

Transient Ipsilateral Lower Limb Paresis after Interscalene Brachial Plexus Block

O. H. Idehen*, F. E. Amadasun

Department of Anaesthesia, University of Benin Teaching Hospital, Benin City, Nigeria
Email: idehenhanson@yahoo.com

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Abstract

We describe a partial motor block of ipsilateral lower limb after interscalene block with the injection of 40 ml of 0.5% bupivacaine and 2% lignocaine with adrenaline. Immediately after the block, patient reported a right motor hemi syndrome which was transient. We concluded that the neurological symptom was caused by the technique of interscalene brachial plexus block may be as a result of excessive lateral deviation of the neck with a compromised collateral circulation.

Keywords

Interscalene Block; Transient; Ipsilateral; Lower Limb Paresis

1. Introduction

Interscalene approach to brachial plexus blockade results in consistent anaesthesia of the shoulder, arm and elbow. It is not recommended for hand surgery. It relies on dispersion of large volume of local anaesthetic within the interscalene groove to accomplish blockade of the brachial plexus.

Complications commonly associated with interscalene block include infection, hematoma, vascular puncture, nerve injury, total spinal anaesthesia, Horner's syndrome, diaphragmatic paralysis etc. due to the proximity to the neurovascular structures in the neck. Major neurologic sequelae or neuropathy that occurs after interscalene block is rare [1]-[3]. Interscalene neurologic complications are either associated with or not to the interscalene blockade.

The incidence of transient ipsilateral lower limb paresis is extremely rare, the only reported case following interscalene block was with the use of catheter and continuous local anaesthetic infusion. We report the same following single shot interscalene block.

*Corresponding author.

2. Case History

A 30-year-old male patient, who was involved in a road traffic accident and sustained fracture of the right proximal humerus, he was billed for open reduction and internal fixation of the right humerus. Pre-operative evaluation of the patient revealed a conscious patient, afebrile, not pale, anicteric. Clinical examination revealed pulse rate of 75 b/min, blood pressure of 110/60 mmHg, respiratory rate of 18 cycles/min, vesicular breath with no added sounds. He was assigned a mallampatti score of 2, based on visualization of oro-pharyngeal structures and was classified as ASA1 on physical fitness status.

Results of laboratory investigations were all within normal range, PCV 36%, and normal electrolyte and acid-base values (urea 17 mg/dl, Na 135 mmol/l, K 3.8 mmol/l, HCO₃ 24 mmol/l, Cl 95 mmol/l. Creatinine 0.9 mg/dl). Coagulation profile was normal.

The patient was transferred to the operation theatre and standard monitors attached (SpO₂, non-invasive blood pressure monitors, and electrocardiogram). The baseline vital sign parameters were pulse rate 85/min, blood pressure 117/72 mmHg and SpO₂ was 98%. Intravenous access gained using a 18 G cannula.

The right side of the neck was prepared for an interscalene block aseptically. Patient was placed supine with pillows beneath the scapular, with the head facing away from the side to be blocked. The landmarks for the interscalene block were identified and the needle insertion point was a point where a lateral line from the cricoid cartilage meets the interscalene groove, using a nerve stimulator (life-Tech EZstim 11) and an initial current of 1 mA (2 Hz, 100 µsec). Bicep contraction was elicited when needle was about 2.5 cm deep and 60° to the sagittal plane. The current was then gradually reduced to about 0.4 mA with sustained biceps contraction and 40 ml (20 ml of 0.5% bupivacaine and 20 ml of 2% lignocaine adrenaline) deposited after confirmation of correct needle placement by aspiration to rule out blood or CSF. There was positive Raj response when few ml of local anaesthetic agent was injected. Injection of local anaesthetic continued while making sure patient was not experiencing excruciating pains and the assistance also took note of any increased resistance when depositing the drug from a syringe loaded with local anaesthetic.

Five minutes after the block was established, patient experienced gradual loss of temperature, sensory and motor sensation and ability to abduct the affected arm. Shortly after, the patient complained of inability to raise the right lower limb (no Horner syndrome, hoarseness of voice, respiratory distress). The patient was then instructed to raise the said right lower limb, and was unable to do so. Motor power was 1 in the Bromage scale. Neurological examination was done to rule out cervical subarachnoid /sub dura deposition or spinal cord injury. Contra lateral upper and lower limb motor power and sensation were intact. Hemodynamic values were pulse rate 101/min, blood pressure 150/91 mmHg and SPO₂ was 98%. The patient was reassured and after 5 mins, paresis was no more and no other significant intraoperative events.

Complete onset of block was 15 minutes thereafter; block was excellent for the desired surgery, with no rescue blocks administered. Vital signs remained stable intraoperatively. The post operative period was normal. Analgesia lasted for 7 hours 20 minutes and the patient was discharged home after 10 days on admission.

3. Discussion

Ipsilateral lower limb paresis following interscalene brachial plexus block is a rare but known complication; because of its rarity only one study has reported similar presentation [4]. In the report, it occurred following the use of catheter for continuous local anaesthetic infusion for post operative analgesia unlike in this index report which occurred after single shot deposition of local anaesthetic.

Interscalene block is said to have numerous advantages, among which are lower VAS pain score, decreased opioid requirement, fewer opioid related side effects, an improved joint range of motion for a better post operative physiotherapy and higher patient satisfaction [5].

However, one systemic review noted that the interscalene block also has the highest incidence of permanent neurological complication of all peripheral nerve blocks [6]. Complications due to interscalene block are related to the spread of the local anaesthetic agent through the facial sheath to the surrounding anatomical structures [7]. These structures include the brachial plexus, stellate ganglion, phrenic nerve, the recurrent laryngeal nerve, the epidural and subarachnoid space and vertebral artery. The complications that can occur due to this anatomical relationship include brachial plexus nerve injury [8], Horner syndrome (Ipsilateral ptosis, hyperaemia of the conjunctiva, nasal congestion), diaphragmatic paralysis with 100% incidence [9], hoarseness, epidural and spinal anaesthesia [1] [2], infection, hematoma, local anaesthetic toxicity, which can lead to cardiac arrest. Even, a

Table 1. Intraoperative vital signs.

	Heart Rate	Blood Pressure	SpO ₂
5 min	101/min	150/91 mmHg	98%
10 min	103/min	137/83 mmHg	99%
15 min	104/min	132/84 mmHg	99%
20 min	104/min	126/82 mmHg	98%
25 min	101/min	151/96 mmHg	99%
30 min	103/min	135/91 mmHg	99%

rare presentation of transient hypertension after interscalene block has anatomical explanation [10], being ascribed to the blockade of the carotid baroreceptors.

Epidural injection of the local anaesthetic during interscalene block is one of the speculated mechanisms for the ipsilateral lower limb sensory and motor loss [4]. Epidural injection will result in cervical epidural anaesthesia that is characterized by bilateral cervical and thoracic blockade with difficulty in breathing and hemodynamic instability [2]; this was not the presentation as reported by Faust *et al.* [4] and also there was no hemodynamic instability in this report (see **Table 1**). Previously, published reports on epidural placement of catheter after interscalene block did not present with Ipsilateral lower limb paresis [11] [12]. Cook also described signs of cervical epidural blockade characterized by numbness spreading to the contralateral shoulder with postural hypotension [11]. Complications resulting from the use of the interscalene perineural catheter are rare; interscalene catheter placement as well as the prolonged administration of 0.2% ropivacaine does not increase the rate of complication [13].

Transient cerebral ischemic attack with hemi paresis or ipsilateral paresis secondary to interscalene block can cause a diagnostic dilemma especially with the anaesthetized upper limb. It becomes difficult to categorically state which is responsible. In the case reported there were no cranial nerve involvements like loss of vision (amaurosis) and difficulty in speaking (dysphasia). Thereby the most likely cause is due to the interscalene plexus block and not due transient ischemic attack.

It is possible that the position the patient assumed during interscalene block (excessive lateral deviation of the neck) led to the occlusion of the contralateral vessels with a compromised collateral circulation, causing transient paresis or a neurological complication resulting from interscalene block. Most neurological symptoms are due to causes unrelated to nerve blocks [14].

The limitation of this case presentation is the absence of CT scans evaluation of a possible cerebral lesion and an Echo Doppler to access the integrity of the carotids and vertebral arteries that patient could not afford. However more appropriate and proper examination should be taken into consideration in cases like this to determine the most probable reasons for occurrence of this phenomenon. Patient recovered almost immediately with no residual neurologic deficit till discharge from the hospital after 10 days.

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