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COMPARATIVE EVALUATION OF ADDING CLONIDINE V/S DEXMEDETOMIDINE DURING BIER'S BLOCK

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KEYWORDS :

Bier's block is an ideal technique for short operative procedures of anticipated duration of 60-90 minutes on extremities, performed on day care basis. Bier's block or intravenous regional anaesthesia (IVRA) is technically simple and reliable, with success rates between 97-98%.

Moreover the cost of anaesthesia and recovery using IVRA for outpatient hand surgery is less than general anaesthesia and also it is more effective in speeding recovery and minimising postoperative complications. Biers block was first described by August K. G. Bierin 1908 by injecting a solution of Prilocaine into one of the subcutaneous veins that were exposed between two constricting bands. This technique was not widely used until Holmes reintroduced the technique with lignocaine in 1963. Lignocaine remains the standard local anaesthetic agent in many countries. Advancements in the field of IVRA have been primarily aimed at reducing the tourniquet pain, increasing tourniquet tolerance, improving the overall quality of intra-operative and post-operative analgesia and reducing the drug related adverse effects. Local anaesthetics alone are not able to bestow all such attributes to the bier's block solution, hence a multitude of adjuncts have been used. Several local anaesthetics adjuncts that have been used are, opioids like fentanyl , pethidine , tramadol , NSAIDs like ketorolac , acetylsalicylate , lornoxicaine; and muscle relaxant e.g. atracurium , neostigmine , and ketamine. Lately magnesium have also been tried. However none of them have proved to be ideal.

Recently, α-2 adrenergic receptor agonists have been the focus of interest for their sedative, analgesic and peri-operative sympatholytic and cardiovascular stabilising effects in addition to their general anaesthetic sparing effects and ability to prolong local anaesthetic induced analgesia when used in regional blocks. Clonidine is a selective partial α-2 agonist. Clonidine has been added to local anaesthetics for various nerve blocks, resulting in improved anaesthesia and analgesia. Studies investigating the addition of clonidine to local anaesthetic in IVRA have demonstrated reduced tourniquet pain and improved post-operative pain relief. Dexmedetomidine, a potent α-2 adrenoreceptor agonist, is approximately 8 times more selective towards the α-2 adrenoreceptors than clonidine. Dexmedetomidine- lignocaine mixture has been used recently to provide Bier's block and has been shown to improve the quality of anaesthesia ,reduce tourniquet pain and post-operative analgesic requirement.

Esmaoglu observed that addition of dexmedetomidine to local anaesthetic solution in IVRA improved the quality of anaesthesia and decreased analgesic requirements, but had no effect on the sensory and motor blocks onset and regression times. Reuben SS observed that the addition of 1μg/kg clonidine to lignocaine 0.5% for IVRA in patients undergoing ambulatory hand surgery improves post-operative analgesia without causing significant side effects on the first postoperative day.

These reports suggest that dexmedetomidine would be better adjuvant to lignocaine in providing biers block than clonidine. However there is no direct comparison between these two drugs to favour one α2 blocker over the other in patients of ASA grade I, II and III. Therefore, present study was carried out to evaluate the effects of adding either clonidine or dexmedetomidine to lignocaine for bier's block or intravenous regional anaesthesia.

MATERIAL AND METHODS

Present study was a prospective, randomized double blind study in which 60 patients in the age group of 18-65 years of either sex, with American Society of Anesthesiologists (ASA) class I, II admitted to hospital, attached to institute and scheduled for elective upper limb orthopaedic surgeries were included after the approval of institution's ethical and scientific committee and taking the informed consent.

Appropriate dose of lignocaine in mg/kg was calculated according to the weight of the patients. Adequate volume of 2% lignocaine, according to dose in mg/kg was diluted with distilled water to four times the original volume to make the solution, 0.5% lignocaine. Identical syringes containing each drug were prepared by personnel blinded to the study.

ALLOCATION OF GROUPS:

Patients were randomly divided into two groups of 30 each.
Group 1 (LC): Clonidine 1μg / kg added to 3mg / kg lignocaine 0.5%.
Group 2 (LD): Dexmedetomidine 1μg / kg added to 3mg / kg lignocaine 0.5%

TECHNIQUE

Baseline ECG, pulse rate (PR), non-invasive systolic and diastolic blood pressure and peripheral arterial saturation (SPO2) were monitored. Before establishing the anaesthetic block, two intra-venous cannula were placed; one, in a vein on the dorsum of the operative hand and the other in the opposite hand for crystalloid infusion. The operative arm was elevated for 3 minutes then exsanguinated with an Esmarch bandage. A pneumatic tourniquet was placed around the upper arm and the proximal cuff was inflated to 100 mmHg more than systolic BP to a minimum of 250 mmHg and the Esmarch bandage was removed. Circulatory isolation of the arm was verified by inspection, absence of radial pulse, and loss of pulse-oximetry tracing of the ipsilateral index finger. IVRA was established using 3 mg/kg of 0.5% lignocaine, diluted with saline to a total volume of 40 ml to which 1 μg/kg of clonidine or 1 μg/kg dexmedetomidine was added. Drug was then slowly injected and patient warned that the limb may start to feel hot and the skin would take on mottled appearance. The sensory block was assessed by a pinprick performed by a 25 G short bevelled needle at every 30 seconds interval. Sites used for sensory testing included the thenar eminence (median nerve), hypothenar eminence (ulnar nerve) and first web space (radial nerve).Sensory regression was assessed at these nerve sites at 1 minute interval after tourniquet deflation. Motor function was assessed by asking the patient to flex and extend his/her wrist and complete motor block was noted when no voluntary movement was possible. The distal tourniquet was inflated to 250 mmHg, after the achievement of sensory and motor block. The proximal tourniquet was released and surgery was allowed to commence. Tourniquet was not deflated before 30 minutes even if surgery finished and was not kept inflated for >1.5 hour. After the completion of surgery, tourniquet deflation was performed by the cyclic deflation-inflation technique. Sensory and motor blocks were then tested and the regression times were noted.

MONITORING:

Continuous multipara monitoring was done for haemodynamic response. Readings were recorded every 5 minutes till the end of surgery and then 1 hourly till 6 hours. Bradycardia (defined as heart rate <60 beats/min) was treated with intravenous atropine, 0.5mg.

Hypotension (defined as systolic blood pressure <20% less than base value) was treated with intravenous ephedrine as and when required. Pain (tourniquet or post-operative) was assessed by using a 10 cm visual analogue scale (VAS). Inj. butorphanol 0.5mg intravenous was given intra-operatively if patient complained of pain (VAS >3). An intramuscular dose of diclofenac sodium 75 mg was given post-operatively as and when required (VAS >3). Intra-operative butorphanol and postoperative diclofenac sodium consumption was recorded and the number of doses of diclofenac, were calculated.

Sedation was assessed on a 1-5 numeric scale. Score were assessed as:
1 - Completely awake.
2 - Awake but drowsy.
3- Asleep but responsive to verbal commands.
4 - Asleep but responsive to tactile stimuli.
5 - Asleep and not responsive to any stimulus.

The VAS for pain and sedation score were measured every 10 minutes during surgery, at 15 minutes, 30 minutes and at hourly intervals until 4 hours then every 4 hourly till 24 hours. Onset and regression time for sensory and motor blocks were noted. Any side effects or complication (systemic or local) were noted. Thenon-parametric data were analyzed using the 'Chi– Square tests' and the parametric data were analyzed using the 'Unpaired "t" test '.The 'p-value' was determined to finally evaluate the levels of significance. The 'p-value' of < 0.05 was considered significant and the 'p-value' of < 0.001 was considered highly significant. The results were analyzed and compared to previous studies.

RESULTS

Both the groups were comparable with respect to age, sex, weight, ASA grade, baseline haemodynamic vitals, duration of surgery and intra-operative and post-operative haemodymanic variables. (Table 1) Sensory block onset and recovery was 4.85±0.49 minutes and 5.9±0.66 minutes respectively in group 1(LC) and 5.01±0.42 minutes and 6.2±0.56 minutes respectively in group 2(LD). Motor block onset and recovery was 10.91±0.6 minutes and 6.83±0.69 minutes respectively in group 1(LC) and 11.2±0.59 minutes and 7.13±0.57 minutes respectively in group 2(LD). Both the groups were comparable with respect to onset and recovery of both sensory and motor block. (Table 2) The VAS score, in intra-operative period, was significantly higher in group 1(LC) at 20 min, 30 min, 40 min and at 50 min time interval, than in group 2(LD). During post-operative period, VAS score was significantly higher at 15 min, 30 min, 1 hr, 2 hr, 3 hr, 12 hr and at 20 hr time intervals, in group 1(LC) than in group 2(LD). (Table 3, 4) The mean rescue analgesia requirement, in intra-operative period, in group 1(LC) was 0.367±0.49 doses, while none of the patients required any rescue analgesia in group 2(LD). During post-operative period mean rescue analgesia requirement in group 1(LC) was 3±0 doses, while in group 2(LD), it was 2.23±0.43 doses, which was significantly higher in group 1(LC) than in group 2(LD). The mean sedation score during intra-operative period in both the groups was equal which was1 (completely awake).During post-operative period significantly higher sedation was observed at 15 min, 30 min and at 1hr interval in group 2(LD) as compared to group 1(LC). The maximum sedation score achieved was 3 i.e. asleep but responsive to verbal commands. The mean duration of analgesia, based on the time for request of first dose supplement analgesic, in group 1(LC) was 2.8±0.66 hr, while ingroup 2(LD) was 8.67±2.12 hr in group 2(LD), which was significantly longer in group 2(LD), than as compared to group 1(LC).The surgeon satisfaction score was equal in both the groups, which was 3(3-3) i.e. 'perfect'. The patient satisfaction was significantly higher in group 2(LD) than as compared to group 1(LC). Quality of analgesia, as determined by the number of rescue analgesic doses during intra-operative and post-operative period, was better in group 2(LD) than, as compared to group 1(LC).

DISCUSSION

The result of this study reveals that the onset and regression times of both the sensory and motor block during bier's block using either clonidine or dexmedetomidine with lignocaine, at the doses used in this study, are similar. However, the dexmedetomidine-lignocaine mixture provided better quality of analgesia with reduction of intra-operative and post-operative rescue analgesic requirement and longer duration of post-operative analgesia. These improvements were associated with more but short-lived sedation. The analgesic sparing effects of dexmedetomidine were not limited to the intra-operative period but extended to the early post-operative period much more than clonidine as evidenced by the lower VAS scores, lesser number of patients requiring rescue analgesia and lesser analgesic consumption in those who had significant pain scores(VAS>3) in the dexmedetomidine group. The limited analgesic sparing effects of clonidine observed in our study confirm the finding of other investigators (Kleinschmidtet al , Gentili et al). The post-operative analgesic sparing effects of dexmedetomidine in the present study confirm the findings of other investigators(Memis et al. Esmaoglu et al.)The analgesic effects of α2blockers appears to be mediated peripherally and not the result of central redistribution. Patients receiving intravenous clonidine failed to demonstrate any additional analgesia compared to lignocaine alone. The precise mechanism by which α2 blockers exerts its analgesic effects remains unknown. Activation of postsynaptic α2 receptors in substantiagelatinosa of the spinal cord is the presumed mechanism by which α2 blockers produces analgesia. α2-blockers enhances peripheral nerve blocks of local anaesthetics by selectively blocking Aδ and C fibres. These may produce a peripheral analgesic effect by releasing encephalin like substances. Dexmedetomidine, a potent α2 receptor agonist, is approximately 8 times more selective towards the α2 receptors than clonidine. It is therefore hardly surprising that dexmedetomidine, that has 8 times the affinity of clonidine for α2 receptors, caused more postdeflation sedation compared to clonidine in the present study. This confirmed the findings of Esmaogluet al , who reported significant post-deflation sedation using the same dose of dexmedetomidine used in present study.

CONCLUSION:

Both the addition of 1mcg/kg of clonidine and 1mcg/kg of dexmedetomidine to 3mg/kg of 0.5% lignocaine are effective, comparable in terms of onset and recovery of sensory and motor blockade, haemodynamically stable and without any side effect and complications.However, dexmedetomidine- lignocaine mixture provides better quality of analgesia and longer duration of analgesia along with short lived post- deflation sedation.