Case report

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Interscalene block - Potential for cardiac arrest

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ABSTRACT

A 52 year Hypertensive female with normal examination and routine investigations including ECG for upper end humerus surgery was given PNS guided interscalene block supplemented with superficial cervical plexus block. After 20 min, due to inadequate block action, routine general anaesthesia was supplemented. At 25 min post induction of GA, just when surgery was to be started, there was sudden unresponsive hypotension with bradycardia culminating in asystole. CPCR started according to ACLS led to ill sustained ROSC followed by pulse less Ventricular tachycardia twice which reverted with prompt defibrillation. We achieved ROSC with sinus tachycardia, RBBB and ST elevation in lateral leads. Inj nikoran dil and enoxaparin were started empirically for ischemic event. After 1 hr of the event ECG done at ICU admission showed normal sinus rhythm with no ischemic changes. Serum electrolytes, ABG, 2D Echocardiography revealed no abnormality. She was extubated fully awake with no neuro deficits. Coronary angiography showed minor block amenable to medical management. She took discharge after 2 days and was subsequently lost to follow up.

Keywords: Cardiac Arrest, Interscalene Block

INTRODUCTION

The risk of perioperative (intraoperative and PACU) cardiac arrest was 5.6 per 10,000 cases with an associated mortality from the arrest of 58.4%. The rate of cardiac arrest increased with age and ASA physical status[1].

Shoulder surgery has since long been performed under Interscalene brachial plexus block supplemented with sedation or general anaesthesia. Semi sitting position is employed to reduce need for traction and hence traction neuropathies. However, simultaneous use of interscalene block, GA and Semi sitting position has been seen to cause hypotension and bradycardia to the extent of imminent cardiac arrest. We report one such case where the patient arrested even before the surgery could be started discussing the differential diagnoses, various theories of causation and prevention and treatment for the same.

CASE REPORT

A 52 year 60 kg ASA2 Hypertensive female on irregular treatment with amlodipine was posted for Right upper end humerus fracture open reduction and plating in supine position. All routine investigations including ECG were normal. Her MET score was 4. Pre-operative Pulse was 84/min and BP was 140/86 mmhg.

After appropriate consent check she was given PNS guided Right sided interscalene brachial plexus block with 15 cc 0.75% ropivacaine + 7 cc 2% xylocaine + 8 cc NS + hyaluronidase

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supplemented with superficial cervical plexus block with 8cc 1% xylocaine with adrenaline. Vitals remained stable throughout the procedure and thereafter, however as the action was found to be inadequate even after 20 min, it was decided to supplement the block with General Anaesthesia.

After preoxygenation and premedication with 0.2 mg glycopyrrolate,100 mcg Fentanyl and 2 mg Midazolam, induction was carried out with propofol 75 mg and 50 mg atracurium. Endotracheal intubation was carried out with 7 number tubes which was duly checked and fixed. Mechanical ventilation was initiated at tidal volume of 400 ml at 12 breaths per minute with 50% nitrous oxide in oxygen and 1% sevoflurane. Pulse was 72, NIBP 110/88, 5 lead ECG, ETCo2 remained stable and within normal limits. After 5 minutes, pt was given 30 degree head up along with lower limb elevation and surgical preparations were started. No changes in vital signs were observed after position change.

At 25 min post induction of GA, just when surgery was to be started, there was sudden hypotension from110/70 to 70/50 mmhg for which 6 mg ephedrine and fluid bolus was given but no response. Further 6 mg ephedrine and sevoflurane dial turned to 0.2 did not help and within a span of seconds the heart rate started dropping from 72–50, 0.6 mg. atropine 100% oxygen without any agents and lowering the head up position was ineffective and severe cardiovascular collapse with asystole occurred. CPR started according to ACLS algorithm and 1mg adrenaline given. Ill sustained Return of spontaneous circulation occurred in 30 sec followed by pulseless ventricular tachycardia which was promptly defibrillated with biphasic 200 J and chest compressions continued. Again, an ill sustained sinus rhythm with a central pulse was achieved within 30 seconds but the pulseless ventricular tachycardia recurred which was again reverted to sinus rhythm with 200j defibrillation followed by chest compressions.

This time we could get a sustained return of spontaneous circulation with sinus tachycardia and ST elevation in lateral leads Bp 180/100. Chest was clear with bilaterally equal air entry. Inj amiodarone 150 mg was given slow IV to prevent further arrhythmia. A 12 lead ECG showed fresh RBBB with lateral lead STT changes. Suspecting a myocardial ischemic event Inj nikorandil 48 mg followed by 2 mg/hr was started and enoxaparin 0.6 mg IV was given. Supportive therapy in the form of 200 mg hydrocortisone, 8 mg dexa were given. CVP was 8-10 cm h20. Radial arterial line inserted for arterial blood pressure monitoring. Surgery was deferred a cast was applied to fractured limb and the patient was shifted to ICU. After 1 hr of the event ECG done at ICU admission showed normal sinus rhythm with no ischemic changes. Serum electrolytes, ABG reports were normal. 2D Echocardiography revealed no abnormality except concentric LVH. Meanwhile the patient was fully awake moving all limbs on command with stable hemodynamics. On extubation, vitals remained stable. She did not have significant pain at fracture site and pin prick sensation and power was reduced in the fractured limb but not completely absent. She refused memory of any event after the injection for block. A coronary angiography done after about 6 hrs revealed 30% block in LAD amenable to medical management and no evidence of coronary vasospasm. She was put on oral statins, regular antihypertensive therapy, aspirin and clopidogrel. She was observed in ICU for 6 hrs thereafter in the ward. The patient took discharge after 2 days and was subsequently lost to follow up.

DISCUSSION

Cardiac arrest in our case could have occurred from any one or a combination of the following

- Block related event
- Positioning related event
- Coronary event
- Anaphylaxis

It has been found that the onset of Local Anaesthetic Systemic Toxicity (LAST) is usually very rapid, following LA injection by 50 secs or less in half of cases, and occurring before 5 mins in three-quarters of the cases[4]. Ropivacaine is known to have a safer cardiac profile than Bupivacaine. Symptoms such as bradycardia and hypotension can persist for several hours after injection of even small amounts of LA, for example, bupivacaine 1 mg/kg[2,3]. In case of a rare delayed presentation, it is recommended that as LAST can present 15 mins after injection, patients who receive potentially toxic doses of LA should be closely monitored for at least 30 mins after injection[4].

Our patient received both lignocaine and ropivacaine in the recommended doses and did not present with symptoms for more than 45 min after injection of the drug, also did not have sustained bradycardia or hypotension, so LAST seems to be unlikely.

Likewise, intrathecal spread of the drug leading to total sympathectomy and parasympathetic over activity would have presented much earlier.

There are several case reports and studies listed in (Table 1) where interscalene block given with or without sedation or GA when combined with semi sitting position for shoulder surgeries have given rise to Hypotensive Bradycardic Events

(HBE) defined as a decrease in heart rate of more than 30/min in less than 5 minutes or any decrease of greater than 50/min, and/or a decrease in systolic blood pressure of more than 30 mm Hg in less than 5 minutes or any decrease below 90 mm Hg[10-15].

Table 1. Incidence of HBE

Study	Sample size	Incidence (%)
Vester Anderson	100	12
D Alessio	103	16
D Alessio	116	17
Ligouri	150	28
Kahn & Hargett	150	13
Seo	63	21

Most cited cases occur approximately 40 to 80 minutes after placement of the interscalene block or 25 to 45 minutes from initiation of the sitting position[5,6].

There are cases which have reported HBEs to have the potential to progress to lethal alterations in hemodynamics and cardiac rhythm sometimes requiring cardiopulmonary resuscitation[7-9].

There are multiple theories of causation mentioned in literature as follows:

1. **Carotid sinus hypersensitivity**
   - Exaggerated response to stimulation of the carotid artery baroreceptors located at the bifurcation of the common carotid arteries resulting in severe bradycardia and/or vasodilation leading to hypotension or syncope.
   - While performing interscalene block even stretching of neck while positioning may inhibit transmission of proprioceptive information from the sternocleidomastoid muscle which would be misinterpreted as hypertension leading to HBE elicited by baroreceptors[6].
   - Direct stimulation of the carotid baroreceptors while performing the interscalene block or due to pressure of local anaesthetic injected may also be responsible for HBEs.
2. **Bezold Jarisch reflex**
   - Cardioinhibitory reflex originating from stimulation of mechano and chemoreceptors in the walls of the ventricles of the heart signaling the medulla to respond by reducing sympathetic tone, causing bradycardia, peripheral vasodilation, and hypotension[16,17].
   - In context of shoulder surgery factors promoting activation of BJR – semi sitting position causing venous pooling in lower extremity augmented by IV sedation or GA, use of NTG for improving surgical vision with low BP thus reduced venous return and relatively empty ventricle.
   - As the patient is put in semi sitting position venous pooling occurs in the lower extremities. This orthostatic hypotension will be augmented by peripheral vasodilation from the effects of IV sedation/ga as also use of vasodilators like nitroglycerin for surgical preference for lower ranges of BP, Adrenaline added to the block solution or irrigation fluid or endogenous from surgical stress response will increase sympathetic tone leading to hypercontractility of a relatively hypovolemic ventricle stimulating the mechanoreceptors in the ventricular walls activating the BJR.
   - Chemoreceptors may also be stimulated by hypovolemia triggering thrombocytes to release serotonin which increases the activity of the vagus nerve, contributing to the parasympathetic response associated with the BJR[18].
   - The assumption of the BJR as the cause of HBEs, however, remains a source of considerable debate[16,19,20].
3. **Vasovagal reflex syncope**

   In the efferent path of this reflex arc are hypotension and bradycardia are due to inhibition of the sympathetic system and to activation of the parasympathetic system, respectively. The afferent pathway may consist of various receptors sensing: 1. fear of bodily injury; 2. painful or noxious stimuli; 3. venipuncture; 4. prolonged standing; 5. heat exposure; 6. exertion; and 7. coughing, swallowing or straining. However, all these are possible only in an awake patient and not in one who is not adequately sedated or anaesthetised.
4. **Orthostatic Hypotension (OH)**

   OH is defined as a reduction of SBP of at least 20 mmHg or diastolic blood pressure of at least 10 mmHg within 3 minutes of standing. Generally, orthostatic syncope occurs when the autonomic sympathetic vasomotor system is incapacitated and fails to respond to the challenges imposed by the upright position causing hypotension. Factors promoting OH perioperatively are fasting status, blood loss during surgery, use of antihypertensives preoperatively and vasodilators intraoperatively.

   Postulated Potential Risk Factors for HBE in association with interscalene block for shoulder surgeries are noted in (Table 2).

Table 2. Risk factors for HBE

Right sided Interscalene block due to spread of LA to Right Stellate Ganglion[18]
Exogenous epinephrine (as additive to LA or in irrigation fluid for arthroscopic surgery)[26,27,28]
Use of vasodilators for improving surgical field[27]
Patient on anti-hypertensives[27]
Use of fentanyl to supplement incomplete block[2-5]

Prevention and treatment of HBE is summarized in Table 3.

Table 3. Prevention and treatment of HBE

Use of GA instead of ISB in patients with known h/o Vaso-vagalsyncope[25]
preoperative appropriate hydration and an intraoperative stocking device[29,30]
Use of beta blockers, ondansetron[31,32]
Ephedrine is the drug of choice to treat the unknown origin of HBEs

Coronary Artery Spasm (CAS) though rare has been implicated as a cause of sudden, unexpected circulatory collapse and death during surgery, cardiopulmonary bypass, and other non-cardiac surgical procedures[21]. It is characterized by typical ECG changes including sudden ST segment elevation in the leads overlying the ischemic region with associated ST segment depression in the reciprocal leads. The resolution phase of coronary spasm is characterized by rapid return to baseline of ST segments.

The cause of perioperative CAS is unknown but potential general mechanisms to explain its occurrence perioperative patients include redistribution of blood flow, altered humoral factors, increased catecholamine response secondary to the level of anesthesia, Disruption of the sympathetic-parasympathetic balance[23] and imbalance of vasoconstrictor-vasodilator forces. More specific factors thought to provoke spasm may interact in the perioperative period, including increases in blood pH, excess α-adrenergic activity, stimulation of the parasympathetic nervous system, physical manipulation of a coronary artery, and release of vasoconstrictor substances by platelets[22].

Prompt coronary angiography is the only definitive modality for early diagnosis and targeted treatment. Pharmacological testing, such as provocation with intravenous ergonovine, should be used only under special conditions and with extreme care. If transfer to the cardiac catheterization lab is delayed, intravenous nitroglycerine has also been reported to reverse intraoperative CAS[22]. However, intravenously administered nitroglycerin is not always effective. Nifedipine, a calcium-channel blocker, is effective in relieving coronary artery spasm[24].

Severe Anaphylaxis can present with sudden cardiovascular collapse and although most drugs used in the perioperative period can cause anaphylaxis, it is fortunately a rare event[33]. Management involves cardiovascular support with adrenaline and fluids to counter the sudden vasodilation and use of corticosteroids[34].

CONCLUSION

HBEs are a well-known entity during shoulder surgery whether open or close occurring due to a combination of physiological changes arising from anaesthesia technique, positioning, patient predisposition and use of certain drugs. The anaesthesiologist managing such a case should be equipped with extreme vigilance to pick it up before it progresses to an irreversible catastrophe.

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