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Dexmedetomidine as a Neuroprotective Sedative Agent in Ultrasound-Guided Ulnar Nerve Block for a Patient with Traumatic Brain Injury: A Case Report

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A B S T R A C T

Introduction: Traumatic brain injury (TBI) often presents alongside extracranial injuries requiring surgical intervention. General anesthesia in such cases poses significant challenges, particularly in patients with concomitant pulmonary complications. This case report describes the successful use of ultrasound-guided ulnar nerve block combined with dexmedetomidine sedation for a patient with moderate TBI and pulmonary contusion undergoing open reduction and internal fixation (ORIF) of a left-hand finger fracture. **Case presentation:** A 50-year-old male presented with moderate TBI, pulmonary contusion, and an open fracture of the fifth digit of his left hand following a motor vehicle accident. Due to the risks associated with general anesthesia, an ultrasound-guided ulnar nerve block was performed using levobupivacaine 0.375%. Dexmedetomidine was used as a sedative agent due to its neuroprotective properties and minimal respiratory depressant effects. The procedure was successful, with the patient maintaining stable hemodynamics and adequate sedation throughout the surgery. **Conclusion:** This case highlights the feasibility and safety of ultrasound-guided peripheral nerve block combined with dexmedetomidine sedation as an alternative to general anesthesia in patients with TBI and pulmonary contusion. Dexmedetomidine's neuroprotective effects and minimal respiratory depression make it a valuable tool in managing such complex cases.

1. Introduction

Traumatic brain injury (TBI) is a significant public health concern, encompassing a spectrum of severities and presenting formidable challenges in clinical management. It is a leading cause of mortality and disability worldwide, with an estimated annual incidence of 69 million individuals sustaining TBI across all severities. The consequences of TBI are far-reaching, impacting not only the individual but also their families, communities, and healthcare systems. The complexities of TBI management are further compounded when it occurs in conjunction with other bodily injuries, a frequent scenario in trauma cases. These additional injuries often necessitate surgical intervention, introducing a new layer of complexity to anesthetic considerations. The choice of anesthetic technique must carefully balance the need for surgical anesthesia with the potential risks to the already compromised brain. General anesthesia, the traditional mainstay for surgical procedures, presents particular challenges in the context of TBI. It involves the administration of drugs that induce a state of

unconsciousness, amnesia, and analgesia, along with the use of positive pressure ventilation to maintain respiration. While effective for surgical anesthesia, general anesthesia can have unintended consequences for the injured brain. One of the primary concerns with general anesthesia in TBI patients is the potential for increased intracranial pressure (ICP). The injured brain is particularly vulnerable to changes in ICP, and elevations can compromise cerebral blood flow, leading to secondary brain injury. General anesthesia can influence ICP through various mechanisms, including alterations in cerebral blood flow, cerebrovascular reactivity, and cerebrospinal fluid dynamics.[1-4]

Another challenge posed by general anesthesia is the potential for hemodynamic instability. TBI can disrupt the brain's ability to regulate blood pressure and heart rate, making patients more susceptible to fluctuations during anesthesia. These fluctuations can further compromise cerebral perfusion, potentially exacerbating brain injury. Furthermore, general anesthesia often requires the use of medications that can have neurodepressant effects. These medications can interfere with neurological assessment, making it difficult to monitor the patient's neurological status during and after surgery. This can hinder the timely detection of neurological deterioration, potentially delaying critical interventions. In the presence of pulmonary injuries, such as pulmonary contusion, the risks associated with general anesthesia are further amplified. Pulmonary contusion, a common accompaniment to TBI in trauma cases, can impair gas exchange and respiratory function. General anesthesia, particularly the use of positive pressure ventilation, can exacerbate these respiratory challenges, increasing the risk of postoperative pulmonary complications. Given the potential drawbacks of general anesthesia in TBI patients, particularly those with concomitant pulmonary injuries, there has been a growing interest in exploring alternative anesthetic techniques. Regional anesthesia, which involves the administration of local anesthetics to block nerve conduction in a specific region of the body, has emerged as a promising alternative.[5-7]

Regional anesthesia offers several advantages over general anesthesia in the TBI population. It avoids the need for airway manipulation and positive pressure ventilation, reducing the risk of pulmonary complications. It also minimizes the impact on systemic hemodynamics and respiratory function, providing greater stability during surgery. Additionally, regional anesthesia allows for a faster recovery, earlier neurological assessment, and reduced need for opioid analgesics, which can have neurodepressant effects. Among the various regional anesthesia techniques, peripheral nerve blocks (PNBs) have gained popularity for their versatility and effectiveness. PNBs involve the injection of local anesthetic around a specific nerve or group of nerves, providing localized anesthesia to the surgical area. The advent of ultrasound guidance has further enhanced the safety and accuracy of PNBs, allowing for precise needle placement and visualization of the targeted nerve. In this context, the use of dexmedetomidine as a sedative agent in conjunction with regional anesthesia has gained traction. Dexmedetomidine, a highly selective alpha-2 adrenoceptor agonist, possesses unique properties that make it particularly appealing for TBI patients. It provides sedation, anxiolysis, and analgesia while exhibiting minimal respiratory depression. Moreover, dexmedetomidine has demonstrated neuroprotective effects in various experimental and clinical studies, making it a promising agent for mitigating secondary brain injury.[8-10] This case report presents the successful anesthetic management of a patient with moderate TBI and pulmonary contusion undergoing open reduction and internal fixation (ORIF) of a left-hand finger fracture using an ultrasound-guided ulnar nerve block and dexmedetomidine sedation.

2. Case Presentation

This case report details the anesthetic management of a 50-year-old male who presented to our emergency department following a motor vehicle accident. The patient's initial presentation painted a picture of significant trauma with potential multi-system involvement, necessitating a comprehensive and systematic evaluation. The patient's history was limited to the acute event – a motor vehicle accident. No further details regarding the circumstances of the accident, such as the speed of impact or mechanism of injury,

were available at the time of initial assessment. This lack of information underscores a common challenge in emergency medicine, where the immediate priority is stabilizing the patient and addressing life-threatening conditions. However, a detailed history, including pre-existing medical conditions, medications, and allergies, is crucial for informed decision-making and optimal patient care. The initial assessment followed the Advanced Trauma Life Support (ATLS) protocol, focusing on the primary survey (ABCDE) to identify and manage immediate life threats. The patient's airway was patent, ensuring adequate ventilation. Respiratory assessment revealed an elevated respiratory rate (26 breaths/min) with bilateral rhonchi, suggesting potential pulmonary injury. Despite this, oxygen saturation was maintained at 96% on room air, improving to 99% with supplemental oxygen via nasal cannula. This indicated adequate oxygenation despite the underlying pulmonary insult. The patient's circulatory status was deemed stable, with no signs of active bleeding or hemodynamic compromise. Neurological examination revealed a Glasgow Coma Scale (GCS) score of E3V4M5, indicating moderate traumatic brain injury. The patient exhibited purposeful eye-opening to verbal commands, confused verbal responses, and localized pain. Pupils were isochoric and reactive to light, measuring 3 mm in diameter. These findings suggested an intact brainstem function. However, the patient also displayed agitation, a common finding in TBI, which can be attributed to pain, anxiety, disorientation, or underlying cerebral injury. A thorough physical examination revealed an open wound on the left hand, necessitating further investigation. The secondary survey, a more detailed head-to-toe examination, identified an open fracture at the base of the fifth digit on the left hand. This finding, in conjunction with the patient's agitation and potential pulmonary injury, underscored the need for prompt surgical intervention. Laboratory tests were crucial in assessing the patient's overall physiological status and identifying potential complications. A hemoglobin level of 10.5 g/dL (normal range for males: 13.5-17.5 g/dL) indicated mild anemia. This finding was likely attributable to blood loss from the accident and the open fracture. Close monitoring of hemoglobin levels was essential to ensure adequate oxygen-carrying capacity and guide potential transfusion requirements. An elevated white blood cell count of 14,000/µL (normal range: 4,500-11,000/µL) suggested an inflammatory response to the trauma. This finding, while expected in the acute phase of injury, warranted close monitoring for signs of infection. A platelet count of 250,000/µL (normal range: 150,000-450,000/µL) was within the normal range, indicating adequate clotting ability. This was reassuring, especially in the context of an open fracture and potential surgical intervention. A blood glucose level of 110 mg/dL (normal range: 70-100 mg/dL) showed mild hyperglycemia, a common stress response to trauma. Maintaining normoglycemia is crucial in critically ill patients, as hyperglycemia can exacerbate neurological injury and impair immune function. A normal coagulation profile (Prothrombin time (PT), International normalized ratio (INR), and activated partial thromboplastin time (aPTT) within normal limits) suggested no underlying coagulopathy. This was essential for safe surgical intervention and minimized the risk of bleeding complications. Arterial blood gas analysis revealed a pH of 7.35, $PaCO_2$ of 45 mmHg, PaO_2 of 80 mmHg, and HCO_3 of 24 mEq/L. These findings indicated mild respiratory acidosis with hypoxemia, likely due to the suspected pulmonary contusion. Close monitoring of arterial blood gases was necessary to assess the adequacy of ventilation and oxygenation. Electrolyte levels (Sodium 138 mEq/L, Potassium 4.0 mEq/L, Chloride 100 mEq/L) were within the normal range, indicating no major electrolyte imbalances. Maintaining electrolyte balance is crucial for optimal cellular function and overall physiological stability. Imaging studies played a pivotal role in confirming the clinical suspicions and guiding further management. The CT scan revealed intracranial hemorrhage (ICH) with subarachnoid hemorrhage (SAH) in the left parietal region, intraventricular hemorrhage (IVH) in bilateral lateral ventricles and the third ventricle, and subdural hemorrhage (SDH) in the falx cerebri. These findings confirmed the diagnosis of traumatic brain injury and provided crucial information regarding the location and extent of the hemorrhage. The presence of brain edema further highlighted the severity of the injury. The chest X-ray confirmed the

presence of pulmonary contusion, explaining the patient's respiratory symptoms and the abnormalities observed in the arterial blood gas analysis. The X-ray confirmed the open fracture of the base of the fifth digit, necessitating surgical intervention. Based on the comprehensive clinical, laboratory, and imaging findings, the following diagnoses were established; Moderate traumatic brain injury (TBI): The patient's GCS score, along with the CT scan findings of intracranial hemorrhage and brain edema, confirmed the diagnosis of moderate TBI; Pulmonary contusion: The chest X-ray findings, coupled with the patient's respiratory symptoms and arterial blood gas analysis, confirmed the diagnosis of pulmonary contusion; Open fracture of the base of the fifth digit of the left hand: The physical examination and X-ray findings confirmed the open fracture, requiring surgical intervention. This case presented a complex clinical scenario with the convergence of moderate TBI, pulmonary contusion, and an open fracture. The combination of these injuries posed significant challenges in anesthetic management, necessitating a careful consideration of the risks and benefits of various anesthetic techniques (Table 1).

This section details the peri-operative management of the patient, encompassing the anesthetic strategy, surgical procedure, and post-operative course. Given the patient's moderate TBI and pulmonary contusion, meticulous planning and execution were paramount to ensure safety and optimize outcomes; Preoperative: The patient was assigned an American Society of Anesthesiologists (ASA) physical status classification of III, indicating severe systemic disease. This classification reflected the patient's moderate TBI and pulmonary contusion, which posed significant risks for anesthesia and surgery. After careful consideration of the patient's condition and the potential risks associated with general anesthesia, a decision was made to proceed with an ultrasound-guided ulnar nerve block combined with dexmedetomidine sedation. This approach aimed to minimize the impact on cerebral hemodynamics and respiratory function while providing adequate anesthesia and analgesia for the surgical procedure; Intraoperative: Standard monitoring, including non-invasive blood pressure (NIBP), electrocardiogram (ECG), and pulse oximetry, was implemented to ensure continuous assessment of the patient's vital parameters. Oxygenation was maintained via nasal cannula at a flow rate of 3 L/min. Dexmedetomidine, a highly selective alpha-2 adrenoceptor agonist, was chosen as the sedative agent due to its favorable pharmacological profile in the context of TBI. It provides sedation, anxiolysis, and analgesia while exhibiting minimal respiratory depression. Moreover, dexmedetomidine has demonstrated neuroprotective effects in various experimental and clinical studies, making it a promising agent for mitigating secondary brain injury. A loading dose of dexmedetomidine 1 mcg/kg was administered over 10 minutes, followed by a maintenance infusion of 0.5 mcg/kg/hour. Fentanyl 50 mcg was administered intravenously for analgesia. An ultrasound-guided ulnar nerve block was performed using a linear probe (SonoSite) to visualize the ulnar nerve. A 23G needle (Terumo) was inserted, and 5 mL of levobupivacaine 0.375% was injected after careful aspiration to exclude intravascular placement. The use of ultrasound guidance enhanced the safety and accuracy of the nerve block, ensuring adequate anesthesia for the surgical procedure; Postoperative: Following the completion of the surgical procedure, the dexmedetomidine infusion was discontinued. Intravenous paracetamol 1000 mg was administered for postoperative analgesia; Procedure of Surgery: The surgical procedure involved open reduction and internal fixation (ORIF) of the fifth digit of the left hand using K-wire fixation. The duration of the surgery was 1.5 hours. The patient's hemodynamics remained stable throughout the procedure, and adequate sedation was maintained with dexmedetomidine; Immediate Postoperative Period: The patient's neurological status showed improvement in the immediate postoperative period, with a GCS score of E4V5M6. This indicated a favorable response to the surgical intervention and anesthetic management. Postoperative pain was well-controlled with intravenous paracetamol. The patient maintained adequate oxygen saturation (SpO_2 98%) on room air, and bilateral breath sounds were clear, indicating no respiratory complications. The patient's hemodynamic parameters remained stable in the immediate postoperative period;

24 Hours Postoperative: The patient exhibited further improvement in neurological status, with a GCS score of E4V5M6. Adequate pain control was achieved with oral analgesics. The patient showed no signs of respiratory distress, and oxygen saturation remained at 99% on room air. The surgical wound appeared clean and dry, with no signs of infection; 7 Days Postoperative: The surgical wound continued to heal well. The patient demonstrated continued neurological improvement, achieving a GCS score of 15, indicating a full recovery from the TBI. Hand function gradually improved with physiotherapy. Long-term follow-up with orthopedics and neurosurgery was planned to monitor the patient's recovery and ensure optimal functional outcomes. This case report highlights the feasibility and safety of an ultrasound-guided ulnar nerve block combined with dexmedetomidine sedation in a patient with moderate TBI and pulmonary contusion undergoing hand surgery. This approach successfully avoided the potential risks associated with general anesthesia, providing stable hemodynamics, adequate sedation, and a favorable postoperative course. The patient's neurological status improved throughout the perioperative period, with no evidence of respiratory or hemodynamic complications. This case adds to the growing body of evidence supporting the use of regional anesthesia and dexmedetomidine in the management of TBI patients requiring surgical intervention (Table 2).

Table 1. Anamnesis, clinical findings, laboratory, imaging, and diagnosis.

Category	**Findings**
Anamnesis	50-year-old male. Involved in a motor vehicle accident.
Clinical finding	Primary Survey: - Airway: Clear. - Breathing: Symmetrical chest expansion, RR 26 breaths/min, bilateral rhonchi, SpO_2 96% on room air, 99% with 3 L/min oxygen via nasal cannula. - Circulation: Clear. - Disability: GCS E3V4M5, isochoric pupils 3 mm. - Exposure: Open wound on the left hand. Secondary Survey: - Open fracture of the base of the fifth digit on the left hand. - Agitation.
Laboratory	- Hemoglobin: 10.5 g/dL (normal range for males: 13.5-17.5 g/dL). This indicates mild anemia, possibly due to blood loss from the accident and fracture. - White blood cell count: 14,000/μL (normal range: 4,500-11,000/μL). This signifies leukocytosis, suggesting an inflammatory response to the trauma. - Platelet count: 250,000/μL (normal range: 150,000-450,000/μL). This is within the normal range, indicating adequate clotting ability. - Blood glucose: 110 mg/dL (normal range: 70-100 mg/dL). This shows mild hyperglycemia, which is a common stress response to trauma. - Coagulation profile: Normal (Prothrombin time (PT), International normalized ratio (INR), and activated partial thromboplastin time (aPTT) within normal limits). This suggests no underlying coagulopathy, which is important for surgical intervention. - Arterial blood gas analysis: pH 7.35, $PaCO_2$ 45 mmHg, PaO_2 80 mmHg, HCO_3 24 mEq/L. This indicates mild respiratory acidosis with hypoxemia, likely due to the pulmonary contusion. - Electrolytes: Sodium 138 mEq/L, Potassium 4.0 mEq/L, Chloride 100 mEq/L. These are within the normal range, indicating no major electrolyte imbalances.
Imaging	- CT scan of the head: Intracranial hemorrhage (ICH) with subarachnoid hemorrhage (SAH) in the left parietal region, intraventricular hemorrhage (IVH) in bilateral lateral ventricles and third ventricle, subdural hemorrhage (SDH) in the falx cerebri, brain edema. - Chest X-ray: Pulmonary contusion. - X-ray of the left hand: Open fracture of the base of the fifth digit.
Diagnosis	- Moderate traumatic brain injury (TBI). - Pulmonary contusion. - Open fracture of the base of the fifth digit of the left hand.

Table 2. Anesthesia management, procedure of surgery, and follow-up.

Category	Details
Anesthesia management	**Preoperative:** - ASA physical status classification: III - Plan: Ultrasound-guided ulnar nerve block with dexmedetomidine sedation **Intraoperative:** - Monitoring: Non-invasive blood pressure (NIBP), electrocardiogram (ECG), pulse oximeter - Oxygenation: Nasal cannula 3 L/min - Medications: - Loading dose dexmedetomidine 1 mcg/kg over 10 minutes - Maintenance dose dexmedetomidine 0.5 mcg/kg/hour - Fentanyl 50 mcg for analgesia - Ultrasound-guided ulnar nerve block: - Linear probe, SonoSite - 23G needle, 25 mm (Terumo) - Extension tube 150 cm (Terumo) - Disposable syringe 10 mL (Terumo) - Levobupivacaine 0.375%, 5 mL **Postoperative:** - Dexmedetomidine infusion stopped - Intravenous paracetamol 1000 mg for analgesia.
Procedure of surgery	- Open reduction and internal fixation (ORIF) of the fifth digit of the left hand - K-wire fixation - Duration of surgery: 1.5 hours - Hemodynamics: Stable throughout the procedure - Sedation: Adequate throughout the procedure.
Follow-up	- Immediate postoperative period: - Neurological status: GCS E4V5M6, improved from preoperative status - Pain: Well-controlled with intravenous paracetamol - Respiratory status: SpO_2 98% on room air, clear breath sounds bilaterally - Hemodynamic status: Stable - 24 hours postoperative: - Neurological status: Further improvement in GCS, now E4V5M6 - Pain: Adequate pain control with oral analgesics - Respiratory status: No signs of respiratory distress, SpO_2 99% on room air - Wound: Clean and dry - Discharge planning initiated - 7 days postoperative: - Wound: Healing well - Neurological status: Continued improvement, GCS 15 - Hand function: Gradually improving with physiotherapy - Long-term follow-up with orthopedics and neurosurgery planned.

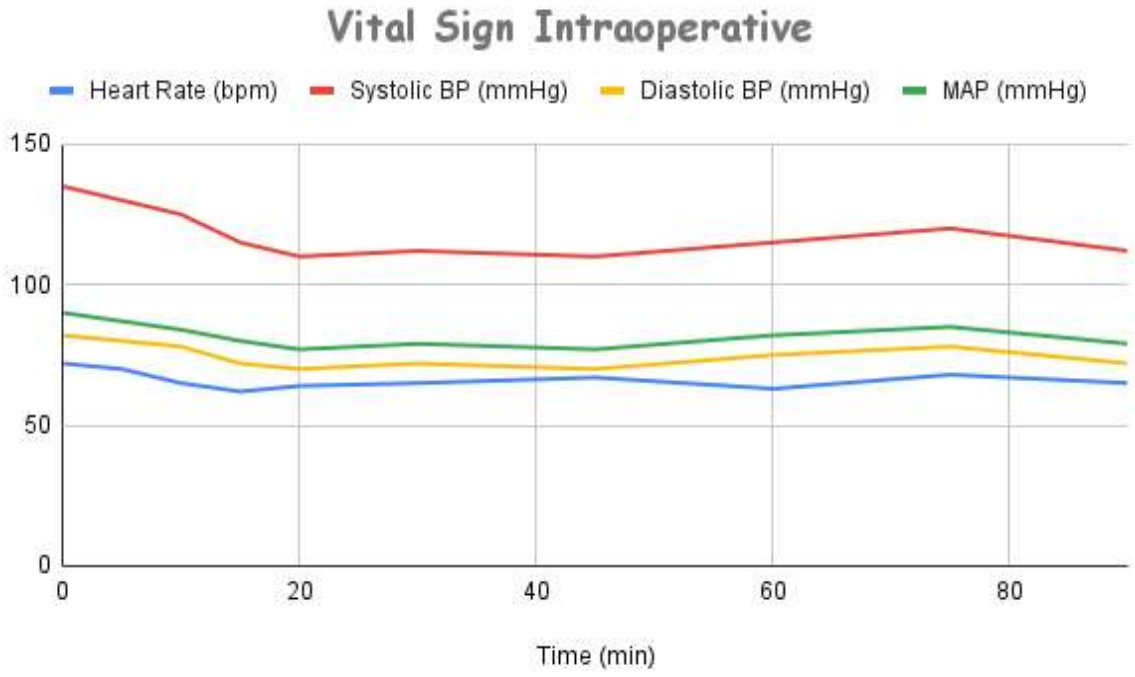

Figure 1. Vital sign intraoperative.

3. Discussion

Traumatic brain injury (TBI) is a complex and multifaceted condition that presents unique challenges for anesthetic management. The injured brain is in a delicate state, vulnerable to secondary insults that can further exacerbate the initial injury. Anesthesia, while essential for surgical procedures, can inadvertently contribute to this secondary injury if not meticulously planned and executed. Understanding the intricate interplay between TBI and anesthesia is crucial for optimizing patient outcomes and minimizing the risk of neurological deterioration. The brain, the command center of the human body, is a highly metabolically active organ with a limited capacity to store energy. It relies on a constant supply of oxygen and glucose delivered through cerebral blood flow to maintain its function. Any disruption to this delicate balance, such as that caused by TBI, can have profound consequences. The injured brain often exhibits impaired cerebral autoregulation, the ability to maintain constant cerebral blood flow despite changes in systemic blood pressure. This makes the brain more susceptible to fluctuations in blood pressure, which can lead to ischemia (inadequate blood flow) or hyperemia (excessive blood flow), both of which can worsen brain injury. TBI can lead to cerebral edema (swelling of the brain tissue), bleeding within the skull, and impaired cerebrospinal fluid drainage. These factors can contribute to increased ICP, which can compress brain tissue and blood vessels, further compromising cerebral blood flow and potentially leading to herniation (displacement of brain tissue). TBI triggers a complex inflammatory response within the brain, involving the release of various inflammatory mediators that can contribute to neuronal damage and cell death. TBI can disrupt the brain's metabolic processes, leading to imbalances in electrolytes, neurotransmitters, and energy substrates. These disturbances can further impair neuronal function and contribute to secondary injury. Anesthesia, while necessary for surgical procedures, can exacerbate these pathophysiological processes if not carefully managed. General anesthesia, the traditional mainstay for surgery, involves the administration of drugs that induce a state of unconsciousness, analgesia, and amnesia, along with the use of positive pressure ventilation to maintain respiration. While effective for surgical anesthesia, general anesthesia can pose significant challenges in the context of TBI. One of the primary concerns with general anesthesia in TBI patients is the potential for increased ICP. Volatile anesthetics, commonly used to induce and maintain general anesthesia, can increase cerebral blood flow and cerebral metabolic rate, potentially leading to increased ICP. Positive pressure ventilation, a cornerstone of general anesthesia, can impede venous return from the brain, further contributing to increased ICP. Hypercapnia (elevated carbon dioxide levels in the blood), which can occur during general anesthesia if ventilation is not carefully managed, causes cerebral vasodilation, increasing cerebral blood flow and ICP. Hypoxia (low oxygen levels in the blood) can also lead to cerebral vasodilation and increased ICP. TBI can disrupt the brain's ability to regulate blood pressure and heart rate, making patients more prone to fluctuations during anesthesia. General anesthesia can further contribute to hemodynamic instability through its effects on the cardiovascular system. Some anesthetic agents can cause hypotension (low blood pressure), which can compromise cerebral perfusion, particularly in patients with impaired cerebral autoregulation. Hypertension (high blood pressure) can also occur during anesthesia, potentially leading to increased cerebral blood flow and ICP. Tachycardia (rapid heart rate) and bradycardia (slow heart rate) can both occur during anesthesia, potentially affecting cardiac output and cerebral perfusion. General anesthesia often requires the use of medications that can have neurodepressant effects. These medications can interfere with neurological assessment, making it difficult to monitor the patient's neurological status during and after surgery. This can hinder the timely detection of neurological deterioration, potentially delaying critical interventions. Volatile anesthetics can depress neuronal activity and impair neurological function. Opioids, commonly used for pain management during and after surgery, can also have neurodepressant effects, including respiratory depression and decreased level of consciousness. In the presence of pulmonary injuries, such as pulmonary contusion, the risks associated with general anesthesia

are further amplified. Pulmonary contusion, a common accompaniment to TBI in trauma cases, can impair gas exchange and respiratory function. General anesthesia, particularly the use of positive pressure ventilation, can exacerbate these respiratory challenges, increasing the risk of postoperative pulmonary complications. Pulmonary contusion can lead to ventilation-perfusion mismatch, where areas of the lung are ventilated but not perfused, or vice versa. This impairs gas exchange and can lead to hypoxemia and hypercapnia. Pulmonary contusion can increase airway resistance, making ventilation more challenging and potentially leading to barotrauma (lung injury caused by high airway pressures). Patients with TBI may have impaired airway reflexes, increasing the risk of aspiration (inhalation of foreign material into the lungs) during general anesthesia. Given the potential drawbacks of general anesthesia in TBI patients, particularly those with concomitant pulmonary injuries, there has been a growing interest in exploring alternative anesthetic techniques. Regional anesthesia, which involves the administration of local anesthetics to block nerve conduction in a specific region of the body, has emerged as a promising alternative. It avoids the need for airway manipulation and positive pressure ventilation, reducing the risk of pulmonary complications, which is particularly crucial in patients with pulmonary contusion. It minimizes the impact on systemic hemodynamics and respiratory function, providing greater stability during surgery, which is essential in patients with TBI who may have compromised cerebral autoregulation. It allows for a faster recovery, earlier neurological assessment, and reduced need for opioid analgesics, which can have neurodepressant effects, facilitating the early detection of any neurological deterioration. Regional anesthesia can attenuate the stress response to surgery, which can be beneficial in TBI patients who may already be in a hypermetabolic state. Regional anesthesia can provide excellent postoperative pain control, reducing the need for opioids and their potential side effects.[11-13]

Dexmedetomidine, a highly selective alpha-2 adrenoceptor agonist, has emerged as a valuable tool in the anesthesiologist's armamentarium, particularly in the context of neuroanesthesia. Its unique pharmacological properties, including sedation, analgesia, anxiolysis, and minimal respiratory depression, coupled with its potential neuroprotective effects, make it an attractive alternative to traditional sedative agents, especially in patients with traumatic brain injury (TBI). Dexmedetomidine exerts its effects primarily through its interaction with alpha-2 adrenoceptors, which are widely distributed throughout the central and peripheral nervous systems. Activation of alpha-2 adrenoceptors in the locus coeruleus, a brainstem nucleus involved in arousal and vigilance, leads to a decrease in norepinephrine release, resulting in a state of sedation that resembles natural sleep. This sedation is characterized by arousability, allowing patients to be easily awakened and cooperate with neurological assessments. Dexmedetomidine enhances opioid-induced analgesia and provides independent analgesic effects through its action on spinal cord alpha-2 adrenoceptors. This can reduce the need for opioids, which can have neurodepressant effects and contribute to respiratory depression. Activation of alpha-2 adrenoceptors in the amygdala, a brain region involved in fear and anxiety, contributes to the anxiolytic effects of dexmedetomidine. This can help alleviate anxiety and agitation, which are common in TBI patients, and promote a calm and cooperative state. Dexmedetomidine also exerts sympatholytic effects, leading to a decrease in heart rate, blood pressure, and systemic vascular resistance. This can be particularly beneficial in TBI patients who may have compromised cerebral autoregulation and are more susceptible to fluctuations in blood pressure. Beyond its sedative, analgesic, and anxiolytic effects, dexmedetomidine has demonstrated promising neuroprotective properties in various experimental and clinical studies. Excitotoxicity, a process involving excessive glutamate release and neuronal damage, is a key contributor to secondary brain injury following TBI. Dexmedetomidine has been shown to attenuate excitotoxicity by decreasing glutamate release and inhibiting NMDA receptors, which are involved in glutamate-mediated neuronal injury. Neuroinflammation plays a significant role in the pathophysiology of TBI. Dexmedetomidine has been shown to exert anti-inflammatory effects by reducing the production of pro-inflammatory cytokines

and chemokines, which can contribute to neuronal damage and cell death. Oxidative stress, an imbalance between the production of reactive oxygen species and the body's antioxidant defenses, is another important contributor to secondary brain injury. Dexmedetomidine has been shown to possess antioxidant properties, scavenging free radicals and protecting neurons from oxidative damage. Mitochondria, the powerhouses of cells, are particularly vulnerable to injury following TBI. Dexmedetomidine has been shown to preserve mitochondrial function, maintaining cellular energy production and reducing apoptosis (programmed cell death). Neurotrophic factors are proteins that promote neuronal survival, growth, and differentiation. Dexmedetomidine has been shown to modulate the expression of neurotrophic factors, potentially contributing to neuronal repair and regeneration following TBI. Studies have shown that dexmedetomidine sedation in TBI patients is associated with improved neurological outcomes, including reduced mortality, shorter duration of mechanical ventilation, and better cognitive function. Dexmedetomidine has been shown to reduce cerebral edema and ICP in TBI patients, potentially mitigating secondary brain injury. Studies have demonstrated that dexmedetomidine can improve cerebral blood flow and oxygenation in TBI patients, promoting neuronal recovery. Dexmedetomidine has been shown to have anticonvulsant properties, potentially reducing the risk of seizures in TBI patients. Dexmedetomidine is generally well-tolerated, with a favorable safety profile. Its most common side effects include hypotension, bradycardia, and dry mouth. These side effects are usually mild and transient, and can be managed with appropriate monitoring and supportive care. In the presented case, dexmedetomidine was chosen as the sedative agent for the patient with moderate TBI and pulmonary contusion undergoing ORIF of a left-hand finger fracture. Dexmedetomidine's potential neuroprotective effects were considered crucial in this patient with TBI, aiming to minimize the risk of secondary brain injury. Dexmedetomidine's minimal respiratory depressant effects made it a safe option in this patient with pulmonary contusion, who may have had compromised respiratory function. Dexmedetomidine's sympatholytic effects helped maintain hemodynamic stability during the procedure, which is essential in TBI patients with potentially impaired cerebral autoregulation. Dexmedetomidine's unique sedative properties allowed for ongoing neurological assessment, enabling the early detection of any neurological deterioration. The successful outcome of this case, with the patient exhibiting neurological improvement and no evidence of respiratory or hemodynamic complications, further supports the use of dexmedetomidine in this patient population.[14,15]

The management of traumatic brain injury (TBI) patients requiring surgery presents a unique challenge for anesthesia providers. The delicate balance between providing adequate anesthesia for the surgical procedure and protecting the injured brain from further insult necessitates a careful and nuanced approach. In recent years, the synergistic combination of regional anesthesia and dexmedetomidine sedation has emerged as a promising alternative to traditional general anesthesia, offering a multitude of benefits that align with the specific needs of this vulnerable patient population. Regional anesthesia, which involves the administration of local anesthetics to block nerve conduction in a specific region of the body, has long been recognized for its ability to provide excellent surgical anesthesia while minimizing the systemic effects associated with general anesthesia. Dexmedetomidine, a highly selective alpha-2 adrenoceptor agonist, has gained prominence in anesthesia practice due to its unique pharmacological properties, including sedation, analgesia, anxiolysis, and minimal respiratory depression, coupled with its potential neuroprotective effects. The combination of these two techniques creates a synergistic approach that capitalizes on their respective strengths while mitigating their limitations. Regional anesthesia, such as the ultrasound-guided ulnar nerve block used in the presented case, provides targeted anesthesia to the surgical site, effectively blocking pain signals and allowing the surgical procedure to proceed without the need for general anesthesia. General anesthesia typically requires endotracheal intubation and positive pressure ventilation, which can have detrimental effects on the injured brain. Positive pressure ventilation can

increase intracranial pressure (ICP) and impede cerebral venous drainage, potentially exacerbating brain injury. Regional anesthesia avoids these risks, allowing for spontaneous ventilation and maintaining physiological respiratory patterns. General anesthesia can cause fluctuations in blood pressure and heart rate, which can be particularly detrimental in TBI patients with compromised cerebral autoregulation. Regional anesthesia, on the other hand, has minimal impact on systemic hemodynamics, promoting greater stability and reducing the risk of cerebral hypoperfusion or hyperemia. General anesthesia often involves the use of medications that can have neurodepressant effects, making it difficult to assess neurological status during and after surgery. Regional anesthesia avoids the use of these medications, allowing for a clearer neurological picture and facilitating the early detection of any neurological deterioration. Dexmedetomidine complements regional anesthesia by providing sedation, anxiolysis, and analgesia without causing significant respiratory depression. This is particularly important in TBI patients who may already have compromised respiratory function due to associated injuries, such as pulmonary contusion. Dexmedetomidine's unique sedative properties allow patients to remain arousable and cooperative, facilitating neurological assessment and minimizing the need for deeper levels of sedation. One of the most compelling aspects of this synergistic approach is the potential for neuroprotection. Excitotoxicity, a key contributor to secondary brain injury, involves excessive glutamate release and neuronal damage. Dexmedetomidine attenuates excitotoxicity by decreasing glutamate release and inhibiting NMDA receptors, which are involved in glutamate-mediated neuronal injury. Neuroinflammation plays a significant role in the pathophysiology of TBI. Dexmedetomidine exerts anti-inflammatory effects by reducing the production of pro-inflammatory cytokines and chemokines, which can contribute to neuronal damage and cell death. Oxidative stress, an imbalance between the production of reactive oxygen species and the body's antioxidant defenses, is another important contributor to secondary brain injury. Dexmedetomidine possesses antioxidant properties, scavenging free radicals and protecting neurons from oxidative damage. Mitochondria, the powerhouses of cells, are particularly vulnerable to injury following TBI. Dexmedetomidine preserves mitochondrial function, maintaining cellular energy production and reducing apoptosis (programmed cell death). Neurotrophic factors are proteins that promote neuronal survival, growth, and differentiation. Dexmedetomidine modulates the expression of neurotrophic factors, potentially contributing to neuronal repair and regeneration following TBI. By incorporating dexmedetomidine into the anesthetic plan, this synergistic approach not only provides comprehensive anesthesia but also offers an additional layer of protection to the injured brain, potentially mitigating secondary brain injury and improving neurological outcomes. Hemodynamic stability is paramount in TBI patients, as fluctuations in blood pressure and heart rate can compromise cerebral perfusion and exacerbate brain injury. Regional anesthesia avoids the systemic effects of general anesthesia, which can include hypotension, hypertension, and tachycardia/bradycardia. Dexmedetomidine exerts sympatholytic effects, leading to a decrease in heart rate, blood pressure, and systemic vascular resistance. This can be particularly beneficial in TBI patients with impaired cerebral autoregulation, as it helps maintain a stable cerebral perfusion pressure. The stress response to surgery can trigger the release of catecholamines, which can increase heart rate and blood pressure. Regional anesthesia and dexmedetomidine can attenuate this stress response, further contributing to hemodynamic stability. Early neurological assessment is crucial in TBI patients, as it allows for the timely detection of any neurological deterioration and prompt intervention. General anesthesia often involves the use of medications that can have neurodepressant effects, making it difficult to assess neurological status. Regional anesthesia and dexmedetomidine avoid these medications, allowing for a clearer neurological picture. Dexmedetomidine's unique sedative properties allow patients to remain arousable and cooperative, enabling them to participate in neurological assessments and provide valuable information about their condition.[16-18]

The case presented in this report serves as a compelling testament to the feasibility, safety, and efficacy of the synergistic approach combining regional anesthesia and dexmedetomidine sedation in the management of patients with moderate traumatic brain injury (TBI) and concomitant pulmonary contusion. This particular case involved a 50-year-old male who sustained these injuries following a motor vehicle accident and required open reduction and internal fixation (ORIF) of a left-hand finger fracture. The moderate TBI, evidenced by a Glasgow Coma Scale (GCS) score of E3V4M5 and confirmed by computed tomography (CT) findings of intracranial hemorrhage, necessitated careful consideration of anesthetic agents and techniques to avoid exacerbating the brain injury. The presence of pulmonary contusion, confirmed by chest X-ray, further complicated the anesthetic management. Pulmonary contusion can impair gas exchange and respiratory function, increasing the risk of respiratory complications associated with general anesthesia, particularly those related to positive pressure ventilation. The open fracture of the left-hand finger required surgical intervention, adding another layer of complexity to the anesthetic plan. Recognizing the unique challenges posed by this patient's condition, the anesthesia team opted for a patient-centered approach, carefully considering the potential risks and benefits of various anesthetic techniques. Regional anesthesia, in this case, an ultrasound-guided ulnar nerve block, provided excellent surgical anesthesia while avoiding the potential detrimental effects of general anesthesia on the injured brain. By avoiding airway manipulation, positive pressure ventilation, and the use of volatile anesthetics, this approach minimized the risk of increased intracranial pressure (ICP), hemodynamic instability, and neurodepression. The use of regional anesthesia also avoided the potential respiratory complications associated with general anesthesia in the presence of pulmonary contusion. By allowing for spontaneous ventilation, this approach minimized the risk of ventilation-perfusion mismatch, barotrauma, and aspiration. Dexmedetomidine, chosen as the sedative agent, offered the added benefit of potential neuroprotection. Its ability to reduce excitotoxicity, attenuate neuroinflammation, combat oxidative stress, preserve mitochondrial function, and modulate neurotrophic factors potentially mitigated secondary brain injury and promoted neuronal recovery. Dexmedetomidine's unique sedative properties allowed the patient to remain arousable and cooperative, facilitating ongoing neurological assessment and enabling the early detection of any neurological deterioration. The combination of regional anesthesia and dexmedetomidine promoted hemodynamic stability, which is crucial in TBI patients with potentially impaired cerebral autoregulation. The successful outcome of this case, with the patient maintaining stable hemodynamics and adequate sedation throughout the surgical procedure, is a testament to the efficacy of this tailored anesthetic approach. The patient's neurological status improved throughout the perioperative period, with no evidence of respiratory or hemodynamic complications. The ultrasound-guided ulnar nerve block provided precise and effective anesthesia to the surgical site, ensuring patient comfort and allowing the surgical procedure to proceed smoothly. The combination of regional anesthesia and dexmedetomidine maintained hemodynamic stability, protecting the injured brain from fluctuations in blood pressure and heart rate. The avoidance of general anesthesia and positive pressure ventilation minimized the risk of respiratory complications associated with pulmonary contusion. Dexmedetomidine's potential neuroprotective effects likely contributed to the patient's neurological improvement and the absence of neurological deterioration. The ability to perform ongoing neurological assessments facilitated the early detection of any subtle changes in the patient's neurological status, allowing for prompt intervention if necessary. This successful case highlights the importance of individualizing anesthetic management in patients with TBI, particularly those with concomitant injuries. The traditional reliance on general anesthesia is gradually shifting towards a more nuanced approach that prioritizes patient safety and neurological well-being. The synergistic combination of regional anesthesia and dexmedetomidine offers a compelling alternative, particularly in patients with pulmonary injuries, providing hemodynamic stability, pulmonary

protection, and the opportunity for early neurological assessment.[19,20]

4. Conclusion

This case report underscores the feasibility and safety of utilizing an ultrasound-guided ulnar nerve block with dexmedetomidine as a sedative agent in a patient with moderate TBI and pulmonary contusion undergoing hand surgery. This approach effectively circumvented the potential risks associated with general anesthesia, ensuring hemodynamic stability, adequate sedation, and a favorable postoperative recovery. Notably, the patient's neurological status progressively improved throughout the perioperative period, without any signs of respiratory or hemodynamic complications. Our findings contribute to the growing body of evidence supporting the use of regional anesthesia and dexmedetomidine in managing TBI patients requiring surgical intervention. The neuroprotective properties of dexmedetomidine, coupled with its minimal respiratory depression, make it a particularly attractive option for this patient population. The success of this case highlights the importance of tailoring anesthetic management to the unique needs of TBI patients, particularly those with concomitant injuries. By combining regional anesthesia techniques with the judicious use of dexmedetomidine, anesthesia providers can optimize patient outcomes, minimize the risk of secondary brain injury, and ensure a safe and effective surgical experience.

5. References

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