

UNEXPECTED BRADYCARDIA AND CARDIAC ARREST UNDER SPINAL ANESTHESIA:

- Case Reports And Review Of Literature -

DEVARAJ NIDAGATTE DYAMANNA*, SANATH KUMAR BS**
AND BINOY THOMAS ZACHARIA***

Abstract

Spinal anesthesia has been regarded as safe and simple technique since its introduction into anesthesia practice. Bradycardia and hypotension under spinal anesthesia is a known phenomenon. However sudden unexpected bradycardia and cardiac arrest under spinal anesthesia is considered as rare and uncommon manifestation. On the contrary, as per current reviews, severe bradycardia and cardiac arrest under spinal anesthesia occurs more frequently in healthy, young and vagotonic patients. It is often associated with higher mortality. However, appropriate risk stratification, careful monitoring and structured management plan will have favorable outcome in these patients. We report successful management of two cases of unexpected cardiac arrest under spinal anesthesia and briefly reviewed the literature.

Key words: Spinal anesthesia, unexpected bradycardia, sudden cardiac arrest

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Introduction

Ever since August Bier administered first clinical spinal anesthesia more than a century ago, it has become an integral part of the modern day anesthesia practice. Although considered simple to perform and relatively safe technique, life threatening complications do occur under spinal anesthesia. Bradycardia and cardiac arrests during spinal anesthesia are described as very rare and unexpected, but are not uncommon. As per current literature the incidence of cardiac arrest under spinal anesthesia (neuraxial blockade) varies from 1.3 to 18 per 10,000 cases¹.

We report occurrence of severe bradycardia followed by asystole under spinal anesthesia in two patients, who were otherwise young and healthy. This communication is to emphasize the importance of vigilant monitoring and protocol based treatment in the management of severe bradycardia and cardiac arrest under spinal anesthesia.

* MD, EDRA, Senior Registrar

** MD, Registrar.

*** MD, Senior Registrar.

Affiliation: Department of Anaesthesia and Intensive Care, Sultan Qaboos University Hospital, Muscat, Oman.

Corresponding author: Dr. Devaraj Nidagatte Dyamanna, Senior Registrar, Department of Anaesthesia and Intensive Care, Sultan Qaboos University Hospital, Al-Khoud, P.O Box: No: 38, Muscat, Oman, Tel: 00968-96917685, Fax: 00968-24144710. E-mail: devrajnd@gmail.com

Case 1

23yrs old healthy male weighing 60kg was scheduled for left inguinal hernia repair. Routine preoperative assessment was done. Patient denied any medical illness and the laboratory reports were unremarkable. Anesthetic plan was discussed and patient consented for spinal anesthesia. He was advised overnight fasting and premedicated with oral midazolam 7.5mg.

Upon arrival to the operating theatre, the patient was calm and adequately sedated (Ramsay sedation score 2). Baseline blood pressure of 140/80mmhg, heart rate 72/min and oxygen saturation 97% on room air were recorded. Intravenous access (18G cannula) was obtained and preloading was done using 15ml/kg of lactated Ringers solution. Spinal anesthesia was performed under strict aseptic technique with the patient in the sitting position. Through L3/4 interspace, hyperbaric bupivacaine 12.5mg and fentanyl 20mcg was injected into the subarachnoid space using 25 G Whitacre-type spinal needle. Block level was assessed using pinprick and sensation for cold. At 10 min, maximum sensory block up to T6 was noted. Patient was continuously monitored. In the first 20 min, blood pressure and heart rate remained stable. While patient was verbalizing with the anesthetist, and without any prodromal symptoms he developed sudden bradycardia (heart rate <30/min). Subsequently, intravenous (IV) atropine 0.5mg was administered. Intravenous fluid was also given as rapid infusion. Suddenly patient became unresponsive with asystole. Immediately IV ephinephrine 1ml (1:10000) was given and cardiopulmonary resuscitation (CPR) initiated as per ACLS (American Heart Association) guidelines. After 2 min of CPR, patient was successfully revived without any residual effect. The surgical procedure was performed as planned. Postoperative electrocardiogram and cardiac enzymes were unremarkable. The patient was transferred to high dependency unit for observation and cardiology review.

Case 2

This 26yrs old male, weighing 74kg, with ureteric stones, was scheduled for ureteroscopy. During

routine preoperative visit, he was found healthy and denied any co-morbidity. His blood investigations were unremarkable. Patient accepted spinal anesthesia technique and consent was obtained. Patient was advised to fast as per guidelines and received oral midazolam 7.5mg as premedication.

On arrival to operation theatre patient was adequately sedated (Ramsay sedation score 2). Initial readings were 130/85mmHg for blood pressure, 70/min for heart rate and 99% for oxygen saturation on room air. Intravenous access was achieved using 18G intravenous cannula. Fluid preloading was done with 15ml/kg lactated Ringers. Under strict aseptic precautions with patient in the sitting position, spinal anesthesia was performed through L3-4 interspace using 25G Whitacre needle. We used 12.5mg of hyperbaric bupivacaine plus fentanyl 20mcg for subarachnoid injection. Vital signs were continuously monitored. Block level was assessed at regular intervals using pinprick and sensation for cold. At 15 min, the upper level of sensory block was T5 and patient was positioned in the lithotomy position. After 5 min post positioning, the complained of nausea. However his vital signs remained stable. While in conversation, he had abrupt bradycardia with heart rate down to 35/min. Immediately IV Atropine 0.5mg was administered and patient was repositioned in the supine position. Crystalloid rapid infusion was continued, but the patient suddenly became unconscious and developed asystole. Arrest code was activated and IV epinephrine 1ml (1:10,000) was given. He was revived after 3min of CPR and was fully awake with normal sinus rhythm. It was decided to continue with the planned surgical procedure. Post operative 12-lead electrocardiogram and cardiac enzymes were unremarkable. Cardiologist consultation was sought and patient was monitored in the high dependency unit.

Discussion

Bradycardia and cardiac arrest under spinal anesthesia is not an uncommon manifestation. It remained under reported. Ever since Caplan et al² reported 14 cases of cardiac arrest during spinal anesthesia in a American Society of Anesthesiologists

closed claim analysis, numerous case reports and reviews have been published³⁻⁶.

The mechanism that triggers severe bradycardia and cardiac arrest under spinal anesthesia remains controversial and unclear. Over sedation, respiratory arrest, unintentional total spinal, myocardial infarction and local anesthetic toxicity were hypothesized as the causative factors²⁻⁵. However, contribution of intrinsic cardiac mechanisms and autonomic imbalance with the background of parasympathetic predominance may provide more convincing and physiological explanation for the occurrence of abrupt severe bradycardia and cardiac arrest under spinal anesthesia^{7,8}.

The protective cardiac reflexes triggered by hypovolemia resulting in bradycardia include, 1) right atrial stretch reflex 2) firing of low pressure baroreceptors in right atria and venacavae and 3) the paradoxical Bezold-Jarisch reflex, due to stimulation of left ventricular mechanoreceptors^{4,8,9}. Bradycardia represents one end of the spectrum with cardiac arrest at the other end and may also be associated with vagal symptoms including sweating, nausea and syncope. Thus onset of bradycardia may be well thought of as the warning sign of severe bradycardia or impending cardiac arrest.

Our patients were comfortable, hemodynamically stable and well oxygenated, except for the second patient who had nausea prior to bradycardia. No ischemic changes were noticed in the electrocardiogram. Causative factors like myocardial infarction, respiratory depression, local anesthetic toxicity, subdural injection and high level of spinal anesthesia were considered and excluded by the sequence of events and laboratory investigations. Thus we attributed autonomic imbalance with intrinsic cardiac reflexes as the primary trigger resulting in bradycardia and asystole in our patients.

Autonomic imbalance with background vagal dominance may intensify any tendency to bradycardia, that might otherwise been more benign, transient, or possibly unnoticed. There exist a number of risk factors (Table1) with variable impact on the occurrence of severe bradycardia and cardiac arrest under spinal anesthesia^{8,10,11}. These factors may identify the vulnerable patients. However presence of two or more listed factors may place these patients

at high risk for bradycardia and cardiac arrest under spinal anesthesia⁸. Due to inconsistent reporting, risk factor association with the occurrence of bradycardia and cardiac arrest under spinal anesthesia still remains uncertain and contradictory.

Table 1

Risk factors for bradycardia and cardiac arrest during spinal anesthesia

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| <ol style="list-style-type: none"> 1. Age <50 years 2. Baseline heart rate <60/min 3. ASA physical status I and II 4. Use of beta blockers 5. Sensory level blockade above T6 6. Prolonged PR interval 7. Vagotonia |
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Hypovolemia with decreased preload may precipitate vagal symptoms and cardiac arrest in otherwise healthy patients¹³. Certain perioperative events are known to decrease preload or may cause vagal stimulation. Factors like surgical positioning, tissue retraction, bone cementing, reaming of long bones, membrane rupture and vasovagal syncope have been documented in the literature¹.

Our patients were young, healthy and with sensory block level T5/T6. There were no features suggestive of vagal predominance. Practically during routine anesthesia, it seemed unjustifiable to consider them as high risk for developing sudden and severe bradycardia under spinal anesthesia. Unexpected adverse events are known to occur under anesthesia, however being vigilant and use of a structured approach in the management of such an event still remains overemphasized.

Specific strategies to anticipate and prevent vagal predominance forms the mainstay in the management of severe bradycardia and cardiac arrest under spinal anesthesia are presented in Table2. Appropriateness of spinal anesthesia in patients at risk must be evaluated carefully. Alternative anesthetic techniques should be considered whenever intraoperative massive blood loss or vasodilatation is anticipated. Adequate preloading and replacement of volume loss has been emphasized in number of studies¹²⁻¹⁴.

Table 2

Management strategies for bradycardia and cardiac arrest during spinal anesthesia

<p>Prevention:</p> <ol style="list-style-type: none"> 1. Appropriate patient selection for spinal anesthesia when two or more risk factors are present (Table 1) 2. Maintaining adequate preload 3. Prompt replacement of fluid and blood loss. 4. Vigilant during patient positioning <p>Treatment of Bradycardia:</p> <ol style="list-style-type: none"> 1. Mild to moderate bradycardia (HR 30-60/min)-stepwise escalation of therapy <ol style="list-style-type: none"> a. Inj Atropine 0.4-0.6mg, IV b. Inj Ephedrine 25-50mg, IV c. Inj Epinephrine 0.2-0.3mg, IV 2. Severe bradycardia or cardiac arrest <ol style="list-style-type: none"> a. Advanced Cardiac Life Support guidelines to be followed b. Early administration of epinephrine known to improve outcome <p>Management of associated factors:</p> <ol style="list-style-type: none"> 1. Rapid fluid infusion 2. Patient repositioning 3. Avoid surgical manipulation

Whenever early vagolysis is required or vagal predominance continues, atropine and ephedrine must be administered. When the bradycardia is profound and unresponsive or a full cardiac arrest ensues, the early administration of epinephrine and effective cardiac compressions will be critical to maintain coronary perfusion and improve outcome. In addition, acute reduction in preload occurs during patient positioning, tourniquet release and acute blood loss. Thus rapid infusion of fluids and patient repositioning must be considered simultaneously. However, few questions still remain unanswered: 1) reliability and predictability of the stated risk factors 2) the extent of contribution of individual risk factors and 3) duration of post spinal anesthesia monitoring.

Conclusion

From literature review and our experience we conclude that bradycardia and cardiac arrest under spinal anesthesia is more common than once believed. However judicious patient selection, careful monitoring, early detection and prompt treatment may avert catastrophic outcome in these patients.

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