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IN EARLY DIAGNOSIS OF PSYCHOTIC DISORDERS: A REVIEW

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ABSTRACT

Schizophrenia could be a long-term and severe disturbance characterised by abnormalities in thinking, perception, emotions, language, sense of self, and behaviour. This report presents the Clinical pharmacists' role within the management of a patient diagnosed with schizophrenia with symptoms of psychosis. A 20-year-old African male adult who was rumoured to be wandering around the city from one bank to another was in remission. The patient was admitted to a hospital's medical speciality unit and diagnosed with schizophrenia. Key interventions offered enclosed speedy tranquilization, electroshock, and psychotherapy. On admission, the patient was given IV benzodiazepine, IM major tranquillizer, IV club drug, IM flupentixol, olanzapine tablets, and trihexyphenidyl tablets. Problems raised by clinical pharmacists throughout the patient's admission included a lack of various medication for rapid tranquilization, a lack of initial investigations and documentation of the patient's vital organ, the initiation of major tranquilliser medical aid without initial observance and screening for abuse, an inappropriate dose at the initiation of major tranquilliser medications, an untreated indication, and the occurrence of incomprehensible doses. Clinical pharmacist interventions aided in the improvement of the patient's symptoms prior to hospital discharge. The case demonstrates the importance of clinical pharmacists being included in the multidisciplinary team during the management of patients with mental illness.

KEYWORDS: Schizophrenia, Nanoparticle, Central nervous system, etc.

INTRODUCTION

Schizophrenia is a persistent and severe mental illness that affects more than 21 million people worldwide which is characterised by cognitive, perceptual, and emotional distortions language, sense of self, and behaviour. Common experiences include hallucinations mostly involving hearing voices or seeing things that do not exist and delusions which involve having fixed, false beliefs. Since because schizophrenia is a chronic condition that affects nearly every element of a person's life, treatment planning has three purposes. Which are to decrease or eliminate symptoms to maximize life satisfaction and adaptive functioning and to promote and maintain recovery from the debilitating effects of illness to the maximum extent possible. Medications are invaluable in the management of patients with mental illnesses. Pharmacists are thus essential in enhancing the quality of service provided to patients suffering from mental illnesses such as schizophrenia, thereby contributing to the reduction of the myriad problems linked with and experienced by patients suffering from mental disorders. Management of patients with conditions such as schizophrenia is generally a collaborative effort which encompasses incorporation of skills of a myriad of health care professionals involved in patient care. For almost 30 years, clinical pharmacists have played important roles as educators, advisers, and providers. Because pharmacists are experts in pharmaceutical care, they use their complementary skills and knowledge to manage patients with mental diseases in collaboration with other health care providers on the multidisciplinary team. Clinical pharmacists contribute to patient care by assisting in the discovery, resolution, and prevention of medication-related issues. In order to ensure the safe and efficacious use of medications, clinical pharmacists are also pivotal. Furthermore, pharmacists are accessible to provide detailed pharmacological information to patients suffering from mental diseases such as schizophrenia, their families, and other health care professionals involved in patient management. Pharmacists are in charge of drug adherence as well as primary prevention of mental diseases, health promotion, and lifestyle adjustment. In a review of clinical pharmacy services offered in mental health care conducted by Richardson et al. hospital-based studies were identified in which interventions conducted by clinical pharmacists during patient medication chart

reviews, laboratory investigation result assessment, and prescription of medications were identified.[1]

Oligodendrocytes in Schizophrenia

Despite numerous neuroimaging studies indicating grey matter volume deficits in schizophrenia, there is no conclusive evidence. Compelling post-mortem there is no evidence to show neuronal loss in schizophrenia, nor is there a clear or particular profile of grey matter abnormalities. Instead, no localised lesion that characterises grey matter appears to have been observed. abnormalities in schizophrenia. Instead, there appears to be no observed focal lesion that characterizes gray matter pathology in schizophrenia. This state of affairs has resulted in a shift from viewing schizophrenia as the outcome of a single injury to viewing schizophrenia as the result of aberrant communication between brain regions. Bleuler (1911) proposed aberrant connection between brain areas. What is new is an understanding that brain regions that are not physically proximal may be functionally coupled into neural networks, and that in order to understand altered neural connectivity, we must first understand the connections. Between brain regions which are made possible by white matter, the main infrastructure in the brain that makes possible long distance communication among neurons. As a result, a focus on white matter connections in the brain has grown in importance in schizophrenia research, particularly in imaging investigations, post-mortem studies, and new animal models of schizophrenia. This special issue focuses on schizophrenia and oligodendrocytes, a kind of neuroglia that produces myelin. The major goal of this special issue is to better understand and clarify white matter pathology in schizophrenia, as well as how it may lead to disconnectivity among brain regions, resulting in the observed cognitive, behavioural, and clinical symptoms in this disorder.[2]

To recapitulate, relative to the emphasis on grey matter and neurons, research on white matter and myelin/glial in schizophrenia has been limited. Neuroimaging discoveries in this special issue reveal that myelin abnormalities in schizophrenia may underpin the white matter abnormalities detected using diffusion tensor imaging techniques. Post-mortem data indicate the occurrence of oligodendrocyte and myelin abnormalities in schizophrenia and mood disorders. Oligodendrocytes provide protection and increase communication between brain regions, while oligodendroglia-producing myelin is maturing at the same time as symptoms appear. Furthermore, normal age-associated white matter increases and concomitant grey matter alterations that predate the white matter changes are observed. deregulated in schizophrenia and may contribute to the clinical symptomatology of the disease.

Thyroid Hormones and the Relationship Between Neurotransmitter Systems and Neural Networks in Schizophrenia

The connection between TH and schizophrenia is significant. In reality, multiple groups have tested TH levels and other thyroid-related parameters in schizophrenia patients (both hospitalised and outpatients), reporting on their findings. On several abnormalities, rom our analysis, to date, independent studies of human population cohorts have been published addressing the role of TH in schizophrenic patients, with assessments and/or measurement of TH status. It is of mention that prior to the mid-1980s the lack of high-sensitivity assays for measurement of TH, specifically for free TH, was a handicap. Nonetheless, earlier reports already made mention of thyroid function abnormalities in schizophrenic patients and their loved ones, as well as on the resemblance between the psychotic symptoms of people with severe hypo- and hyperthyroidism and those of schizophrenic patients. Altogether, from the literature analysis, a dynamic relationship emerged.[4]

1) Dopaminergic System:- Dopamine was the first neurochemical to be associated with schizophrenia, in part due to the efficacy of antipsychotic drugs that block dopamine D2- like receptor in alleviating the hallucinations and delusions of patients. Additionally, neuroimaging studies have revealed enhanced activity a component of the nigrostriatal dopamine system, albeit a hypo functionality of the mesoprefrontal cortical system, in schizophrenic individuals.[4]
2) Serotonergic System:- Serotonin (5-hydroxytryptamine, 5-HT) is an essential neurotransmitter. Curiously, it was first thought to have a role in schizophrenia given its similarity to lysergic acid diethylamide (LSD), a compound that competes for and occupies serotonin's receptor sites, resulting in psychotic symptoms.[4]
3) Glutamatergic System:- The glutamatergic theory of schizophrenia is based on the discovery that psychotomimetic drugs such as ketamine and phencyclidine generate a glutamatergic response. neurocognitive deficiencies and psychotic symptoms, similar to those of schizophrenia, through blockage of the neurotransmission at N-methyl-D-aspartate-(NMDA-) type glutamate receptors.[4]
4) GABAergic System:- The role for the GABA (δ-aminobutyric acid)-ergic system in the pathogenesis of schizophrenia derives mostly from neuropathologic studies. Specifically, the chandelier neurons, a subtype of GABA interneurons, have decreased immunostaining for the GABA transporter, possibly related to decreased BDNF signalling or NMDA receptor hypofunction. Furthermore, upregulation of the postsynaptic GABA-A receptors, together with reduction of both glutamic acid decarboxylase (GAD) 67 and reelin (a protein that colocalizes with GABAergic interneurons), was described in schizophrenic patients. GAD67 and reelin are involved in the glutamate conversion to GABA and in synaptic plasticity and/or neuromigration.[4]

1. Myelination and Cytokines:-The TH involvement in the administration of myelination and/or oligodendrocytes' functionality, central processes in the modulation of neural networks, is of interest in schizophrenia, where involvement of white matter has been implicated.[4]
2. Thyroid Hormones as Neurotransmitters:-The role of TH in the pathophysiology of schizophrenia is more so noteworthy when considering the possible function of TH as neurotransmitters. The breakthrough hypothesis of a neurotransmitter role for T3 was put forward in the endocrinology field in the 1970s by Dratman and collaborators, based on the colocalization of TH with the noradrenergic system Given T3's numerous roles in the brain, this is hardly surprising. Among others, T3 promotes differentiation in astrocytes, mediates cerebellar astrocyte and neuronal proliferation, and participates in the organization of extracellular matrix molecules via astrocytes.[4]

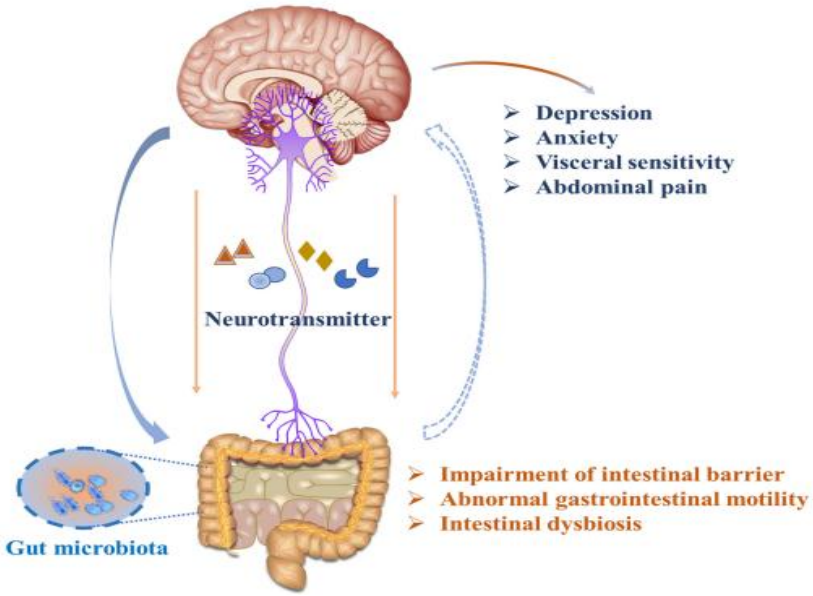

Figure 1: Relationship between Thyroid Hormones and Schizophrenia with Neurotransmitter Systems and Neural Networks.

❖ Schizophrenia and Thyroid Hormones:: Human Studies Considerations

1) Effect of Antipsychotic Medication on Thyroid Hormone Status. The literature reports on the effect of neuroleptic medication on deiodinases activities, as well as on the Nglucuronidation of TH and its impact on TH levels. Specifically, the regularly used antipsychotic haloperidol can increase type 2 deiodinase, whereas clozapine reduces type 2 but not type 1. increases type 3 deiodinase activity in several brain regions.[4]
2) Serum and CSF Thyroid Hormone Level Assessments. The measurement of TH levels in CSF samples would be more inclined to represent TH brain homeostasis. This type of research would not only supplement previous efforts to diagnose schizophrenic sickness.[4]
3) Familial, Prenatal, Neonate, and Early Childhood Thyroid Status. During development TH play a vital part in CNS development, including in cerebral cytoarchitecture, neural growth, and synaptogenesis.[4]

Tackling Negative Symptoms of Schizophrenia with Memantine

Negative symptoms are a mainstay of chronic schizophrenia and constitute a cause of severe disability for the patients. The etiology of negative symptoms is complex; could be because of the disease itself, secondary to positive symptoms, or due to medication's side effects; additional causes are depression and institutionalization. Negative symptoms are resistant to the current pharmacological treatments. Even after the discovery of the novel or "atypical" antipsychotics, negative symptoms remain mostly refractory to treatment. Various medications have been tried as add-on therapies to atypical antipsychotics with modest benefit, at best: antidepressants, cholinesterase inhibitors, selegiline, Ginkgo biloba, modafinil, and armodafinil. A recent research hypothesis regarding the etiology of schizophrenia suggests that one of its main causes is glutamate excitotoxicity; as a consequence, glutamatergic antagonists could hypothetically not only provide symptom relief but also be disease-modifying. Among the glutamate antagonists, memantine a drug used in modest to severe Alzheimer's disease has been tried as an adjunct medication.[7]

Glutamate is the main excitatory neurotransmitter in the central nerve system. According to a current research hypothesis, the glutamatergic system and specifically the Nmethyl-D-aspartate (NMDA) receptors are hypofunctional in schizophrenia. It is likely that the hypofunctional NMDA receptors cause compensatory

excessive glutamate release in an attempt to compensate for the loss; reversing this trend may be beneficial in lowering schizophrenia symptoms. Furthermore, NMDA-receptor hypofunctioning may reduce central gamma-aminobutyric acid (GABA) tone and cause a disproportionate release of glutamate into the synapse, resulting in widespread neuronal death. Memantine is an NMDA-receptor antagonist that partially blocks NMDA receptors thus preventing a toxic influx of calcium and the resultant cell death. It has been hypothesized that it could ameliorate schizophrenia symptoms the negative ones among them. Memantine has shown to improve agitation and delusions in patients with Alzheimer's dementia. Furthermore, it does not seem to worsen the positive symptoms of patients with schizophrenia. As a result, memantine may be useful in maintaining and, in any case, not worsening the antipsychotic therapy-induced improvement in favourable symptoms. In our situation, the small improvement in the SAPS likely indicates this action. Furthermore, when serotonin binds to the serotonin 5HT-2A receptors, it accelerates glutamate release. As a result, atypical antipsychotics that are also serotonin 5HT-2A antagonists, such as risperidone, have the ability to reduce toxic glutamate hyperactivity via serotonin 5HT2A antagonism and, in turn, reducing mesolimbic dopamine release.[7]

Schizophrenia, Diffusion Tensor Imaging, and Structural Connectivity

DTI stands for Diffusion Tensor Imaging enables inferences to be made in terms of the integrity and orientation of fiber tracts on the basis of patterns of water diffusion. DTI is noteworthy in that it can provide information in terms of WM anatomy that is simply not accessible with any other method either in vivo or in vitro. Diffusion otherwise known as Brownian motion refers to particle movement at random, such as water molecules, as a result of unpredictable, thermally driven molecular collisions. In an unrestricted medium, a water molecule is equally likely to move in one direction as another. However, in brain tissue particle movement at random is restricted by obstacles in the local environment such as cell membranes, myelin sheaths, and macromolecules. The extent to which diffusion is restricted differs between the different tissues of the brain. In cerebral spinal fluid (CSF), for example, there are relatively few obstacles to diffusion, and hence the average shape of the resultant diffusion is approximately spherical: this is known as isotropic diffusion. In contrast, the tightly packed and homogeneously oriented bundles of myelinated axons in a WM fibre bundle provide a considerable barrier to water diffusion.[8]

MD is greatest in tissues with low obstacles to water diffusion (e.g., CSF), and lowest in tissues where diffusion is restricted at least one direction (e.g., WM). Although FA Despite the fact that and MD are (nearly) mathematically independent, they are often shown to be negatively connected in the brain, with tissue exhibiting highly anisotropic diffusion (such as WM) generally shows low MD. Mode is a relatively new measure that provides more information about the 3D geometry of the diffusion ellipsoid than FA. Roughly speaking, for a given FA value, Mode describes whether the diffusion ellipsoid is shaped like a cylinder (i.e., having highly "tubular" anisotropy) or like a disk (i.e., having highly "planar" anisotropy). When considered in combination with FA, the Mode of a diffusion ellipsoid provides unique information as to the microstructural features of the underlying WM. For example, the presence of fiber crossings has been associated with reductions in Mode. Hence, the finding that Mode in the corpus callosum in schizophrenia patients has been hypothesised to represent a reduction in the the density of fibers adjacent to this fasciculus.[8]

Time Psychopathology in Brain Disease and Schizophrenia

Neurologists and psychiatrists have mostly ignored the psychopathology of time as a research area. Compared with, say, the volume of articles and bewildering array of terms which attempt to convey the ways in which the spatial fabric of our existence may change (Critchley, 1953; Cutting, 1990), the literature on temporal alterations to our world is meagre in the extreme. This is surprising, if only for the reason that space and time are, in a philosophical sense, equal components in our world-view. According to Kant (1787), for example, both are "necessary a priori representations which underlie all other intuitions". Or, according to Jaspers (1959), both share the quality of "investing all objectivity". The purpose of this article is three-fold: first, to draw together a scattered literature from neurological, psychiatric and psychological sources, in the hope of establishing a correspondence between patterns of altered time sense and sites of brain Second, to call the reader's attention to a neglected literature on the subject by German neurologists and psychiatrists between the two World Wars. Wars; thirdly, to analyse the subjective experience of time in schizophrenics. Two general theses will be argued. One is that our sense of time is just as determined by focal representations within the brain as any other quality of the external world-colour, shape, space, etc.; and that it can be selectively disrupted by brain disease just as our sense of any of these other qualities can be-as in colour agnosia, visual-object agnosia, visuospatial agnosia, respectively. A second thesis is that there are similarities between the subjective experience of time in subjects with right hemisphere brain damage and the experience of schizophrenics, an observation which supports the views of one of the authors that right hemisphere dysfunction is the underlying pathogenic substrate for schizophrenia itself' (Cutting, 1990).[9]

A Model for Decision Support in Early Diagnosis of Psychotic Disorders

There are various stages in the Decision-Making Process Support. Main stages of the decision support process, according to, are as follows.[10]

1. Structuring: it deals with the problem formulation and identification of goals. This phase seeks to identify, characterize, and organize the factors considered important in the decision support process.
2. Evaluation: it enables the subdivision on a subphase partial evaluation of actions (alternatives) based on each point of view (criteria) and an overall evaluation considering several partial reviews
3. Recommendation: in this phase sensitivity analyses and robustness are made to verify those changes, in the parameters of the evaluation model, affecting the final result. It is a key phase that helps to generate knowledge about the problem and thus increases confidence in the results obtained from the decision maker.

Decision Support in the Field of Mental Health. Initially, it would be worth noting that, searching the literature of decision support in health, some studies reported hybrid models used to aid decision-making and finding diagnostics for diseases. [10]

DISCUSSION

In the context of damage to the left temporal and parietal lobes, a schizophrenia-like illness in this patient with no family history of the disease (or a schizoid personality) suggests symptomatic schizophrenia.

Nanotechnology has emerged as an interesting and promising new platform for treating neurological illnesses, with considerable promise to overcome issues associated with traditional therapy procedures.

CONCLUSIONS

Despite significant advances in medical technologies and healthcare facilities, there has been a consistent clinical failure to find a permanent solution for the majority of CNS illnesses. Among the different current therapeutic options, nanomedicine-based has given rise to unique nanoscale targeting approaches, ushering in a new era in which active agents with promising pharmacokinetics can be delivered to ameliorate the status of these diseases. The failure of CNS medication development is attributable to the disease's intricacy and the present challenges of Alzheimer's disease neuropharmacology research.

REFERENCE

1. Case Report. A Case of Schizophrenia in a Young Male Adult with no History of Substance Abuse: Impact of Clinical Pharmacists' Interventions on Patient Outcome.
2. Oligodendrocytes in Schizophrenia Haiyun Xu, 1 Vahram Haroutunian,2 George Bartzokis,3 and Martha E. Shenton.
3. Mercy N. A. Opare-Addo,1 Josephine Mensah, 2 and Grace Owusu Aboagye.
4. Revisiting Thyroid Hormones in Schizophrenia Nadine Correia Santos, Patrıcio Costa, Dina Ruano, Antonio Macedo, Maria Joao Soares, Jose Valente, Ana Telma Pereira, Maria Helena Azevedo, and Joana Almeida Palha.
5. Schizophrenia as a Disorder of Communication Margaret A. Niznikiewicz, Marek Kubicki, Christoph Mulert, and Ruth Condray.
6. Symptomatic Schizophrenia with Moya Moya Disease. GERARD McDADE.
7. Case Report Tackling Negative Symptoms of Schizophrenia with Memantine. Antonios Paraschakis.
8. Diffusion Tensor Imaging, Structural Connectivity, and Schizophrenia Thomas J. Whitford, Marek Kubicki, and Martha E. Shenton.
9. Psychopathology of Time in Brain Disease and Schizophrenia. JOHN CUTTING and HERTA SILZER.
10. Handling Diagnosis of Schizophrenia by a Hybrid Method Luciano Comin Nunes, Plácido Rogério Pinheiro, Tarcísio Pequeno Cavalcante, and Mirian Calíope Dantas Pinheiro.
11. Diagnosis of Schizophrenia Based on Deep Learning Using fMRI JinChi Zheng , XiaoLan Wei, JinYi Wang, HuaSong Lin, HongRun Pan, and YuQing Shi.
12. Review, Applications of nanotechnology in drug delivery to the central nervous system Majid Saeedia, Masoumeh Eslamifarb , Khadijeh Khezric, Solmaz Maleki Dizaj.
13. Review, Nanotechnology-based drug delivery for central nervous system disorders Thuy Trang Nguyen, Ph.Da, Thi Thuy Dung Nguyen, MS b , Tuong Kha Vo, MD.,Ph.Dc, Nguyen-Minh-An Tran, Ph.Dd , Minh Kim Nguyen, Ph.De , Toi Van Vo, Ph.Df,i, Giau Van Vo, Ph.D.