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Case Report

The Use of Ketamine in Traumatic Brain Injury

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Abstract

Ketamine, initially developed as an anesthetic agent, has gained significant attention for its potential therapeutic effects in various neurological conditions, particularly in patients with traumatic brain injury (TBI). The use of ketamine is controversial, especially due to concerns that it may increase intracranial pressure in patients with TBI. In this case report, a 29-year-old male with a diagnosis of severe head injury complicated by hemorrhagic shock from multiple traumas presented with decreased consciousness, active bleeding from the extremities, and hemodynamic instability. Rapid Sequence Intubation (RSI) was performed, during procedure which the patient was administered ketamine at a dose of 1 mg/kg, lidocaine 1.5 mg/kg, and rocuronium at 1 mg/kg. Fluid resuscitation with 1000 ml of crystalloid solution and norepinephrine drip 0.1 mcg/kg/min was also initiated. Post-resuscitation, the patient's hemodynamics were monitored and found stable. A literature search revealed systematic reviews from 2020 and studies from 2022 that focused on outcomes related to intracranial pressure and mortality in TBI patients receiving ketamine. The use of ketamine did not demonstrate evidence of harm in patients with traumatic head injury.

Keywords: Intracranial pressure, ketamine, traumatic brain injury

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Introduction

Ketamine, initially developed as an anesthetic, has gained significant attention for its potential therapeutic effects in various neurological conditions, particularly in patients with traumatic brain injury (TBI).[1] TBI encompasses a spectrum of injuries that can lead to severe cognitive, physical, and emotional impairments, posing significant public health challenges. Recent research suggests that ketamine may offer neuroprotective properties, which are particularly relevant in the context of TBI.[2] Its ability to modulate glutamate activity, reduce inflammation, and enhance neurogenesis, positioning ketamine as a candidate to mitigate secondary brain injuries—a critical phase that can exacerbate initial trauma. Despite these promising attributes, the use of ketamine in TBI is still under investigation, particularly concerning optimal dosing strategies, timing, and long-term outcomes. As research evolves, ketamine could emerge as a valuable tool in the multidisciplinary management of TBI, contributing to improved recovery outcomes and quality of life for affected individuals, maintaining hemodynamic stability, and enhancing postoperative pain quality.[3]

Case

A 29 years old male was diagnosed with hemorrhagic shock due to multiple trauma, severe head injury. Patient arrived at the Cipto Mangunkusumo hospital emergency department (ED) one hour after the traffic accident. He was riding a motorcycle when struck by a truck, falling on the asphalt on his right side and without using a helmet. Upon arrival at the ED, trauma code was

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Table 1. Primary Survey

Primary Survey	
Airway	Clear
Breathing	Respiratory rate: 35 x/minute
	Oxygen saturation: 95% on non-rebreather mask 15 L/minute
Circulation	Heart rate: 122 x/minute
	Blood pressure: 91/67 mmHg
Disability / Neurologic Assessment	GCS 10 (E3M3V3)
Exposure	Open fracture in the right lower extremity and closed fractures in the left lower extremity and right upper extremity

Table 2. Secondary Survey

Secondary Survey	
Allergies	None
Medication	None
Past Medical History	None
Last Meal	Unknown
Event	Traffic accident involving riding a motorcycle then struck by a truck, falling on the asphalt on the right side and without using a helmet.
Head	Anemic conjungtiva, sclera not icteric, no ottorhea, no deviated septum, equal pupil (isochore) 3 mm/3 mm, reactive pupils
Thorax	
Pulmonary	Symmetrical chest movement, no crepitation, bilateral vesicular breath sounds, no rales or wheezing
Cardiac	S1S2 normal, no gallop or murmur
Abdomen	No hematoma on abdomen and flank area, supple, bowel movement normal
Extremities	Open fracture in the right lower extremity and closed fractures in the left lower extremity and right upper extremity, cold extremities, capillary refill time more than 2 seconds

activated. From alloanamnesis with the parents, the patient had no history of previous illness such as hypertension, diabetes, pulmonary or cardiac disease. Patient also had no history of allergies or asthma, as well as no history of prior surgeries.

Patient presented with decreased consciousness and active bleeding from the extremities. There was an open fracture in the right lower extremity and closed fractures in the left lower extremity and right upper extremity. There were no signs of internal bleeding. The patient had a Glasgow Coma Scale (GCS) score of 10, responded to pain stimuli, with gasping breaths, and experienced seizure on arrival. Hemodynamics was unstable with a blood pressure of 91/67 mmHg, pulse rate of 122 beats per minute (bpm), respiration rate of 35 breaths per minute, and SpO_2 of 95% on 15 L/min non-rebreather mask (NRM). From the alloanamnesis with the witness, it was reported that the patient experienced vomiting after the collision and had decreased consciousness. Peripheral access of 18G was inserted on left upper extremity and femoral access of 16G was also inserted on left femur. Blood sample was taken for further investigation. Fluid resuscitation was initiated with 1000 milliliters of crystalloid. Due to unstable hemodynamics, decreased consciousness and ineffective respiration, intubation was performed using Rapid Sequence Intubation (RSI). Ketamine was administered at 1 mg/kg, Lidocaine at 1.5 mg/kg, and rocuronium

Table 3. Laboratory Results

Laboratory Results	
Hematology	Hemoglobin: 7.7, Hematocrit: 21.5, Leucocyte: 9560, Thrombocyte: 174.000, Mean Corpuscular Volume (MCV): 82.1, Mean Corpuscular Hemoglobin (MCH): 28.5
Blood Gas Analysis	pH: 7.310, pCO_2: 25, pO_2: 258, HCO_3: 20, Saturation O_2 96.3%, BE: -2.50
PT/APTT	PT: 11.5 (11.3), APTT: 30.7 (30.1)
Electrolytes	Natrium: 138, Potassium: 3.9, Chloride: 110
SGOT/SGPT	SGOT: 32, SGPT: 40
Ureum/Creatinine	Ureum: 24.5, Creatinine: 0.7
Glucose	119
Lactate	3.4

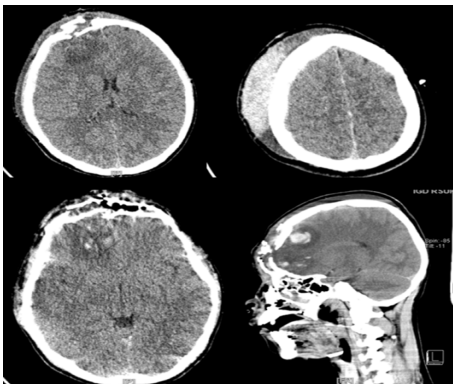

Figure 1. Head CT-Scan without contrast

Table 4. Primary Survey after Intubation

Primary Survey Post Intubation	
Airway	clear on ETT
Breathing	Respiratory rate: 14 x/minute
	Oxygen saturation: 97-99% on Pressure Control with P insp 14, Rate 14, PEEP 5, FiO_2 60%
Circulation	Heart rate: 130 x/minute
	Blood pressure: 88/50 mmHg
Disability / Neurologic Assessment	Sedated, equal pupil (isochore) 3 mm/3 mm, reactive pupils
Exposure	Open fracture in the right lower extremity and closed fractures in the left lower extremity and right upper extremity

at 1 mg/kg. Post-intubation, hemodynamics remained unstable with BP 88/50 mmHg and HR 130 bpm. Fluid resuscitation with crystalloids was continued, and norepinephrine drip was started at 0.1 mcg/kg/min. The patient was stabilized in the ED while awaiting ICU availability. Head CT-scan without contrast was done and found there were subdural bleeding in right fronto-temporo-parietal region, subarachnoid bleeding in right frontal region and intracerebral bleeding with perifocal edema in cortical-subcortical right frontal lobe with estimated volume of 23.1 ml which causes subfalcine herniation to the left in anterior frontal lobe region.

Discussion

Literature search was done using database on PubMed® and Cochrane Library® using keywords “Ketamine”, “traumatic brain injury”, and “intracranial pressure”. All study in English from last ten years were included and checked for double. Two studies were found to be appropriate for analysis, both were systematic reviews which included all significant studies regarding our topic.

The unique pharmacokinetics and pharmacodynamics of ketamine make it a potential neuroprotective agent in TBI management.[1] The two studies reviewed indicate that ketamine may have beneficial effects in managing intracranial pressure (ICP) and reducing mortality in TBI patients. Ketamine's ability to modulate neurotransmitter activity and reduce excitotoxicity could help stabilize or lower ICP, a critical factor in preventing further brain damage from intracranial hypertension.[3] While some studies suggest that ketamine use in

Table 5. Study results

Author	Outcome	Results	Conclusion
Zanza C, Piccolella F, Racca F, Romenskaya T, Longhitano Y, Franceschi F, et al.[4]	Intracranial pressure	The systematic review examined the use of ketamine and its effect on intracranial pressure outcomes by including 11 case reports. These case reports indicated that no pathological intracranial pressure was observed. There was a brief period of decreased intracranial pressure and an increase that did not reach life-threatening levels. It was also found that in the pediatric population, ketamine could reduce intracranial pressure.	Due to its pharmacokinetic and pharmacodynamic characteristics, including its neuromodulatory properties, ketamine is a safe medication that can be used alone or in combination with other sedatives in patients with moderate to severe traumatic brain injury requiring mechanical ventilation.
Gregers MCT, Mikkelsen S, Lindvig KP, Brochner AC.[5]	Intracranial pressure	This systematic review included 7 articles addressing intracranial pressure outcomes. The articles reported that 3 showed no change in intracranial pressure, 1 observed increases of 5 and 8 mmHg, and 1 demonstrated a decrease in intracranial pressure after 2 minutes of ketamine bolus administration, followed by an increase after 30 minutes. However, all articles indicated that there was no life-threatening increases in intracranial pressure or effects on mortality.	None of the studies in the report showed evidence of harm from ketamine use in TBI patients.

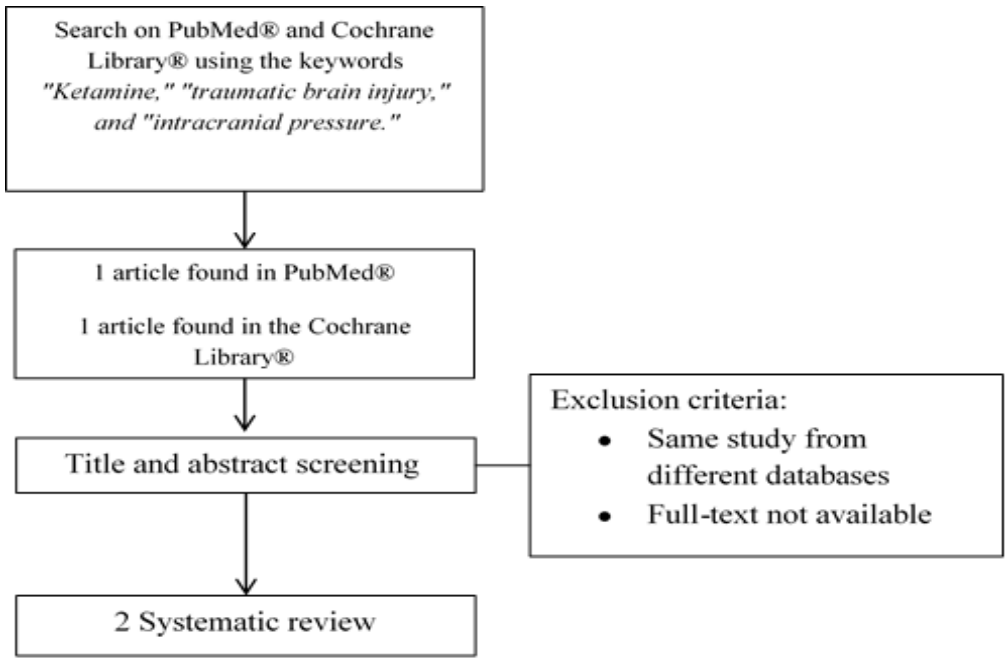

Figure 1. Flow Diagram of Literature Search

TBI might be associated with reduced mortality, the data is still limited and varies based on patient characteristics and injury severity.[3-4] Despite the potential benefits, ketamine's use must be carefully managed due to possible side effects, such as dissociation, increased blood pressure, and risk of abuse.[6] The two studies reviewed presented varied outcomes, but both indicated that ketamine does not pose significant harm in TBI patients. The systematic reviews included studies with weak quality, making strong recommendations difficult. Nonetheless, no studies demonstrated evidence of harm from ketamine use in TBI patients. In the clinical scenario presented, where the patient required life-saving intubation due to decreased consciousness from a traffic accident, ketamine's use is supported by evidence-based medicine, indicating no life-threatening increases in ICP or negative impact on mortality. Hemodynamics of the patient was relatively similar before and after the use of ketamine. No changes were found in pupil size and reaction after intubation, suggesting no increased in ICP or slight increase but does no give negative effect.

However, changes in the severity of ICP were only evaluated clinically in this scenario because there were no CT-scan prior to intubation with ketamine. Ketamine's neuroprotective effects are supported by evidence suggesting it can modulate neurotransmitter release and improve cerebral blood flow, particularly beneficial in traumatic brain injury (TBI).[8,9] According to one study stated ketamine shows promise in reducing intracranial pressure (ICP) without exacerbating mortality rates, making it a valuable option in the acute phase of severe TBI.[7] However, variability in patient response and study quality necessitates further research to confirm its efficacy and establish clear clinical guidelines.[7,10] Further research is essential to clarify the optimal dosing and timing of ketamine administration to maximize its benefits.

Conclusion

Ketamine shows promise as a therapeutic agent in TBI management, particularly in controlling intracranial pressure and potentially reducing mortality. Its neuroprotective mechanisms, including reducing excitotoxicity and inflammation, are crucial in preventing further brain injury. Early administration of ketamine in TBI patients may contribute to better clinical outcomes, though the data is limited, and further research is necessary. While promising, ketamine's use must be cautiously approached, with strict monitoring and evidence-based application. Overall, ketamine has potential as part of a TBI treatment strategy, but more research is needed to fully determine its effectiveness and safety in this context. Ketamine shows promise as a therapeutic agent in TBI management, particularly in controlling intracranial pressure and potentially reducing mortality. Its neuroprotective mechanisms, including reducing excitotoxicity and inflammation, are crucial in preventing further brain injury. Early administration of ketamine in TBI patients may contribute to better clinical outcomes, though the data is limited, and further research is necessary. While promising, ketamine's use must be cautiously approached, with strict monitoring and evidence-based application. Overall, ketamine has potential as part of a TBI treatment strategy, but more research is needed to fully determine its effectiveness and safety in this context.

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