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A Review on Schizophrenia

Mohan Kumar M

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ABSTRACT
Schizophrenia is a most common Chronic psychotic disorder. The psychopathological signs and symptoms of schizophrenia are clustered into three principal categories: positive, negative and cognitive. Schizophrenia may result in hallucinations, delusions, and disorganizedthinking whichimpairs the daily activities and can be disabling. Environmental and social factors may also play a important role in the development of schizophrenia..To obtain long term outcome both pharmacological and Non Pharmacological treatment is required.Positive and Negative Syndrome Scale is a gold standard measure of cure efficacy which is used to measure the clinical response to pharmacological treatment. Antipsychotic drugs are classified into two categories First Generation Antipsychotic drugs and Second Generation Antipsychotic drugs. First generation antipsychotics act on the brain by blocking dopamine D2 receptor. FGAs drugs are Chlorpromazine , Perphenazine , thiothexine , Fluphenazine , Haloperidol. Second generation antipsychotics drug such as Risperidone , Olanzapine, Quetiapine , Ziprasidone, Aripiprazole , Clozapine.

KEYWORDS: Schizophrenia, positive symptoms, negative symptoms, First generation antipsychotics, Second generation antipsychotics.

I. INTRODUCTION

Schizophrenia is a complex, persistent intellectual mental disorder characterised by means of an array of symptoms, inclusive of delusions, hallucinations, disorganized speech or behavior, and impaired cognitive ability [1].

The psychopathological signs and symptoms of schizophrenia are clustered into three principal categories: positive, negative and cognitive. Disability is a frequent outcome from each poor signs and symptoms (characterized by way of loss or deficits) and cognitive symptoms, such as impairments in attention, working memory, or government function. In addition, relapse may also take place due to the fact of positive symptoms, such as suspiciousness, delusions, and hallucinations [1,2]. Environmental and Genetic factors can also additionally play a function in the development of schizophrenia. Environmental stressors linked to schizophrenia encompass childhood trauma, minority ethnicity, house in an city area, and social isolation[3]. In 1950s, two generations of antipsychotic agents have been developed, which are D2 receptor blockers. The mechanisms by means of which D receptor blockers exert their therapeutic properties are no longer as clear, however dopamine D receptor antagonism is regarded as a unifying property of most antipsychotic drugs[4]. Treatment had been begun initially with the “First Generation Antipsychotics (FGAs)”, the first of which Chlorpromazine was discovered in 1952, and In 1996, the first "Second-generation antipsychotic (SGA)" Risperidone bought in market. Both FGAs and SGAs had been focusing on psychopathology and fine signs and symptoms[5].Individuals with schizophrenia lead a bad exceptional of life, due to bad scientific attention, homelessness, unemployment, economic constraints, lack of education, and bad social skills[6].

ETIOPATHOGENESIS

Neurodevelopmental hypothesis

Based on early studies, it was considered that the structural Brain modifications that happen in schizophrenia had been triggered by early prenatal or perinatal insults, which can exist as a predisposing factor for development of schizophrenia. Complications in being pregnant can alter the axonal connection patterning in synaptic projections via affecting neuronal cell proliferation, migration and apoptosis which are equally required for ideal Central Nervous System (CNS) development. As early as 1976, it was once suggested that cerebral ventricles or cortical sulci are enlarged in early stage of patient suffering from Schizophrenia[7].

Contributing environmental factors

According to epidemiologic studies and research from discordant equal twins, there exists pronounced effect on early stage of brain

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development due to environmental factors in patients with Schizophrenia. Viral infections such as influenza and poliovirus, bad prenatal nutrition, unfavorable obstetric occasions and smoking at some stage in adolescence, are all examples of environmental factors, which can also be the reason for development of schizophrenia. It has been recommended that environmental conditions combined with a genetic predisposition result in schizophrenia[8].

Impairments in cognitive function

Schizophrenia is also developed via extreme cognitive dysfunction or impairment. Specifically, people with schizophrenia are unable to assume clearly, have troubles with memory, thinking and solving problem, and have dysfunction in the capability to speech. Early studies considered that development Schizophrenia is the result of structural abnormalities, which finally lead to cognitive deficits. Currently, investigators are using imaging equipments to understand well about schizophrenia. Functional magnetic resonance imaging (fMRI), blended with different diagnostic equipment such as the electroencephalogram (EEG) have allowed for the unique examination of most important psychiatric illnesses. Functional neural imaging, specifically MRI, is one of the most important tool to understand schizophrenia as this approach permits for excessive spatial and temporal resolution in research analyzing cognitive dysfunction and mapping of affected person's brain[9].

Oligodendrocytic computation capability theory

White matter abnormalities in the Brain have additionally been correlated with schizophrenia. The end result of these abnormalities is particular defects in intelligence lateralization. Some investigators have cautioned that broken or immature oligodendrocytes can stop axonic formation. Based on this, Mitteraue postulated the oligodendrocytic computation capability theory, decomposition of the oligodendrocyte-axonic machine may also responsible for the symptoms of schizophrenia[10].

Genetic inheritance in schizophrenia

Schizophrenia manifestations are greater frequent in some families. Although now not strictly due to heredity, more modern fashions have been proposed that recommend that unique allelic inheritance may additionally make contributions to the development of schizophrenia. Recent research of twins and adoption research suggests that Schizophrenia is a genetic disorder[11].

Reduction in neuropeptide Y

Several research have proven a clear relationship between decreased stages of neuropeptide Y (NPY) in the brain and the pathophysiology of schizophrenia. Independent two groups have mentioned that there is a decreased NPY content in the brain of patient with Schizophrenia[12,13].

Alterations in neurotransmission

There has been tremendous proof that glutamatergic N-methyl-D-aspartate (NMDA) neurotransmission is additionally noticeably disrupted in schizophrenia. Spinophilin, a neuronal protein implicated in the NMDA signaling, was once additionally stated to be downregulated in the striatum after repeated phencyclidine (PCP) treatment. These effects verified that repeated therapy PCP drugs, an NMDA receptor antagonist, leads to cognitive deficits that are related with differences in gene expression in brain areas which plays a important role in the pathophysiology of schizophrenia[14].

DIAGNOSIS

Diagnostic principles play a necessary position in the treatment and management of schizophrenia patients . Most of the attributes defining schizophrenia are self-reported subjective evidence[15]. Two or more symptoms should be present during a time period of atleast one month duration (or much less if efficaciously treated): (a) delusions, (b) hallucinations, (c) disorganized speech, (d) grossly disorganized or catatonic behavior, or (e) Negative symptoms. PANSS(Positive and Negative Syndrome Scale) are used in measuring the clinical response to pharmacological treatment and it is very beneficial in medical research, so PANSS is described as “gold standard measure of cure efficacy.” PANSS is comprised of 30 awesome gadgets prepared into three impartial subscales with scoring that degrees from 30 to 210 points[16]. The 24-item Brief Psychiatric Rating Scale (BPRS, version4.0) permits the rater to measure psychopathology severity. An exploratory component evaluation of the 24-item BPRS produced a six-factor answer labelled Mood disturbance, Reality distortion, Activation, Apathy, Disorganization, and Somatization[17].

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PSYCHIATRIC COMORBIDITY

Psychiatric comorbidities are most common among patients with schizophrenia. Substance misuse is additionally common; conservative estimates recommend atleast half of sufferers are affected. In 50% of patients it is common to have comorbidity. Anxiety problems (particularly panic disorder, post-traumatic stress disease and obsessive-compulsive disorder) can additionally be existing to various degrees[18].

SYMPTOMS

The Diagnostic and Statistical Manual, fifth Edition (DSM-5), is a medical aid that practitioners use to diagnose intellectual fitness conditions. As with different conditions, Schizophrenic patient should be diagnosed with special clinical criteria[19].

Schizophrenia signs and symptoms are categorised into two groups: Positive and Negative. Positive signs and symptoms are these which includes an extra or disturbance of daily function, including:

Delusions — delusions can be somatic (involving false beliefs about bodily illnesses), grandiose (containing beliefs of self-importance and having distinct powers or abilities) or paranoid (where there are beliefs of persecution).

Hallucinations — hallucinations can be auditory, tactile, visual, olfactory or gustatory, characterised by means of experiences when there are no exterior stimuli.

Thought problems — thinking ailment is characterised by using disorganized speech, which is believed to be due to atypical thoughts; ideas can be blocked (where little or no ideas occur), or can show up to have been inserted into, or withdrawn from, the thinking by way of others.

Reference include — thoughts of reference happen when a individual believes that sure exterior phenomena such as TV, radio or newspaper articles are reporting about them or speaking without delay to them (ideas of reference can additionally be viewed delusions if there are beliefs that exterior happenings relate immediately to the individual).

Negative signs are these that lead to a minimize or loss of regular function, inclusive of lack of emotion, apathy, terrible or non-existent social functioning, lack of motivation, decreased speech, lack of initiative, sluggish moves and negative self-care.

It is frequent for human beings with schizophrenia to lack perception to such an extent that they do not consider they are sick[20].

MANAGEMENT

NON PHARMACOLOGICAL THERAPY

The goals in treatment of Schizophrenia is concentrated on symptoms, stopping relapse, and growing adaptive functioning so that the affected person can be built-in returned to the community. To obtain long term outcome both pharmacological and Non Pharmacological treatment is required . Psychotherapeutic processes is additional, which is divided into three categories: Individual, Group, and Cognitive behavioral. Nonpharmacological management can be used as an addition to medications, not as an alternative to them .In addition patient family support can be encouraged to minimize rehospitalization and to enhance social functioning[21]. It is necessary to provide knowledge to the patient about their sickness and about the risk and effectiveness of treatment. Family members or care taker can be educated how to monitor the patient and report if any adverse effect is produced to the physician[22].

Some psychotherapies can educate about the significance of taking their medications. These initiatives encompass cognitive behavioral therapy (CBT), private therapy, and compliance therapy. Most psychotherapies promote family involvement[23].The Evidence-based non-pharmacological interventions which is most commonly used for schizophrenia is encomposed of cognitive-behavioral therapy (CBT), cognitive remediation, psychoeducation, social and coping skills, family interventions, and Assertive Community therapy (ACT)[24].

Diet

It used to be observed that men and women with schizophrenia consume less fiber, retinol, carotene, nutrition C, diet E, fruit, and vegetables[25].

Administration of folic acid dietary supplements might ameliorate the positive and negative symptoms in schizophrenia. Vitamin C, E, and B (including B12 and B6), had been additionally discovered to be most effective in managing schizophrenia[26].

Yoga

Combination of Pharmacological treatment with yoga therapy is most effective in treating Schizophrenia[27].

Pharmacological intervention along can't be effective in managing schizophrenia symptoms, particularly negative symptoms . Yoga as an add-on to antipsychotic medications, helps deal with

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positive and negative symptoms, extra than medicines alone. Furthermore, pharmacological interventions frequently produce weight problems in schizophrenia .Yoga therapy can decrease weight which may caused due to the administration of antipsychotic medications. Pharmacological treatment can lead to endocrinological and menstrual dysfunction which might be reduced via yoga therapy[28].

PHARMACOLOGICAL THERAPY

The treatment for acute psychotic episode such as administration of drug with appropriate dose, dose are titrated according to the patient response , Maintenance therapy for improving self care and increasing socialization [28,29].

ANTIPSYCHOTIC DRUGS :

Typical Antipsychotics

It is also known as first generation antipsychotics act on the brain by blocking dopamine D2 receptor. FGAs drugs are Chlorpromazine , perphenazine , molinelone , thiothexine , fluphenazine , Haloperidol .In mesolimbic pathway ,there is a lack of selectivity in dopamine receptor so the FGAs drug cause more side effects. The extrapyramidal symptoms are the major ADR such as dyskinesia , Tardive dyskinesia , akathisia or parkinsonian like movements by blocking D2 receptor in the nigrostriatal pathway. High dose of typical antipsychotic can inhibit dopamine receptor in the mesocortical pathway and induce negative and cognitive symptom where as Hyperprolactinemia occurs by the increasing the release of prolactin in the pituitary gland by blocking the tuberoinfundipular pathway[30].The high-potency drugs of FGAs such as fluphenazine, haloperidol, loxapine, pimozide, and thiothixene are usually associated with severe risk of EPS. 21 to 31 percent of patients who is treated with haloperidol for three to eight weeks experienced drug-induced EPS which is found in systemic review study .The low-potency medications such as chlorpromazine and thioridazine are less likely to cause EPS than high-potency drugs[31,32].The most common adverse effects of the first generation or typical antipsychotics was Tardive Dyskinesia(TD)[33]. Tardive dyskinesia is the condition of involuntary movements, most probably tongue and mouth with twisting of the tongue, chewing, and frown movements of the face. Tardive dyskinesia was developed after chronic adminstration of antipsychotic drugs for about six months [34]. Within first few days after the initiation of the antipsychotic drugs a condition called acute dystonia is occured and can be prevented or reversed with biperiden which is an anticholinergic agent [35,36]. Acute dystonia is most common with FGAs such as haloperidol and less common with SGAs [37].

ATYPICAL ANTIPSYCHOTICS

It is also known as second generation antipsychotics. SGAs drug such as Risperidone , olanzapine, Quetiapine , ziprasidone, aripiprazole , clozapine. According to American psychiatric association ,SGAs are the first choice of treatment for schizophrenia with the exclusion of clozapine increase the risk of agranulocytosis[28,29].SGA had fewer extrapyramidal symptoms than the FGAs . SGAs shows metabolic side effects such as weight gain , hyperlipidemia and diabetic mellitus. To improve metabolic side effects the drugs are given in combination. The use of clozapine alone shows less benefit where as administration of aripriprazole and clozapine worsen the positive and general symptom of schizophrenia[38].

Some antipsychotic drug cause neuroleptic malignant syndrome such as fever , rigidity, confusion and autonomic instability and management include discontinuing antipsychotics , supportive care and other intervention [39].Acute dystonias, Akathisia, Parkinsonism, and tardive dyskinesia (TD)were the developed EPS. EPS sometimes causes severe adverse effect and patient with EPS developement require additional pharmacotherapy. EPS develop into two phases as Early Acute EPS and Later onset EPS. Early acute EPS develop commonly at the beginning of treatment with antipsychotics or when the doses of antipsychotics is increased. Later-onset EPS mostly occurs after prolonged treatment with antipsychotic and present as tardive dyskinesia (TD). The motor manifestations include akathisia a condition of restlessness and pacing, acute dystonia is a condition of sustained abnormal postures and muscle spasms, especially of the head or neck ,and Parkinsonism causes tremor, skeletal muscle rigidity, and/or bradykinesia[40,41].

II. CONCLUSION

Schizophrenia is a complex disorder which should be treated or managed at the first signs and symptoms of a psychotic episode.Without proper treatment, this could cause many serious problems as social withdrawal, delusions, hallucinations, disorganized speech, etc. Clinicians should reflect onconsideration for

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nonadherence and treatment-related side effect while preparing treatment regimen. Schizophrenic patient can improve the adaptive functioning via pharmacological and nonpharmacological therapy options.

REFERENCE

[1]. Patel KR, Cherian J, Gohil K, Atkinson D. Schizophrenia: overview and treatment options. Pharmacy and Therapeutics, 2014 Sep;39(9):638.

[2]. Dominguez Mde G. Viechtbauer W, Simons CJ, van Os J. Krabbendam L. Are psychotic psychopathology and neurocognition orthogonal? A systematic review of their associations. Psychol Bull, 2009; 135: 157.71.

[3]. Krishna R. Patel, Jessica Cherian, Kunj Gohil, and Dylan Atkinson. P T. 2014 Sep; 39(9): 638–645. PMID: 25210417

[4]. Crismon L, Argo TR, Buckley PF. Schizophrenia. In: DiPiro JT, Talbert RL, Yee GC, et al., editors. 1Pharmacotherapy: A Pathophysiologic Approach. 9th ed. New York, New York: McGraw-Hill; 2014. pp. 1019–1046.

[5]. Cetin M. Treatment of schizophrenia: past, present and future. KlinikPsikofarmakolojiBülteni-Bulletin of Clinical Psychopharmacology, 2015;25(2):95-9.

[6]. Children PronabGanguly, Abdrabo Soliman and Ahmed A. Moustafa. Front. and Health Holistic Management of Schizophrenia Symptoms Using Pharmacological and Non-pharmacological Treatment. Public Health, 2018. DOI: https://doi.org/10.3389/fpubh.2018.00166.

[7]. Johnstone EC, Crow TJ, Frith CD, Husband J, Kreel L. Cerebral ventricular size and cognitive impairment in chronic schizophrenia. Lancet, 1976; 2: 924-926.

[8]. Murray RM, Lewis SW: Is schizophrenia a neurodevelopmental disorder?. BMJ, 1987; 295: 681-682. DOI: 10.1136/bmj.295.6600.681.

[9]. Honey GD, Fletcher PC, Bullmore ET. Functional brain mapping of psychopathology. J Neurol Neurosurg Psychiatry, 2002; 72: 432-439.

[10]. Mitterauer B. The incoherence hypothesis of schizophrenia: based on decomposed oligodendrocyte-axonic relations. Med Hypotheses, 2007; 69: 1299-1304. 10.1016/j.mehy.2007.03.024.

[11]. Pearlson GD, Folley BS: Schizophrenia, psychiatric genetics and Darwinian psychiatry: an evolutionary framework. Schizophr Bull, 2008; 34: 722-733. DOI: 10.1093/schbul/sbm130.

[12]. Frederiksen SO, Ekman R, Gottfries CG, Widerlov E, Jonsson S. Reduced concentrations of galanin, arginine vasopressin, neuropeptide Y and peptide YY in the temporal cortex but not in the hypothalamus of brains from schizophrenics. Acta PsychiatrScand, 1991; 83: 273-277. DOI: 10.1111/j.1600-0447.1991.tb05539.x.

[13]. Gabriel SM, Davidson M, Haroutunian V, Powchik P, Bierer LM, Purohit DP, Perl DP, Davis KL: Neuropeptide deficits in schizophrenia vs. Alzheimer's disease cerebral cortex. Biol Psychiatry, 1996; 39: 82-91. 10.1016/0006-3223(95)00066-6.

[14]. Beraki S, Diaz-Heijtz R, Tai F, Ogren SO: Effects of repeated treatment of phencyclidine on cognition and gene expression in C57BL/6 mice. Int J Neuropsychopharmacol. 2008, 12 (2): 243-255. DOI: 10.1017/S1461145708009152.

[15]. Assen Jablensky. Dialogues Clin Neurosci. 2010 Sep; 12(3): 271–287. DOI: 10.31887/DCNS.2010.12.3/ajablensky. PMID: 20954425.

[16]. Suneeta Kumari, Mansoor Malik, Christina Florival, PartamManalai, and SnezanaSonje. An Assessment of Five (PANSS, SAPS, SANS, NSA-16, CGI-SCH) commonly used Symptoms Rating Scales in Schizophrenia and Comparison to Newer Scales (CAINS, BNSS). J Addict Res Ther. 2017; 8(3): 324. DOI: 10.4172/2155-6105.1000324. PMID: 29430333.

[17]. Adriano Zanello, Laurent Berthoud, Joseph Ventura, Marco C G Merlo. The Brief Psychiatric Rating Scale (version 4.0) factorial structure and its sensitivity in the treatment of outpatients with unipolar depression. Psychiatry Res., 2013;210(2):626-33.

[18]. Buckley P, Miller B, Lehrer D, et al. Psychiatric comorbidities and schizophrenia. Schizophrenia Bulletin 2009;35:383–402.

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[19]. Jodi Clarke. Signs and Symptoms of Schizophrenia, 2022.
[20]. Gelfer M, Harrison P, Cowen P. Shorter Oxford Textbook of Psychiatry 5th edition. Oxford: Oxford University Press, 2006.
[21]. Crismon L, Argo TR, Buckley PF. Schizophrenia. In: DiPiro JT, Talbert RL, Yee GC, et al., Pharmacotherapy: A Pathophysiologic Approach. 9th ed. New York, New York: McGraw-Hill; 2014. pp. 1019–1046.
[22]. Rummel-Kluge C, Kissling W. Psychoeducation for patients with schizophrenia and their families. Exp Rev Neurother, 2008;8(7):1067–1077.
[23]. Dickerson FB, Lehman AF. Evidence-based psychotherapy for schizophrenia: 2011 update. J NervMent Dis. 2011;199(8):520–526.
[24]. Malla AK, Norman RM. Early intervention in schizophrenia and related disorders: advantages and pitfalls. CurrOpin Psychiatry, 2002;15:17–23. doi: 10.1097/00001504-200201000-00004.
[25]. Kalaydjian AE, Eaton W, Cascella N, Fasano A. The gluten connection: the association between schizophrenia and celiac disease. Acta Psychiatr Scand,2006;113:82–90. doi: 10.1111/j.1600-0447.2005.00687.x
[26]. Brown HE, Roffman J. Vitamin supplementation in the treatment of schizophrenia. CNS Drugs, 2014; 28:611–22. DOI: 10.1007/s40263-014-0172-4.
[27]. Jha A. Yoga therapy for schizophrenia. Acta PsychiatScand, 2008;117:397–403. DOI: 10.1111/j.1600-0447.2008.01151.x
[28]. Lehman AF, Lieberman JA, Dixon LB, et al. American Psychiatric Association Practice Guidelines; Work Group on Schizophrenia. Practice guideline for the treatment of patients with schizophrenia. Am J Psychiatry. (2nd ed) 2004;161(suppl 2):1–56.
[29]. Moore TA, Buchanan RW, Buckley PF, et al. The Texas Medication Algorithm Project antipsychotic algorithm for schizophrenia: 2006 update. J Clin Psychiatry. 2007;68(11):1751–1762.
[30]. Stępnicki P, Kondej M, Kaczor AA. Current concepts and treatments of schizophrenia. Molecules. 2018 Aug 20;23(8):2087.
[31]. Antipsychotic-induced extrapyramidal side effects in bipolar disorder and schizophrenia: a systematic review.Keming Gao et al. J Clin Psychopharmacol. 2008 Apr.
[32]. Comparative efficacy and tolerability of 15 antipsychotic drugs in schizophrenia: a multiple-treatments meta-analysis.StefanLeucht et al. Lancet. 2013.
[33]. National Institute for Health and Care Excellence . Psychosis and schizophrenia in adults. Quality standard. London: NICE; 2015. 1
[34]. Jeste DV, Caliguir MP. Tardive dyskinesia. Schizophrenia Bull. 1993;19:303–15.
[35]. A. A. Shirzadi and S. N. Ghaemi, "Side effects of atypical antipsychotics: extrapyramidal symptoms and the metabolic syndrome," Harvard Review of Psychiatry, vol. 14, no. 3, pp. 152–164, 2006.
[36]. M. Raja, "Managing antipsychotic-induced acute and tardive dystonia," Drug Safety, vol. 19, no. 1, pp. 57–72, 1998.
[37]. A. F. Lehan, J. A. Lieberman, J. A. Dixon et al., Practice Guideline for the Treatment of Schizophrenia, American Psychiatric Association, Washington, DC, USA, 2nd edition, 2004.
[38]. Muscatello MRL, Bruno A, Pandolfo G, Micò U, Scimeca G, Di Nardo F, et al. (2011). Effect of aripiprazole augmentation of clozapine in schizophrenia: a double-blind, placebo-controlled study. Schizophr Res. (2011) 127:93–9. doi: 10.1016/j.schres.2010.12.011
[39]. Strawn JR, Keck PE Jr, Caroff SN. Neuroleptic malignant syndrome. Am J Psychiatr 2007;164(6):870-876.
[40]. D. E. Casey, "Implications of the CATIE trial on treatment: extrapyramidal symptoms," CNS Spectrums, vol. 11, no. 7, pp. 25–31, 2006.
[41]. D. E. Casey, "Pathophysiology of antipsychotic drug-induced movement disorders," Journal of Clinical Psychiatry, vol. 65, no. 9, pp. 25–28, 2004.