

## Case report

# A Case report of a cardiac arrest under a total spinal anesthesia

### ABSTRACT:

Total spinal anesthesia, while infrequent, may manifest during spinal anesthesia procedures. It is typically characterized by a sudden hypotension, difficulty of breathing, rapidly increasing motor block, loss of consciousness, dilated pupils, apnea, and in severe cases, cardiac arrest. Given its rarity and profound impact on patients, it's crucial to document and report these uncommon and potentially preventable incidents in clinical practice.

We document an instance of unexpected cardiac arrest subsequent to spinal anesthesia in a 45 years old male, ASA I. Fortunately, prompt and appropriate cardiopulmonary resuscitative interventions led to successful resuscitation of the patient. Subsequently, he underwent left inguinal hernia repair, experiencing a favorable post-operative course and recovery.

It is often associated with higher mortality. However, employing suitable risk stratification, meticulous monitoring, and a well-organized management strategy can lead to positive outcomes for these patients.

**Key words:** Cardiac arrest; Total spinal anesthesia ; Intraoperative complications, Resuscitation.

### INTRODUCTION:

Since August Bier administered the first clinical spinal anesthesia over a century ago, it has become an indispensable component of modern anesthesia practice. Widely adopted worldwide, spinal anesthesia offers a swift, profound, and predictable block, characterized by a straightforward administration process and a high success rate. However, its utilization is subject to contraindications, and patients should be informed about potential complications associated with its use.

High neuraxial anesthesia is also a frequently encountered issue, albeit rare, with the potential for a complete spinal block, which represents a life-threatening complication unless promptly recognized and addressed [1].

Bradycardia and cardiac arrest during spinal anesthesia are often characterized as very rare and unexpected but are not uncommon occurrences. In the literature, the reported incidence of cardiac arrest during spinal anesthesia (neuraxial blockade) ranges from 1.3 to 18 per 10,000 patients [2].

We report a case of sudden unexpected cardiac arrest of a young man, ASA 1, scheduled for left inguinal hernia repair under a spinal anesthesia. This communication serves to underscore the crucial significance of vigilant monitoring and adherence to protocol-based treatment in effectively managing sudden cardiac arrest occurring under spinal anesthesia.

## **CASE PRESENTATION:**

We present the case of a 45-year-old male with no significant medical history or prior surgeries, who was scheduled for a left inguinal hernia repair.

The patient was seen in pre-anesthetic consultation. He was in good general condition, his weight was 82 Kg, his height was 176cm, his BMI was 26.4. He had no dyspnea at rest or during exercise, no chest pain, his functional capacity was > 4 METs.

The cardiorespiratory examination was normal, there were no heart murmurs. His vitals were normal with BP 132/68, HR 67Bpm, respiratory rate 16 and SpO<sub>2</sub> 98%. His preoperative workup was strictly normal, with a hemoglobin level of 13.7 g/dl, normal renal function, and other blood investigations were unremarkable.

His ECG was also normal, with a regular sinus rhythm, HR at 68 bpm, he had no conduction or repolarization disorder. His frontal lung x-ray was normal, with a cardiothoracic index < 0.5.

The patient was not taking any long-term treatment, so he was classified as ASA I, scheduled for a low-risk cardiovascular surgery. Anesthetic plan was discussed and patient was consented for spinal anesthesia. He was advised overnight fasting and premedicated with oral midazolam.

The patient was admitted to the operating room, installed on an operating table, was monitored with heart rate 78 beats/min, electrocardiogram, noninvasive blood pressure (BP) 120/78 mmHg, and pulse oximetry with SpO<sub>2</sub> at 99% in room air. An intravenous access (18G cannula) was secured and preloading was done using 500 ml of normal saline solution.

Then the patient was installed in a sitting position, with all necessary aseptic measures in place, a spinal anesthesia was realized by an anesthetist at the level of the L3-L4 space, by injection of 7.5mg of hyperbaric bupivacain, with 25micrograms of fentanyl as an adjuvant.

Five minutes after transitioning the patient to the supine position, it was observed that the sensory block level had reached T6.

Verbal contact was maintained with the patient, who responded perfectly to the questions of the anesthesiologist. The patient remained hemodynamically stable and did not present a post-spinal hypotension.

While preparing the abdomen's skin, approximately 10 minutes after the subarachnoid injection, and without any prodromal symptoms the patient developed sudden bradycardia (heart rate <30/min). Subsequently intravenous atropine 0.5mg was administered. Intravenous fluid was also given as rapid infusion.

Suddenly he becomes; apprehensive, had chest heaviness, and he became apneic and unresponsive with asystole.

Immediately cardiopulmonary resuscitation (CPR) was started with direct IV injection of 1mg of adrenaline, orotracheal intubation was performed, and ventilation initiated with 100% Oxygen.

After 5 min of resuscitation effort, the patient regained spontaneous circulation, and vasoactive drugs were required to ensure hemodynamic stability. In a joint decision, the surgical team and the anesthesiologist opted to delay the surgical procedure by 2 hours until the spinal effects regress.

Then he was operated as planned and the remainder of the anaesthetic course was uneventful.

The patient was transferred to the intensive care unit (ICU) at the end of the procedure, intubated, and put on a ventilator for further management. Approximately 24 hours after admission to the ICU, Trans thoracic echography, Postoperative electrocardiogram and cardiac enzymes were unremarkable. The patient was extubated successfully and maintained the hemodynamic parameters up to a discharge from the ICU to the ward the next day.

## DISCUSSION:

According to the literature review, total spinal anesthesia can manifest during a range of regional anesthesia methods, such as epidural anesthesia, spinal anesthesia, caudal anesthesia, paravertebral block, interscalene brachial blocks, stellate ganglion block, and other techniques performed near the vertebral column. Clinical signs typically encompass sudden hypotension, rapid onset of motor block, difficulty of breathing, unconsciousness, dilated pupils, apnea, and in severe instances, cardiac arrest [3,4].

While spinal anesthesia is generally considered safe, it can potentially lead to complications, with cardiopulmonary arrest being the most severe, albeit uncommon [5, 6].

The two largest prospective studies aimed at assessing the incidence of complications during spinal anesthesia documented two arrests among 1,881 patients [7] and 26 arrests among 40,640 patients [8], resulting in an overall incidence of seven arrests per 10,000 (0.07%) spinal anesthetics.

Although regional anesthesia typically carries lower rates of morbidity and mortality, it can still pose troubling or even life-threatening risks. One particularly feared complication is total spinal anesthesia, marked by the inadvertent upward diffusion of local anesthetic agents into the subarachnoid space, impacting the brainstem and cranial nerves, ultimately leading to loss of consciousness.

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There are several contributing factors to total spinal anesthesia: [9]

- Local anesthetic dosage
- Patient positioning
- Pre-existing epidural block
- Unrecognized dural puncture and intrathecal injection
- Accidental subdural block
- Accidental intradural space

Several risk factors (as outlined in Table 1) have varying degrees of influence on the incidence of severe bradycardia and cardiac arrest during spinal anesthesia. Carpenter et al. [10] found that a baseline pulse rate of less than 60 bpm was linked to a fivefold increase in the likelihood of developing moderate bradycardia during spinal anesthesia. Typically, younger patients exhibit strong vagal tone, with ASA physical status I patients showing a threefold increased risk of moderate bradycardia under spinal anesthesia. Additionally, current treatment with beta-blockers or a block height above T6 were identified as significant risk factors for bradycardia in this study. Others have reported that patients under the age of 50 and those with first-degree heart block also face an increased risk of moderate bradycardia during spinal anesthesia.

A single risk factor from Table 1 does not make it certain that a patient will experience severe bradycardia or cardiac arrest. However, The presence of two or more of the listed factors may indicate a high risk for bradycardia and cardiac arrest during spinal anesthesia in these patients [11].

*Table 1*  
*Risk factors for bradycardia and cardiac arrest during spinal anesthesia*

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

Patients frequently present various clinical symptoms before complete spinal block, which often depend on the distribution level of local anesthetic agents. Hypotension, with or without bradycardia, is a result of venous and arterial vasodilation, causing diminished venous return, cardiac output, and systemic vascular resistance, as well as direct blockade of cardioaccelerating fibers (T1-T4) [12,13]. Mild dyspnea is frequently reported, attributed to the inhibition of abdominal and intercostal respiratory accessory muscles (T1-T12). Additionally, sensations of numbness or weakness in the arms, shoulders, and trunk (C5-T1) may be experienced.

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Following these, patients may experience nausea, with or without vomiting, attributed to cerebral hypotension, respiratory arrest due to diaphragmatic paralysis (C3-C5), and involvement of the brainstem [12,14]. In certain instances, cardiac arrest may follow, either due to hypoxemia and hypotension or unopposed vagal dominance [14,15].

The management is based on symptomatic treatment with effective cardiopulmonary resuscitation. External cardiac massage should be started as soon as possible according to the recommendations. Mechanical ventilation is usually performed in controlled assisted ventilation mode

Early detection is crucial, as the advancement of the block can be managed by adjusting the patient's positioning. In the event of total spinal anesthesia, placing the patient in a Trendelenburg position can enhance venous return and improve cardiac output. However, if the patient experiences a high spinal (as opposed to a total spinal), the Trendelenburg position is not advisable as it may exacerbate the block, potentially leading to a total spinal.

Sedation and mechanical ventilation should be sustained until signs of block regression emerge, indicated by sufficient spontaneous respiratory function and stable hemodynamic parameters. [16,17–18,19,20].

Specific strategies to anticipate and prevent severe bradycardia and cardiac arrest during spinal anesthesia are outlined in (Table 2) [7] :

*Table 2*  
*Management strategies for bradycardia and cardiac arrest*  
*during spinal anesthesia*

<p>Prevention:</p> <ol style="list-style-type: none"><li>1. Appropriate patient selection for spinal anesthesia when two or more risk factors are present (Table 1)</li><li>2. Maintaining adequate preload</li><li>3. Prompt replacement of fluid and blood loss.</li><li>4. Vigilant during patient positioning</li></ol> <p>Treatment of Bradycardia:</p> <ol style="list-style-type: none"><li>1. Mild to moderate bradycardia (HR 30-60/min)-stepwise escalation of therapy<ol style="list-style-type: none"><li>a. Inj Atropine 0.4-0.6mg, IV</li><li>b. Inj Ephedrine 25-50mg, IV</li><li>c. Inj Epinephrine 0.2-0.3mg, IV</li></ol></li><li>2. Severe bradycardia or cardiac arrest<ol style="list-style-type: none"><li>a. Advanced Cardiac Life Support guidelines to be followed</li><li>b. Early administration of epinephrine known to improve outcome</li></ol></li></ol> <p>Management of associated factors:</p> <ol style="list-style-type: none"><li>1. Rapid fluid infusion</li><li>2. Patient repositioning</li><li>3. Avoid surgical manipulation</li></ol>
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Precautionary measures should be taken during the administration of spinal anesthesia to prevent such dramatic occurrences: [9]

-Consider the level (and hence the dose of local anesthetic) required for surgery.

-Patient Position: Block height may be manipulated for up to 30 minutes with hyperbaric

-Patient Characteristics: In susceptible patients, such as those in obstetrics, with short stature, advanced age, or obesity, it is advisable to reduce the volume of local anesthetics injected to decrease the incidence of high neuraxial blockade [21].

-Technique: Take into account the effects of injection speed and Avoid excessive backflow.

-If performing spinal anesthesia after an epidural, dose reduction may be necessary based on the existing block level (reductions to 1-1.5 ml of local anesthetic have been suggested after failed epidural top-up); there is no clear consensus on this.

## **CONCLUSION:**

Although uncommon, the occurrence of total spinal anesthesia can have fatal consequences. It's crucial to implement precautionary measures during the administration of regional anesthesia for high-risk patients, prioritizing early identification and immediate intervention.

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