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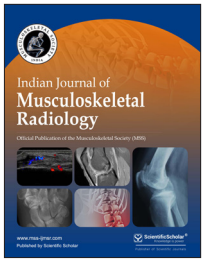

Editorial

Local anesthetics in musculoskeletal practice: Their use and safety

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A quest to control the pain has been a matter of interest for the mankind since ages, way back to Homer's Iliad, that relate the use of herbal remedies for pain control to the discovery of first local anesthetic (LA), cocaine, in South America. The clinical usefulness of cocaine was not appreciated until 1884, when Koller reported upon topical anesthesia of the eye. A major breakthrough in the chemistry of LA agents occurred during World War II, in 1943, when Loefgren synthesized Lidocaine. Synthesized in 1957, Bupivacaine was of special interest because of its long duration of action and history of clinical application, but because of its central nervous system (CNS) and cardiovascular (CV) toxicity, its use became restricted. Numerous experimental studies were conducted to identify the cellular mechanism of this toxicity, which refines our understanding of the action of LAs. The identification of Ropivacaine, a levo-enantiomer of Bupivacaine, whose usefulness and toxicology were selectively and extensively studied before its introduction in the market in 1996, was another landmark in this direction.

LA is extensively used for the diagnosis and treatment of pain associated with a variety of musculoskeletal conditions. LA works by binding to and inhibiting voltage-gated sodium channels on nerve cell membranes, thereby preventing development of an action potential and blocking nerve transmission. They are divided into two main categories: esters and amides. Lidocaine, Bupivacaine, and Ropivacaine which are examples of amides, are the most commonly used LA in musculoskeletal interventions.

The clinical usefulness of LA in musculoskeletal intervention is determined by its potency, toxicity, onset of action, and duration of action. The onset of action of LA depends on its pKa value. Lidocaine has the shortest pKa therefore the fastest onset of action (<2 min) and shortest duration of action (30–120 min). In contrast, Bupivacaine has the highest pKa value, therefore, slowest onset of action (2–10 min), and longest duration of action (180–360 min). Ropivacaine has similar onset of action to Bupivacaine, given their similar pKa values, although its duration of action generally falls between that of ropivacaine and Bupivacaine (140–200 min).

The potency of the LA is directly related to lipid solubility, which is lowest in Lidocaine and highest in Bupivacaine. However, lipophilicity and potency of Bupivacaine is responsible for the propensity for CNS toxicity as compared to Lidocaine, which has the lowest. As the pathomechanism of LA is to block sodium channels of nerve membrane to prevent nerve transmission, Bupivacaine also blocks the cardiac sodium channels with greater affinity thus responsible for cardiac toxicity as compared to Lidocaine. Ropivacaine was created because of Bupivacaine cardiac toxicity and has lowest potency at myocardial sodium channels, thus least

cardiotoxic. Compared to Lidocaine and Bupivacaine, Ropivacaine has a higher CV collapse to CNS ratio, which is "the ratio of drug dose required to cause catastrophic CV collapse to the drug dose required to produce seizures," meaning that CNS features will be detected earlier and allow for treatment of systemic toxicity prior to CV compromise.

The chondrotoxicity and tenocyte toxicity of LA are one of the primary concerns in musculoskeletal interventions. Ropivacaine has the least chondrotoxicity at doses ≤0.5%, while Bupivacaine has the most chondrotoxicity at doses ≥0.5%. Lidocaine chondrotoxicity at doses >1% is comparable to Bupivacaine. These effects are dose-and time-dependent and worsened by the addition of corticosteroids.

The potential for tenocyte toxicity appears to be greater in Lidocaine than other LA. Ropivacaine alone is not significantly tenocyte toxic; however, addition of Dexamethasone potentiates Ropivacaine tenocyte toxicity at higher doses of Ropivacaine. This effect is, however, not seen with Lidocaine when used in combination with Dexamethasone. The overall toxicity is dependent on exposure time and concentration, with 0.2% Ropivacaine being least toxic and Lidocaine being very toxic even at lower concentrations.

The maximum permissible dose of LA without epinephrine in musculoskeletal interventions is 3–5 mg/kg of 1% Lidocaine (20 mg/mL), 2 mg/kg of 0.25% Bupivacaine (2.5 mg/mL), and 3 mg/kg of 0.5% Ropivacaine (5 mg/mL). The typical doses that are commonly used in musculoskeletal intervention are 3–5 mL of 1% Lidocaine, 0.5–2 mL of 0.25% Bupivacaine, and 2–4 mL of 0.5% Ropivacaine.

LA are important diagnostic and treatment tools for multiple musculoskeletal pathologies. While Lidocaine, Bupivacaine, and Ropivacaine are the most commonly selected LA for musculoskeletal interventions, the choice of specific LA is largely dependent on the procedure type, dose and concentration, and cost. Bupivacaine has the highest potency and provides the longest duration of action, although it has been shown to have an increased risk of adverse effects, including cardiac, CNS, and chondrocyte toxicity. Lidocaine has been shown to have a high potential for tenocyte toxicity even at low concentrations. Ropivacaine has generally been found to be least toxic to chondrocytes and tenocytes among the three most widely used LA and, therefore, may be a more desirable choice. However, addition of Dexamethasone to Ropivacaine has shown tenocyte toxic. In addition, availability and cost factors may also limit its widespread use.

In view of these potential side effects and ever-increasing use of LA in guided musculoskeletal interventions, proper documentation of the type, volume, and concentration of the LA along with the type and dose of steroid/other medication used should be encouraged. This could be improved by adding World Health Organization patient safety checklist, preform as for musculoskeletal injections and departmental guidelines regarding use of type and dose of LA being administered. Timely departmental audits would further strengthen a safety check on appropriate use of these drugs in musculoskeletal radiology practice.

RESOURCES

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