To determine the effect of tramadol and dexamethasone added as an adjuvants to local anesthetic agents in Supraclavicular Brachial plexus block

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Abstract: This is a prospective randomized controlled study to determine the post operative analgesia following supraclavicular brachial plexus block with tramadol or dexamethasone as an adjuvant to bupivacaine and lignocaine in upper extremity elective orthopedic surgery. 60 patients of ASA I and II undergoing upper extremity surgery under brachial plexus block with local anesthetic agents were randomly divided into three groups with 20 patients in each groups. One group received Tramadol (2mg/kg) maximum 100 mg, second group received dexamethasone (8mg) and controlled group received 2 ml normal saline as an adjuvants to lignocaine and bupivacaine. The duration of postoperative analgesia was recorded in three groups with pain Visual Analog score. Which was determined by maximum VAS score of 8-10. The mean duration of postoperative analgesia in the control group 396.12 minutes, Tramadol group 453.21 minutes and in the dexamethasone group it was 1028.34 minutes. Conclude

Dexamethasone with local anesthetics prolongs postoperative analgesia significantly than tramadol (P0.05) when used as an adjuvants to local anesthetic agent in supraclavicular Brachial plexus block for upper extremity surgery.

Keyword: supraclavicular block, Brachial plexus, Dexamethasone, Tramadol

Introduction: William Steward Halsted first performed brachial plexus block is 1885. In 1911 Kulenkampff and Hirschel described the first percutaneous brachial plexus block by supraclavicular and axillary routes respectively. Regional anesthesia offers as the name implies is the blocking of peripheral nerve conduction in a reversible manner by using local anesthetic agents thereby one region of the body is made insensitive to pain and is devoid of reflex response to surgical stimuli. The central nervous system is spared, so the patient is conscious during the surgical procedure. Many advantages over general anesthesia for surgery on upper extremities.

ANATOMY:
The brachial plexus is formed by the union of anterior primary rami of C5 to C8 and T1 with occasional contributions from the C4 (prefixed) above and T2 (post fixed) below. These nerves unite to form trunks, which lie in the neck above the clavicle. Its roots pass through the fascia enclosed space between the scalenus anterior and the scalenus medius accompanied by the subclavian artery and invaginate the scalene fascia to form a neurovascular bundle.

### Roots
- Anterior primary rami of C5-C8 and T1 (Occasionally C4 & T2)

### Trunks
- Ø Upper trunk – anterior rami of C5 and C6
- Ø Middle trunk – anterior rami of C7
- Ø Lower trunk – anterior ramus of C8 and T1

### Divisions
- Behind the clavicle, each trunk divides into anterior and posterior divisions.

### Cords
- Ø Lateral cord - Anterior divisions of upper and middle trunks (C5-C7)
- Ø Medial cord - Anterior division of lower trunk (C8-T1)
- Ø Posterior cord - Posterior divisions of all the three trunks (C5-T1)

### Supraclavicular block anatomy:

### Supraclavicular functional anatomy:

### Landmarks: Needle Puncture:

In classic approach, the needle insertion site is approximately 1 cm superior to the clavicle at the clavicular midpoint. If the artery is palpable in the supraclavicular fossa, it can be used as a landmark. From this point, the needle and syringe are inserted in a plane approximately parallel to the patient’s neck and head, taking care that the axis of the syringe and needle does not aim medially toward the cupola of the lung. The needle should be a 22-gauge, 5-cm needle that typically contacts the rib at a depth of 3 to 4 cm. The initial needle insertion should not proceed past 3 to 4 cm until a careful search in an anteroposterior plane does not identify the first rib. During insertion of the needle and syringe, the assembly should be controlled with the hand. The operator’s hand can rest lightly against the patient’s supraclavicular fossa because with elicitation of a paresthesia patients often move their shoulder.
After injecting the local anesthetic the block is tested for both sensory (using Pin prick) and motor (using muscle power) and is compared with same stimulation or power in the contra-lateral arm. Motor block was evaluated by thumb abduction (Radial Nerve), thumb adduction (ulnar nerve), thumb opposition (Median Nerve) and flexion of the elbow in supination and pronation of the forearm (musculocutaneous). The Hollmens scale is used in the study for assessing both sensory and motor blockade.

**Hollmen's Scale**

**Sensory blockade (Grade)**
- 0 - Normal sensation of pin prick
- + - Pin prick felt as sharp pointed but weaker compared with the same area in other extremity
- ++ - Pin prick felt as touch with blunt object
- +++ - No perception of pin prick. Onset of blockade means minimum grade 2 and complete blockade means minimum grade

**Motor Blockade (Grade)**
- 0 - Normal muscle function
- + - Slight depression in muscle function as compared with pre-anesthetic power
- ++ - Very weak muscle action persisting in muscle
- +++ - Complete block with absent muscular function.

Onset of blockade means minimum grade 2 and complete blockade means minimum grade 3.

**Nerves studied in the block**

- Sensory: Median Nerve, Ulnar Nerve, Radial Nerve
- Motor: Median Nerve, Radial Nerve

**Ulnar Nerve**

**Musculocutaneous Nerve**

Evaluation was carried out for every minute after completion of injection and the time of onset was noted both for sensory and motor blockade. Sensory and motor scores were at least grade 3 in Hollmens scale. Only patients with complete block are included in the study.

Duration of sensory blockade was considered as the time interval between local anesthetic administration and the onset of paresthesia, while the duration of motor block was defined as the time interval between local anesthetic administration and recovery of the block.

Sedation was assessed using Sedation scores by Culebras et al where sedation was graded on a scale of 1-5 as follows.

- Awake & alert
- Sedated, responding to verbal stimulus
- Sedated, responding to mild physical stimulus
- Sedated, responding to moderate or severe stimulus

. Not arousable Pharmacology:

Dexamethasone: It is a potent synthetic glucocorticoid which has anti-inflammatory. The use of corticosteroids as an adjuvant to local anesthetic for peripheral nerve blocks rarely has been described. Corticosteroids cause skin vasoconstriction on topical application. The vasoconstriction effects of topical steroids are mediated by occupancy of classical glucocorticoid receptors rather than by nonspecific pharmacological mechanisms.
Steroids bind to intracellular receptors and modulate nuclear transcription. In our study, dexamethasone produced a relatively rapid effect which cannot be explained by the above mechanism. Therefore, vasoconstriction, the presumed mechanism of action for epinephrine's adjunctive effect on local anesthetics, is probably not responsible for block prolongation by dexamethasone. Corticosteroids may have a local effect on the nerve; the dexamethasone effect may be related to this action. It block the nociceptive impulse transmission along myelinated C fibers, immunomodulatory and analgesic effect. Tramadol: Tramadol is an analogue of codeine and has central nervous system (CNS) mediated properties with a low affinity for opioid receptors. It is antagonized by a2-adrenocceptor antagonists, as well as opioid antagonists. The central analgesic, tramadol, is successful in the management of pain. It is formulated as a racemic mixture with each enantiomer displaying different opioid-receptor binding properties, monoaminergic reuptake inhibition, and metabolic pathway. Tramadol has a methyl group substitution on the phenolic moiety, which explains its weak affinity for opioid receptors. It was initially reported to lack selectivity for mu, kappa, or delta receptors but in a more recent study, it has been demonstrated to be selective for the mu receptor. Furthermore, the O-desmethyl tramadol metabolite of tramadol, which is produced by O-demethylation, shows higher affinity for opioid receptors than the parent drug. The O-desmethyl-tramadol is catalysed by cytochrome P450(CYP)2D6. Tramadol and its metabolite excreted by the kidney. Elimination t ½ is about 6 hours. Tramadol inhibits norepinephrine and serotonin reuptake in rat brain synaptosomes. Moreover, pretreatment with yohimbine and idazoxan (both a2-adrenocceptor antagonists) can significantly reduce the antinociceptive effect of IV tramadol. Based on this profile of action, we hypothesized that the effect of adding tramadol to local anesthetics for brachial plexus blockade is mentioned previously, tramadol influences the monoaminergic system, with pain-modulating effects. It inhibits norepinephrine and 5-hydroxy-tryptamine reuptake. The mechanism of action of tramadol as adjuncts to local anesthetics for peripheral nerve block is a local effect on the nerve that is not mediated via receptors. This suggests that tramadol might modify the action of local anesthetic at the sodium channel either directly or indirectly. Aim of the study: This is a prospective randomized double blind controlled study to determine the effect of adjuvants. Tramadol(2mg/kg) or dexamethasone (8mg) in combination with local anesthetic agents lignocaine and bupivacaine into brachial plexus sheath via supraclavicular approach. Background: Adding adjuvants to local anesthetics for peripheral plexus blockade may enhance the quality and duration of anesthesia and post operative analgesia. Study design: Prospective randomized double blind controlled study and after obtaining ethical committee approval from kilpauk medical college and informed consent obtained from all the patients. Inclusion Criteria: ASA I and II Age 18-65 years Both sex Elective upper limb orthopedic surgery Without any co-morbid illness
Exclusion Criteria:
ASA III-IV
Allergy to local anesthetics
Morbid obese
Known COPD
NM diseases and coagulopathies Patchy or inadequate anesthesia Diabetes Patient refusal

Groups:
Group C (control group): 20 patients receiving 10ml 2% Lignocaine+20ml 0.5% Bupivacaine + 2ml of NS
Group T (Tramadol group): 20 patients receiving 10ml 2% Lignocaine+20ml 0.5% Bupivacaine + 2ml Tramadol (100mg)
Group D (dexamethasone group): 20 patients receiving 10ml 2% Lignocaine+20ml 0.5% Bupivacaine + 2ml Dexamethasone (8mg)

Monitoring:
All the cases under the study belonged to ASA I and ASA II so only non invasive monitors were used and the invasive cardiac output monitors were avoided since the one of the upper extremity was being operated and usage of invasive line on the other limb would impair the functionality of the patient.

- Pulse oximetry
- NIBP
- ECG
- Respiratory rate

Methodology:
For all patients, IV line secured, monitors attached. Landmarks identified and marked. All patients under aseptic precaution received brachial plexus block via classical supraclavicular approach. Paresthesia as an end point. Onset of sensory block assessed by Hollmen’s sensory scale. Onset of motor block by motor scale saturation <94%. Hemodynamics, Sedation score

Post operative analgesia and adverse effects are evaluated.
Patients will receive sedatives on demand basis. Patients will receive O2 through mask if Supraclavicular block provide anesthesia of the entire upper extremity. It is the most effective block for all portion of the upper extremity and it is carried at the Division level of the brachial plexus. currently available local anesthetic can provide analgesia for limited period of time when used as a single injection. In this study Tramadol and dexamethasone was used as adjuvant to local anesthetics. Steroids blocks the transmission of impulse in nociceptive C fibers, thus when used with local anesthetics steroids prolongs the duration of local anesthetic block. Tramadol used with local anesthetic inhibit the reuptake of serotonin from nerve ending and potentiate the block effect. These two drugs lack respiratory depressant effect. The onset of motor block was faster than the sensory block in the dexamethasone group compared with control groups but it is statistically insignificant. The onset of sensory block was faster in the dexamethasone group compared with control groups which was statistically significant. The mean duration of return of motor block in control group was 178.93 minutes, In tramadol group 202.93 minutes and in dexamethasone group it was 393.03 minutes which was statistically significant (P<0.05). Duration of analgesia was determined by(Visual Analog Score 8-10). Score 8-10 was regarded as the end point for total analgesia assessed by the observer blinded to the study. In dexamethasone group total analgesia duration was 1028.34 minutes which was statistically significant.
significant compared to control groups. During the study 7 patients were excluded from the study due to inadequate blockade and converted to General Anesthesia. 5 cases in the Tramadol group had nausea in the early postoperative period. 2 cases had nausea twice. The nausea was corrected on its own. None of the patients had respiratory complication in the postoperative period. The block prolonging effect of dexamethasone is due to its local action and not a systemic action. It has been found this effect of steroid is mediated through steroid receptors. when steroid alone was used to block the nerves the effect was not prolonged. The action of steroid has been related with the alteration of function of K+ channels.

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Conclusion:
Addition of Tramadol or Dexamethasone to local anesthetics in brachial plexus block prolong postoperative analgesia duration (mean 1028.34 min) which was significantly higher in comparison with tramadol (mean 453.21 min) and control group (mean 396.12 min) as adjuvant to local anesthetic. This help to minimize the cost and provide patient comfort.

References:


11 Droger C, Benziger D, Gao F, Berde CB. Prolonged intercostals nerve blockade in sheep using controlled-release of bupivacaine and


