Application Of Cognitive Therapy And Pharmacotherapy In Treating Psychosis

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Abstract

This research contains a summary of the key medications employed in the treating of psychosis, a description as to how cognitive therapy may imitate the impacts of pharmacotherapy, as well as an analysis of how to combine cognitive therapy with pharmaceutical management. When treating schizophrenia, cognitive therapy is often only effective when patients are also taking antipsychotic medication. It is helpful for therapists to have a broad understanding of how these drugs operate as well as what potential side impacts, they may induce in order to support good dialogue between the counselor and the psychiatrist. In a similar vein, if psychiatrists were to acquire some fundamental cognitive skills, as well as really discuss and implement such strategies with their patients, then they would have a greater grasp of the job that therapists do. The patient will advance more quickly if there is effective collaboration between the two types of therapy they are receiving.

Keywords: Cognitive Therapy, Pharmacotherapy, Psychosis

Introduction

Even if cognitive therapy and other psychosocial treatments are being used in the management of schizophrenia, it is still necessary to take medication in order to alleviate the manifestations of this illness. This cannot be avoided at this time. Cognitive therapy is designed to do two things: discuss the psychological interpretation underneath psychotic symptoms to identify, in a manner that is often lacking in straightforward treatment plans, the human face affiliated with the perceptions of hallucinations, delusions, disorganized thinking, and the negative syndrome; and augment the use of medication in order to lessen the impact and intensity of these symptoms.

When adequate psychotherapy is offered for the treatment of schizophrenia, there are others who say that medication is not necessary and that it is unnecessary to do so. It is possible that this is the case for certain other types of psychosis, but it is very unlikely that this is the scenario for schizophrenia. It is not acceptable to deny medication treatment to patients who could gain from this form of treatment and who may encounter adverse implications if they are denied it due to the personal priority of a specialist to prevent the use of medication if that practitioner prefers to mitigate the use of medication. It is possible that cognitive therapy and other types of treatment may reduce the required dose of medicine, but this will not remove the need entirely. In the future, when our knowledge of schizophrenia has progressed further, there may be treatment procedures that do not include the use of medication; however, this is not the case right now.

The belief that the ability of substances that alter neurophysiology (such as psychiatric meds) to produce shifts in cognition, feelings, and actions in those with schizophrenia implies that the etiology is entirely based in neurophysiology represents the opposite end of the biopsychosocial spectrum. It is not the case that schizophrenia is caused by an imbalance in these substances only because there are effective antipsychotic drugs that raise serotonin levels or lower dopamine levels, respectively. According to this line of reasoning, the fact that a pain medicine is helpful after inadvertently lifting a heavy burden does not suggest that the source of the pain is a deficiency in opioid neurotransmitters. Psychosocial conditions such as acute traumatic experiences and/or years of lesser stressful situations, as well as psychological impact to those stressors, have the potential to produce psychiatric conditions such as schizophrenia, which can then be treated by altering the brain chemistry of the patient. These stresses and the responses to them either change the levels of specific neurotransmitters or they had some impact that may then be partly restored by chemically modifying the levels of some neurotransmitters. In the case of back pain, lifting is the cause of the pain, pain neurotransmitters (material P) are the ones that mediate the pain, and the pain may be alleviated by enhancing the impact that opioid neurotransmitters have by delivering exogenous opioids. To reiterate, the discomfort is not the result of an inadequate supply of opioids, just as the psychosis may be the result of anything other than only an excess of dopamine. The fact that medications that change specific neurotransmitters have the ability to ameliorate psychotic symptoms has, without a doubt, helped to direct studies into the etiology and progression of schizophrenia. These medications have also helped to ease the burden that is caused by this condition, at least in part. On the other hand, our working hypothesis is that both biological and psychological variables play a role in the development of schizophrenia as well as in its treatment.

The Development of Neuroleptics

Antipsychotic pharmaceuticals, like many other categories of psychotropic drugs, were found by accident while researchers were looking for something else entirely. Henri Laborit, one of the French specialists, employed chlorpromazine (also known as Thorazine) like a sedative for routine surgery in 1952. During this time, he noticed that patients were less interested in their surroundings as a result of taking the drug. This discovery eventually resulted in a paradigm shift in the way schizophrenia is treated, and it was the impetus for the use of the drug in psychiatric patients. As a result of chlorpromazine and the myriad of other treatments that soon accompanied suit, psychiatric hospitals, also known as "insane asylums," were able to begin releasing some of their patients back into the general population. This was made possible by a reduction in the intensity of the patients' psychotic symptoms.

These antipsychotic drugs have also been referred to as neuroleptics (because to the impact they have on motor control) and large tranquilizers (in contrast to the lesser tranquilizers, including the benzodiazepines, which include clonazepam, alprazolam, and diazepam, respectively). The participants of this older group were renamed first-generation, traditional, classical, or customary antipsychotics following the introduction of a newer version of antipsychotic medication in the 1980s. This was done in order to differentiate them from the more recent second- and third-generation antipsychotics, also known as atypical antipsychotics. This occurred as a result of the use of a newer version of antipsychotic medication.

It was established in the middle of the 1970s that the initial antipsychotics had their impact by inhibiting dopamine receptors, namely the D2 subtype; hence, the term dopamine antagonists antipsychotics was coined to describe these medications (DAAs). The dopamine hypothesis of schizophrenia was developed as a result of this attribute, as well as the fact that the usage of these drugs proved to be effective in the management of schizophrenia. As was just said, therapy does not always reflect an underlying cause. The finding that first-generation antipsychotics inhibit D2 receptors, on the other hand, prompted a comprehensive investigation into the ways in which

dopamine transmission could be connected to the symptoms of schizophrenia. If preventing the transfer of dopamine through one nerve cell to another helps reduce psychosis, then excessive dopamine transmission has to be the major psychophysiological basis of schizophrenia. This was a sensible assumption to make, and it was supported by evidence. Despite this, this idea has not been verified at this time. Blocking postsynaptic D2 receptors is the mechanism that is thought to be responsible for the antipsychotics' therapeutic efficacy.

Even though the rapid sedative impacts of several of these antipsychotics are beneficial in treating the acute agitation that is frequently observed in psychiatric emergency departments and inpatient units, the delayed antipsychotic consequences are due to the dopamine constriction and the reactionary up-regulation of postsynaptic D2 receptors that occurs three to six weeks later. The reactive up-regulation of postsynaptic D2 receptors is defined as a rising prevalence of receptors in response to the blockade. According to positive emission tomography, in order to produce clinically relevant outcomes, a receptor occupancy of 65–70% in the D2 subtype is required.

On the basis of the chemical structures that they possess, DAAs are subdivided into a few different groups. The half-lives of these several drugs range anywhere from Sixteen to 45 hours. The length of time it requires for 50% of the medicine to be removed from the body is referred to as its "half-life." This removal often takes place in the liver, where it takes the form of either demethylation or hydroxylation, and it may also take place via the kidneys or the digestive system. Some antipsychotic medications, such as haloperidol and fluphenazine, come in depot injectable forms with a decanoate connection that extend the drug's half-life to anywhere from two to six weeks. As a result, patients only need to get injections every two weeks or once per month. Because DAAs have such a significant affinity for albumin and other proteins present in the circulation, we say that they have a high protein-binding capacity. They too are lipophilic, which means that they are capable of binding to lipids (lipids). Because of this, they are found in relatively high quantities in the brain.

Despite the fact that DAA drugs have alleviated the pain of a great number of people and the agony of their families, these medications have a lot of drawbacks and negative effects. To begin, not every person who has schizophrenia will benefit from taking these medications. There are some people whose psychotic manifestations are not considerably influenced by any medicine, despite the fact that this is the case in the majority of instances where one medication is ineffective, another medication may be so. Second, DAAs typically only treat the positive symptoms of schizophrenia, which include hallucinations, delusions, and disorganized thinking. They do not treat the negative symptoms of schizophrenia, with the exception of those aspects that stem from responses to the occurrence of favorable symptoms, nor do they treat cognitive deficits. Last but not least, just like with any other drug, there is a wide range of possible adverse effects. The tardive dyskinesia, which is a late-onset involuntary movement disease, is one of these conditions. The neuroleptic malignant syndrome is another. Both of these conditions have the potential to cause death. It is useful to have some background knowledge of the physiology associated with the effects of these drugs before attempting to get an understanding of these limits.

A discussion on the pharmacodynamics of the DAAs

The neurotransmitter systems that are affected by DAAs may be used to explain both the advantages and the adverse effects of these drugs. In addition to dopamine, the muscarinic variant of the neurotransmitter acetylcholine, the 1-adrenergic system, as well as the histaminergic-1 (H1) framework are also engaged as neurotransmitter systems.

The dopamine system is comprised of four primary routes in the brain in addition to its five neurotransmitter subtypes (D1–D5). Both the mesolimbic as well as mesocortical tracts have their origins with in ventral tegmental area (VTA), which is located in the middle of the brain. From

there, they make their way to the nucleus accumbens, which is a component of the limbic system. It is thought that the positive symptoms of schizophrenia, such as hallucinations, delusions, and mental disorder, are brought on by an excessive amount of dopamine activity in the mesolimbic system, whereas the negative symptoms are brought on by a decreased amount of dopamine behavior in the mesocortical system. The substantia nigra is the starting point for the nigrostriatal tract, which continues on to the basal ganglia and plays a role in the regulation of movement. Parkinson's disease is a condition that may be caused by degeneration in this system. The hypothalamus is connected to the anterior pituitary by the tuberoinfundibular tract, which acts to suppress the production of prolactin, a hormone that stimulates the production of milk.

Although blocking dopamine in the mesocortical transmission (in addition to blocking it in the mesolimbic projection) is helpful in reducing positive psychotic symptoms, there is a risk that it will make the patient's negative symptoms even more severe. Reducing dopamine transmission in the nigrostriatal tract by blocking at least 80percent of the available postsynaptic neuron can lead to the idiopathic intracranial hypertension system (EPS) side effects. These side effects include parkinsonian-like symptoms such as original intent tremors (tremors all through initiated movements), postural instability, and disguised facies. Acute dystonia (spasms or prolonged tensed muscles), akathisia (motor hyperactivity), akinesia (lack of motion), and dyskinesia (TD), a severely disabling syndrome characterized by unconscious choreoathetoid (dance-like or squirming) movements, are some of the additional side effects that can occur. The development of TD is thought to be caused by an up-regulation of presynaptic D2 receptors; it may take place years after the beginning of therapy with DAAs; and it has no connection to the dosage used.

In the striatum, the dopaminergic neurons of the nigrostriatal pathway are responsible for inhibiting the acetylcholine neurons. Therefore, since antipsychotics inhibit dopamine, they cause an increase in the release of acetylcholine, which results in the EPS negative effects. Therefore, anticholinergic drugs, which impede the transmission of acetylcholine, may assist to counteract the increased amount of acetylcholine that is released. On the other hand, this may result in anticholinergic adverse effects, which are already present as a consequence of the anticholinergic action of DAAs themselves.

The dopamine blockage in the sar tract disinhibits the withdrawal of prolactin, which leads to hyperprolactinemia. Hyperprolactinemia can lead to different fatigue (lactation unrelated to miscarriage), decreased sexual desire or function, hormonal problems (male breast enlargement), pregnancy complications, and amenorrhea. All of these conditions can be caused by hyperprolactinemia (lack of menstruation).

The neuroleptic malignant syndrome is yet another serious adverse effect that poses a risk to the patient's life (NMS). The symptoms of NMS, which include severe bodily stiffness and high body temperature, are thought to be induced by the actions of dopamine on the mechanism that controls temperature regulation in the hypothalamus. Treatment in an emergency room is necessary to alleviate NMS symptoms. In addition to the negative consequences that are brought on by unintentional dopamine barriers, there are also negative effects that are brought on by the antagonistic effects that DAAs have on other neurotransmitters. One of them is the muscarinic (M1) variant of the cholinergic neurons, which may result in potential adverse effects such as dry mouth, impaired vision, constipation, urine retention, drowsiness, and a slowing of cognitive function. Other neurotransmitters that are inhibited include the alpha-adrenergic (a1) as well as the histaminergic (H1) systems. In the case of the former, this results in sedation as well as orthostatic hypotension, which is a drop in blood pressure that occurs when the patient moves from lying down to sitting to standing up, and it frequently causes dizziness. In the case of the latter, this results in sedation as well as weight gain.

Both the positive and negative effects of DAAs may be organized into categories depending to the drug's strength. It is generally agreed that haloperidol, range of advantages, thiothixene, and haloperidol are examples of high-potency DAAs; phenelzine, mesoridazine, as well as thioridazine are examples of low-potency antipsychotics; and the majority of other antipsychotics fall somewhere in the middle of these two extremes. Due to the fact that having a high potency is a result of having a higher affinity for Nmda receptor, medications that have a potent activity also carry with them the negative effects of dopamine blockage. These negative effects include an increased likelihood of experiencing EPS side effects, as well as TD and NMS. They also have substantial side effects on the alpha-1 adrenergic system. In contrast hand, when high-potency DAAs are used, the adverse effects that are linked to the H1 and M1 receptors are far less severe. The converse is true with low-potency antipsychotics, which have less adverse effects associated with EPS, TD, NMS, and 1-adrenergic activity and more side effects associated with histaminergic and muscarinic activity.

The Role of Cognitive Behavioral Therapy in Pharmacotherapy

The brain is capable of translating physiological alterations (such as the use of medications, illicit drugs, toxins, or electroconvulsive therapy) into changes occurring, as well as transforming psychological events (such as long-term circumstances or acute situations) into metabolic responses (such as the formation of new synapses, the strengthening of existing synapses, and cell death) (e.g., thoughts, emotions, behavior). In point of fact, any sensory input (such as a conversation, a sunset, or the temperature) provokes physiological changes that are at least of a short-term nature, if not of a longer-term nature. These physiological changes can be attributed to other physiological changes, which can lead to psychological changes in our opinions, emotions, and behaviors. There is no reason to reject the hypothesis that the waves of psychic stimuli provided by psychotherapy, which lead to changes in thoughts, feelings, and behavior, would also involve physiological modifications in the brain. These changes would be a direct result of the changes brought about by the psychotherapy. A handful of brain imaging experiments have previously established physiological alterations in brain metabolism in particular brain areas as a result of the application of cognitive therapy in the treatment of depressive and anxious disorders. Based on these results, cognitive treatment, which is a subset of psychotherapy, may be conceptualized in the same way as pharmacology and chemotherapy are. Regardless of whether the physiologic changes do not continue to be present after the treatment has been stopped, this would not be significant departure from the impacts of medicine, which normally need to be taken on a regular basis in order to keep their physiological (and mental) effects. In point of fact, it is more probable that cognitive treatment would lead to longer-lasting physiological changes. These changes would be a reflection of the learning of new abilities in judging events differently from one's normal method of interpretation.

However, early research is being carried out by Silbersweig and others, and further studies are anticipated to appear over the next decade. There haven't been any brain scans investigations of the impact of cognitively therapy in the management of schizophrenia up to this point. It is possible to anticipate that cognitive treatment will result in a variety of physiological brain alterations in a number of different ways. These include the development of synapses in neural pathways that indicate new ways of thinking about how to perceive environmental inputs, a reduction in the frequency of firing in neural pathways that represent acute stress reactions, and a reduction in the buildup of long-term brain reactions to stress.

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