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Bradycardia, low blood pressure, and spinal anesthesia

Case report

We report the case of a 34-year old woman undergoing the resection of a right foot arthrosynovial cyst under spinal anesthesia. Her medical history consisted of morbid obesity (140kg, 168cm, BMI 50), active smoking (3.5 UAP), treated and stable chronic high blood pressure, hypothyroidism, and depression. Ongoing medications included combined antihypertensive medications (linisopril associated with hydrochlorothiazide), nebivolol, L-thyroxine, escitalopram, and zolpidem. She already had two interventions under locoregional anesthesia, namely a cesarean section, and a right carpal tunnel intervention. Moreover, clinical examination revealed the presence of difficult airway management criteria, including small mouth opening, and reduced neck mobility.

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On the day of surgery, she received 0.5 mg alprazolam in addition to her usual treatment except for her combined antihypertensive medication. At 8 am, upon arrival in the operating theatre, the patient was placed in the sitting position, and monitored with pulse oximetry, 3 lead electrocardiogram, and non-invasive blood pressure monitoring. A peripheral 18 gauge intravenous catheter was placed in her forearm, and used to deliver 500 milliliters of crystalloids. Spinal anesthesia was performed at the L3-L4 intervertebral level, using a 25 gauge withacre needle. We injected 2.2 milliliters of 0.5 % hyperbaric bupivacaine. Subsequently, the patient was placed on her right side for 10 minutes. Only 10 minutes after the intrathecal injection, the blood pressure dropped to 78 / 52 mmHg, and measured heart rate was 70 bpm. The patient was then positioned in the dorsal decubitus position, and fitted with a 40 % Venturi mask. After obtaining a T6-T7 sensory block, at 8:29, surgery could begin. At 8:53, surgery was completed, our patient transferred to bed with a slight head elevation of 30 °. Before transfer, blood pressure was 100 / 37, heart rate 53 bpm, and oxygen saturation level 99 %.

Upon leaving the operating room, our patient signaled discomfort without nausea, and rapidly lost consciousness. She was quickly pushed back in the operating room and given oxygen. A dose of 10 mg of ephedrine was instantly administered, without monitoring. First pulse oxymeter measured heart rate showed 30 bpm, which led to the subsequent administration of 0.5 mg of atropine and another 10 mg ephedrine. Within a few seconds, the patient regained consciousness, a heart rate of 60 bpm, and blood pressure could finally be measured at 142/85 mmHg.

Discussion

During spinal anesthesia, sympathetic blockade is the first event to occur, and the last to disappear. This blockade causes hemodynamic instability, such as hypotension and delayed

bradycardia, which is critical to prevent and recognize early, in order to avoid dramatic consequences such as cardiac arrest. Sympathetic block generally extends 2-6 dermatomes above the sensorial blockade (1-2).

The hemodynamic consequences mainly result from sympathetic blockade on venous territories. It triggers the distension of the capacitive system, and storage of blood in dependent parts, resulting in decreased venous return and a reduction of cardiac output. If the sensitive block is below T10, there are little variations of systemic vascular resistances and, therefore, few hemodynamic consequence (3-4). When the level is higher or equal to T6, blocking splanchnic nerves leads to pooling of blood in the hepatosplanchnic venous areas, with a significantly higher decrease in blood volume. In addition, delayed emergence of splanchnic vasoconstriction may cause secondary hypotension (1-3-5).

At the cardiac level, the cephalic extension of the block leads to the blockade of the sympathetic cardioaccelerator fibers from T4 to T1, resulting in a progressive decrease in heart rate, due to the predominance of the parasympathetic nervous system. Bradycardia may also occur for lower block levels, for example, in a sedated patient. It can also occur in an awake patient, because of the significant decrease in heart preload (5). This can trigger different reflexes that result in severe bradycardia or asystole (2, 5). One of these reflexes is triggered by mechanoreceptors located in the left ventricule. Those receptors are activated by a rapid decrease in ventricular volume and feedback occurs through the vagus nerve, which triggers a vasodepressor response by increasing the activity of the parasympathetic system. As a consequence, systemic vasodilatation, hypotension, and bradycardia occurs. This reflex is called the Bezold-Jarisch reflex, or vasovagal syncope, or neurocardiogenic syncope. It can be triggered centrally by psychological stress, pain, or changes in position, but can also be peripherally initiated by a decrease in venous return (2-6).

It should be emphasized that about 7 % of the population are known as "vagotonic". In these individuals, the activity of the sympathetic and parasympathetic nervous systems shows an imbalance, in favor of the latter one. These patients are usually young healthy men (2-7).

To prevent the potentially disastrous consequences of spinal anesthesia, it is necessary to recognize the predictive factors of these complications. Indeed, the occurrence of bradycardia is increased in case of sensory block cephalic extension (sensory block > T5), in young « vagotonic » individuals, in patients below the age of 50, in the presence of an atrioventricular block, baseline heart rate below 60 bpm, or in case of ongoing beta-blocker therapy. Recommendations suggest that, if two of these factors are present, patients are at high risk of developing bradycardia and / or cardiac arrest during spinal anesthesia (2-7). The occurrence of severe hypotension depends on the cephalic extension of the block, which depends on dose, volume, density, and type of injected local anesthetic agent, age , elevated abdominal pressure (pregnancy, obesity), height, injection technique (speed of injection, direction of the bevel of the needle), injection site, and posture during and after injection (4-5). The latency of a block is due to the time required for the cephalic migration of the local anesthetic agent into the cerebrospinal fluid in an sufficient amount to generate a sensory block and the onset of action of the local anesthetic agent on the neural structure (8).

Frequently, we allow surgery to begin when an adequate level of sensory blockade is achieved, approximately 5 to 10 minutes after our injection. However, the block level achieved 10 minutes after injection does not correspond to the highest sensory blockade, which depends on the various factors discussed above. Several case reports described cardiac arrest occurring within 10 to 70 minutes after skin puncture and occurring a few seconds after a change of position. Cardiac arrest may also occur in the recovery room. The mean interval to develop cardiac arrest is 58 minutes after injection (7). The authors found that the absence of orthostatic hypotension monitored twice at 30-minute intervals was a better criterion for discharge from the postoperative recovery area than obtaining a sensory level below T10 associated with an active mobilization of the toes (9).

The development of hypotension and bradycardia during spinal block anesthesia can be prevented. Indeed, using more adequate anesthesia techniques reduces the extension of the sympathetic block. Unilateral spinal anesthesia, continuous spinal anesthesia, reduction of local anesthetic agent doses, addition of adjuvants in the solution, intravenous infusion of phenylephrine, or patient position adjustment are all preventive measures (4).

An initial volume preload with crystalloid or colloid does not effectively prevent hypotension caused by spinal anesthesia (1-5-7-8-11). However, to reduce the incidence and severity of hypotension, rapid fluid administration appears to be effective when given concomitantly to the spinal injection of local anesthetic agents (1-5). Nevertheless, volume loading may not be enough to reverse the vagal symptoms in response to a decreased preload. Therefore, in patients with multiple risk factors, it is often necessary to administer vasopressors concomitantly, in order to prevent cardiac arrest. The use of mixed sympathomimetic agents is most suitable in the management of hemodynamic changes. ephedrine, as an alpha and beta agonist agent, is effective in both increasing blood pressure and correcting bradycardia, which mostly arises from a decrease in venous return. However, during a vasovagal syncope or profound bradycardia, administration of atropine is often necessary. If bradycardia persists despite our best efforts, small boluses of adrenalin should be effective. Adrenalin, which is a more potent α/β agonist, increases coronary perfusion pressures and allows effective cardiopulmonary resuscitation if needed. Pollart et al. advise a stepwise treatment: atropine 0.4-0.6 mg, ephedrine 25-50 mg, and adrenaline 0.2-0.3 mg (2).

Our patient presented multiple risk factors of hypotension and bradycardia such as obesity, age below 50 years, and beta-blocker treatment. In addition, surgery took place quite quickly. Therefore, if preoperative blood volume had been successfully restored before injection, and effectively compensated preload changes during surgery, bradycardia could be related to position changes. Indeed, complications occurred a few seconds after transfer to her bed and a 30 ° head elevation. In this patient, hypotension and bradycardia may have been caused by severe hypotension due to decreased venous return, but also by a vasovagal syncope.

Conclusion

This case reminds us of the importance of identifying risks factors of bradycardia and hypotension before performing spinal anesthesia. The choice of the most suited anesthetic

technique could also be questioned, as well as best preventive strategies to avoid such consequences. Unfortunately, these complications cannot always be linked to a decreased cardiac output. Noteworthy, standard monitoring should be maintained until the patient reaches postoperative care unit and during careful mobilization of the patient. Finally, pain as well as anxiety should be properly treated.

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