Case report

Development of third-degree heart block due to thoracic epidural anaesthesia

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1. Introduction

Neuraxial anaesthesia can have profound effects on cardiovascular physiology. Severe bradycardia, asystole, and third-degree heart block have been documented following spinal anaesthesia [4,11–13]. Development of third-degree heart block can occur in patients receiving the combination of AV node blocking drugs, general anaesthesia, and epidural anaesthesia [7,15]. However, due to these confounding factors, the contribution of epidural anaesthesia itself cannot be determined. To our knowledge, the complication of third-degree heart block in a patient receiving thoracic epidural anaesthesia alone has not previously been reported. We present a case of an 82-year-old man with multiple rib fractures who developed third-degree heart block following initiation of thoracic epidural anaesthesia. Pretreatment with an anticholinergic medication or the use of thoracic paravertebral nerve blocks may decrease the risk of this rare but potentially life-threatening complication. The patient provided consent for the publication of this report.

2. Report

An 82-year-old man was admitted to the trauma ICU following a motor vehicle collision. His injuries consisted of right clavicular and scapular fractures, a right pulmonary contusion, and fractures of right-sided ribs #1 and #5–8. His past medical history included mild dementia, hypertension, hyperlipidemia, and first-degree heart block with a PR interval of 250 ms (Fig. 1) but no other known cardiac disease. The patient was not taking beta-blockers, calcium channel blockers, or other anti-arrhythmic or AV-node blocking medications.

On the day after admission the patient continued to experience right-sided chest pain on deep inspiration and was able to achieve only 800 ml on incentive spirometry. A thoracic epidural at the T7/T8 vertebral interspace was placed to provide analgesia. Preprocedure blood pressure was 126/84 mmHg and heart rate 100 beats/min with first-degree heart block. No sedation was given prior to the procedure. Following subcutaneous injection of 2 mL of 1% lidocaine, a 17 Ga Tuohy needle was inserted via a right paramedian approach and a 19 Ga epidural catheter was threaded easily into the T7/T8 epidural space without immediate complications. Aspiration was negative for blood and CSF and a 3 mL test dose of 1.5% lidocaine with 1:200,000 epinephrine was negative. The epidural was then dosed with 5 mL of 0.2% ropivacaine with 50 mcg of fentanyl through the catheter, however, continuous infusion of local anaesthetic/narcotic solution was not started. EKG monitoring, non-invasive blood pressure measurements, and pulse-oximetry were performed continuously during and after the procedure.

Hypotension following this first dose was treated with infusion of 500 mL of hetastarch solution and periodic boluses of intravenous phenylephrine (80–160 mcg boluses for a total of 480 mcg over a period of 30 min). Fifteen minutes after the initial epidural bolus, a T4–T8 sensory level was confirmed via pinprick. The patient reported decreased pain with inspiration and was able to achieve 1100 mL on incentive spirometry. Twenty-seven minutes following the initial bolus, the patient’s blood pressure was measured at 103/65 mmHg with a heart rate of 83 beats/min. Three minutes later, the patient experienced loss of consciousness and was noted to have developed third-degree heart block (Fig. 2) without palpable carotid or femoral arterial pulses. Third-degree heart block persisted, but after 20 s the patient developed a ventricular escape rhythm with a rate in the 30 s and spontaneously regained consciousness. Atropine 0.5 mg was then administered intravenously which resulted in development of a sinus rhythm with a rate in the 90 s. External pacemaker pads were placed on the patient prophylactically. An electrolyte panel and complete blood count sent after the event showed no significant abnormalities and the patient did not experience an increase in cardiac troponin levels. He was evaluated by the electrophysiology service and had a dual-chamber pacemaker successfully implanted the following day.

3. Discussion

In achieving the desired analgesic effect, epidural anaesthesia results in an obligatory sympathetic block. This occurs because the sympathetic nerves are most sensitive to the effects of local anaesthetics, due in part to their small diameter and lack of myelination. Blockade of these sympathetic fibres causes significant venodilation and contributes to hypotension with epidural...
anaesthesia regardless of the level of epidural catheter placement. During thoracic epidural anaesthesia, blockade of the T1–T4 cardioaccelerator fibres contributes to bradycardia via a reduction in sympathetic outflow to the sinus node. These same fibres modulate intracardiac conduction velocity through stimulation of $\beta_1$ receptors in the cardiac conduction system including the AV node. Blockade of these fibres during thoracic epidural anaesthesia has been shown to prolong AV node conduction time in dogs [9]. The patient exhibited a T4 sensory level on exam. This level of analgesia corresponds to blockade of the majority of the cardioaccelerator fibres as sympathectomy has been shown to occur two to three dermatomes above the level of anaesthesia to pinprick [5,8]. Additionally, because the patient’s T4 sensory level was confirmed only 15 min after the initial bolus of ropivacaine, continued cranial spread of local anaesthetic effect and sympathectomy likely occurred. By 30 min following the initial dose of epidural solution, sufficient blockade of the cardioaccelerator fibres had developed to completely impede conduction through the AV node and result in the development of third-degree heart block.

This patient may have been at increased risk for progression of AV node conduction defects due to baseline first-degree heart block. First-degree heart block has multiple causes including drug effects, ischemia, and degeneration of the cardiac conduction system. The patient was taking no medications associated with first-degree heart block and he had no history of myocardial ischemia. Therefore his heart block likely represented age-associated changes in the AV node. This preexisting impairment in AV node conduction may have increased the risk for a higher degree of heart block in the setting of reduced sympathetic stimulation as has been suggested with spinal anaesthesia [4]. Other mechanisms for dysrhythmias due to neuraxial anaesthesia have been proposed, including reduced stimulation of intracardiac stretch receptors from low cardiac filling pressures causing a parasympathetically mediated decrease in heart rate [2,10]. This mechanism is unlikely to explain the changes seen in this patient because at the time of development of third-degree heart block he had already received 500 mL of hetastarch solution with an improvement in blood pressure and the last recorded blood pressure before the event, 103/65 mmHg, was within his range of blood pressures during the prior 24 h. Additionally, no evidence of increased vagal tone such as gradually developing sinus bradycardia was seen prior to the abrupt onset of third-degree heart block.

The degree of heart block can progress for reasons unrelated to neuraxial anaesthesia, however, these are unlikely to have played a role in this patient. First, the patient had a negative history for coronary artery disease and there was no rise in cardiac troponin levels following the event. This makes ischemia of the AV node an unlikely explanation. Drugs that prolong AV node conduction time are often avoided in patients with first-degree heart block due to the risk of inducing higher degrees of heart block, and third-degree heart block has been reported in patients receiving AV node blocking medications whilst undergoing combined general/epidural anaesthesia [7,15]. However, this patient was not receiving any medications prior to or during admission that are commonly known to slow AV node conduction. Hence, the direct effect of the thoracic epidural was observed. Finally, local anaesthetics can induce cardiovascular arrhythmias when injected in toxic doses. This too is an unlikely explanation for the events seen in this patient because the dose of ropivacaine he received, 8 mg, was well below the recommended maximum single dose of 200 mg [1]. Additionally, intravascular placement of the epidural catheter was doubtful due to negative aspiration for blood and a negative test dose.

Chest trauma resulting in rib fractures is common following motor vehicle collisions and these fractures lead to significant morbidity and mortality, particularly in the elderly [3,17]. The goal of analgesia in these patients is not simply to alleviate pain, but also to decrease the splinting and atelectasis that accompany these injuries in the hope of reducing resultant respiratory complications. In this patient, the goal of analgesia was successfully achieved via thoracic epidural anaesthesia as shown by a reduction in the patient’s reported pain score and an improved volume on incentive spirometry. However, thoracic epidural anaesthesia is not the only regional anaesthetic technique for rib fractures. Thoracic paravertebral blocks are also effective in patients with rib fractures and are associated with less hypotension than thoracic epidural anaesthesia, likely due to their unilateral sympathetic blockade [6,14,16]. Because of this reduced degree of sympatholysis, thoracic paravertebral blocks may be preferable to thoracic epidural anaesthesia in patients at risk for heart block. If thoracic epidural anaesthesia is the method of analgesia chosen, premedication with an anticholinergic medication prior to the initial local anaesthetic bolus may be considered to reduce the risk of heart block.

4. Conclusion

This report demonstrates that thoracic epidural anaesthesia alone can induce third-degree heart block. This is a serious and
potentially life-threatening complication rarely considered by physicians caring for trauma patients. First-degree heart block may increase the risk of this event. For this reason, patients with preexisting first-degree heart block must be carefully evaluated and the risks and benefits of thoracic epidural anaesthesia should be weighed. Prophylactic administration of an anticholinergic medication should be considered in patients deemed at risk of high-degree heart block, or paravertebral nerve blocks can be chosen in place of a thoracic epidural in order to minimize sympathetic blockade. Cardiac monitoring during and after epidural placement is mandatory in these patients.

Conflict of interest statement

The authors have no personal or financial conflicts of interest in relation to this case report.

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References