Case Report

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Anaesthesia Section

Transient Hypertension after an Interscalene Block-The Presentation of a Rare Complication with an Anatomical Explanation

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ABSTRACT

We would like to report 2 cases where we encountered a rare complication of hypertension without tachycardia after the administration of an interscalene block. We have tried to explain the same with the anatomical correlations.

Key Words: Interscalene block, Hypertension, Carotid receptors

INTRODUCTION

Case capsule 1:

A 55 year old male patient had a history of the fall of a coconut on his left arm and he had sustained a fracture on the mid shaft of the humerus. He was posted for the open reduction and the internal fixation of the left humerus. The preoperative evaluation of the patient revealed a pulse rate of 62 per min, a regular and a blood pressure of 170 / 90 mm Hg and a respiratory rate of 18/ min. Electrocardiography revealed left ventricular hypertrophy by the voltage criteria and 2 D echocardiography showed a grade I diastolic dysfunction, no regional wall motion abnormality, and a trivial aortic, mitral and tricuspid regurgitation with no clots in the chambers. The other systemic examinations were unremarkable. The laboratory investigations revealed anaemia (a haemoglobin concentration of 7.1), which was optimized with blood transfusions. The other investigations were unremarkable. The patient was optimized with the cardiac medications; Tab. Atorvastatin 20 mg OD and Tab. Amlodipine 2.5 mg OD for a week and he was then taken for surgery.

He was premedicated with Tab. Ranitidine 150 mg, Tab. Metoclopramide 10 mg and Tab Diazepam 10 mg along with cardiac medications on the day of the surgery. The patient was transferred to the operation theatre and the standard monitors were attached (SpO2, non-invasive blood pressure and the electrocardiogram). The baseline vital parameters were a pulse rate of 85/ min, BP of 140/90 mm Hg and SpO2 of 98%. The left side of the neck was prepared for an interscalene block. Under ultrasound guidance (Sonosite), the landmarks for the interscalene block were identified and 15 ml of 0.5% bupivacaine with 15 ml of 2% adrenalized lignocaine was infiltrated. The spread of the local anaesthetic agent through the plexus sheath was confirmed by doing an ultrasound. The sensory and the motor blockade of the left extremity was confirmed with the loss of temperature sensation and the inability in abducting the arm.

Five minutes after the block, we noticed that the patient had hoarseness of voice and a difficulty in speaking. He was conscious and oriented and he responded to the verbal commands. There was no evidence of any respiratory distress. The blood pressure suddenly increased to 202/114 mm Hg with a heart rate of 90/ min and a normal sinus rhythm of the ECG on the monitor. The hypertension persisted for another 20 minutes, during which the patient continued to have high blood pressure (170 – 190 / 106 – 110 mm Hg) without a significant increase in the heart rate. Inj. Midazolam 2 mg and Inj. Fentanyl 100 µg were intravenously administered to the patient. The blood pressure reached a level of 150/90 mm Hg and the heart rate was 92/min. General anaesthesia, with a cuffed oral endotracheal tube was administered, as the surgical procedure was planned in the right lateral position and the ensuing unstable haemodynamics. As the surgery was in the arm, the usage of a tourniquet was not contemplated. The vital parameters remained stable intraoperatively. The postoperative period was normal.

Case capsule 2:

A 45 year old male was posted for the reconstruction of a shattered elbow. The pre anaesthetic check up did not reveal any systemic illness. He did not have any other injuries. The pulse rate was 72/minute, with a blood pressure of 130/82 mm Hg. All the routine investigations were normal. The patient was transferred to the operation theatre and the standard monitors were attached (SpO2, non-invasive blood pressure and the electrocardiogram). The pulse rate was 70/min,. with a blood pressure of 130/80mmHg. The premedication was 1.5 mg intravenous midazolam. He was administered an interscalene block in the classical paraesthaesia technique, with 35 ml of 1.5% lignocaine with 1 in 4 lakh adrenaline. In this case, after 2 minutes, a sensation of numbness started in the forearm, with no evidence of the Horner's syndrome. The blood pressure started to increase to 186/110 mm Hg in the next 2 minutes, with a stable pulse rate of 75/minute. The systolic blood pressure remained at high levels of around 170- 180 mmHg, with a slow decline to 140 mmHg in around 30 minutes. The pulse rate remained at 70 to 75 /minutes. The surgery was done uneventfully without the usage of a tourniquet. The immediate postoperative course was normal.

DISCUSSION

Alon P. Winnie described the method of the interscalene block for injecting a local anaesthetic drug into the facial sheath of the brachial plexus in the neck, in the 1970s [1]. Since then, this method

of the brachial plexus block has been widely practised for surgeries of the shoulder joint and the upper arm. This block has been stated to have various advantages like lower visual analogue scale pain scores, decreased opioid requirements, fewer opioid-related side effects, an improved joint range of motion for a better postoperative physiotherapy and higher patient satisfaction [2,3]. The interscalene block is not left with noticeable complications too. These include pneumothorax, persistent neurological sequele (hypesthaesia, paresthaesias, pain/dysesthaesias, and motor weakness), phrenic nerve palsy, total spinal anaesthesia and cervical cord injuries [4–6].

The complications which are related to the interscalene block are related to the spread of the local anaesthetic agent through the facial sheath to the surrounding anatomical structures [7]. These anatomical structures can be the stellate ganglion, the phrenic nerve, the recurrent laryngeal nerve, the epidural and the subarachnoid space and the contralateral interscalene plexus [4,6,8,9]. The clinical correlation of the complications of the blockade of the anatomical structures varies e.g. the phrenic nerve blockade is revealed as a unilateral hemi diaphragmatic paralysis or the stellate ganglion blockade is revealed as the Horner's syndrome [8].

The disturbances in the autonomic nervous system after an interscalene block due to the blocked carotid body baroreceptors have been described, which clinically correlate as severe hypertension [10]. The hypertension without tachycardia is observed with a carotid sinus blockade, which is due to the local anaesthetic agents experimentally [11]. Chakithandy et al., described a case report with a hypertensive crisis after an interscalene blockade. The hypertension remained to be persistent due to the failure of a contralateral carotid sinus body reflex compensation [10]. The blood pressure did not remain persistently high in our cases, which reflected the compensatory control mechanism of the contra lateral carotid body receptor. In our first case, which was a hypertensive patient, one could expect hypertension which was associated with the stress of the anaesthesia, but it was unlikely to be without concomitant tachycardia, especially without beta blockers. The antihypertensive drug was amlodipine, which causes tachycardia. The second case was normal, without any preoperative drugs which could influence the haemodynamics. In the first case, we resorted to the institution of general anaesthesia for the reasons like apprehensions about the position, Horner's syndrome and the possible unstable haemodynamics. In the second one, as the position was supine, we continued with the regional technique. Hence, we propose that the hypertensive response without tachycardia in our patients could be due to the involvement of the carotid baroreceptors. Severe hypertension without an increase in the heart rate after an interscalene block, can be considered as a complication which is not well documented in the medical literature [12].

CONCLUSION

To conclude, hypertension without tachycardia can occur as a rare complication after an interscalene blockade, which is due to a blockade in the carotid baroreceptors. This is transient because of the compensatory mechanisms from the contralateral side.

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