

Case Report

Complete Heart Block After Administration of Intravenous Pethidine

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Abstract

Pethidine is used by anesthesiologists to provide pain relief during the perioperative period, labor, and trauma, but it is not frequently used nowadays because of the availability of more potent shorter-acting opioids. But even today it is commonly used by many to alleviate shivering in surgical patients. Because of these actions on the autonomic nervous system, pethidine may produce changes in the heart rate in combination with other medications or interventions. We encountered ill-sustained complete heart block in a patient who received 25 mg pethidine intravenously to alleviate shivering. No other treatment except 0.2 mg glycopyrrolate was required as the patient had hemodynamic stability.

Key words: Complete heart block, pethidine, shivering

INTRODUCTION

Pethidine exerts its analgesic effect via the μ receptors. It has structural similarities to atropine and other tropane alkaloids, and may have some of their effects and side effects. In addition to these opioidergic and anticholinergic effects, it has local anesthetic activity related to its interactions with sodium ion channels. Considering these pharmacological actions of pethidine, it is indeed surprising that its actions on the conduction system are rarely reported. This case is being reported because of paucity of such information in the literature.

CASE REPORT

A 59-year-old gentleman, weighing 58 kg, was scheduled to undergo a revision knee replacement. He was a diabetic on oral hypoglycemic agents and a well-controlled asthmatic. Pre-anesthetic evaluation and preoperative laboratory values were normal. The patient had undergone knee replacement under spinal anesthesia earlier and preferred to have the same for this procedure as well. The planned anesthetic technique was sciatic and femoral nerve block under ultrasound guidance for providing postoperative pain relief, followed by subarachnoid block (for intraoperative anesthesia). The patient was administered oral alprazolam 2 mg on the night prior to surgery as per the hospital

protocol. On the day of surgery, in the operation theater, electrocardiogram, noninvasive blood pressure, and pulse oximetry monitoring commenced. Under local anesthesia, using ultrasound guidance, 18 gauge femoral catheter was placed. Femoral and sciatic block was administered with 20 ml of 0.25% bupivacaine for each block. Subarachnoid block with 3 ml 0.5% bupivacaine heavy was administered using 27 gauge spinal needle. Onset and action of the subarachnoid block was satisfactory. Surgery was commenced after preparing the part and draping. During the procedure, there was no hypotension or bradycardia. After about an hour of surgery, the patient started shivering. Despite using warm blankets and having a normal core temperature, the patient continued to shiver. Twenty-five milligrams of pethidine was administered intravenously. Within a minute of its administration, the cardiac rhythm, which was hitherto normal sinus, changed to complete heart block [Figure 1]. An abrupt decrease in the blood pressure was also noticed. A bolus of 0.2 mg of glycopyrrolate (preloaded) was administered and this intervention converted the rhythm back to sinus in about 5 min. Episodic heart blocks occurred for short duration of time even after this; during the heart block,

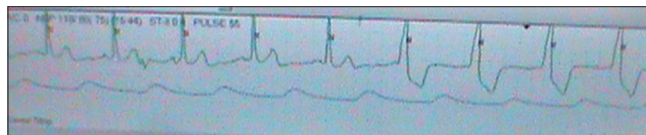


Figure 1: Figure showing the abrupt change of electrocardiogram from normal sinus rhythm to complete heart block.

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blood pressure decreased significantly. Normalcy was restored after about 30 min. No other treatment such as defibrillation or pacing was required to achieve hemodynamic stability. There were no arrhythmias after this. Rest of the procedure went off without events. The patient was transferred to the postanesthesia care unit after completion of the surgery. No further investigations were carried out, considering the fact that the patient was stable.

DISCUSSION

The use of pethidine to provide analgesia is declining because of the availability of superior analgesic agents such as fentanyl and remifentanyl. Despite this, several anesthesiologists, especially in our country, continue to use pethidine to provide perioperative analgesia and when the patient gets shivering. The anti-shivering effect of pethidine is believed to be caused by its agonistic “k” opioid receptor action.^[1] Because of the similarities to atropine and other tropane alkaloids in structure, its role in increasing the refractory period in rat heart has been implicated.^[2,3] Zhang and coworkers reported negative inotropic action with pethidine, but not with morphine.^[4] Complete heart block following administration of pethidine has not been reported in the literature. The response to the bolus of glycopyrrolate suggests sympathetic block mediated bradycardia/heart block. Such events might prove very costly in patients with pre-existing conduction defects. One could argue that the bradycardic effect experienced by this patient may have been the delayed manifestation of spinal anesthesia or due to the systemic toxic effect of the local anesthetic administered for sciatic and femoral block. This aspect could have been confirmed if serum levels of bupivacaine had been measured. We could not get that done

because of logistic reasons. The possibility of toxic dose of bupivacaine causing the heart block may not be ruled out considering that the total dose of bupivacaine administered had approached approximately 2 mg/kg. However, the short time gap between administration of pethidine and manifestation of heart block led us to believe that it could most probably be pethidine induced. The other probable reasons such as delayed manifestation of adverse effect due to spinal anesthesia and toxicity of bupivacaine also should be borne in mind. This report is being published to warn the users of pethidine to be aware of the possibility of heart block after its administration and to avoid its use in patients with pre-existing conduction defects, especially when administered concomitantly with local anesthetic agents.

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