St Segment Elevation During a Non Cardiac Surgery under General Anaesthesia after a Failed Attempt of Spinal Anaesthesia

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Abstract

Introduction: For safe lower abdominal and lower limb surgeries, spinal anesthesia is commonly used. Failure of Spinal Anesthesia can occur even in the expert hands. Causes of failed subarachnoid block includes technical difficulties, poor patient positioning, incorrect insertion of the spinal needle.

Resistance to local anesthetic is difficult to diagnose. One of the etiology for Local Anesthetic resistance is history of scorpion bite in the past. Here we report one such case where spinal anesthesia was administered and later upon noticing a failed block, a history of scorpion sting was elicited in operating room and the procedure was carried out under general anesthesia.

Case Report: A 55 year old male diagnosed with Open type III B both bone fracture of right leg. Patient gave an alleged history of RTA.

With anesthetic plan of Subarachnoid block, 18G IV Cannula was secured. Lumbar puncture was done at L3-L4 Level with 23G Quincke's Needle in sitting position. 3.5ml (15mg) of Bupivacaine and 60mcg of bupreonorphine was injected. Even after waiting for 20 minutes the patient had no sensory or motor block. There was no fall in blood pressure. During the wait, a further detailed history was taken which revealed that patient had a history of scorpion bite 5 years ago and was treated in a local hospital. Hence the surgery was done under general anesthesia.

During the surgery, sudden elevation in blood pressure and ST segment elevation in chest leads was observed. Suspecting intraoperative Myocardial Infarction Inj. Loxicard 20mg and Tab. Ecospirin 150mg was given. ST segment elevations were reverted. ABG which was done to rule out any acid base disorder was within normal limits.

After completion of surgery, Reversal (Inj. Neostigmine 3mg + Inj. Glycopyrolate 0.6mg) was given and patient was extubated.

Once the patient was stabilised, patient was shifted to ICU for observation. Post operative orders for a chest X-ray, SOB profile, 2D ECHO for intraoperative changes were given and were normal.

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Conclusion: During routine preanaesthetic evaluation, we never ask about previous exposure to toxins, as in our patient it was discovered later. Now we have made it part of questionnaire in preanesthetic check.

The patient was monitored carefully, therefore early detection of ST-segment elevation was possible. The presence of an anesthetic team favored the early diagnosis, so the patient was given a quick and safe intervention.

Keywords: Spinal Anesthesia; Scorpion sting; General Anesthesia; ST Segment Elevation; Takatsubo Cardiomyopathy.

This work is licensed under a Creative Commons Attribution-NonCommercial-ShareAlike 4.0. **Key Messages:** Failed Spinal Anesthesia can be due to technical reasons or drug issue. We encountered a rare undiagnosed patients of scorpion strings, who may present resistance to local anesthetist.

Initially during our pre-anesthetic evaluation, the history of scorpion bite was not being elicited. Now, with a history of we made it a part of standard pre-anesthetic evaluation.

We are reporting a case of failed spinal with a history of scorpion bite successfully managed.

INTRODUCTION

For safe lower abdominal and lower limb surgeries, spinal anesthesia is commonly used. Failure of Spinal Anesthesia can occur even in the expert hands. Causes of failed subarachnoid block includes technical difficulties, poor patient positioning, incorrect insertion of the spinal needle, spinal abnormalities, obesity, pseudo successful lumbar puncture, errors in the injection of drug, wrong dose selection, misplaced injection, inadequate spread of the drug intrathecally, ineffective drug action, chemical incompatibility caused by adjuvant and finally the local anesthetic 'resistance'.

This resistance to local anesthetic is difficult to diagnose. One of the etiology for Local Anesthetic resistance is a history of scorpion bites in the past. Pumping mechanism of sodium channels in the nerve fibers, involved in the basic mechanism of action of local anesthetic agents, are affected by the scorpion venom. Here we report one such case where spinal anesthesia was administered and later upon noticing a failed block, a history of scorpion stings was elicited in operating room and the procedure was carried out under general anesthesia.

CASE REPORT

A 55-year-old male diagnosed with Open type III B both bone fracture of right leg, posted for Knee sparing External Fixator Application as an emergency. Preanesthetic evaluation was done to assess patient's ability to respond to physiological challenges encountered intraoperatively. NPO was maintained for both solids and liquids. Patient gave an alleged history of RTA, due to which he sustained an injury to the right leg. Patient was not a known case of and comorbidities. Patient had no history of any previous surgeries or hospital admissions. Patient had a mouth opening of three fingers, one finger temporomandibular joint, three finger thyromental distance. And grade IV Mallampati grading. Blood work up of the patient

were within normal limits except an elevated total leucocyte count.

With anesthetic plan of Subarachnoid block,18G IV Cannula was secured, fluids initiated at the rate of 15ml/kg and patient was shifted to operation theatre. Lumbar puncture was done at L3-L4 Level with 23G Quincke's Needle in sitting position. 3.5ml (15mg) of Bupivacaine and 60mcg of bupreonorphine was injected after confirming free flow of CSF on aspiration. The patient was made to lie down. Even after waiting for 20 minutes the patient had no sensory or motor block. There was no fall in blood pressure. During the wait, a further detailed history was taken which revealed that patient had a history of scorpion bites 5 years ago and was treated in a local hospital. Hence the surgery was done under general anesthesia.

During the surgery, sudden elevation in blood pressure and ST segment elevation in chest leads was observed. Suspecting intraoperative Myocardial Infarction Inj. Loxicard 20mg and Tab. Ecospirin 150mg was given. ST segment elevations were reverted. ABG which was done to rule out any acid base disorder was within normal limits.

After completion of surgery, Reversal (Inj. Neostigmine 3mg + Inj. Glycopyrolate 0.6mg) was given and patient was extubated.

After extubation, patient developed a bradycardia, wheeze and increased work of breathing, Inj. Hydrocort 100mg, Inj. Magnesium Sulphate 1gm in 100ml Normal Saline, Inj. Adrenaline 1:1,00,000 subcutaneously were given in view of bronchospasm.

Once the patient was stabilised, he was shifted to ICU for observation. Post operative orders for a chest X-ray, SOB profile, 2D ECHO for intraoperative changes were given and were normal.

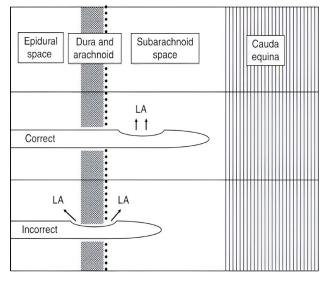
DISCUSSION

Many practitioners have considered that failure rate of spinal anaesthesia is less than 1%. Failure of anaesthesia can be avoided.³

Failure of spinal anesthesia can be due to the following reasons:

Failed lumbar puncture

- Positioning
- Insertion of Needle
- Adjuncts
- Pseudo-successful lumbar puncture
- Solution Injection errors
- Misplaced injection



Even when all the above reasons for failed spinal anaesthesia were conquered, Studies were conducted to show that scorpion sting showed spinal anaesthesia failure and delayed effect of anaesthesia.⁴

Sodium channel consist of alpha, beta-1 and beta-2 subunits. The alpha subunit has four homologous domains (I-IV) and each of domains is madeof six trans-membrane segments (S1-S6). Local anaesthetic action is due to an interaction of the local anaesthetic with the 6th segment of domain four of the alpha subunit (IV-S6).

Local anesthetic resistance can be related to the alpha subunit of the sodium channel and more specifically to the 6th segment of domain four of this subunit.³ Local anesthetics reach Sodium channels by intracellular and extracellular pathways. They act by binding with Na+ channels in the closed, open and inactivated states.

One hypothesis for resistance to local anesthetics is the combined flow of Na+ and local anesthetic in a closed channel. This explains contradicting evidence on how outer pore mutations, tetrodotoxin and μ -conotoxin binding affect the entry and exit of local anesthetics.³ Voltage gated sodium channels help to generate, propagate and transmit action potentials in excitable cells. Neurotoxins target these channels by binding too many receptor sites on the pore-forming α-subunit. Scorpion poisons have a predilection for specific sodium channel subtypes in mammals and invertebrates.⁴ The antigenic nature of scorpion venom could trigger a strong antigen-antibody reaction, particularly if the same person has been attacked several times. Our idea is that these antibodies circulating even during the administration of local anesthetics may have created competitive antagonism with them at the "receptor site" of sodium channels (6th segment of domain four of the alpha subunit (IV-S6), where the Local anesthetics act.³

Scorpion bites are rather prevalent in subtropical areas such as India. Clinicians do not consider the history of the scorpion bite to be a relevant or consequential factor. If the patient has come for an unrelated medical/surgical problem, even a short mention of this fact may be noted and forgotten. A typical scorpion's venom contains various poisons, biogenic amines, enzymes, salts, unidentified compounds and water. Toxins are classified into two classes based on their composition: buthidae and chactoids. Buthidae is more significant to physicians because these toxins have been shown to influence sodium ion channels, potassium ion channels and calcium ion channels in terms of electrolyte balance.

The permeability of sodium and calcium has an effect on the heart. Sodium and potassium influence nerve transmission and cell membrane integrity. Sodium impacts the kidney's equilibrium. Calcium influences the muscles and serves as an important secondary messenger.⁴

Scorpion venom's neuromuscular intoxication could be attributed to its capacity to act on exposed fibers or muscles directly or via motor neurons. Because an intact nerve trunk appears to be resistant to venom, the venom enters the nerve tissue through the exposed pre-synaptic terminals at the neuromuscular junction. Muscular twitchings and fibrillations may arise from the transmitter chemical being released. Scorpion neurotoxins are thermostable, basic proteins with a modest molecular weight that have the general ability to depolarize excitable membranes. This is brought on by a decrease in the rate and extent of sodium inactivation as well as an increase in the resting membrane's sodium permeability. It has been proposed that the venom of scorpions may alter both the passive and active sodium permeability systems and the sodium-pumping mechanism within fibers. The significance of scorpion venom lies in its antigenic character, which can trigger a strong antigen-antibody response.³

A shared binding site is suggested by the literature search, which also indicates that there is sufficient data to support the hypothesis that bupivacaine may partially protect against irreversible inhibition of scorpion venom. This supports our theory that, because the binding site is so ubiquitous, the earlier envenomation from a scorpion bite may actually interfere with the local anesthetic's ability to work.³

Takotsubo cardiomyopathy (TCM)

Takotsubo cardiomyopathy refers to a syndrome characterized by transient left ventricular (LV) dysfunction but without evidence of obstructive coronary artery disease (CAD). The term "takotsubo" refers to a Japanese ceramic pot used to trap octopuses, which resembles the most common LV conformation associated with this disorder, with apical akinesis and basal hyperkinesis. This condition was first described in the 1990s in Japan, delineating a stunned myocardium in the setting of multivessel coronary artery spasm. This cardiomyopathy has since been identified in more people globally and it is now known by several names, including stress-induced cardiomyopathy, apical ballooning syndrome and broken heart syndrome. This cardiomyopathy can be driven on by both physical and emotional stressors. It has been observed in critical care units, surgical settings and outpatient and inpatient scenarios.²

Management and Prognosis²

While most TCM patients who survive the acute episode have a good prognosis and recover quickly, it's crucial to remember that TCM patients have a mortality risk that is comparable to that of AMI patients. 5.9% was the 30-day risk of serious adverse cardiac and cerebrovascular events, according to Templin et al. In addition, there was a 1.7% annual rate of stroke or transient ischemic attack and a 5.6% annual mortality rate. Research shows that the yearly recurrence rate of TCM is between 1% and 2%, with ACEi/ARBs having a greater effect on recurrence reduction than beta-blockers.

CONCLUSION

Individuals who had previously been bitten by a scorpion seemed to be resistant to bupivacaine spinal block. This resistance showed up as insufficient block or block failure that needed general anesthesia to be used. In contrast to insufficient spinal block, several scorpion bites and more recent bites are linked to spinal block failure.

We are now more skilled at treating and diagnosing TCM in general and perioperative TCM in particular as our body of knowledge has grown. To evaluate different diagnosis and treatment plans, however, carefully thought-out controlled clinical trials are required. In addition to specific criteria for the perioperative context, clinical studies must also yield enough data to allow consensus on a single set of criteria for defining TCM. The creation of guidelines with treatment recommendations tailored to the level of hemodynamic stability of each patient would be made possible by such thorough testing. In addition to treating more patients more quickly and effectively if these principles are widely adopted by practitioners, there would also be a decreased chance of potentially fatal cardiac and cerebrovascular events following TCM. In addition to helping to prevent TCM perioperatively by customizing anesthetic management, concurrent research to better understand the cardioprotective effects of volatile agents and any potential genetic links may also help identify individuals who may be susceptible to this syndrome in the future.

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