Prozac [fluoxetine hydrochloride] term paper sample

Health & Medicine

Introduction

Prozac [fluoxetine hydrochloride] is commonly regarded as a first-line therapeutic medication for adolescents due to its supposedly better risk ratio than other antidepressants (Meeker, et al., 2015; Capitao, et al., 2015). It is also currently the most commonly used among selective serotonin reuptake inhibitor (SSRI) active ingredients, more than sertraline, escitalopram, venlafaxine (Wagner, 2015).

However, its link to suicide cannot be ignored; or to birth defects and low libido (Naish, 2013). Despite the euphoria over it, a dampening cloud can be felt in the background. Is it worth risking adverse effects with Prozac? Is the risk for death or deteriorated mental condition worth the promises marketers make about it?

The medication is also prone to abuse, especially in locations where patients have no alternative therapeutic options other than pharmacological therapy, and perhaps even self-medication. In Brazil, Prozac has been abused in the treatment of depression among older adults, oftentimes as an all-purpose medication amidst psychosocial problems, inadequate social support, and limit access to adequate psychiatric services (Wagner, 2015). Especially among the elderly people whose activities had been cut down to a minimum far less than they used to do during their active years, inadequate and unsatisfactory social support can bring them into bouts of depression, anxiety, and meaninglessness or lack of purpose.

This paper attempts to understand Prozac, its known psychopharmacological effects, its indications, its advantages and disadvantages as a psychopathological therapy, and its efficacy. The aim is to provide a wider

understanding of this highly popular and top-selling antidepressant in the global markets. However, there will be no attempt to create a comprehensive and complete profile of the product beyond what is available in the limited sources, primary and secondary, selected for this paper. Nonetheless, it will attempt to present both the positive and the negative outcomes from research and clinical reports.

Psychopharmacological effects

While antidepressants are considered “ not stimulants” (Ponterotto, 1985), Murray (2006) finds it capable of activating properties, which causes a “ stimulant syndrome,” an undefined condition that is characterized by high-strung behavioral dysfunction. For Prozac, these activating side-effects include agitation, anxiety, insomnia, tremors, and adverse gastrointestinal reactions. The U. S. FDA (2015) also mentioned other adverse effects, such as aggressiveness, akathisia (restlessness with compelling need to move constantly), hostility, impulsivity, irritability, hypomania, panic attacks, and mania. Hypomania is a mood state wherein the person persistently exhibits lack of inhibition and pervasive euphoria or irritability; but generally less severe than full mania.

The mechanism of action revolves primarily the neurotransmitter called serotonin. Serotonin (chemically, 5-hydroxytryptamine [5-HT]) in the CNS (largest concentration in the brain) neurons up-regulates mood, appetite, memory, learning and sleep; thus significantly increasing the sense of well-being as well as of happiness (Prabhakar, et al., 2015). The 5-HT system regulates emotional and behavioral activity. It has a vasoconstrictor function, which supports a positive affective sense. Its multiple roles of serotonin in

the functions of the brain involves behavior, sleep, pituitary hormone release regulation, temperature regulation, pain response, and many more (Fuller & Wong, 1977).

Secreted primarily in the Raphe nuclei, it is released into the inter-neuronal space (synaptic cleft) from where it diffuses into the neuronal membrane and activate its receptors situated there. Its neurotransmission ends when reuptake of serotonin occurs in the synapses, dissipating its chemical action in the area. Depletion of serotonin in the brain due to the reuptake mechanism will inevitably lead to mood deterioration, which may often lead to anxiety disorders and MDD (major depressive disorder). The reuptake system on the neuronal membrane inactivates the serotonin that can be found in the synaptic cleft. SSRIs are specific inhibitors that do not inhibit norepinephrine-acting neurons available in the area (Fuller & Wong, 1977). Fluoxetine is one of these specific inhibitors.

Fluoxetine maintains and boosts serotonin activity by blocking the reuptake pump, which transports serotonin to another location. The mechanism effectively decrease serotonin turnover and prevent the consequent reduction of the firing rate of the neuronal units in the raphe area of the brain, allowing rich levels of serotonin to stay in the synaptic cleft and continue neurotransmission (Fuller & Wong, 1997). The inhibitory action occurs through a desensitizing mechanism of the 5-HT receptors, particularly the serotonin 1A (Stahl, 2015), which prevents serotonin binding to its transporters (5-HTT). This mechanism theoretically creates the overall feeling of heightened euphoria.

In addition, fluoxetine has antagonistic effect on 5HT2C receptors (usually at

extra high doses of 60 to 80 mg), which could simultaneously increase neurotransmission involving norepinephrine and dopamine and contribute to the antidepressant effect of fluoxetine. Moreover, fluoxetine has also a stimulatory effect on neurosteroids, particularly allopregnanolone, which is a potent allosteric stimulant of the GABAα receptor (Pinna, Costa & Guidotti, 2009). Reduced level of allopregnanolone has been associated with depressive and anxiety conditions.

Thus, fluoxetine has inhibitory effect on serotonin reuptake pumps, antagonistic action against 5HT2C receptors, and stimulatory activity on allopregnanolone. This triple-target action of fluoxetine significantly enhances its antidepressant effect, making it popular among prescribing professionals. Moreover, the onset of therapeutic effect takes 2 to 4 weeks to appear; although, early increased energy occurs after the initial treatment (Stahl, 2015). If no symptomatic improvement occurs within 6 to 8 weeks, an increase in dose may be needed or it may never work.

Indicated psychological conditions

The U. S. FDA approved Prozac for the treatment of major depressive disorder (MDD), obsessive-compulsive disorder (OCD), bulimia nervosa (not approved for children), panic disorder (not approved for children), depressive episodes related with Bipolar I disorder (often with olanzapine [Zyprexa]), dysphoric premenstrual disorder, and treatment-resistant depression (TRD), also taken together with olanzapine (not approved for children). TRD is a depression that showed no improvement even after treatment with other two medications. However, Jang and colleagues (2015) reported Prozac as

the only SSRI (selective serotonin reuptake inhibitor) that the USFDA approved for the treatment of MDD in patients 17 years and younger.

Advantages and disadvantages

Advantages: Fluoxetine, like other SSRIs, is regarded as a first-line treatment for MDD due to its supposedly favorable risk-benefit ratio compared to tricyclic antidepressants and serotonin and norepinephrine reuptake inhibitors (SNRIs) (Meeker, et al., 2015; Capitao, et al., 2015). In long-term treatment regimen, fluoxetine has less adverse events and its dropout rates much lower than first-generation antidepressants and SNRIs. Calil (2001) reported it also safe in overdose and in such special groups as pregnant women.

Disadvantages: One important disadvantage with Prozac involves its many adverse effects that are life threatening, such as suicidal thoughts or actions; serotonin syndrome; severe allergic reactions; abnormal bleeding (especially when interacting with warfarin, non-steroidal anti-inflammatory drugs [NSAIDs], or aspirin); seizures/convulsions; manic episodes (i. e. heightened energy level, severe insomnia, racing thoughts, reckless behavior, strangely grand ideas, irritability or euphoria [excessive happiness], and fast-talking); appetite and/or weight changes (particularly among children and adolescents); hyponatremia (dangerous for the elderly); and abnormal cardiac electrical activity (e. g. arrhythmia, apnea, or dizziness/fainting) (U. S. Food and Drug Administration [USFDA], 2015).

Another disadvantage is the presence of withdrawal syndrome. Patients who stop taking Prozac abruptly may experience severe symptoms such as mental instability (i. e. anxiety, irritability, high or low mood, restlessness,

confusion, and/or insomnia) and physical disturbances (e. g. headache, sweating, queasiness/nausea, dizziness, electric shock-like sensations, and tremor/shaking) (USFDA, 2015; Meeker, et al., 2015).

Prozac is also not to be prescribed for children and adolescents due to highly pronounced adverse incidents (USFDA, 2015). A limited number of children and adolescents (up to 24 years) had exhibited suicidal tendencies or even commit suicide after taking this medication; more pronounced when they have a history of using antidepressants.

Efficacy

Outcomes in adolescents: An adolescent (age 16 years) with temporal lobe (recurrent grand mal) epilepsy with left mesial hippocampal sclerosis [symptoms include persistent suicidal and homicidal ideation, auditory and visual hallucinations, episodic memory loss, and persistent depression] experienced improved psychiatric symptoms (e. g. depression, suicidal ideation, homicidal ideation) after taking for seven days a combination of fluoxetine (30 mg) and aripiprazole (2-10 mg) (Jang, et al., 2015). Initial treatment with fluoxetine (e. g. 10-40 mg) alone for 20 days resulted to frequent episodes of memory loss at the highest dose. Fourteen days after the combination treatment, most symptoms had significantly improved. After discharge, a regular visit to her should remission of depression and suicidal/homicidal ideation. This outcome indicates the efficacy of Prozac when modulated by aripiprazole, a USFDA-approved adjunctive drug for MDD, to control inherent adverse effects (e. g. memory loss episodes).

Moreover, Capitao and others (2015) reported that adolescents (age 18 – 21 years) better accuracy in identifying anger and sadness after taking an acute

single-dose (20 mg) of Prozac at 6-hour post-treatment measurement. No overall significant effects were observed on the mood's subjective ratings. Outcomes on post-menopausal women: Macias-Cortes and colleagues (2015) reported the safety of fluoxetine in climacteric women; but not less effective in improving menopausal symptoms (e. g. significantly less fatigue, hot flushes, anxiety, depression and low quality of life experience) compared to homeopathy.

Conclusion

The assertion of Capitao and others (2015) of Prozac's gold standard status in treating adolescents used to be disavowed by the U. S. FDA (2015) due to the vulnerability of this age group to mental instability. The latter requirement of a medication guide insert may technically inform the patients on the risks. However, patients do not always understand how these risks work in their respective cases. A psychiatrist needs to provide them a satisfactory explanation for the risks to be taken and the options, of course. While the mechanism of action appears straightforward, there are details that remained to be scientifically verified though. Still, it cannot be denied that the popularity of fluoxetine may reflect its positive outcomes in most of patients in psychopathology. That is ground enough to be generally optimistic about the medication. However, it should be noted that the first-line status of fluoxetine is relative to other classes of antidepressants (i. e. tricyclics and SNRIs). It is not a reputation built upon a factual safety-efficacy standard that is beyond question. The permission to use it in adolescents is one point to be careful about due to its negative history with this age-group. If there is a better option in mild cases, perhaps the alternative may be

preferred as the least harmful, assuming that efficacy is at least comparable to fluoxetine. Macias-Cortes (2015), for instance, found homeopathy better.

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