A Case of Success of Subarachnoid Block Using 0.75% Hyperbaric Ropivacaine in Previous Multiple Failed Attempts at Spinal Anaesthesia

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Abstract: This report explores the impact of scorpion bites on the effectiveness of Sub-Arachnoid blocks (SAB) in surgical patients, primarily in the rural Indian subcontinent. It is observed that the scorpion venom interferes with the action of local anaesthetics by affecting voltage gated sodium channels, resulting in a resistance to these drugs. A case report of a 45-year-old woman who underwent fistulectomy in a Mumbai based hospital is presented. Despite her history of scorpion bites and previously failed subarachnoid blocks, she successfully received SAB with 0.75% hyperbaric Ropivacaine, achieving sensory and motor blockade for up to three hours. This case suggests the potential of newer anaesthetic molecules to overcome resistance caused by scorpion venom, though further research is required for definitive conclusions.

Keywords: Scorpion bite, Sub arachnoid block, local anaesthetic resistance, voltage gated sodium channels, Hyperbaric Ropivacaine

1. Introduction

Scorpion bite is of common incidence majorly in the rural Indian subcontinent, with majority elective surgical patients giving a history of grade 1 or 2 sting. Not only a failure of subarachnoid block/ central nervous blockade, but a hearsay resistance to multiple local anaesthetic has been documented on numerous occasions during peripheral nerve block or local infiltration in these patients.

2. Case Report

A 45-year-old lady presented with complaints of bleeding and pus discharge from a swelling in the perianal area in the last one week. She was posted for Fistulectomy. On preoperative evaluation, patient had nil co-morbidities. Her general examination was normal. Laboratory investigations were within normal limits. Her Mallampati grade was III. She had a positive surgical history of lower segment Csection 20 years ago under General anaesthesia, tubal ligation under general anaesthesia 15 years ago and Fistulotomy 2 months prior to currently posted surgery under Total intra venous anaesthesia (TIVA). Further enquiry revealed that she was administered Sub arachnoid block for all previous surgeries but because of failure of the block, the procedures were converted to general anaesthesia or TIVA. On probing further, patient revealed a positive history of scorpion bite on 5 different occasions in the past, twice on her left foot when she was 15 years of age, once on her right arm around 18 years of age, one over her right side of face at 24 years of age and the latest over her left hand at around 30 years of age. The patient hails from a village in rural Maharashtra in India. Patient gave a verbal history and did not have any previous documentation of a failed subarachnoid block. During the pre-anaesthetic evaluation, after explaining to the patient her special condition, after obtaining consent, we administered a ring block on her right index finger using Inj Lignocaine 2% with a total drug volume of 5cc. Patient was observed for 2 hours post block. There was failure to achieve both sensory and motor blockade after 2 hours.

After taking written, informed consent, patient was taken inside the operating room. Three parametric monitoring including ECG, Pulse oximetry and non- invasive blood pressure (NIBP) were started. A 20-guage IV line was secured and IV fluid started. Under all aseptic precautions, lumbar puncture was performed at the level of L3-4 interspace with a 25-gauge Quincke's spinal needle in sitting position. After confirmation of free flow of CSF on aspiration, a drug volume of 2.8cc of 0.75% hyperbaric ropivacaine was injected into the sub-arachnoid space. The patient was made supine with a 10-degree head down tilt of the table. Sensory, motor, and autonomic blockade was assessed from the time of spinal injection till 20 mins from administration. Hemodynamic monitoring was done every 3 minutes from time of supine positioning till 20 minutes and every 5 minutes thereafter till completion of procedure. Motor blockade as power 0 was achieved at 4 minutes from SAB administration and sensory blockade at 7 minutes at dermatomal level of T10. Autonomic block of hypotension & tachycardia was observed at 3 minutes after SAB administration (Figure 1). The procedure duration was 160 minutes (Figure 2). The sensory and motor block receded completely by 3.5 hours and patient was shifted to the ward after attainment of Bromage (1) grade III. Patient was followed up till the next 24 hours which was uneventful.

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Figure 1: Vitals post induction



Figure 2: Vitals after procedure completion

3. Discussion

As early as 1922, Gaston Labat made a statement: "two conditions are absolutely necessary to produce spinal anaesthesia; puncture of the dura mater and subarachnoid injection of an anaesthetic agent." ⁽²⁾ In 2009, the subject of failed spinal anaesthesia enjoyed its first large review, "Failed spinal anaesthesia: mechanisms, management, and prevention" by Fettes. ⁽³⁾

The causes related to spinal failure can be enumerated as ⁽⁴⁾

A) Operator Related Failure

- Improper drug dose or volume
- Improper assessment of block
- Inappropriate positioning
- Failure to counsel and communicate
- Seniority and personal experience
- **B)** Technique Related Failure
 - Faulty technique
 - Difficult back (anatomical deformities)
 - Misplaced injectate
 - Pseudo puncture
- C) Equipment Or Drug Related Failure.
 - Block needle
 - Use of pencil point needles
 - Drug potency
 - Wrong drug
 - Drug resistance.

The sodium channel has been shown to consist of alpha, beta-1 and beta-2 subunits. The alpha subunit involves four homologous domains (I-IV), and each of these domains is made up of six transmembrane segments (S1-S6). Local anaesthetic action is believed to be due to an interaction with the sixth segment of domain four of the alpha subunit (IV-

S6), involving sites of phenylalanine and tyrosine amino acid residues. $^{(5,6)}$

One of the postulated mechanisms for local anaesthetic resistance is receptor mutation associated with sodium channel abnormalities. An atypical receptor site might result from genetic variation in the amino acid sequence within the sodium channel. Therefore, it is possible that genetic variation that alters the site of action as stated above can be the cause of resistance to local anaesthetics. ⁽⁶⁾ Panditrao et al⁽⁷⁾ had described the resistance to intrathecal bupivacaine in patients with a history of scorpion sting, and postulated that scorpion toxin itself or the antibodies against the toxin are responsible for the development of resistance to intrathecal bupivacaine. The mean time to onset and completion of sensory and motor blockage was more in patients with history of multiple (>2) stings as compared to single sting. This may be due to the antibodies against scorpion venom that had accumulated with multiple stings as postulated. (7,8)

The scorpion venom is a weak acid (pH 6.5) and highly complex mixture of salts, nucleotides, biogenic amines, enzymes, mucoproteins, and neurotoxins, acting on ion channels specifically voltage gated sodium channels (VGSC). Out of various scorpion toxins, alpha and beta toxins are known to bind to mammalian VGSC. The alpha toxin binds extracellularly to S3-S4 loop in domain IV and extracellular part of segment S5-S6 of domain I. The beta toxin binds to extracellular part of segment 4 of domain II.

Bupivacaine is an amino-amide local anaesthetic. Ropivacaine is a piperidine-carboxamide based amide, prepared as pure S-enantiomer. Molecular modelling of local anaesthetic binding with VGSC has demonstrated the differences in the relative alignment of aromatic part of ropivacaine as compared to other local anaesthetics on VGSC. The aromatic part of ropivacaine aligns towards the outer side of VGSC whereas the aromatic part of bupivacaine aligns towards the inner side of the channel. (10,11) This differential alignment of aromatic ring may contribute to the difference in resistance of the two local anaesthetics caused by scorpion sting. Further, action of ropivacaine on gamma aminobutyric acid A (GABA-A) and N-methyl-D-aspartate (NMDA) receptors (12,13) facilitates its local anaesthetic action, thereby decreasing the chances of its resistance in patients with a scorpion sting. Thus, differences in the three-dimensional structures of ropivacaine and bupivacaine may confer differences in the activity of their enantiomers in the complex biological environment of the receptors ⁽¹⁴⁾ and may be responsible for the success of intra-thecal ropivacaine in patients with scorpion stings.

4. Conclusion

While a history of scorpion bite in patients receiving subarachnoid block is significant, we have possible now found newer molecules that can overcome the said resistance to local anaesthetic drugs. However, more research into the drug is required to establish this as an absolute alternative to above.

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