DOI: 10.21088/ijaa.2349.8471.12125.6

Indian Journal of Anesthesia and Analgesia
Volume 12 Number 1 January - March 2025

Case Report

A Case Report of Resistance to Intrathecal Anaesthesia following Scorpion Bite

Dinesh Kumar T.[1], Suresh Kumar N.[2], Huchappa K.V.[3]

How to cite this article:

Dinesh Kumar T., Suresh Kumar N., Huchappa K.V. A Case Report of Resistance to Intrathecal Anaesthesia following Scorpion Bite. Ind J Anesth Analg. 2025; 12(1): 35-38.

ABSTRACT

Introduction: Spinal anaesthesia is a widely used anaesthetic technique. But sometimes the failure of spinal anaesthesia occur because of reasons like obesity, poor positioning, spinal deformity like kyphosis, misplaced injection and local anaesthetic resistance. Resistance to local anaesthetic is one of the reason. The mechanism of action of local anaesthetics is through sodium channels and mutation in the sodium channel could be one of the possible reasons for the resistance.

Case Report: A 52-year-old man who has been experiencing excruciating stomach ache for three days. After receiving a diagnosis of acute appendicitis, he was scheduled for an open appendectomy while under spinal anesthesia. His laboratory results and general physical examination during the pre-anesthesia evaluation were normal, with an ASA grade I. A 25G Quincke's spinal needle was used to do a lumbar puncture at the L3-L4 area while the patient was seated and under aseptic precautions. The subarachnoid area was injected with 30 mcg of buprenorphine and 0.5% hyperbaric bupivacaine. The patient was then placed in a supine position. General anesthesia was administered for the procedure after two successive attempts at spinal anesthesia failed, as demonstrated by the pinprick method or by motor block, as demonstrated by lower limb movements.

The surgery and immediate post-operative period was uneventful. The patient described a scorpion bite that occurred a year ago when asked about his medical history.

Conclusion: Scorpion stings can lead to resistance to local anesthetics due to the venom's effect on sodium channels, potentially causing failure or delay in spinal anesthesia. Awareness of a patient's history of scorpion envenomation is crucial for effective anesthetic management.

KEYWORDS

• Spinal anaesthesia • Scorpion bite • Local anesthesia

Author's Affiliation:

[1] Junior Resident, Department of Anaesthesiology, Sri Devaraj Urs Medical College, SDUAHER, Kolar, Karnataka, India.
[2] Professor and HOD, Department of Anaesthesiology, Sri Devaraj Urs Medical College, SDUAHER, Kolar, Karnataka, India.
[3] Senior Resident, Department of Anaesthesiology, Sri Devaraj Urs Medical College, SDUAHER, Kolar, Karnataka, India.

Corresponding Author:

Suresh Kumar N., Professor and HOD, Department of Anaesthesiology, Sri Devaraj Urs Medical College, SDUAHER, Kolar, Karnataka, India.
E-mail: drskumar6@gmail.com

➢ **Received:** 11-10-2024 ➢ **Revised:** 19-12-2024 ➢ **Accepted:** 29-01-2025

KEY MESSAGES

Since spinal anesthesia is the most often utilized anesthetic technique, it is important to clearly elicit the reasons why spinal anesthesia fails before proceeding with the surgery. Finding these causes might be aided by a thorough medical history.

INTRODUCTION

Spinal anaesthesia is a widely utilized anaesthetic technique due to its high efficacy and relatively low risk profile.[1,3] However, in some instances, the procedure may fail to achieve the intended outcomes, resulting in inadequate analgesia or anaesthesia. The failure rate for spinal anaesthesia has been reported to range from 0.72% to 16% 4, demonstrating that it is not always completely reliable. There are several factors that can contribute to the failure of spinal anaesthesia, and recognizing these factors is important for enhancing patient care and minimizing complications.

Key contributors to failure include poor patient positioning, which can affect the precise placement of the needle, and obesity, which may alter the anatomical landmarks and complicate needle insertion. Mistakes in the insertion of the spinal needle, such as improper angle, depth, or misplacement, can also lead to suboptimal anaesthesia outcomes. Spinal abnormalities, such as kyphosis, scoliosis, ligament calcification, or disc fusion, can present additional challenges by affecting the normal structure of the spinal column and interfering with the spread of the anaesthetic. Furthermore, mistakes in the administration of the anaesthetic, such as incorrect drug choice, dosage, or injection technique, can result in inadequate anaesthesia. An inaccurate dose or improper distribution of the anaesthetic agent within the intrathecal space may also hinder the effectiveness of the procedure. In some cases, resistance to local anaesthetics might develop, preventing the drugs from achieving the desired effect[1,2.]

Local anaesthetics exert their effect by targeting sodium channels, which are integral to the transmission of nerve signals. By blocking these channels, local anaesthetics inhibit the propagation of pain signals, thereby inducing the desired anaesthetic effect. However, one potential reason for the failure of spinal anaesthesia could be mutations in the receptors that regulate sodium channels. These mutations can disrupt the normal functioning of the channels, leading to reduced sensitivity to the anaesthetic agent and contributing to the phenomenon of local anaesthetic resistance[1,2.]

CASE REPORT

A case of 52 year male came with complaints of severe abdominal pain since 3 days. He was diagnosed with acute appendicitis and posted for open appendicectomy under spinal anaesthesia. He was non hypertensive and non-diabetic and he had no other comorbidities. During the pre-anaesthetic examination, he weight of 56 kg. he was full conscious and oriented and vitals stable. There were no signs like cyanosis or pedal edema. On auscultation the lung fields were clear with bilateral equal air entry and no added sounds. Cardiovascular examination was normal. Upon neurological examination his motor power in both the lower limbs was 0/5 with no sensory deficit. His airway examination showed Mallampati grade 3 with and no restricted neck movements.

All his lab parameters were within normal limits. A peripheral line with 18G cannula was secured in the preoperative room and administered with Metoclopramide 10 mg and Ranitidine 150 mg and fluids were started and patient was shifted to OT. Monitors conected for continuous monitoring of heart rate, blood pressure, SpO2 and ECG. Patient was made to sit and under strict aseptic precautions parts were painted and draped. After identifying L3-L4 intervertebral space, 25 G Quincke spinal needle was used and lumbar puncture was done and after confirming negative aspiration of blood and free flow of CSF. Injection 0.5% hyperbaric Bupivacaine 2 ml and inj. Fentanyl 0.2 ml (15mcg) was administered.

The patient was carefully positioned in the supine position, and the level of sensory block

was assessed by using the pinprick method along the mid-axillary line after waiting for 3 minutes. However, despite waiting for 15 minutes, there was no noticeable improvement in the sensory block. As a result, a second attempt at spinal anesthesia was performed, but unfortunately, no signs of anesthesia developed even after this second attempt. Consequently, general anesthesia was administered in order to proceed with the surgical procedure. The surgery was carried out without any complications, and the immediate postoperative period was also uneventful, with no issues arising. During the process of taking the patient's medical history, it was revealed that the patient had experienced a scorpion bite approximately one year ago.

DISCUSSION

The phenomenon of resistance to local anesthetics, particularly in the context of spinal anesthesia, is a significant concern that can result in incomplete blocks, delayed onset, or even total failure of the intended anesthetic effect. While various factors can contribute to this phenomenon, there is growing evidence that scorpion stings may play a crucial role in the development of such resistance, as observed in certain patients with a history of scorpion envenomation.

Mechanism of Action of Local Anesthetics and Sodium Channels-Local anesthetics, such as lidocaine and bupivacaine, exert their effects by blocking sodium channels, which are crucial for nerve signal transmission. The sodium channel is composed of two subunits: the α subunit and the β subunit. The α subunit has four homologous domains, each containing six transmembrane segments (S1-S6). Local anesthetics are known to interact specifically with the sixth segment of the fourth domain of the α subunit, binding to phenylalanine and tyrosine residues, which disrupt the normal ion flow and inhibit nerve conduction. This interaction is central to the ability of local anesthetics to produce sensory and motor block during regional anesthesia.

Impact of Scorpion Venom on Sodium Channels- Scorpion venom is a complex mixture of toxins, enzymes, salts, biogenic amines, and water, and its composition can vary significantly depending on the species of scorpion. The toxins in scorpion venom are known to target sodium, potassium, and calcium ion channels. In particular, scorpion toxins can bind to the fourth domain of the sodium channel, which modifies the activation process of the channel and, consequently, the way local anesthetics interact with these channels. This binding alters the function of the sodium channels, making them less susceptible to the usual inhibitory effects of local anesthetics, leading to a phenomenon known as resistance to local anesthetics.

This resistance is thought to occur through the development of circulating antibodies to the venom. These antibodies can competitively antagonize the local anesthetic's binding site on the sodium channel, particularly the sixth segment of the fourth domain of the α subunit. Over time, repeated exposure to scorpion venom, such as through multiple stings, can augment this immune response, further decreasing the efficacy of local anesthetics.

Genetic Factors and Resistance-In addition to the direct effects of scorpion venom, genetic factors may also contribute to variations in individuals' responses to local anesthetics. For example, it has been observed that certain individuals, such as those with red hair, exhibit an increased resistance to local anesthetics, though the exact reasons for this remain unclear. Genetic variation in the expression or function of sodium channels could potentially play a role in how the body responds to both local anesthetics and toxins such as those from scorpions.[4]

Clinical Implications of Scorpion Bite History-The development of resistance to spinal anesthesia following scorpion bites can manifest in several ways, including delayed onset of sensory and motor block, incomplete block, or complete block failure. The severity of this resistance can vary depending on factors such as the duration since the scorpion sting and the number of stings the patient has experienced. Studies have shown that the longer the time since the scorpion sting, the less pronounced the resistance is to local anesthetics. However, the number of stings does not necessarily correlate with the extent of sensory and motor block failure.

In clinical practice, especially in areas where scorpion stings are common, it is essential for anesthesiologists to be aware of a patient's history of scorpion envenomation. Detailed

history-taking, including the number of stings and the time since the last sting, should be part of the preoperative assessment. If a patient with a history of scorpion bite is scheduled for surgery, regional anesthesia may be avoided, or alternative anesthetic techniques, such as general anesthesia, should be considered.[5]

CONCLUSION

Resistance to local anesthetics following scorpion envenomation represents an important, yet often underrecognized, cause of failure of spinal anesthesia. The interaction between scorpion venom and sodium channels, as well as the potential for antibody-mediated interference, can diminish the effectiveness of local anesthetics, leading to incomplete or delayed blocks. Awareness of this phenomenon, particularly in regions where scorpion stings are common, is crucial for anesthesiologists in planning appropriate anesthesia techniques. By incorporating questions about scorpion stings into the preoperative assessment, anesthesiologists can better anticipate and mitigate the risks associated with regional anesthesia in these patients. This small but crucial step can significantly improve patient care and reduce the likelihood of unnecessary complications.

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