

Ketamine: Anti-Depressant Effects

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Abstract

This review looks at the drug Ketamine and delves into some of the clinical situations where Ketamine can be used outside of its traditional use of induction. To challenge the traditional thinking of how Ketamine can be applied clinically this review will mainly touch upon Ketamine's use in the psychotherapeutic realm. This review will discuss Ketamine's role as an antidepressant, used in PTSD, used for the treatment of all types of addiction, and the abuse potential of using ketamine. the efficacy of the use of Ketamine This review will also briefly touch upon other areas where Ketamine has been used that are nontraditional. To determine the traditional methods of treating those issues, this review will incorporate research where statistical analysis has been done where Ketamine was added treatment plan where it would have otherwise. The results of the finding with the use of Ketamine will be compared to the results without the use of findings to see if there was a significant difference in the quality of care when Ketamine was applied. This review is intended to bring awareness to the multifaceted use of Ketamine in Clinicals and where using it could produce better outcomes.

Introduction

Ketamine is a drug whose traditional use is for induction. Originally available in 1970, Ketamine was targeted as a rapidly acting, nonbarbiturate general anesthetic for the short procedure (Clark, 2020). Ketamine has grown into a drug with so much more applications. Ketamine is a unique drug when it comes to induction agents used in anesthesia. Ketamine is the only induction drug out of pentothal, propofol, and etomidate that does not affect the Gabba receptors or the Reticular Activating System. Instead, Ketamine inhibits reflexes in the spinal cord as well as excitatory neurotransmitter effect in the brain, by binding non-competitively to the phencyclidine site on N-methyl-D-aspartate receptors (McGuinness et al., 2011). This uniqueness is what makes Ketamine a drug that can be used in a wide variety of settings. The one unique feature that this review will focus on is ketamine's ability to produce a disconnected dreamlike state at a subanesthetic dose (Trujillo, 2020). The use of Ketamine at a subanesthetic state was influenced by the search for a drug that could provide long-lasting antidepressant properties for patients who might be resistant to traditional antidepressants.

Anti-Depressant/PTSD MOA

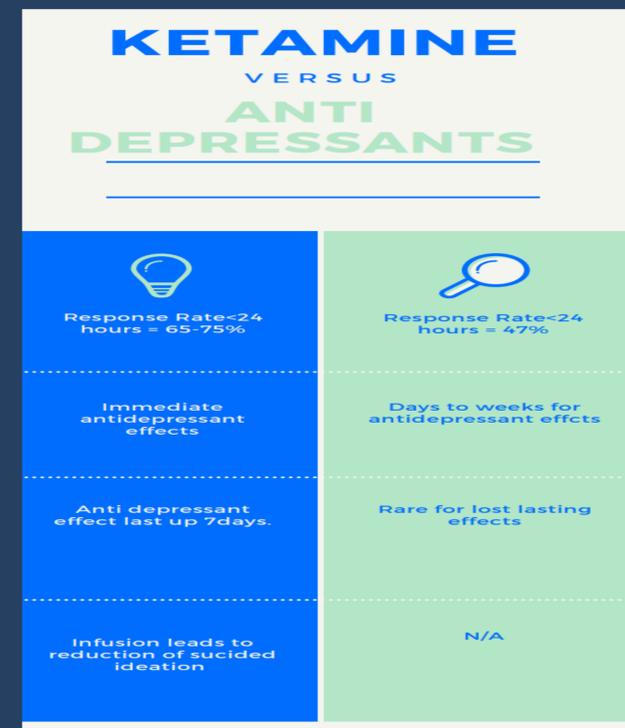
Ketamine works on the brain-derived neurotrophic factor(BDNF), α -amino-3-hydroxy-5-methyl-4-isoxazole propionic acid (AMPA) receptor and mammalian target of rapamycin (m Tor) signal pathway are involved in the antidepressant effects (EliLavender et al., 2020). At a chemical level, depression is caused by decreased BDNF levels in the mPFC and hippocampus, and increased levels in nucleus accumbent are related to depression-like behaviors in rodents (EliLavender et al., 2020). Ketamine produces increased levels of BDNF in the hippocampus that may promote long-term- anti-depressant actions. Ketamine's effects on PTSD are dose-dependent. Ketamine helps treat PTSD by the same mechanism of action as it treats antidepressants Current research has shown that Ketamine has been effective for alcohol dependence, heroin dependence, and reducing the craving and self-administration of cocaine (Ezquerro-Romano et al., 2018).

Ketamine VS Conventional Anti-Depressants

If Ketamine is given to treat depression the response rate is typically between 65--70% within 24 hours. If the same person was treating depression with conventional antidepressants the response rate would be 47% but that response rate is after taking the medication for weeks and months.(Lener et al., 2017; Trivedi et al., 2006). ketamine's antidepressant actions are almost immediate and last for approximately a week (Lener et al., 2017; McGirr et al., 2015). Most conventional anti-depressive are taken each day and they take weeks to produce an effect and they do not provide long lasting effects. (Lingford-Hughes et al., 2012). Studies have show that the thought of committing suicide was reduced when taking a ketamine infusion for several days (Ballard et al., 2014; DiazGranados et al., 2010; Larkin and Beautrais, 2011; Rebecca et al., 2009)

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Authors	Etiology	Dosage	Effect
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Conclusion

Ketamine use goes outside of just being used for its anesthetic and sedative properties during induction. Ketamine has been shown to be a beneficial drug for people who suffer from depression, PTSD, and addiction. While those are the three alternative uses of Ketamine that were discussed in this review Ketamine's other uses far extend those. Ketamine has been used in patients with burns, Ketamine has been used to treat chronic pain and help try to fight the opioid epidemic that is currently going on in the United States.

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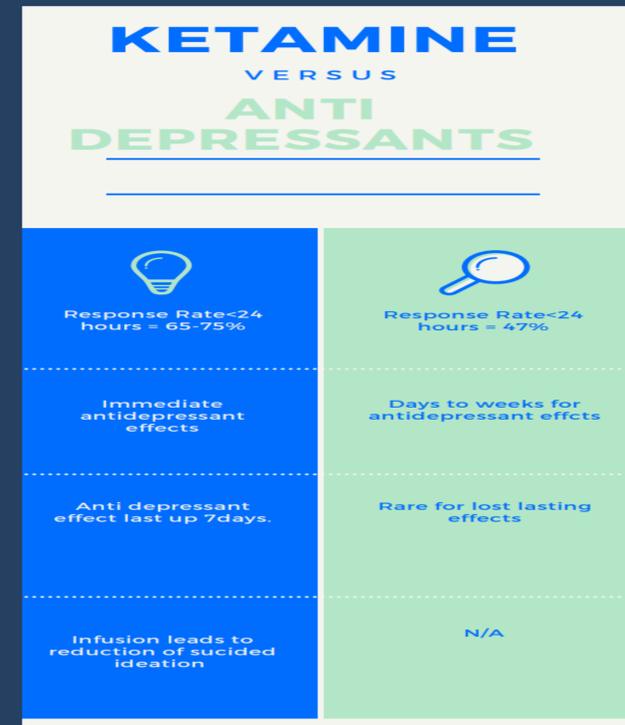
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Questions and Answers

Question 1: Which of the following drugs does not affect the GABA receptors or the Reticular Activating system True or false?

- a. Pentothal
- b. Ketamine
- c. Etomidate
- d. Propofol

Detailed Answers to Question 1: **Option B.** is the correct answer. Ketamine binds non-competitively to the phencyclidine site on N-methyl-D-aspartate (NMDA) receptors. **Option A.** Pentothal Enhances the inhibitory effects of GABA in the reticular activating system (RAS). **Option B.** Etomidate Depresses RAS and Inhibits GABA. **Option D.** Propofol enhancing the inhibitory effects of GABA.

Question 2: The Hippocampus is the main brain structure that contributes to depression at chemical level True or False ?

Detailed Answers to Question 2: Detailed Answers to Question 2: **True** is the correct answer decreased or increased levels of BDNF has been depressant/ anti-depressant effects

Question 3 According to research Ketamine's PTSD dose <0.5 mg/kg True or false?

Detailed Answers to Question 3: False is the Correct answer less <0.5 mg/kg dose of ketamine has been shown to help with anxiety but =>0.5mg/kg has dose needed to treat PTSD.

Question 4: At a chemical level depression is caused by what?

- a. Decreased levels of BDNF
- b. Increased levels of BDNF
- c. AMPA
- d. MTOR

Detailed Answers to Question 4: **Option A.** Is the correct answer. At a chemical level, depression is caused by decreased BDNF levels in the mPFC and hippocampus. Option B increased levels of BDNF is used to treat the depression. **Option C and D.** Are pathways that are involved in producing anti-depressant effects.

Question 5: All of the following are true of Ketamine vs Conventional Anti-depressant medications except?

- a. Takes weeks to produce an effect
- b. Response rate 65-75%
- c. Effects last up to 7 days
- d. Immediate Effects

Detailed Answers to Question 5: **Option A.** is the correct answer. Most conventional anti-depressive are taken each day and they take weeks to produce an effect. **Option B.** Ketamine is given to treat depression the response rate is typically between 65--70% within 24 hours. **Option C.** Ketamine's anti-depressant effects last for approximately a week. **Option D.** Ketamine's anti-depressant effects are immediate.

References

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