

Esketamine Medication Role in Treating Alzheimer's Disease and Side Effects

Sha Zhang*

Department of Neurology, Xiangya Hospital Central South University, Changsha, China

*Corresponding author: Sha Zhang, Department of Neurology, Xiangya Hospital Central South University, Changsha, China, E-mail: hsa@gznh.cn

Received: 28-Jun-2023, Manuscript No. JADP-23-111120; **Editor assigned:** 30-Jun-2023, PreQC No. JADP-23-111120 (PQ); **Reviewed:** 14-Jul-2023, QC No. JADP-23-111120; **Revised:** 21-Jul-2023, Manuscript No. JADP-23-111120 (R); **Published:** 31-Jul-2023, DOI: 10.4172/2161-0460.1000576

Citation: Zhang S (2023) Esketamine Medication Role in Treating Alzheimer's Disease and Side Effects. J Alzheimers Dis Parkinsonism 13:576.

Copyright: © 2023 Zhang S. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

Description

Millions of people, as well as their families, are impacted by Alzheimer's disease. The main feature of Alzheimer's disease is the formation of aberrant protein aggregates in the brain, including beta-amyloid plaques and tau tangles. These aggregates cause neuronal loss, synaptic malfunction and inflammation, which together impair cognitive function [1]. A common psychological symptom of many neurological diseases is depression. Additionally, it increases the risk of developing stroke, Parkinson's disease and dementias including Alzheimer's and others. Ketamine is perhaps best known as a dissociative anesthetic used in medical and veterinary settings. Ketamine's mechanism of action involves modulating the neurotransmitter glutamate, which is implicated in synaptic plasticity, learning and memory. The rapid-acting and long-lasting antidepressant effects of (R,S)-ketamine for severe depression were found accidentally. (R,S) ketamine is a racemic combination of (R)-ketamine (also known as arketamine) and (S)-ketamine (also known as esketamine). (R)-Ketamine has stronger antidepressant-like effects in animal models of depression. It has positive effects in numerous animal models of neurological diseases in addition to these antidepressant-like effects in a number of animal models [2].

Role of esketamine

Esketamine is an antidepressant drug that is often sold under the trade name Spravato. It has received regulatory agency approval for the treatment of specific types of depression, including Major Depressive Disorder (MDD) with acute suicidal ideation or behavior and Treatment-Resistant Depression (TRD). Esketamine is known for having powerful antidepressant effects quickly. Esketamine can relieve depressed symptoms within hours to days, in contrast to standard antidepressants, which might take weeks to months to show visible mood improvements. For people with severe depression who have not reacted well to conventional medications, this quick onset of action can be especially helpful [3-6].

NMDA receptor modulator: The enantiomer of ketamine known as esketamine does certainly modulate the N-Methyl-D-Aspartate (NMDA) receptor. In the brain, NMDA receptors are a subtype of glutamate receptor that are important for synaptic plasticity, learning and memory. Like ketamine, esketamine works by altering these receptors to produce its effects. It works as a NMDA Receptor Antagonist by blocking or inhibiting the activity of NMDA receptors. These receptors are involved in the transmission of signals between neurons in the brain. It also works as Glutamate modulator. The brain's main excitatory neurotransmitter, glutamate, is essential for a variety of cognitive processes. One of the primary receptors where glutamate acts

is the NMDA receptor. Esketamine inhibits NMDA receptors, which controls glutamate signaling. Esketamine can affect synaptic plasticity, the brain's capacity to create new neural connections and alter existing ones, by inhibiting NMDA receptors. This may have effects on memory, learning and stimulus adaptation.

Administration

Esketamine is mainly used to treat people with treatment-resistant depression who have not reacted well to previous antidepressant medications. It is usually administered as a nasal spray in clinical settings.

Side effects

Dissociation: One of the esketamine side effects that is most frequently mentioned is dissociation. This is a feeling of disconnection from reality or from one's own ideas, feelings, or environment. Dissociation can range from mild to severe and it usually happens soon after esketamine is administered. Healthcare professionals frequently keep an eye out for dissociation in patients and make sure it goes away before the patient leaves the treatment center.

Sedation: Esketamine can make the person feel sleepy or sedated and this effect may last for several hours after the treatment. On the day of treatment, patients are typically advised not to drive or use any heavy equipment [7].

Elevated blood pressure: Esketamine has the potential to temporarily raise blood pressure.

Headache: Headache is a relatively common side effect of esketamine treatment.

Nausea and vomiting: Some individuals may experience nausea or vomiting after esketamine administration.

Feeling drunk or "out of it": Some patients report feeling as though they are drunk or have consumed alcohol after taking esketamine.

Anxiety or panic: In some cases, esketamine may trigger feelings of anxiety or panic, especially in individuals prone to these sensations.

Dizziness or vertigo: Sensations of dizziness or vertigo have been reported as side effects.

Blurred vision: Some people may experience temporary blurred vision after esketamine treatment.

Increased salivation: An increase in saliva production has been noted as a side effect.

Conclusion

Esketamine is a significant advancement in treating depression, particularly for treatment-resistant forms. It should be used in consultation with a qualified healthcare provider, considering the individual's circumstances and treatment history. It is not a one-size-fits-all solution and should be based on a thorough evaluation of the patient's needs, potential benefits and risks.

References

1. Andreescu C, Lenze EJ, Dew MA, Begley AE, Mulsant BH, et al. (2007). Effect of comorbid anxiety on treatment response and relapse risk in late-life depression: controlled study. *Br J Psychiatry* 190(4): 344-349.
2. Bremner JD, Krystal JH, Putnam FW, Southwick SM, Marmar C, et al. (1998). Measurement of dissociative states with the clinician-administered dissociative states scale (CADSS). *J Trauma Stress* 11:125-136.
3. Chandler GM, Iosifescu DV, Pollack MH, Targum SD, Fava M (2010). Validation of the massachusetts general hospital Antidepressant Treatment History Questionnaire (ATRQ). *CNS Neurosci Ther* 16(5): 322-325.
4. Fava M, Rush AJ, Alpert JE, Balasubramani GK, Wisniewski SR, et al. (2008). Difference in treatment outcome in outpatients with anxious versus nonanxious depression: a STAR* D report. *Am J Psychiatry* 165(3):342-351.
5. Fava M, Rush AJ, Alpert JE, Carmin CN, Balasubramani GK, et al. (2006). What clinical and symptom features and comorbid disorders characterize outpatients with anxious major depressive disorder: a replication and extension. *Can J Psychiatry* 51(13):823-835.
6. Fawcett J (2001). Treating impulsivity and anxiety in the suicidal patient. *Ann NY Acad Sci* 932(1):94-105.
7. Ionescu DF, Luckenbaugh DA, Niciu MJ, Richards EM, Slonena EE, et al. (2014). Effect of baseline anxious depression on initial and sustained antidepressant response to ketamine. *J Clin Psychiatry* 75(9):5470-5740.