Clinical depression: drug treatment research article analysis

Overview

Clinical depression is a common mood disorder characterized by several symptoms affecting how a person thinks, feels, and behaves. Symptoms are wide-ranging and can include a persistent sad mood or feeling of hopelessness, irritability, less interest in pleasurable activities, difficulty concentrating, appetite and weight changes, suicidal thoughts, over or under sleeping, general fatigue, and physical symptoms including headaches and gastrointestinal distress. Symptoms vary in intensity and duration for every patient and depending on the stage of clinical depression (National Institute, 2018). Mental health professionals typically diagnose a patient with clinical depression. Several of the symptoms of depression must be present nearly every day for at least 2 weeks to be diagnosed with clinical depression (U. S. National, 2019). Clinical depression is one of the most frequently diagnosed mental health disorders in America. Approximately 7% of adults and 13% of adolescents were diagnosed with depression in 2016 (U. S. National, 2019). Although clinical depression can occur at any age, it is most common in late adolescence and early adulthood. Woman are disproportionality diagnosed with clinical depression and are 2 times more often than men (U. S. National, 2019). While a single cause of clinical depression is currently unknown, research suggests that genetic and environmental factors can cause depression (National Institute, 2018). No single gene has been associated with clinical depression. Instead, it is believed that a variation in multiple genes mix to increase the likelihood of developing clinical depression. When a patient has a first-degree family member with clinical depression, they are two to three time more likely to develop the disorder themselves. Additional

risk factors including substance abuse, stressful life events, and chronic physical conditions all increase the likelihood of developing clinical depression (U. S. National, 2019).

Drug Treatment

Escitalopram, marketed under the brand name Lexapro in the United States, is a selective serotonin reuptake inhibitor (SSRI) that is derived from the older SSRI citalopram. Escitalopram works by inhibiting the reuptake of the neurotransmitter serotonin by presynaptic terminals in the central nervous system (CNS). This results in more availability of serotonin in CNS synapses allowing for more reception of serotonin at postsynaptic terminals. Escitalopram specifically targets serotonin reuptake and has little to no effect on norepinephrine or dopamine (Drugs. com, 2019). Escitalopram is a synthetic drug that is developed from its parent drug Citalopram. While Citalopram is a racemic mixture of the S and R enantiomers, escitalopram is made from only the S-enantiomers. The pure S-enantiomer formulation makes escitalopram twice as effective as Citalopram. The active ingredient in escitalopram, escitalopram oxalate, is a white to slightly off-white powder that is soluble in methanol and only slightly soluble in water. Escitalopram is sold as an oral solution and as tablets in the United States with dosages of 5mg/ml, 5mg, 10mg, and 20mg (Dailymed, 2019). Escitalopram is taken orally and has an 80 percent bioavailability 5 hours after consumption. Escitalopram is primarily processed by the liver where two enzymes, CYP3A4 and CYP2C19, break Escitalopram down into S-DCT. Because the liver is the primary metabolizer for escitalopram, patients with renal impairment are given the recommendation not to exceed a dose of 10mg per day. Elderly

patients, those over the age of 65, are also given a maximum recommended dose of 10mg due to a 50 percent increase in escitalopram's half-life (Dailymed, 2019). Escitalopram is prescribed to treat symptoms of acute generalized anxiety disorder, and acute or chronic clinical depression. Escitalopram treats symptoms of clinical depression exclusively through its ability to inhibit the reuptake of the neurotransmitter serotonin in the CNS. Escitalopram is recommended for adolescent, ages 12 to 17, and adults who are diagnosed with clinical depression. The suggested starting dose for both adolescents and adults is 10mg per day. After 3 weeks at the 10mg daily dose, adolescents may be given a maximum of 20mg per day to address symptoms. Adults are also given a maximum of 20mg per day, but only have to wait 1 week from the initial dose (Dailymed, 2019). Escitalopram does result in side effects for some patients with the most common ones being insomnia, sexual dysfunctions including an inability to ejaculate, increased sweating, and sleepiness (Drugs. com, 2019). One particularly severe adverse reaction to escitalopram is the possible development of serotonin syndrome. Serotonin syndrome is potentially fatal and can result in symptoms ranging from hallucinations and falling into a coma to tachycardia, tremors, and vomiting. Serotonin syndrome is most often seen when mixing escitalopram with other SSRI's or monoamine oxidase inhibitors (MAOIs), both of which are not recommended if a patient is taking escitalopram (Dailymed, 2019). While not explicitly tested for addictive properties, there have been no observable drug-seeking behaviors in clinical trials for escitalopram. Overdose of escitalopram has been seen at a 600mg one-time dose in clinical trials. There have been reports of escitalopram overdose in the market at one time doses of 1000mg as well. Death is rare with an

overdose of escitalopram, but patients can experience symptoms including convulsions, vomiting, drowsiness, and falling into a coma. Escitalopram does not have an antidote, so flushing the stomach or the use of activated charcoal is recommended in the case of an overdose (Dailymed, 2019). Withdrawal effects such as anxiety, headache, sadness, and paresthesia can be experienced if escitalopram use is abruptly stopped. Tapering off the medication with a healthcare provider is the recommended way to discontinue treatment (Drugs. com, 2019). Escitalopram's efficacy was confirmed in three different clinical studies where a statistically significant improvement of depression symptoms was shown when compared to placebo (Dailymed, 2019).

Empirical Research Article on Drug Treatment

Researchers were interested in studying the efficacy of escitalopram specifically on depressed patients of Chinese ethnicity. Depression is historically less common among Chinese people versus the western population. Studies were conducted using diagnostic tools similar to western standards, and although the identification of depression was common, it still affected a smaller percentage of the Chinese population. Escitalopram had never been studied on Chinese patients before, but the researchers hypothesized that it would be efficacious in the treatment of depression based on previous studies. This study was a randomized trial run in a double-blind fashion with parallel groups of patients. The trial consisted of 240 Chinese patients between the ages of 18 and 65 who were all diagnosed with clinical depression. Patients were excluded who had other psychiatric disorders, were suicidal, had substance abuse problems within the prior year,

or were taking herbal remedies for depression. Patients were randomly selected for one of two groups: the first group self-administered 10mg of escitalopram or a placebo labeled fluoxetine, and the second group self-administered 20mg of fluoxetine and a placebo labeled escitalopram. Patients were rated on scales for depression at the beginning of the study, and again at 8 weeks after the study. The results of the study showed a statistically significant improvement in depression for both groups who took the SSRI over the placebo. Statistical analysis showed that patients who took escitalopram had statistically significant better scores on reported depressed feelings and interest in work. Researchers noted that patients had a higher remission rate at a lower dosage than European studies and suggested this was due to genetic differences that resulted in a higher plasma concentration of medications for Chinese patients. The researchers concluded that further study of escitalopram for the Chinese population is justified based on the results of their study (Mao, 2008).

I was fascinated to learn that depression is less common among people living in China than it is in other western societies (Mao, 2008). It would be interesting to learn whether this is due to genetic or environmental and cultural factors. I would be interested in seeing a study of depression rates in people of Chinese ethnicity who have spent their whole lives living in western society to discover if genetics play a role. I thought that this study was well run except for a couple of limitations that were identified by the researchers. First, some patients self-reported adherence to their medication protocol in the study (Mao, 2008). While there wasn’t necessarily a reason for patients to, self-reporting is not as trustworthy as the researchers

administering the medication themselves. One significant flaw to the study was the selection of participants and whether they were representative of the typical population of people with clinical depression. The researches disqualified any patients who had co-occurring disorders like substance abuse and anxiety, as well as any patients with serious medical conditions (Mao, 2008). It is common for depression to co-occur with a serious illness like cancer (National Institute, 2018), and I believe that it is common for depression to present with substance use disorder and anxiety. Since this study disqualified such patients, the real efficacy of escitalopram and fluoxetine on the general population of clinically depressed Chinese patients can't be discerned. I agree with the researcher's conclusion that further studies on escitalopram should be conducted to address these concerns (Mao, 2008). I am diagnosed with clinical depression and have been treated with a combination of talk therapy and fluoxetine to varying effect. After learning about the better scores on two key symptoms of depression with escitalopram, I am going to conduct additional research to see if these results have been replicated in other studies with different ethnic populations. If so, I plan on speaking with my psychiatrist about the option to try escitalopram in the future.

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