Intramyometrial vasopressin as a haemostatic agent: Is it really safe?

INTRODUCTION

Vasopressin when given intramyometrially during myomectomy results in good surgical haemostasis.[1,2] At the same time, there are published reports of adverse cardiac events following vasopressin injection.[3,4] Resuscitation measures following such adverse cardiac events require understanding of pharmacodynamics of vasopressin administration.[5] We present a case of successful resuscitation of complete heart block

following intramyometrial vasopressin injection during myomectomy and the associated implications.

CASE REPORT

A 31 year old, 50 kg American Society of Anaesthesiologists (ASA) physical status-I female patient was posted for open myomectomy. In the operating room standard monitoring with 5-lead electrocardiogram, pulse oximetry, non-invasive blood pressure (NIBP) were attached. After securing intravenous access with 18G cannula, 250 ml of crystalloid was administered. The patient was placed in right lateral position, and subarachnoid block with 26G Quincke® needle was performed at L4-L5 interspace and 2.5 ml 0.5% heavy bupivacaine with 25 μg fentanyl (0.5 ml) was given. NIBP was measured every 3 min and no significant haemodynamic changes were noted following subarachnoid block. Peak cephalad level of sensory blockade (loss of temperature sensation with a alcohol swab) was T6 dermatome at 30 min. Thirty minutes following skin incision, 30 ml normal saline (NS) containing 4 units of vasopressin (20 units vasopressin diluted in 150 ml NS, 0.133 units/ml) was injected intramyometrially after negative aspiration of blood. Two minutes after vasopressin injection patient developed severe conjunctival pallor, hypotension (NIBP - 70/30 mmHg) and complete heart block (heart rate [HR] <40/min) with absent peripheral pulses. Three doses of injection atropine 0.6 mg (30 s between doses) administered intravenously and oxygen 6 L/min was supplemented through face mask.[6] HR and blood pressure were restored with palpable peripheral pulses. The total duration of the event was <2 min and throughout this period the patient remained conscious with palpable central pulses, normal respiration and oxygen saturation. Sensory blockade at this point remained at T6 level. Arterial blood gas analysis performed 15 min later showed no significant abnormality.

Since the patient remained haemodynamically stable, surgery was resumed and completed with no further complications. Post-operatively electrocardiography, cardiac enzymes, echocardiogram, and renal function tests were done and found to be normal. After an uneventful post-operative stay of 5 days, the patient was discharged.

DISCUSSION

One of the main considerations for the surgeon during resection of significantly vascular tumours like uterine myomas (fibroid) is excessive bleeding that may be even life-threatening. Various mechanical and pharmacological interventions are used to minimise blood loss during myomectomy. Vasopressin as a haemostatic agent has been in use for more than 50 years, and there are published reports supporting the use of vasopressin to reduce blood loss during myomectomy and hysterectomy.[1,2] At the same time, there are published reports of adverse cardiac events including cardiac arrest following vasopressin.[3,4]

Vasopressin is primarily an antidiuretic hormone (via V2 receptors) released in response to increases in plasma osmolarity or decreases in blood volume.[5] At higher concentrations, it acts on V1 receptors and produces generalised constriction of most blood vessels including coronary vasculature. It also acts on the brain (area postrema) to decrease cardiac output.[5] The effects of vasopressin on the heart include reduced cardiac output and HR resulting from coronary vasoconstriction, decreased coronary blood flow, altered sympathetic tone and potentiated baroreflex in response to generalised vasoconstriction. These cardiac effects are potentiated in patients with coronary insufficiency. Even though, vasopressin produces generalised vasoconstriction, clinically it will manifest as global hypotension with absent or low volume peripheral pulses.[4] Failure to identify this will lead to treatment with vasopressors that can worsen the cardiac complications.

Our patient also exhibited these undesirable physiological manifestations such as pallor, bradycardia and hypotension. Cutaneous vasoconstriction manifested as conjunctival pallor while the bradycardia possibly occurred due to potentiated baroreflex and altered sympathetic tone.[7] The cause of hypotension could be multifactorial; reduced cardiac output and HR, altered sympathetic tone, or it could be peripheral vasospasm mimicking hypotension in NIBP measurement.[4] The treatment includes cardiac life support measures in the event of cardiac arrest, oxygenation, atropine for bradycardia and absolute avoidance of vasopressors.[4,6,8] The short biological half-life of vasopressin (18 min) with early and correct resuscitation will result in successful outcome in these patients.[5]

The haemostatic property of vasopressin is well addressed in many studies, but the safe total dose and concentration is still controversial.[9] An

interesting review article by Frishman recommended intramyometrial dose of 4–6 U at a concentration 0.2 U/ml to be safe, based on assumption that low dose and low concentration is equally effective in achieving haemostasis without cardiac complications.[9] However, haemostatic and cardiac complications can occur via V1 mediated vasoconstriction at similar doses.[5] Hence, if vasopressin can cause haemostasis at a fixed dose, it can cause cardiac complications at the same dose and concentration. This was proved in our case where adverse cardiac event happened even when the vasopressin dose and concentration used was within the recommended limits. With no large-scale trials on intramyometrial dose and concentration available, the safe dose and concentration of vasopressin suggested in previous articles look arbitrary.

CONCLUSION

After reviewing the literature, we conclude that even though intramyometrial vasopressin has proven haemostatic property, there are increasing reports of adverse cardiac complications following its use. The safe dose and concentration of intramyometrial vasopressin is still controversial. Till conclusive studies on safety profile, dose and concentration on intramyometrial vasopressin are available, routine use of vasopressin as a haemostatic agent should be reconsidered.

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