**Review Article**

**Sedation for non-invasive ventilation after blunt chest trauma**

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**ABSTRACT**

Chest trauma remains an issue for health services for both severe and apparently mild trauma management. Severe chest trauma is associated with high mortality and is considered liable for 25% of mortality in multiple traumas. Blunt traumas are commonly secondary to motor vehicle accidents (MVAs), falls and crush or blast injuries. Notably, pain control seems a crucial endpoint in our success to deliver non-invasive ventilation to patients with chest trauma, when feasible as a pivotal component of patient care after chest trauma, along with non-invasive ventilation. In this context, dexmedetomidine could be an alternative to improve NIV tolerance that provides sedation and analgesia with no significant respiratory depression and a reduced risk of delirium. In addition, Ketamine has several advantages compared with conventional sedatives such as preserving pharyngeal and laryngeal protective reflexes, lowering airway resistance, increasing lung compliance, and being less likely to produce respiratory depression. Therefore; this review article will briefly discusses the safety and efficacy of Dexmedetomidine versus Ketamine in non-invasive ventilation after blunt chest trauma.

**INTRODUCTION**

Trauma is the third leading cause of death in all age groups after cardiovascular diseases and cancer.\(^1\) Chest trauma remains a serious problem as high-speed vehicle accidents increase. Thoracic trauma occurs in approximately 60% of patients with polytrauma and has a mortality of 20%–25%. Trauma can be divided into two types: penetrating and blunt.\(^2\)

This review discusses blunt chest trauma using Dexmedetomidine versus Ketamine in non-invasive ventilation as pain tolerance.

1. **Blunt Chest Trauma**

Blunt traumas were defined as injuries that organs and structures were injured without disrupting tissue integrity. Blunt traumas can also be classified according to their mechanisms. The mechanism of blunt trauma can be listed as motor vehicle accident, occupational accident and fall.\(^3\)

1.1. **Clinical findings of Blunt Chest Trauma**

In a blunt trauma, clinical findings revealed that all structures in the thorax can be damaged, such as chest wall tissues, thoracic cage, ribs, lung, pleura, large vessels, diaphragm, heart and mediastinal structures.\(^3\) Early diagnosis and treatment are important for the prevention of the mortality and complications in patients with blunt thoracic

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trauma. Advanced examination and imaging methods are very valuable and more sensitive for diagnosis.

1.2. Acute Respiratory Distress Syndrome (ARDS)

Among different mechanisms, the early use of positive end-expiratory pressure after chest trauma, when feasible, seems mandatory to optimize oxygenation and improve clinical outcomes. Indeed, interventions aimed at preventing acute respiratory distress syndrome (ARDS) after chest trauma carry the greatest potential to reduce the substantial morbidity, mortality, and resource utilization associated with this syndrome. Patients may present with tachycardia, tachypnoea, hemoptysis, hypotension, confusion, hypoxemia, hypercarbia and increased work of breathing.

1.3. The importance of adequate pain control

Untreated pain can have adverse effects on the hemodynamic stability, gastrointestinal system and renal functions and may lead to conditions such as tachycardia and hypercoagulability.

Nonsteroidal anti-inflammatory drugs (NSAIDs), systemic opioids or regional analgesia methods such as epidural analgesia, intrapleural analgesia, intercostal nerve block, and thoracic paravertebral block can be used for pain control.

2. Non-Invasive Ventilation After Blunt Chest Trauma

Blunt chest trauma in civilian populations is commonly seen after vehicular collisions and falls and is associated with significant mortality and morbidity. Recently the use of non-invasive ventilation (NIV) in a subset of patients has been suggested as an alternative to these traditional respiratory support strategies.

3. Dexmedetomidine and Ketamine to improve patient tolerance for Non-Invasive Ventilation in blunt chest trauma

3.1. Ketamine

Ketamine HCl is a rapidly acting general anesthetic with sedative and analgesic properties. The dissociative anesthetic action of ketamine provides excellent analgesia and anesthesia, with retention of protective pharyngeal and laryngeal reflexes and without depressing respiration. Numerous small series have documented its favorable effects on cardiovascular and pulmonary parameters.

Ketamine HCl is a rapidly acting general anesthetic with sedative and analgesic properties. It is rapidly acting with a short half-life, and the dissociative effects allows for excellent analgesia and anesthesia, with retention of protective pharyngeal and laryngeal reflexes and preserved respiratory effort.

As a phencyclidine derivative, ketamine is in a unique class that induces a functional disorganization between the thalamoneocortical and limbic systems, producing a “dissociative state”. The primary anesthetic properties of ketamine are due to its antagonism of Central Nervous System (CNS) N-methyl-D-aspartate (NMDA) receptors.

Ketamine is also a potent analgesic and has been used effectively for post-surgical analgesia, spinal analgesia, and in chronic pain management settings. Its analgesic mechanism is multifactorial and is in part due to its effects on mu and kappa opioid receptors. Ketamine also promotes central sympathetic stimulation and inhibition of neuronal catecholamine uptake. Moreover, it has also been reported to have immune modulating effects including the inhibition of TNF-a, IL-1, and IL-6 effects to endotoxemia.

Ketamine’s popularity as an agent for maintenance sedation has been related in part to its favorable effects on the pulmonary and cardiovascular systems. Ketamine has been successfully used to treat patients with severe bronchospasm, refractory to conventional
bronchodilators. The ability of ketamine to antagonize antigen-induced bronchospasm may be related to its vagolytic and direct smooth muscle relaxant effects.  

In addition to its efficacy in relieving bronchospasm via bolus administration, continuous infusion of ketamine has also been shown to safely improve pulmonary function. Ketamine improves air entry and decreases wheezing in patients with refractory bronchospasm, as well as improving other objective measurements of respiratory dynamics.  

In addition to preserving respiratory tone, ketamine also augments thoracic compliance. Dynamic compliance (Cdyn) is an important factor in the successful management of status asthmatics, especially in asthmatics on mechanical ventilation.

In addition to its favorable respiratory dynamics profile, the hemodynamic effects of ketamine for maintenance sedation are also promising. In tachycardic and hypotensive patients, the heart rate and systolic blood pressure have been reported to improve following ketamine infusion. Additionally, vasopressor requirements during ketamine maintenance infusion have variably been reported to be decreased or unchanged.

Ketamine should be avoided in decompensated heart failure or cardiogenic shock. Whereas ketamine has a favorable cardiovascular profile related to central sympathetic stimulation and inhibition of neuronal catecholamine uptake which counteracts its direct negative inotropic effects, this favorable profile may not be seen in patients with decompensated heart failure.

In such patients, ketamine’s negative inotropic effects may be unmasked, resulting in deterioration in cardiac performance and cardiovascular instability. Furthermore, since ketamine has been reported to cause hypertension and supraventricular tachycardia in patients on thyroxine re-placement; clinicians should employ additional caution with such patients.

Ketamine has been shown to have beneficial pulmonary and hemodynamic effects when administered as maintenance sedation via continuous infusion. In patients with refractory bronchospasm, ketamine has been reported to decrease audible wheeze, decrease bronchodilator requirements, and improve respiratory rate and oxygenation, and decrease hypercarbia when administered via continuous IV infusion.

Additionally, it offers a favorable hemodynamic profile compared to fentanyl, midazolam, or propofol in that it does not result in significant blood pressure, heart rate, or vascular resistance perturbations. The available evidence suggests that ketamine may be a safe and effective tool for maintenance sedation; however a large prospective clinical trial is necessary to further clarify the utility of ketamine as an agent for continuous IV maintenance sedation of patients on mechanical ventilation.

The use of non-invasive mechanical ventilation (NIV) in patients with severe blunt chest trauma has been associated with reduced tracheal intubation rate, length of intensive care unit (ICU) stay and/ or incidence of ventilator-associated pneumonia. However respiratory comfort and control of chest wall pain, agitation and anxiety is a prerequisite to reduce the incidence of NIV failure in this trauma population. Thoracic epidural analgesia for blunt thoracic trauma is the most examined technique. One alternative is the use of hypnotics and/or opioids, inducing respiratory depression and possibly aggravating the morbidity of chest trauma.

3.2. Dexmedetomidine

Dexmedetomidine could be an alternative to improve NIV tolerance. Dexmedetomidine is a short-acting alpha-2 adrenoreceptor agonist that provides sedation and analgesia with no significant respiratory depression and a reduced risk of delirium. It was found to improve patient ability to communicate with their caregiver and reduce intubation duration in patients with agitated delirium.
Dexmedetomidine-induced effect on NIV duration was not reproducible during a subsequent session. This adds to the recent guidelines suggesting the preferential use of dexmedetomidine over benzodiazepines for mechanically ventilated patients.16

A TTSS of more than 6 was found to have an impact on outcome after chest trauma. With no significant pain at rest as prerequisite, patients received dexmedetomidine (or placebo) at a rate of 0.7 mcg/kg/h one hour prior to starting the first NIV session in order to optimize the drug effect. Although this sequential order seemed as the most efficient to maximize the NIV tolerance, two patients refused the second NIV session of the second cycle.17

Respiratory distress following chest trauma is multifactorial and includes pain, rib fractures, flail chest, pleural effusions, and lung contusions. Placing the patient under invasive mechanical ventilation increases the risk of ventilator-associated pneumonia. The use of NIV in cooperative patients was proposed to overcome this issue.17

In the three randomised studies having tested NIV in chest trauma, pain was controlled with epidural analgesia or intravenous remifentanil infusion, or with a midazolam infusion and a morphine-PCA device.18

As an alternative, dexmedetomidine has already been tested for its ability to improve NIV tolerance in non-trauma patients. In patients who refused to continue NIV due to discomfort, dexmedetomidine was shown to achieve the expected level of sedation and improve NIV tolerance. This drug was associated with a lower tracheal intubation rate compared to midazolam in patients who had discomfort with previous NIV sessions.18

Patients in acute respiratory failure were shown to tolerate NIV for longer durations following dexmedetomidine compared to placebo. This was associated with a reduced respiratory discomfort while differences in RASS scores could be viewed as mild.19

Despite the improved NIV tolerance, dexmedetomidine had no effect on pain scores and morphine consumption. This is not surprising given that dexmedetomidine was administered in patients in whom the control of chest wall pain at rest was prerequisite and who had received multimodal analgesia prior to initiating dexmedetomidine.

CONCLUSION

Dexmedetomidine and Ketamine can be used to improve patient tolerance for non-invasive ventilation after blunt chest trauma

REFERENCES