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A Case Report on Dexmedetomidine's Unique Role in Management of Postoperative Muscle Spasm

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Introduction

Painful muscle spasm is a common complaint in the postoperative period. Postoperative muscle spasms are mostly experienced by patients who have had laparotomies and cervical and lumbar laminectomies[8,13]. Moreover, muscle spasms can be exacerbated in patients with histories of cervical sprain or lumbosacral strain, torticollis, and various other orthopedic conditions[9].

Skeletal muscle relaxants are widely used in treating musculoskeletal conditions. Comparison studies have not shown one skeletal muscle relaxant to be superior to another due to limited availability of comparable effectiveness data. In general, muscle relaxant selection is based on side-effect profile, patient preference, abuse potential, and possible drug interactions[15]. In the postoperative anesthesia care unit (PACU), selection of skeletal muscle relaxants is mostly based on their adverse effects, route of administration, and drug interactions. For example, administration of intravenous diazepam in a patient recovering from anesthesia may pose a huge concern. Coadministration of benzodiazepines and opioids have been associated with significant morbidity and mortality in the form of respiratory depression and respiratory failure, respectively[7].

Centrally acting skeletal muscle relaxants such as cyclobenzaprine (Flexeril), metaxalone (Skelaxin), carisoprodol (Soma), and methocarbamol (Robaxin) are used for muscle spasm. The utility of these drugs has been limited due to their side effects such as sedation, dizziness, drowsiness, and blurry vision[16]. Unlike other centrally acting anti-spasmodic agents, tizanidine is a centrally acting alpha2-adrenergic agonist. It is commonly prescribed to reduce spasticity related to cerebral or spinal cord injury, multiple sclerosis, and other spastic disorders by increasing pre-synaptic inhibition of motor neurons. Tizanidine exerts these effects at both the level of the brain and the spinal cord[10]. Unfortunately, its use is also restricted by its sedative properties.

Because centrally acting muscle relaxants are mostly available in oral formulations, their utility in the PACU is not ideal. Slow onset and aspiration of oral formulations make immediate administration of these medications difficult in the PACU setting. Similarly to tizanidine, dexmedetomidine is also an alpha2-adrenoceptor agonist, but with one key advantage: its ability to be administered in an intravenous formulation. According to Pichot et al., dexmedetomidine can also be combined with analgesics to exert its superior effects. These effects include reducing the incidence of postoperative agitation

and muscle spasm[12]. Herein, we report a positive response to dexmedetomidine and how it was utilized to manage a patient's severe cervical muscle spasm after undergoing orthopedic shoulder surgery.

Case Presentation

Our patient is a 46-year-old obese male (106.6kg; BMI 34.0kg/m^2) with a past medical history of anxiety, depression, post-traumatic stress disorder, chronic neck pain, obstructive sleep apnea, and cervical spinal fusion who presented for elective right shoulder arthroscopy. Patient's history of chronic neck pain was controlled with home medications such as oxycodone-5mg/acetaminophen-325mg, oxycodone 10mg extended release and pregabalin 150mg. Prior to the procedure, patient consented and agreed to a preoperative ultrasound-guided interscalene nerve block for intraoperative and postoperative pain management. Patient was premedicated with 2mg of Midazolam and 100mcg of Fentanyl for anxiety prior to the block. An additional 100mcg of Fentanyl was administered three minutes after the initial dose due to high opioid tolerance and continued discomfort during the block injection. The interscalene nerve block was achieved with 20mL of Bupivacaine 0.5% and 1:200,000 Epinephrine deposited perineurally at C5 and C6 nerve roots. Appropriate nerve block was confirmed by clinical assessment of decreased tactile sensation to dermatomes C5 and C6 and patient's inability to lift his right arm. Patient remained in the preoperative holding area for at least 1.5 hours before he was transported to the operating room.

Upon entering the operating room, the patient received another 2mg of Midazolam due to history of severe PTSD and his expression of immense anxiety when introduced to the operating room staff members. Monitoring consisted of pulse oximetry, electrocardiogram, noninvasive blood pressure, temperature, bispectral index of the electroencephalogram (BIS monitoring; Vista, Aspect Medical Systems Inc., Newton, Massachusetts, USA) and end-tidal CO2 monitoring. After 8-10 minutes of preoxygenation, the patient was induced with Fentanyl 100mcg, Lidocaine 2% 60mg, Propofol 200mg and Rocuronium 60mg. Mask ventilation was maintained for approximately 1 minute and intubation was achieved with Glidescope and 8.0 mm endotracheal tube. The patient was placed in beach chair position with back of the bed raised to 60 degrees, nonoperative arm secured to arm board, head immobilized with head strap and airway accessibility, neck aligned in neutral position, patient's body secured with safety-belt and tape, and bed rotated 60 degrees away from the anesthesia machine.

Anesthesia was maintained with 2% sevoflurane. Right diagnostic shoulder arthroscopy with arthroscopic posterior labral capsulorrhaphy and labral debridement were conducted within 45 minutes without any complications. Mean arterial blood pressure was maintained above 65mmHg with small boluses of Phenylephrine and Vasopressin throughout.

Within 20 minutes of suspending sevoflurane, the patient was administered the standard 2mg/kg dose of Sugammedex for reversal. Although there were no contraindications to using neostigmine, Sugammadex was chosen as the reversal agent for neuromuscular blockade because it has been demonstrated in the STRONGER trial to be associated with clinical and statistically significant lower incidence of major postoperative pulmonary complications[6]. After about one minute of administering Sugammadex, the patient experienced hypotension with inadequate ventilatory drive resulting in hypoxia with pulse oximetry reading as low as 60%, high peak airway pressure of 38, and sudden loss of tidal volume. Bronchospasm secondary to Sugammadex hypersensitivity was suspected due to temporal presentation of symptoms after Sugammadex administration. Reduced respiratory function secondary to preoperative administration of fentanyl was of low suspicion since one elimination half-life has already elapsed and patient was able to spontaneously ventilate with adequate tidal volume prior to receiving Sugammadex. Oxygen saturation improved with manual and positive pressure ventilation. Patient's blood pressure was also supported with 20mg of Ephedrine. Upon the patient resuming spontaneous ventilation with tidal volumes of 400-500mL, sustained head lift, and following simple commands of mouth opening, the patient was extubated. Patient was moved to the recovery room on simple mask and was hemodynamically stable.

In the PACU, portable chest radiograph was obtained for dyspnea and hypoxia observed during emergence from anesthesia. Chest x-ray was reviewed at bedside showing clear lungs with elevated right hemidiaphragm consistent with interscalene nerve block. After approximately 20 minutes in the PACU, the patient endorsed severe neck muscle spasm and pain with limited cervical mobility. This pain was exacerbated upon right cervical lateral flexion and rotation. Numeric pain score remained 8-10 despite the administration of Fentanyl 100mcg and acetaminophen 1000mg. He denied right shoulder pain and admitted to mild pain relief with warm compress applied to his neck.

Given a history of PTSD and anxiety, postoperative agitation may be of concern. Conversely, given the patient was able to verbally localize his pain to the lateral neck, place his hand over his neck as the primary pain generator, and exhibit limited cervical range of motion on clinical exam, postoperative agitation was less likely. Additional narcotic was not administered because of concerns for respiratory complications. According to Poppen et al., even though large doses of narcotics may relieve nonradicular pain, it

has little or no relaxation of palpable paravertebral muscle spasm. This was shown in patients with moderate to severe paravertebral and trapezius muscle spasm after cervical or lumbar laminectomies[10]. The patient's neck muscle spasm could be a result of cervical sprain secondary to beach-chair malpositioning, acute exacerbation of underlying chronic pain, pathology in the facet joints, cervical vertebrae, or intervertebral disks[14]. Other variables associated with the development of persistent neck pain and muscle spasm include: trauma, psychopathology (e.g., depression, anxiety, poor coping skills), sleep disorders, sedentary lifestyles, and obesity[2].

Oral and IV skeletal muscle relaxants were not given at this time due to the delayed onset of action as well as the lack of their immediate availability in our PACU setting, respectively. Moreover, a Ramsay score of -1 put him at risk for aspiration for anti-spasmodic oral formulations. Although agents such as diazepam and methocarbamol are available in our central pharmacy, they were not chosen for the following reasons: diazepam would worsen respiratory depression and methocarbamol's effectiveness is questionable in patients with musculoskeletal conditions[1]. Chou and colleagues performed a systematic review of existing literature and concluded that there is fair evidence in favor of cyclobenzaprine, carisoprodol, orphenadrine, and tizanidine compared with placebo for patients with musculoskeletal neck pain. Chou et al. noted limited or inconsistent data for metaxalone, methocarbamol, chlorzoxazone, baclofen, or dantrolene[1].

Given dexmedetomidine's availability in the anesthesia setting, its IV formulation, and its similar actions to tizanidine, dexmedetomidine was administered with the expectant goal of decreasing the muscle spasm. Due to limited studies on dexmedetomidine's role in management of muscle spasm, proper drug dosage remains unclear. For that reason, dexmedetomidine was titrated to therapeutic effect. In doing so, drug response variability and adverse effects were avoided. Dexmedetomidine was given in incremental boluses of 8mcg IV every 1 to 2 minutes for a total of 100mcg. At that time, a reduction in muscle spasm was observed without compromising hemodynamic stability and over sedation. Unlike benzodiazepines and other anti-spasmodic agents, dexmedetomidine is a great agent in this setting. Dexmedetomidine exerts superior properties in that respiration is minimally affected and the patient remains rousable in a dose dependent manner[17]. While the patient was receiving dexmedetomidine, he demonstrated marked improvement with cervical range of motion and his pain score decreased to zero. The patient was hemodynamically stable and rousable throughout the time of drug administration. He remained in Phase I PACU for an additional 30 minutes until he met the requirements of the Aldrete's scoring system. He was then transported to Phase II PACU where he remained for approximately 40 minutes. Subsequently, he was discharged home without any additional narcotic requirements and denied residual cervical muscle spasm.

The patient was followed up three months later via telephone and E-mail to obtain authorization for publication of this case report. During these interactions, the patient expressed, "Although I don't remember being administered the medication [dexmedetomidine], I do remember how I felt afterwards. I have been on opioids and benzodiazepines to manage this [neck pain] for 10 years…however, nothing compared to this drug you gave me. It lasted into the night, and never had so much relief."

Discussion

Painful muscle spasm is a common complaint after surgery. Selection of an anti-spasmodic agent in the postoperative period remains difficult as this drug class is limited by adverse effects and route of administration. In this case report, we were able to elicit dexmedetomidine's unique role in managing postoperative muscle spasm. Its quick onset, convenient intravenous route of administration, familiarity with anesthesiologists, ability to be titrated, and minimal effect on respiratory physiology makes dexmedetomidine an ideal choice in managing muscle spasm in patients who are recovering from anesthesia. Although dexmedetomidine is a selective alpha-2 adrenergic receptor agonist like tizanidine, its exact mechanism and dosage for treatment of muscle spasm remain unclear. Through its interaction with alpha-2 receptors, tizanidine can inhibit the release of excitatory amino acids from spinal interneurons and consequently enhance the presynaptic inhibition of motor neurons[4]. As for dexmedetomidine, its synaptic interference and polysynaptic interaction are still not fully understood.

Girgin et al. demonstrated the use of dexmedetomidine infusion in six patients with tetanus in the ICU. Here they showed a decrease in frequency and severity in muscle spasm. Moreover, they showed dexmedetomidine reduced the need for additional sedatives, analgesics, and skeletal muscle relaxants to control muscle spasms[5]. Miya et al. demonstrated the concomitant use of dexmedetomidine and propofol as a promising choice of pharmacological management for severe tetanus[11]. Recently, dexmedetomidine has been shown to alleviate paroxysmal rigidity and myoclonus seen in progressive encephalomyelitis. It does this by decreasing noradrenergic neuronal activity. This results in attenuation of antibody-mediated disinhibited increased motor and sympathetic activity[3].

This case report demonstrates a positive response to dexmedetomidine in a patient with postoperative neck muscle spasm. As a case report, further comprehensive

studies would be needed to evaluate its usefulness in clinical management of muscle spasm. We hope to encourage ongoing research to investigate and study the efficacy and exact mechanism of dexmedetomidine's role in managing skeletal muscle spasm as well as its utility in the PACU setting.

Conflict of Interest

The authors declare no affiliation with any organization or is involved with any financial or non-financial interest in the subject matter discussed in this case report.

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