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Case report

IMPENDING CARDIAC ARREST DURING SPINAL ANESTHESIA: REPORT OF A CASE WITH SUCCESSFUL RESUSCITATION

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Abstract Cardiac arrest during spinal anesthesia is dangerous. The incidence is rare and unpredictable. Early diagnosis and prompt treatment are very important. The author reports a case of young pregnant woman who developed severe bradycardia and signs of cerebral ischemia during cesarean section under spinal anesthesia and successful resuscitation. **Chiang Mai Med Bull 2006;45(2):78-91.**

Keywords: Cardiac arrest, spinal anesthesia

Spinal anesthesia was first introduced in 1899 and has gained in popularity since 1905.[1] The ease and long history of spinal anesthesia give the impression that it is a simple technique with little sophistication.[2] Hypotension and bradycardia are common cardiovascular complications of spinal anesthesia.[3,4] Cardiac arrest is serious but infrequently reported in previous literature (Table1).[5-9] We reported a case of impending cardiac arrest during spinal anesthesia for cesarean section.

Case report

A 30-year-old Thai term pregnant woman, with diagnosed pregnancy induced hypertension (PIH), was scheduled for cesarean section under spinal anesthesia. Her preoperative blood pressure was 160/90 mmHg, heart rate 89 beats/minutes and oxygen saturation 98%. Spinal anesthesia was performed with a 27-gauge Quincke-type needle at the L3-4 interspace, with a rapid preload volume of 800 ml of Lactate Ringer Solution. After that, 10 mg of 0.5% hyperbaric bupivacaine was administered slowly to the subarachnoid space. Then, the patient was turned into the supine position with the left uterine tilted. The upper level of the sensory block to cold temperature was T4. No sedative was given. The blood pressure gradually decreased to 110/80 mmHg during the first 20 minutes, while the intravenous fluid was still given rapidly.

The baby was born at 20 minutes after the onset of spinal anesthesia. Immediately after

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Table 1. Report of incidence and conclusion of severe bradycardia and cardiac arrest during spinal anesthesia.

Authors	Year	Number of cases	Event	Conclusion
Caplan RA, et al.[5]	1988	14	cardiac arrest	Excessive sedation and unrecognized hypoxemia might be the causes of cardiac arrest.
Mackey DC, et al.[6]	1989	3	cardiac arrest bradycardia	Bezold-Jarisch reflex induced acute bradycardia or asystole. Preexisting autonomic dysfunction might increase the incidence and/or severity of this reflex response.
Carpenter RL., et al.[7]	1992	123	bradycardia	Blockade of sympathetic cardiac accelerator fibers and decreased venous return to the heart were the causes of bradycardia; and a baseline heart rate < 60 beats/min, beta-blockade and ASA physical status I were the strongest predictors for bradycardia during spinal anesthesia .
Lovstad RZ, et al.[8]	2000	5	cardiac arrest bradycardia	The events were not related to respiratory depression, hypoxemia, hypercarbia, or the level of sensory block and they might occurr at different time intervals after the onset of spinal anesthesia (10-70 min).
Pollard JB.(9)	2001	26	cardiac arrest	Three reflexes could cause severe radycardia after a decrease in preload: 1) pacemaker stretch, 2) firing of low-pressure baroreceptors in the right atrium and vena cava, and 3) paradoxical Bezold-Jarisch reflex.

the child birth, the patient complained of chest discomfort, despite adequate spontaneous breathing. This was followed immediately by spastic contraction of both upper extremities, persistent upward movement of both eyeballs, and loss of consciousness, The heart rate of 45 beats/min was shown on an electrocardiogram. Control ventilation via a face mask was performed. Intravenous atropine at 0.6 mg was immediately injected. The blood pressure was 50/30 mmHg. The patient was then put into the Trendelenburg position and 100 μg of Epinephrine was injected intravenously.

The patient regained consciousness in one minute and could perform a tight handgrip on command. Her heart rate increased to 120 beats/min and blood pressure rose to 130/80 mmHg. Later on, the patient developed occasional mild hypotension that responded to intermittent intravenous injections of 3 doses of ephedrine at 6 mg. Infusion of 500 mL of colloid solution stabilized the patient's condition before the operation was finished.

The baby weighed 3,190 gm. The amount of amniotic fluid was about 1,000 mL, and estimated blood loss was 300 mL.

Rechecked Anesthetic level was at T2 dermatome bilaterally. The patient was admitted to the Intensive Care Unit postoperatively for one night, during which time her blood pres-

sure rose to the preoperative level. The patient's postoperative course was uneventful and she was discharged from the hospital in good condition.

Discussion

The cardioinhibitory receptors of the Bezold -Jarisch reflex may be responsible for bradycardia during spinal anesthesia. The receptors in the walls of ventricles are both machanoreceptive (pressure, inotropism, volume) and chemosensitive (veratrum alkaloids, adenosine tri-phosphate venoms from snakes etc.). Effects of the reflex are bradycardia, peripheral vasodilatation and hypotension.[10] (Fig.1)

Spinal anesthesia causes peripheral vasodilatation, and decreased venous return and volume in the ventricles. Activation of Bezold-Jarisch reflex predominates in the cases of inadequate preload volume. Risk factors for bradycardia and cardiac arrest during spinal anesthesia are sensory block at the level above the T6 dermatome, which includes cardiac accelerator fibers; high vagal tone such as that in a young age group (< 50 years); ASA physical status class I; and a baseline heart rate < 60 beats/min. The patients treated with beta-blocking drugs have a prolonged PR interval on an electrocardiogram.[6,7,9]

These risk factors might occur at different time intervals after the onset of spinal anesthesia (10-70 min), but resuscitation can be easily performed with the prompt administration of atropine and ephedrine.[8] Outcomes are usually good, but delays in instituting corrective treatment and resuscitation may cause permanent cerebral damage or death.[11]

The associated signs and symptoms of cardiac arrest during spinal anesthesia, authorized and publicized by the Royal College of Anesthesiologists of Thailand, are as follows: 1. Seizures, 2. Spastic contraction of the upper extremities, 3. Persistent upward movement of the eyeballs, 4. Bradycardia, and 5. Hypotension non-responsive to vasopressor drugs.

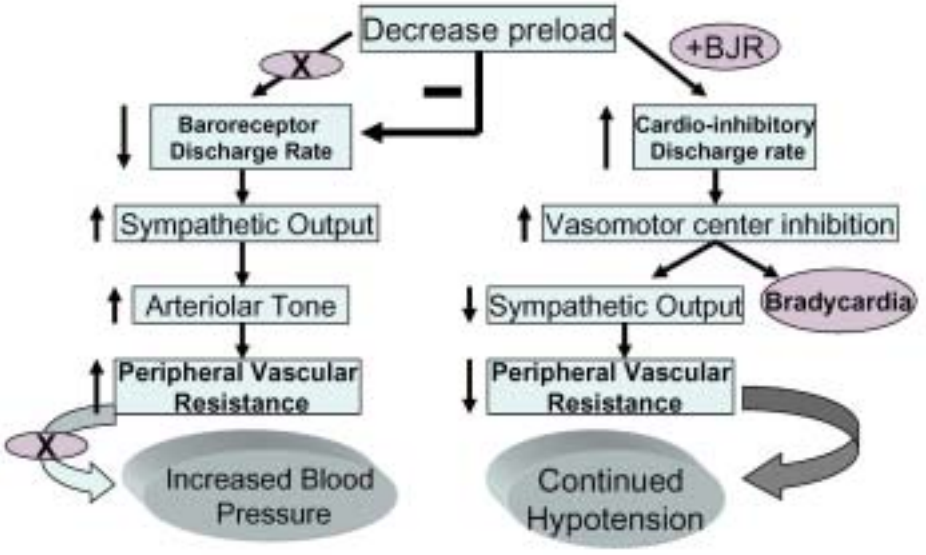

Figure 1. Activation of the Bezold-Jarisch reflex (BJR)

The preoperative heart rate in this case was 89 beats/min, and in the normal physiology of pregnancy, the parasympathetic tone might be decreased. Hypovolemia in pregnancy induced by hypertension might aggravate the decrease in venous return to the heart at childbirth, and the preload volume of 800 mL would be considered inadequate. Sudden decrease in intra-abdominal pressure at the birth of a big baby and 1,000 mL of amniotic fluid loss, might reduce systemic vascular resistance greatly. In this case, Bezold-Jarisch reflex was activated and the heart rate decreased to 45 beats/min.

The chest discomfort could have been a symptom of myocardial ischemia, which can occur for a few seconds before the signs of cerebral ischemia such as seizure, spastic contraction, upward movement of eyes and unconsciousness. Hypotension was severe and the onset rapid, so our patient did not have any signs of mild hypotension like nausea or vomiting before loss of consciousness.

The head down position to rapidly increase venous return combined with intravenous administration of atropine and adrenaline should be performed to stop the reflex before cardiac arrest occurs. Cerebral and myocardial ischemia would be irreversible if the treatment is delayed.

Conclusion

Cardiac arrest during spinal anesthesia is an emergency situation that can cause morbidity and mortality in young healthy patients who are unexpectedly under risk. Electrocardiogram, Non Invasive Blood Pressure Monitoring and Oxygen saturation should be monitored for early detection of an abnormality. Resuscitating drugs such as atropine and epinephrine should not only be available, but also used promptly to treat this life-threatening condition.

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การแก้ไขภาวะหัวใจเต้นช้าขั้นรุนแรงจากการฉีดยาชาเข้าไขสันหลังก่อนเกิดภาวะหัวใจหยุดเต้น

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เรื่องย่อ ภาวะหัวใจหยุดเต้นเป็นภาวะแทรกซ้อนของการฉีดยาชาเข้าไขสันหลัง ที่มีอุบัติการณ์ต่ำ คาดเดาล่วงหน้าได้ยาก และอันตรายมากที่สุด การวินิจฉัยที่รวดเร็วและการเตรียมพร้อมเป็นสิ่งสำคัญที่จะช่วยให้การรักษาประสบความสำเร็จ ผู้รายงานได้รายงานผู้ป่วย 1 ราย ซึ่งเกิดภาวะหัวใจเต้นช้าและมีอาการแสดงของสมองขาดเลือด ซึ่งเป็นอาการนำของภาวะหัวใจหยุดเต้น ขณะผ่าตัดคลอดโดยได้รับการฉีดยาชาเข้าไขสันหลัง และได้รับการรักษาจนกลับมาเป็นปกติ ก่อนเกิดภาวะหัวใจหยุดเต้น **เชียงใหม่เวชสาร 2549;45(2):87-91.**

คำสำคัญ: ภาวะหัวใจหยุดเต้น การฉีดยาชาเข้าไขสันหลัง