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Case Report

Scorpion Envenomation: The Cause of Inadequate Subarachnoid Block - A Case Series

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ABSTRACT

Background: Failure of neuraxial or regional anaesthesia can result from factors such as drug errors, technical inefficiencies, and poor patient positioning. While these causes are well-known, resistance to local anaesthetic action due to mutations in sodium channels or scorpion sting is a lesser-known contributor to block failure. In India, a tropical country with a significant number of patients presenting for surgical procedures, a history of scorpion bites is not uncommon.
Case: We observed seven cases of failed regional anesthesia who had history of scorpion sting. All the patients received intrathecal bupivacaine by experienced anesthesiologists, of seven patients five patients did not develop sensory or motor block. One patient had delayed successful subarachnoid block after second attempt and one patient had successful block at first attempt.
Conclusion: Our observations revealed instances of failed spinal blocks, despite adequate drug dosages and experienced anesthesiologists performing the procedures, in patients with a history of scorpion envenomation. Accordingly, our study concludes that obtaining a thorough scorpion sting history during pre-anesthesia check-ups, particularly in endemic areas, can effectively prevent unnecessary repeated pinpricks, escalating dosages, patient and surgeon discomfort, and skepticism towards the skills of anesthesiologists.

Keywords: Spinal anesthesia, local anesthetic resistance, scorpion envenomation

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INTRODUCTION

Central neuraxial block is one of the most frequently used anesthesia technique, especially in developing and resource limited locations. Hence achieving successful block is of utmost importance in areas or cases where general anesthesia is not desirable. Failure of spinal anesthesia is attributed to many reasons, including incorrect needle placement, incorrect drug dosage, displacement of needle during drug injection, poor patient positioning, abnormalities of spine, ineffective drug action and local anesthetic resistance.[1] Scorpion bite history is a rare cause for local anesthetic resistance. There are more than 1500 species of scorpions worldwide and 86 in India. Of these *Mesobuthus tamulus* (Indian red scorpion) and *Heterometrus swammerdami* (Giant Forest scorpion) are of medical importance.[2] The most common symptoms of scorpion bite are severe pain and burning at the bite site, although systemic complications can ensue. The development of local anesthetic resistance is most often diagnosed retrospectively after failure of spinal block. Here we present a case series of 7 patients in whom a positive history of scorpion bite was elicited after failed subarachnoid block.

CASE

Case 1

A 50-year female patient, classified according to American Association of Anesthesiologist (ASA)-I, presented for infraumbilical incisional hernia repair. A single shot subarachnoid block (SAB) was administered by experienced anesthesiologist, but no sensory or motor blockade was observed within 15 minutes post-SAB. The patient revealed a previous failed spinal block during surgery four years ago, which required general anesthesia. Further inquiry unveiled a history of scorpion bite 20 years ago.

Case 2

A 25-year male patient, classified as ASA-I, presented for inguinal hernioplasty, encountered failed subarachnoid block using 0.5% heavy Bupivacaine. Repeated attempts also failed. Upon further investigation, the patient revealed that he had scorpion bites twice in the past, 8 years and 2 years prior. General anesthesia was then administered. On the second postoperative day patient developed post-dural puncture headache which relieved by sphenopalatine ganglion block and supportive treatment.

Case 3

A 45-year male patient, classified as ASA-II, presented for right inguinal hernioplasty. Initial subarachnoid block with 0.5% heavy Bupivacaine was failed, but a repeat attempt after 20 minutes resulted in an adequate level of block within 15 minutes. A history of scorpion envenomation 15 years prior was obtained.

Case 4

A 32-year female patient, classified as ASA- II, presented for post-natal sterilization. She had previous three normal vaginal deliveries without labor analgesia. A subarachnoid block using 0.5% heavy Bupivacaine was attempted but failed. Subsequent inquiry revealed a positive history of a scorpion bite two years ago. General anesthesia was later administered for the surgery.

Case 5

A 25-year male patient, a farmer by occupation and classified as ASA – I, presented for radiofrequency ablation of varicose veins. Spinal anesthesia using 0.5% heavy Bupivacaine was attempted twice by experienced anesthesiologist, but both attempts resulted in block failure. Upon further investigation, the patient revealed that he had scorpion bites occurring two years and one year prior. General anesthesia was then administered for the surgical procedure.

Case 6

A 50-year male patient, classified as ASA -II, presented for left inguinal hernioplasty. Combined spinal epidural anesthesia was administered. After 5 minutes of subarachnoid drug administration using 0,5% heavy Bupivacaine, neither sensory nor motor block was observed, an attempt to rescue the block using 8 ml of 2% lignocaine via epidural catheter, resulted in an incomplete block. Upon further inquiry, the patient revealed a history of scorpion bite during childhood. The neuraxial anesthesia was then converted to general anesthesia.

Case 7

A 41-year female patient, classified as ASA – II, presented for umbilical hernia repair under spinal anesthesia. She has a history of scorpion bite 30 years prior. Spinal anesthesia using0.5% heavy Bupivacaine was administered, and it was successful.

DISCUSSION

This was a series of seven cases collected retrospectively over a period of one year in a tertiary care teaching hospital. Subarachnoid block is the most frequently administered form of anesthesia. The spinal anesthesia failure rate as mentioned in literature varies between 1-17%, though on an average its less than 4%.[3] Local anesthetic resistance after scorpion bite is one of the reasons for spinal block failure. The literature review suggested isolated case reports of spinal block failure after scorpion bite. In tropical and sub-tropical regions envenomation by scorpion sting is one of important health care problems. Envenomation by scorpions can result in a wide range of clinical effects, including, cardiotoxicity, neurotoxicity, and respiratory dysfunction.[4] Western Maharashtra, Karnataka, Andhra Pradesh, Gujarat and Tamil Nadu (Figure 1) are endemic areas for scorpion bites in India.[5]

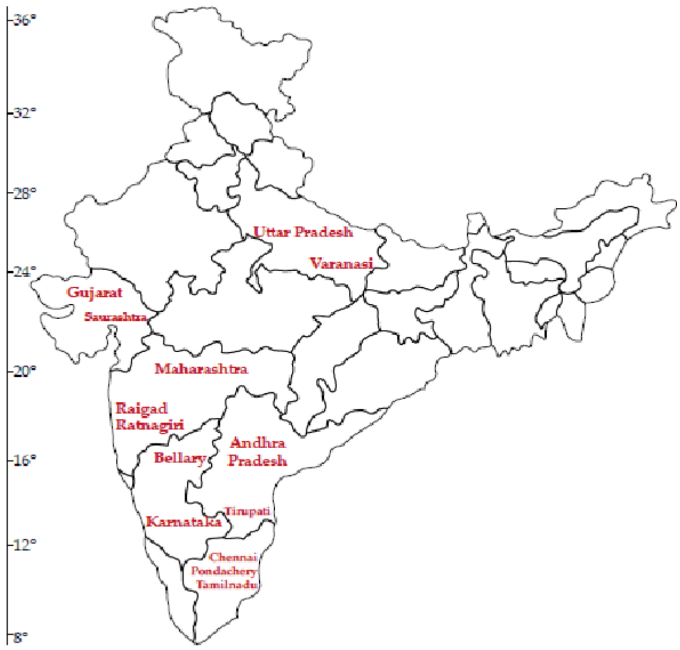

Figure 1. Endemic regions of Scorpion Sting in India [5]

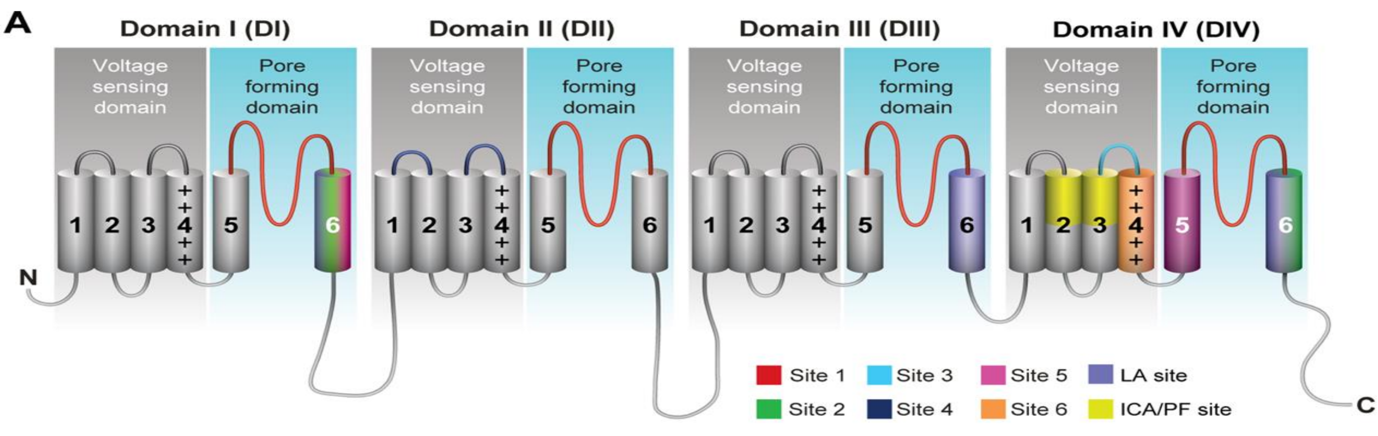

Figure 2. Structure of Voltage gated sodium channel. α - subunit with four domains marked in roman numerical, each domain having six segments marked in numbers[7]

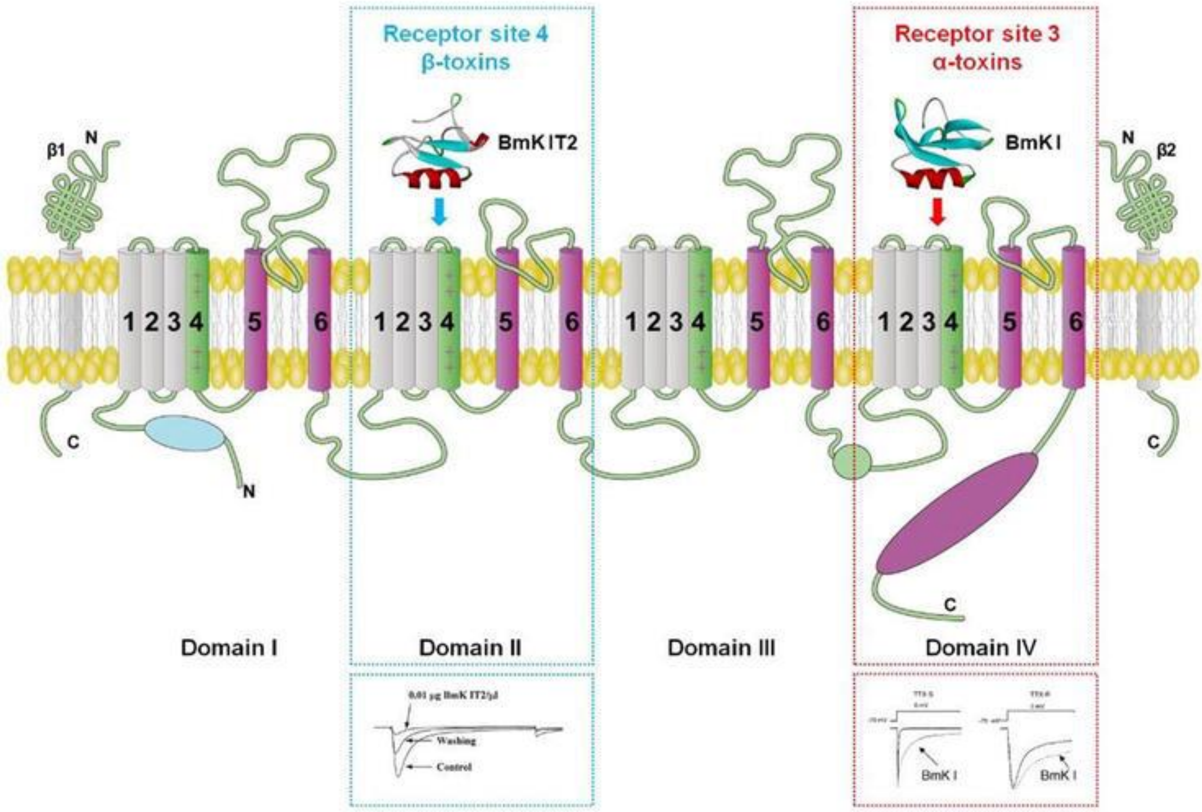

Figure 3. Receptor site for scorpion toxins on voltage gated sodium channel [9]

Scorpions venom is a cocktail of several low molecular weight basic proteins, neurotoxins, nucleotides, amino acids, oligopeptides, cardiotoxins, nephrotoxin, hemolytic toxins, phosphodiesterase, phospholipase A and hyaluronidase. Scorpion venom neurotoxin acts on voltage gated sodium channels and is divided into Alpha and Beta toxins. There are two schools of thought to explain the local anesthetic resistance following scorpion sting. One being mutation in sodium channels and the other being development of antibodies which competitively antagonize action of local anesthetics on sodium channel.[6] The local anesthetics act by blocking voltage gated sodium channel. The sodium channels are composed of α, β_1 and β_2 subunits. Each subunit has 4 domains (D1-4), each containing 6 transmembrane helices (S1-6). The mechanism of action is mainly by an interaction with the sixth segment of domain four of the Alpha (α) subunit (DIV - S6) (Figure 2). [7]

Scorpion toxins show a preference for specific sodium channel subtypes of mammals or insects. [8] Scorpion toxins (ScTxs) block or modify the function of voltage gated ion channels (VGIC). The long-chain scorpion toxins mainly act on voltage- gated sodium channels (VGSCs), while the short-chain scorpion toxins generally target potassium or chloride channels. Based on their physiological effects on VGSC gating and binding properties, the long-chain toxins can be further classified into two categories: α-toxins and β-toxins. The α-neurotoxin targets receptor site 3 of the VGSC, with inhibitory effects on the fast inactivation of VGSCs (Figure 3).

ß-toxins, which bind to receptor site 4 suppresses transient currents of VGSCs. [9] Ts1, which has also been classified as a β-scorpion toxin, is an allosteric modulator of voltage-dependent gating of Sodium channels.[10]

The other postulated theory is development of antibodies to highly antigenic scorpion venom which competitively inhibit binding of local anesthetic at sixth segment of domain four of the Alpha (α) subunit (DIV - S6).[11]

Kosam D and colleagues in their study found that patients with history of scorpion bite < 6 months back had complete failure of spinal anesthesia requiring administration of general anesthesia. While adequate subarachnoid block was noted in those patients who had history of scorpion sting > 12 months back. They concluded, the effect of scorpion sting on spinal anesthesia is more pronounced in recent bites and gradually wanes off after 12 months.[12] Panditrao et.al, in their prospective, case control study involving 70 patients, on effect of previous scorpion bite(s) on the action of intrathecal bupivacaine noted that history of multiple and recent scorpion bites (less than 8 months) was associated with complete failure of the spinal block and required general anesthesia whereas patients with scorpion bites older than a year showed delayed onset for sensory block or the motor block or both. [13]

Other causes for local anesthetic resistance like genetic A572D mutation in the SCN5A gene encoding for Nav1.5, type III Ehler Danlos Syndrome and redheads are rare but have been documented.[14,15]

In our case series we observed varied duration of history of scorpion bite from 1 - 30 years and failure of spinal anesthesia. One of the patients had history of scorpion sting 30 years ago and achieved adequate block after spinal anesthesia. This can be due to waning off the effect of toxins or that it was a non-poisonous scorpion. A global appraisal of scorpion envenomation reported 0.27% mortality with more than 1.2 million being stinged annually. Our hospital serves to population which falls into endemic region for scorpion sting and most patients coming from an agricultural background. The frequent occurrence of failed spinal anesthesia even in hands of experienced anesthesiologist, in spite of adequate drug dosage led us to enquire about history of scorpion sting which was elicited in all these patients. The nerve conduction studies could have been helpful to know any abnormal conduction studies, but it was not feasible to do at our institute. This case series aims to highlight that in at risk areas, it should be made a practice to elicit history of scorpion bite in pre-anesthesia check-up and if possible, patients be tested for local anesthesia sensitivity by subcutaneous instillation of local anesthetic agent.[16] This would be an economical yet robust evidence of local anesthesia resistance if present.

CONCLUSION

Scorpion envenomation is common in tropical countries like India. Often, the diagnosis is retrospectively made following failed neuraxial or regional blocks. Obtaining a thorough history of scorpion envenomation prior to regional block procedures enables anesthesiologists to anticipate potential complications. This proactive approach helps prevent unnecessary repeated pinpricks, escalating dosages, patient and surgeon discomfort, and skepticism towards the skills of anesthesiologists.

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CONFLICT OF INTEREST

The author declares there is no conflict of interest.

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