Case Report

Sudden cardiac arrest during spinal anaesthesia in a case of vaginal hysterectomy: a case report

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ABSTRACT

Spinal anesthesia is an important and commonly practiced anesthetic technique by the anesthesiologists worldwide. Though being considered a safe technique, it is not devoid of fatal complications and cardiopulmonary arrest (cardiac arrest) being the most serious among all. Cardiac arrest during spinal anaesthesia is a multi-factorial outcome, but vagal response to decreased preload often plays a key role. Common practice of preloading with IV fluid is suggested to not to be omitted before initiating spinal anaesthesia. If the situation warrants emergent, stepwise escalated resuscitative measures for treatment of bradycardia need to be instituted. A close supervision of the patients clinical parameter during spinal anaesthesia is a mandatory requirement to avert any untoward complication in such patients. In case of severe bradycardia or cardiac arrest, full resuscitative measures need to be promptly instituted.

Keywords: Bradycardia, Cardiac arrest, Spinal anaesthesia

INTRODUCTION

Spinal anesthesia is an important and commonly practiced anesthetic technique worldwide. Though considered safe, it is not devoid of risks or adverse events and cardiac arrest being of grave concern among all. Cardiac arrest during spinal anaesthesia is a rare but not so uncommon event. The incidence varies between 1.3 to 18/10000 cases. However, its exact frequency and predisposing factors remain undefined. We report a case of sudden cardiac arrest following spinal anaesthesia in a young healthy patient.

CASE REPORT

A 38 years old 50Kg healthy multiparous woman; case of 6-8 weeks fibroid of uterus with cervicitis was scheduled for elective vaginal hysterectomy, with pulse rate of 78beats/min, BP-130/70mmHg and SpO2-98%. Nothing abnormal was detected during her pre-anaesthetic examination. Her laboratory investigations were in normal range. Ultrasonographic examination confirmed a small intramural fibroid in the uterus.

On the day of surgery, patient was looking slightly anxious though adequately counseled. She was preloaded with 1000ml Ringer Lactate and multipara-patient monitor with real time ECG lead-II was attached. Under strict asepsis spinal anaesthesia was established using 3.0 ml of 0.5% Bupivacaine HCl (heavy) through L3-4 interspinous space in midline in left lateral decubitus position using 25G spinal needle, thereafter positioned supine with slight head down. Adequate analgesia up to T10 level was ascertained and the surgery allowed to proceed at around 1135 h.

Around 1200h, when surgeon applied more traction on uterus to operate trans-vaginally, the patient complained of epigastric discomfort, hence, 25mg Inj Ketamine was
administered intravenously along with oxygen through face mask. Around 1220h patient suddenly became unconscious, non-responsive and stopped breathing spontaneously. Carotid pulse was non-palpable, heart sounds were not audible, and ECG displayed a flat line. Immediately patient was intubated using 7.5mm internal diameter, PVC, cuffled, endotracheal tube and ventilated with 100% Oxygen. Inj. Atropine Sulphate 0.6mg was administered intravenously and cardiopulmonary resuscitation initiated with a thump on her chest. Within few seconds patient’s cardio-respiratory system was revived, the carotid pulsation re-appeared, and multipara-patient monitor started displaying sinus-tachycardia(132/min), BP-147/97mmHg and SpO₂-100%. Patient also started responding and generating spontaneous breathing.

Patient was thereafter converted to General anaesthesia with muscle relaxant, using Oxygen, Nitrous Oxide and Halothane through endotracheal tube. Around 1230h, surgeon was requested to continue with surgery. Surgery continued uninterrupted thereafter, and we very gently extubated her on conclusion of surgery. She remained stable throughout in post-operative period and recovered smoothly without any residual feature of cerebral hypoxia or anoxia. Her post-operative ECG showed normal cardiac activity without any sign of ischemia. As there were no complaints about her physical and mental soundness, she was discharged on 8th post-operative day.

Intra-operatively total blood loss was 400ml (approximately) and 1500ml Ringer Lactate solution was transfused.

**DISCUSSION**

Spinal anaesthesia associated cardiac arrests though are termed as rare, unusual and unexpected but are not so uncommon in practice.⁴ Even in the hands of experienced anaesthesiologists, the chain of events occurs very quickly and suddenly progresses in seconds. Cardiac arrest in association with spinal anaesthesia was first reported in 1940.⁵ The incidence of cardiac arrest during neuraxial anaesthesia ranges between 1.3 and 18 in 10,000 cases.⁵ However, predisposing factors, short and long-term survival remain contradictory.⁶ A recent literature review identified; vagal response to decreased preload to be a key factor and suggested that patients with high vagal tone are particularly at risk.⁷ Though multiple mechanisms interplay but the common mechanism is vagal-predominance (Vagotonia).⁸ Patients with increased vagal tone are at an elevated risk of developing severe bradycardia and cardiac arrest.⁹ Worsening bradycardia has often been noted before the onset of cardiac arrest during spinal anaesthesia. Therefore, it provides important clue towards the etiology and at the same time the etiological treatment for these arrests.⁴ Bradycardia thus serves as an important marker for extensive sympathetic blockade as well as for patients with excessive vagal tone due to other causes viz. fear, visceral traction, central volume depletion or athletic heart syndrome.⁵ When two or more of the factors listed in Table 1 are present, the patient may be considered at high-risk. Reduction in preload may also trigger reflexes resulting in severe bradycardia, and even asystole under spinal anaesthesia. Therefore, patient should be preloaded with adequate fluid (10-20ml/kg within 30 minutes).⁹ Maintaining adequate preload and prompt replacement of fluid losses is a key factor in decreasing the risk of bradycardia and cardiac arrest during spinal anaesthesia. If severe bradycardia (>40 beats/min) persists then prompt cardiopulmonary resuscitation (BLS and ACLS protocols) and pharmacological therapy need to be instituted promptly.⁹ Atropine is recommended to treat bradycardia or asystole during spinal anaesthesia because Glycopyrrolate is ineffective in this setting. Atropine (0.4-0.6mgI/V), Ephedrine (25-50mgI/V), and Epinephrine (0.2-0.3mgI/V) may be used in stepwise escalation.¹⁰ Response of cardiopulmonary resuscitation (CPR) is more difficult in cardiac arrests during spinal anaesthesia due to secondary preload reduction because at least 15 mmHg of coronary perfusion pressure is required for an effective CPR. According to Rosenberg et al, the dose of adrenaline necessary to maintain coronary perfusion pressure between 15 to 20mmHg during spinal anaesthesia ranges between 0.1 to 0.1mg/kg.⁵

<table>
<thead>
<tr>
<th>Risk factors of bradycardia.</th>
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<td><strong>Risk factors for moderate bradycardia (pulse &lt; 50 bpm) during spinal anaesthesia</strong></td>
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<tr>
<td>Baseline heart rate</td>
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<td>Asa physical status</td>
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<td>Use of beta-blocking drugs</td>
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<td>Sensory level</td>
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<td>Age</td>
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<td>Prolonged PR interval</td>
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</table>

Unfortunately, not all the cardiac arrests occurring during spinal anaesthesia are successfully resuscitated, fatal arrests still occur in healthy patients. Catecholamine deficiency is an important mechanism which explains the development of refractory cardiac arrest during spinal anaesthesia.

**CONCLUSION**

Currently, spinal anaesthesia figures among the commonly practiced anaesthetic techniques. Unquestionably, it is a very safe technique, however, not devoid of severe complications even in the apparently healthy patient. Cardiac arrest during spinal anaesthesia is a multi-factorial outcome, but vagal response to decreased preload often plays a key role. Common practice of preloading with IV fluid is suggested not be omitted before initiating spinal anaesthesia. If necessary, stepwise escalated dosage of atropine, ephedrine and epinephrine may be administered for treatment of bradycardia. In case of severe bradycardia or cardiac
arrest, full resuscitative measures need to be promptly instituted. Studies demonstrate recovery without cardicerebral sequelae in patients who developed bradycardia or asystole during spinal anaesthesia, when early and aggressive treatment was instituted. However, the possibility of a tragic outcome always exists. Hence, anaesthesiologists need to balance potential advantages and disadvantages diligently while practicing spinal anaesthesia in high risk population.

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REFERENCES
