Cardiac Arrest Due to Severe Vasovagal Response After Spinal Anaesthesia in Caesarean Section: Case Report

CASE REPORT

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ABSTRACT

Introduction and importance: The vasovagal response is a reflex action that causes cardiovascular depression, bradycardia, and loss of consciousness. It is important to be aware of the risk factors for the vasovagal response and the pathophysiology that must be managed once it occurs.

Case presentation: A 28-year-old mother was admitted to Ambo University Hospital for an emergency cesarean section. She received spinal anesthesia with bupivacaine 12.5 milligrams isobaric, and after the T4 blockade was reached, surgery was initiated. A few seconds later, the patient's heart rate, consciousness, oxygen saturation, and blood pressure dropped then immediately cardiopulmonary resuscitation was started adrenaline and fluid was administered. After a few minutes, the patient's vital signs recovered. And with careful monitoring in the intensive care unit for 24 hours, the patient was discharged to the obstetrics department and discharged to her home 2 days later without leaving any specific sequelae. This case demonstrates the potentially serious risk of vasovagal damage with spinal anesthesia during cesarean section, particularly during placental expulsion.

Discussion: Vasovagal response secondary to spinal anesthesia and uterine retraction is a rare phenomenon, which results in a decrease in patient consciousness, heart rate oxygen saturation, blood pressure, and finally cardiac arrest. So it is important to know warning signs of the reflex and the early beginning of recitations.

Conclusion: The unanticipated vasovagal response is secondary to spinal anesthesia and uterine retraction can potentially cause severe consequences. Therefore, the lessons we learned from this case were careful history-taking and preoperative evaluation; proper management of preload, early recognition of vasovagal responses through careful monitoring, and timely treatment of hemodynamic collapse.

Highlight

- The cause of the vasovagal response may be uterine manipulation.
- Vasovagal reflexes can cause cardiac arrest.
- Spinal anesthesia sympathetic nervous system block can cause severe hypotension.

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INTRODUCTION

Vasovagal reflex denotes a reflex that causes cardiovascular depression that gives rise to loss of consciousness with bradycardia and extreme vasodilation. This response commonly occurs during regional anesthesia [1]. There are many causes for vasovagal reflex-like previous history of vasovagal syncope, anxiety, pain, hemorrhage, vena cava compression in pregnancy, uterine manipulation by a gynecologist, and regional anesthesia [2]. Changes in circulatory response are from dysregulation of the sympathetic-parasympathetic autonomic nervous system [3]. When vasovagal reflex happens ephedrine is the drug of the first choice because of its combined action on the heart and peripheral blood vessels. Epinephrine must be used early in established cardiac arrest, especially after high regional anesthesia. This case demonstrates the serious potential risk of a vasovagal response superimposed on nerve axis anesthesia during cesarean section, particularly during placental expulsion [4].

CASE PRESENTATION

A 28-year-old pregnant mother with 74 kilograms of body weight & height of 147 centimeters, previously healthy was admitted to the operation theater for an emergency Cesarean section due to Cephalopelvic Disproportion secondary to macrosomia plus obstructed labor during pre-anesthetic evaluation, it was noted the patient had no medical history, no known allergies, and systemic infection. Physical examination of the airway, respiratory and cardiovascular systems were normal. Laboratory result of hematocrit: 32.3, hemoglobin: 10.8, Platelet count of 288* 10^3. Informed consent was obtained from the patient after the procedure was explained and the patient was advised about the advantages and disadvantages of the procedure. All available monitors pulse oximetry, Electrocardiography, and non-invasive blood pressure monitoring were applied, and vital signs were 109 bpm, normal ECG trace, and 129/70 mm Hg, respectively. Preloading with normal saline of 500 ml was done before spinal anesthesia decision was made to administer single-shot spinal anesthesia. Following a strict aseptic technique cleaning the site of the procedure with Iodine and alcohol, a single attempt at L3–L4 with Tuohy # 23 gauge needle lumbar punctures was performed by bachelor degree anesthetist. The subarachnoid space was identified by the free flow of cerebrospinal fluid from the needle and checked by aspirating the flow of cerebrospinal fluid.

A total dose of 2.5 cc bupivacaine of 0.5% (12.5 mg) was administered with aspiration and slow injection. A T4 sensory level was achieved, loss of sensation was checked by pinprick, motor block was also confirmed by in ability to lift her legs, and the cesarean section began by gynecology. An additional IV line was opened with 18 gauge IV cannula and IV fluid added. A 4 kg baby was born 4 min later with an Apgar score of 1min that was 7. Immediately after baby was out, patient developed darkening of blood and the monitor then indicated the following vital sign: BP 45/29. Immediately became pulseless, unconsciousness. Cardiac arrest was evidenced with no pulse or breathing. The patient was intubated immediately after administering succinylcholine 100mg for muscle relaxation. CPR was started with endotracheal ventilation and adrenaline 1mg was given as I v bolus. CPR continued while the gynecology stabilized hemostasis and controlled bleeding. A bolus of lactated Ringer's and normal saline solution was given in fast drip and increased FiO2 and the pulse was restored immediately with 159 beat minute rates (***Table 1***). We tried to stabilize the patient during the procedure, all vital signs became normal and the procedure finished, the patient was extubated with saturation of 96. After stabilization, the patient was transferred to the intensive care unit for post-cardiac arrest care and follow-up. After a 1 day stay in the ICU, the patient was discharged to the ward with all stable vital signs and discharged to her home 2 days later without leaving any specific sequelae. This case has been reported according to Scare Guideline 2020 [5].

	LABOR WARD	OR	DURING SA	1 MIN AFTER SA	ARREST	1 MIN	2 MIN	3 MIN	4 MIN	5 MIN	AICU
HR	109	90	95	98	Asystole	CPR	CPR	159	145	150	140
SBP	129	128	130	100	45	55	65	85	110	130	125
DBP	70	72	79	65	29	37	46	50	65	75	70
SPO_2	NA	95	94	95	NA	NA	NA	NA	75	88	95

Table 1 Perioperative vital signs of our case.

HR: heart rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, OR: operating room, SA: spinal anesthesia, SpO2: oxygen saturation by pulse oximetry, AICU: Adult intensive care unit, CPR: cardiopulmonary resuscitation, NA: not available.

DISCUSSION

The vasovagal response is simply a reflex, creating it difficult to predict; hence, difficult to prevent. The hemodynamic collapse throughout a severe vasovagal response takes place in an instant; in the present case, the cardiopulmonary arrest occurred in lower than 5–10 seconds [5]. In our patient hemodynamic collapse and asystole happened less than ten seconds at once when the baby was delivered.

The differential diagnosis for this scenario could be high spinal anesthesia; spinal anesthesia induces sympathetic loss associated with a vasovagal reflex. The vasovagal response could cause cardiac slowing and arteriolar dilation. Vasovagal responses reflect autonomic neural changes: bradycardia is the result of a sudden increase in efferent vagal activity, and hypotension is the result of a sudden reduction or cessation of sympathetic activity and relaxation of arterial resistance vessels. Two different neural pathways are thought to be involved, one originating from the hypothalamus, the other from the heart. Direct activation of the hypothalamus of spinal cardiovascular centers triggered by emotional distress or pain elicits a vasovagal (central-like) response [2, 3, 6]. A vagal response can be triggered by an external stimulus (eg, venipuncture or visual input). An alternative mechanism is stimulation of the Bezold-Jarisch reflex, in which low ventricular volumes cause high intraventricular pressures caused by the contraction of the empty ventricle against its own (***Figure 1***). This reflex causes parasympathetic activation and sympathetic abstinence resulting in hypotension and bradycardia. The presence of sympathetic abstinence is supported by the fact that administration of atropine can prevent bradycardia but not hypotension [7].

The mixture of a reduced central blood volume secondary to blood vessel pooling or blood loss, associated with a multiplied inotropic state of the center, might stimulate cavity mechanoreceptors and provoke vasodilatation and cardiac arrhythmia (peripheral type). Vessel afferents originating from stretch receptors in numerous components of the vascular tree typically induce opposite reflexes in comparison with those from the ventricular sensory. The depressor reflex concerned within the peripheral variety of vasovagal response originates in the heart itself and overrides traditional baroreflex circulatory management; an antagonism between the control of volume and pressure on the filling aspect of the heart and also the system of blood pressure becomes apparent. Vasovagal responses don't seem to be essentially abnormal; the neural pathways concerned within the vasovagal response are a most likely gift all told healthy subjects who severally chiefly dissent invulnerability [3, 8–10]. Neuraxial physiological conditions for abdominal delivery might activate a vasovagal response, because of many risk factors; high spinal block, fast hemorrhage, aortocaval compression, serosa manipulation, and emotional stress [5].

LEARNING POINTS

First, a careful history of fainting or syncope and adequate preoperative assessments, such as the tilt-up test, would have reduced the risk of an unprepared vasovagal reaction. Second, careful and slow induction of spinal anesthesia would have reduced the risk of high neuraxial anaesthesia. Third, maintaining adequate preload with sufficient hydration, prior to neuraxial anesthesia, and expulsion of the placenta would have prevented a sudden decrease in venous return. Fourth, intravenous administration of ephedrine, rather than phenylephrine, after neuraxial anesthesia-induced hypotension would have prevented severe reflex bradycardia by stimulating both the alpha and beta-adrenergic receptor systems.

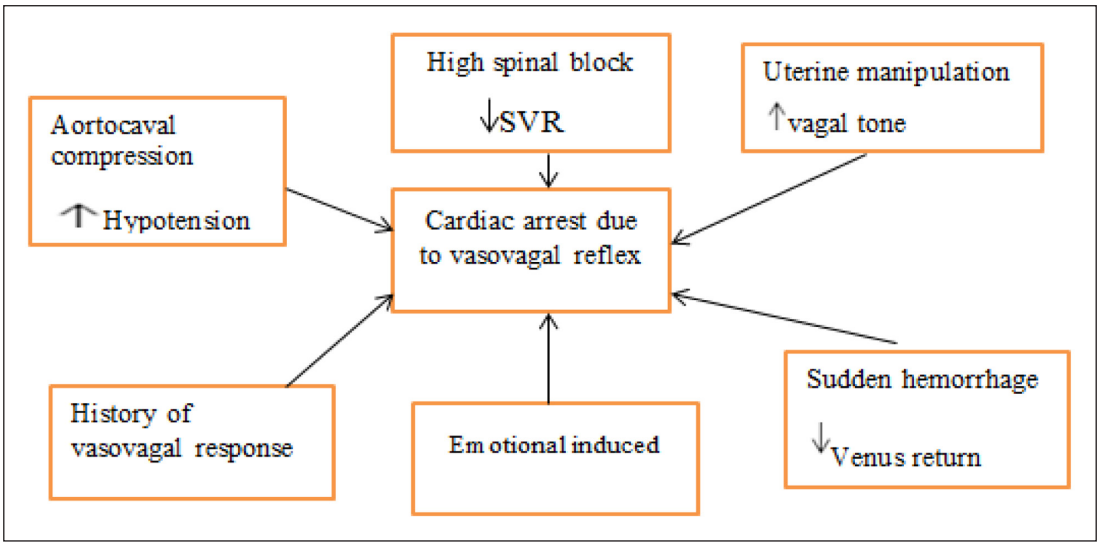

Figure 1 Risk factors for cardiac arrest due to vasovagal reflex during perioperative anaesthesia management of caesarean section under spinal anaesthesia.

Finally, appropriate intraoperative sedation would have reduced the patient's anxiety and the possibility of a vasovagal response. The vasovagal response is only a reflex, which makes prediction difficult; therefore difficult to prevent. Hemodynamic collapse during a severe vasovagal response occurs in an instant; in this case, asystole occurred in less than 10 seconds. Without constant, careful attention and proper preparation, early detection and treatment are unlikely to be successful; this case illustrates a significant potential risk of vasovagal response superimposed on neuraxial anesthesia, throughout a Cesarean section. Vasovagal response ought to be suspected, among the numerous potential causes of cardiopulmonary arrest during a Cesarean section, particularly once cardiac arrest happens during placental expulsion. Careful history taking and surgical evaluation, acceptable preload management, early recognition of vasovagal response by alert monitoring, and prompt treatment of hemodynamic collapse, were vital lessons we have got learned during this case [4, 6–8].

IN CONCLUSION

Vasovagal response causes hypotension, bradycardia, asystole, unconsciousness, so it is important to know the risk factors for vasovagal reflex and pathophysiology so that we can prevent vasovagal response and manage it once it happens to have close monitoring of our patient. The important lessons we learned from this case were careful history taking and preoperative evaluation; proper management of preload, early recognition of vasovagal responses through careful monitoring, and timely treatment of hemodynamic collapse.

RESEARCH REGISTRATION

1. Name of the registry: *Researchregistry.com*
2. Unique identifying number or registration ID: research registry 7316

GUARANTOR

Zenebe Bekele

ETHICS AND CONSENT

In our institution's research, case reports were ethically exempt from consent in cases where the patient consented or warranted.

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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COMPETING INTERESTS

The authors have no competing interests to declare.

AUTHOR CONTRIBUTIONS

ZB and FA contribute equally in Conceptualization, Validation, Data Curation, and Writing – Original Draft, Writing – Review & Editing.

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