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CLINICAL INFORMATION

Hypotension and bradycardia before spinal anesthesia



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KEYWORDS

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Abstract I report a case of hypotension and bradycardia before spinal anesthesia in a pregnant woman with mild to moderate hypertension treated with nifedipine and methyl dopa, scheduled for an elective cesarean delivery. She had the history of neurally-mediated syncope. Two main factors (increased vagal tone and adverse effects of antihypertensive drugs) could explain the hypotension and bradycardia before spinal anesthesia. Monitoring allowed recognizing the problem and corrected it. Thus, it was avoided a disaster in anesthesia, as hemodynamic changes after spinal anesthesia, they would have joined to previous hypotension and bradycardia, which would have caused even a cardiac arrest.

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PALAVRAS-CHAVE

Hipotensão;
Bradicardia;
Tônus vagal;
Medicamentos
anti-hipertensivos

Hipotensão e bradicardia antes da raquianestesia

Resumo Relato de um caso de hipotensão e bradicardia antes da raquianestesia em uma mulher grávida com hipertensão leve a moderada tratada com nifedipina e metildopa, programada para parto cesáreo eletivo. A paciente apresentava história de síncope neuralmente mediadas. Dois fatores principais (aumento do tônus vagal e efeitos adversos de medicamentos anti-hipertensivos) poderiam explicar a hipotensão e bradicardia antes da raquianestesia. O monitoramento permitiu reconhecer o problema e corrigi-lo. Assim, foi evitado um desastre em anestesia; assim como as alterações hemodinâmicas após a raquianestesia, esses fatores teriam se juntado à hipotensão e bradicardia anterior, o que poderia ter causado inclusive uma parada cardíaca.

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Introduction

A wide range of antihypertensive drugs have been proposed to decrease the blood pressure in hypertensive pregnant women, and each of them has different side effects and potential adverse effects. There is no consensus about the definition of hypertensive pregnancy disorders and several classifications have been suggested. Variations in the systems for classification are largely according to which values of blood pressure are considered abnormal. Nevertheless, there is currently an agreement regarding the existence of four categories which include gestational hypertension or pregnancy-induced hypertension.¹

A case of hypotension and bradycardia before spinal anesthesia in a pregnant woman with mild to moderate hypertension, scheduled for an elective cesarean delivery, is reported with the patient's and the Hospital Ethics Committee's written consent.

Case report

A 34-year-old patient was admitted four days before surgery, with a gestational age of 37 weeks by date of last menstruation and pregnancy-induced hypertension. She had two previous Caesarian sections (in 2007 and 2011) which were performed with spinal anesthesia without complications. She was suffering from intermittent bronchial asthma, with a final episode in the year 2013; and reported some episodes of vasovagal and orthostatic syncopes in different times of her life. Her weight and height were 98.8 kg and 1.65 m, respectively. Her physical condition was grade 2 according to American Society Anesthesiologists physical status classification; she was assigned to class 2 of Goldman index of cardiac risk and had a normal electrocardiogram. The laboratory analyses showed a hemogram with normal values (hematocrit 41%, platelets 237,000 mm³); the values of glucose, creatinine, urea, and aminotransferases were normal too. The value of proteins in a 24 h urine was 126 mg (3000 mL of urinary volume).

When the patient was hospitalized, four days before the surgery, her blood pressure was 140/80 mmHg and the heart rate (HR) was 79 beats per minute (bpm). An antihypertensive treatment of 10 mg of nifedipine was administered orally t.i.d. A day later was added methyl dopa 500 mg orally t.i.d. She swallowed food until 5 p.m. and received the last dose of antihypertensive drugs up to 22 h of the previous day to the cesarean section.

The day of surgery the patient did not receive antihypertensive treatment, and the last control of vital signs at 7 a.m. was registered as normal in the hospitalization area. Furthermore, she received a bolus of physiological saline serum (500 mL). None of the previous controls of blood pressure was lesser than 110/70 mmHg until two days before the cesarean section. However the day before, she registered a blood pressure of 90/60 mmHg at 1 p.m.

When the patient entered the operating room at 9:40 a.m., she was lucid and oriented; nevertheless, paleness and anxiety were observed. A non-invasive blood pressure (NIBP) of 73/35 mmHg was measured in the multi-parameters monitor, HR of 37 bpm and a breathing rate of 18 breaths per

minute. The results were corroborated by manual measurements. The patient was asked if she had taken some medicine which she denied.

The patient received an infusion of 500 mL of isotonic saline solution; next a NIBP of 80/42 mmHg was measured. After she was given 1000 mL, her NIBP was 92/64 mmHg. Finally, she gave a volume of 1500 mL and her blood pressure rose to 110/60 mmHg. Atropine (0.5 mg) was given intravenously, which made her HR go up to 82 bpm. At 10 am, I proceeded to administer spinal anesthesia containing 10 mg of hyperbaric Bupivacaine and 10 µg of fentanyl, at the level of the L3–L4 space with needle type Quincke no. 26. We placed the patient in dorsal decubitus with 15° of left inclination from the surgical table. Fifteen minutes after, the surgery started with a NIBP of 90/42 mmHg and HR of 114 bpm. Ten minutes after there was the birth of the fetus with good Apgar scores; in this time it was controlled a NIBP of 79/40 mmHg, so I started intravenous infusion of 3 mg of Etilefrine and two bolus of 1 mg of the same drug with that the NIBP rose to 130/60 mmHg.

No other problems occurred in the remaining period of the surgery. The patient left at 11:05 with isotonic saline solution (total infused volume of 2500 mL) and 20 IU of Oxytocin infused intravenously, without any kind of vasopressor, NIBP of 114/64 mmHg, HR 99 bpm, oxygen saturation on room air 96% and breathing rate of 16 bpm.

Discussion

When I investigated the causes of hypotension and bradycardia at the entrance to the operating room, anaphylaxis was discarded first, because it has not had provided any medication before operating room; then, was considered an excess vagal because she had had some episodes of transient loss of consciousness tone, even though she was aware when she arrived to the operating room. Nonetheless, there are some interesting points about the adverse effects of antihypertensive drugs, too.

Nifedipine, as calcium antagonist antihypertensive, has a short terminal half-life of 1.9 h for a 10 mg tablet, so multiple doses are required to achieve the effective concentration (78 µg/Lt.); being the minimum concentration to decrease diastolic pressure of 10–15 µg/Lt.² In relation to orthostatic syncopes, it has been described four causes of compensatory failure. One of them is the attenuation of vasoconstrictor response to sympathetic stimulation; this attenuated response is primarily caused by the administration of vasodilative agents among which are calcium antagonists as nifedipine.³

Methyl dopa (α -methyl-3,4-dihydroxy-L-phenylalanine), analog of 3,4-dihydroxyphenylalanine (DOPA) discovered in 1960 by Oates,⁴ is a prodrug of central action that acts by the active metabolite α -methylnorepinephrine (which replaces norepinephrine in the secretory vesicles of adrenergic neurons). It causes a decrease of the blood pressure which is a maximum of 6–8 h after an oral or intravenous dose, and mitigates but does not block completely baroreceptors-mediated vasoconstriction. For this reason, is tolerated well during surgical anesthesia and any severe hypotension is reversible with volume expansion, as it happened with the management of this pregnant woman

inside the operating room. Although the maximum plasma concentrations occur after 2–3 h, the maximum effect is delayed 6–8 h, and a single dose usually lasts almost 24 h, which allows providing a dosage one to two times per day.^{5–7} Therefore, in this case, methyldopa should not have been supplied three times per day and could have begun with one lower dose; even more considering that she was already receiving nifedipine.

Among the most common adverse effects from methyldopa include fatigue, xerostomia, decreased libido, parkinsonian signs, hyperprolactinemia, hepatotoxicity, intense bradycardia and sinoatrial asystole, sedation and even depression in higher doses.^{8,9} Hypotension has also been reported as frequent adverse effect, especially in younger patients, lighter subjects, patients with impairment of renal function or in those taking high daily doses.¹⁰ However, it has a safety profile in the treatment of gestational hypertension, currently reaching to constitute the drug of choice for the treatment of non-severe hypertension in this population, used in different regions around the world.^{5,11–13}

In conclusion, two main factors can be observed in this case, in one side a history of increased vagal tone of the patient and in the other, the adverse effects of previous medical therapy. Both of them affected the planned anesthetic scheme for an elective surgery. Monitoring had an important role because it allowed recognizing the problem and corrected it. Thus, it was avoided a disaster in anesthesia, as hemodynamic changes that occur after a spinal anesthesia in a pregnant woman, they would have joined to previous hypotension and bradycardia, which probably would have caused even a cardiac arrest.

Conflicts of interest

The author declares no conflicts of interest.

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