PHD PRIMUM NON NECERA* 47 (2015): 11.
 45. Esscher, et al. "Suicides during pregnancy and 1 year postpartum in Sweden, 1980–2007." *The British Journal of Psychiatry* 208.5 (2016): 462-469.
 46. Jha, Arun. "Yoga therapy for schizophrenia." *Acta Psychiatrica Scandinavica* 117.5 (2008): 397-397.
 47. Sohl, et al. "Development of the beliefs about yoga scale." *International Journal of Yoga Therapy* 21.1 (2011): 85-91.
 48. Vancampfort, et al. "Systematic review of the benefits of physical therapy within a multidisciplinary care approach for people with schizophrenia." *Physical therapy* 92.1 (2012): 11-23.
 49. Feifel, David. "Is oxytocin a promising treatment for schizophrenia?" *Expert review of neurotherapeutics* 11.2 (2011): 157-159.
 50. Chouljian, Tandy L., et al. "Substance use among schizophrenic outpatients: prevalence, course, and relation to functional status." *Annals of Clinical Psychiatry* 7.1 (1995): 19-24.
 51. Mason, et al. "The course of schizophrenia over 13 years: a report from the International Study on Schizophrenia (ISoS) coordinated by the World Health Organization." *The British Journal of Psychiatry* 169.5 (1996): 580-586.
 52. Cuffel, Brian J. "Violent and destructive behavior among the severely mentally ill in rural areas: evidence from Arkansas' community mental health system." *Community mental health journal* 30.5 (1994): 495-504.
 53. Kane John, M. "Improving treatment adherence in patients with schizophrenia." *The Journal of Clinical Psychiatry* 72.9 (2011): 27738.
 54. Wang, et al. "Overexpression of an Arabidopsis peroxisomal ascorbate peroxidase gene in tobacco increases protection against oxidative stress." *Plant and Cell Physiology* 40.7 (1999): 725-732.
 55. Green, Alan I., et al. "Alcohol and cannabis use in schizophrenia: effects of clozapine vs. risperidone." *Schizophrenia research* 60.1 (2003): 81-85.

Cite this article: Saparia, P., et al. Schizophrenia : A Systematic Review. Clin Exp Psychol. 2022, 08 (07), 065-070.