

Introduction

- Brachial Plexus Avulsion (BPA) is a catastrophic nerve injury in which the brachial plexus, responsible for motor and sensory innervation of the upper limb, is torn from the spinal cord.
- The complexity of BPA-related pain triggers maladaptive responses such as cortical reorganization further contributing to persistent refractory pain and dysfunction.

Methodology

- As the cases reported devoid of patient identifiable information, it is exempt from IRB review requirements as per University Hospital policy.

Case 1

- A 34-year-old male. Pmhx: depression, substance use disorder (alcohol and heroin) and left brachial plexus avulsion after MVA.
- Patient reported severe, sharp, intermittent pain radiating down the left arm without a specific peripheral nerve distribution, accompanied by numbness and tingling.
- Acute pain service management:
 - Multimodal analgesic: methadone (10 mg every 8 hours), pregabalin (titrated to 150 mg every 8 hours), lidocaine patches, celecoxib (200 mg twice daily), and acetaminophen (975 mg every 8 hours).
 - Despite these interventions, the patient experienced inadequate pain relief, requiring multiple doses of hydromorphone and oxycodone for breakthrough pain.
 - Ketamine infusion was initiated at 20 mg/hour based on the patient's weight (78 kg). Over five days, the patient demonstrated significant improvement in pain control, as evidenced by restful sleep, increased comfort at the bedside, and improved participation in physical therapy.

Conclusion

- Ketamine mechanism of action includes the inhibition of central sensitization and maladaptive pain processing in the nervous system.
- Ketamine has potential to enhance neuroplasticity and reverse maladaptive pain changes.

References

- 1.- Teixeira, M. J. (2015). Neuropathic pain after brachial plexus avulsion--central and peripheral mechanisms. BMC neurology
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- 3.- Ivan Ezquerra-Romano (2018). Ketamine for the treatment of addiction: Evidence and potential mechanisms. Neuropharmacology