Continuous spinal anaesthesia for abdominal surgery in a patient receiving amiodarone

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The perioperative management of a 57-yr-old patient receiving chronic amiodarone therapy with a continuous spinal anaesthetic for a low anterior resection of the colon is discussed. The most appropriate anaesthetic technique for patients receiving chronic amiodarone therapy remains controversial, but the avoidance of general anaesthesia may be beneficial because of the risk of postoperative pulmonary failure. In this patient continuous spinal anaesthesia was slowly titrated to the desired level, coincident haemodynamic alterations were easily treated as they developed, and high serum local anaesthetic concentrations which occur with other regional anaesthetic techniques were avoided.

La conduite périopératoire d'une anesthésie rachidienne continue lors d'une résection antérieure basse du côlon chez un patient âgé de 57 ans recevant une thérapie chronique à l'amiodarone est décrite. La technique anesthésique la plus appropriée pour les patients sous thérapie chronique à l'amiodarone demeure controversée, mais le fait d'éviter l'anesthésie générale pourrait être bénéfique à cause du risque d'insuffisance pulmonaire postopératoire. Chez ce patient, l'anesthésie rachidienne continue est lentement titrée au niveau désiré et les altérations hémodynamiques furent facilement traitées au fur et à mesure qu'ils apparaissent et l'augmentation des concentrations sériques et des anesthésiques locaux qui surviennent avec d'autres techniques d'anesthésie régionale furent évitées.

Key words

ANAESTHETIC TECHNIQUES: spinal-continuous;

HEART: antiarrhythmics; amiodarone;

INTERACTIONS (DRUG): amiodarone, anaesthetic agents.

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Amiodarone is a class III antiarrhythmic agent whose key actions include prolongation of the action potential and effective refractory period of the heart.¹ Although it is the most effective oral agent available for treating patients with ventricular tachycardia, toxicity has limited its initial use to patients whose arrhythmias are not effectively treated by other agents.

Amiodarone-induced pulmonary toxicity has been reported to occur in 5–28% of patients using this drug chronically.² The clinical course is usually characterized by a slow subacute onset which reverses on reduction of drug dosage, termination of therapy, or corticosteroid administration.

Fulminant life-threatening or fatal adult respiratory distress syndrome has been reported after general anaesthesia in patients receiving amiodarone, even in the absence of apparent preoperative pulmonary damage.^{3,4} As the patient population at risk of developing adverse reactions to amiodarone is not defined, the perioperative management of patients taking this agent remains controversial.⁵ Avoidance of general anaesthesia may be advantageous. This report describes the clinical course of a patient receiving chronic amiodarone therapy in which continuous spinal anaesthesia was utilized for major abdominal surgery.

Case report

A 57-yr-old male receiving amiodarone was scheduled for anterior resection of a partially obstructing adenocarcinoma of the colon. He had a history of congestive cardiomyopathy since 1983 (gated pool LVEF = 34%, RVEF = 52%). Three years before admission, he had an episode of ventricular fibrillation-induced cardiac arrest. The arrhythmias were refractory to multiple conventional antiarrhythmic regimens. Amiodarone (alternating 200 and 400 mg *po* daily) was eventually instituted to suppress the arrhythmias. He had no known primary lung disease, was a non-smoker, and his remaining medical history was noncontributory. He was taking no other medications.

Preoperative physical examination revealed a 175 cm, 72 kg man with blood pressure (BP) ranging from 120/60 to 135/80 mmHg and heart rate (HR) from 65–85

TABLE Haemodynamic changes during abdominal surgery with continuous spinal anaesthesia (bupivacaine) in a patient receiving amiodarone

Events Time	Baseline	Surgery	Phenylephrine	Phenylephrine		Surgery	Phenylephrine D/C'd	Moving knees	Spinal
	Spinal placed 8:38	started 8:53	started at 40 $\mu\text{g} \cdot \text{min}^{-1}$ 9:03	decreased to 20 $\mu\text{g} \cdot \text{min}^{-1}$ 9:13 9:32		completed 9:52	Wiggling toes 10:50	11:09	resolved 11:38
Spinal bupivacaine dose (mg)	5.0	2.5	2.5						
Sensory level	T ₁₀	T ₈	T ₆	T ₆	T ₆	T ₆	T ₈	T ₁₀	
CO	6.1	5.2	5.7	5.4	6.1	5.8	6.3	7.0	6.2
HR	67	65	67	66	70	62	65	66	67
SBP	101	105	75	108	124	124	122	121	109
MAP	67	67	45	76	85	83	93	88	91
DBP	50	48	30	60	65	62	78	72	81
CVP	10	9	9	10	13	9	10	12	12
PAM	18	18	15	22	22	17	19	18	20
PCWP	10	10	10	10	10	10	9	9	11
SVR	747	891	505	977	943	1020	1053	868	1018
PVR	105	123	70	178	157	96	127	103	1116

beats $\cdot \text{min}^{-1}$. Apart from a laterally displaced apical impulse his physical examination was unremarkable.

Electrocardiogram showed sinus rhythm with an occasional ventricular ectopic beat and a first degree atrioventricular block at a rate of 68 beats $\cdot \text{min}^{-1}$. Chest roentgenogram revealed a slightly enlarged cardiac shadow and was free of interstitial infiltrates. Pulmonary function studies showed FEV₁ = 3.49 l. (103% predicted), and FVC = 4.60 l. (75% predicted). Other routine laboratory tests were normal.

Amiodarone was continued until the morning of surgery (200 mg). No premedication was given before the patient's arrival at the operating suite. Following placement of non-invasive monitors and delivery of 2 L $\cdot \text{min}^{-1}$ of supplemental oxygen by nasal cannula, mild sedation was established with 2 mg *iv* midazolam. With ventricular pacing capability readily available, invasive monitoring was established with a radial arterial catheter and a thermodilution pulmonary artery (PA) catheter placed via the right internal jugular vein.

A 20 ga catheter was easily placed 4 cm into the subarachnoid space via an 18 g. Tuohy needle with its needle bevel oriented parallel to the long axis of the dural sac. After obtaining baseline haemodynamic measurements, spinal anaesthesia was induced with 5.0 mg hyperbaric bupivacaine (0.75% in 8.25% dextrose without epinephrine) and slowly titrated over a 15-min period to a sensory level of T₆ using incremental doses of 2.5 mg (10 mg total). Intermittent haemodynamic measurements taken during this period demonstrated a gradual decrease in mean arterial pressure (MAP) after institution of spinal anaesthesia, and a corresponding further decrease in calculated systemic vascular resistance (SVR) below its

initially low baseline level (Table). Titration of a phenylephrine infusion returned MAP and SVR to normal. Minimal changes in cardiac output occurred during these interventions.

The surgical procedure was completed in one hour with an estimated 150 ml blood loss replaced with 1600 ml *iv* normal saline. The anaesthetic level remained at T₆ throughout surgery. The phenylephrine infusion was tapered and discontinued in the recovery room as the segmental analgesic level regressed. The PA catheter was removed prior to discharge from the recovery room.

On the second postoperative day, the patient developed mild symptoms of a typical postdural puncture headache which resolved spontaneously that evening without treatment.

On the third postoperative day, the patient's wound below the umbilicus dehiscd and he was returned to the operating room for wound closure. With knowledge of the patient's response to his previous anaesthetic, it was decided not to replace the PA catheter, and to administer a single injection spinal anaesthetic. Intrathecal injection of 10 mg plain bupivacaine produced a sensory block to T₈ and wound closure was completed uneventfully. Four incremental injections of 50 μg phenylephrine were given during surgery to maintain haemodynamic stability. His subsequent recovery was uneventful and he was discharged ten days after his original surgery in stable health.

Discussion

Amiodarone-induced pulmonary toxicity is well documented. Rapid acceleration of this syndrome with the development of acute fulminant respiratory insufficiency

early after general anaesthesia has recently been described. Tuzcu *et al.* reported a higher incidence of postoperative pulmonary failure and longer duration of tracheal intubation and intensive care stay in postoperative cardiac surgery patients who were treated chronically with amiodarone.⁶ Kay *et al.* reported four patients who developed fatal respiratory failure after noncardiac surgery.³ All did well immediately after surgery, but rapidly developed signs and symptoms of pulmonary insufficiency 18 to 48 hr after tracheal extubation.

The long elimination half-life of amiodarone (52 d) makes the preoperative discontinuation of the drug impractical for many surgical procedures.² Discontinuation of the drug may also result in recurrence of the life-threatening arrhythmias. Furthermore, fulminant pulmonary dysfunction may occur after general anaesthesia, even after lengthy discontinuation of amiodarone.⁴ The use of a regional anaesthetic technique and avoidance of general anaesthesia could therefore be beneficial to these patients.

Severe pharmacodynamic interactions have been described between amiodarone and type I antiarrhythmic agents, such as lidocaine. Additive prolongation of the QT interval, development of torsades de pointes, and a 30–50% increase in serum antiarrhythmic concentration have been reported.⁷ Several cases of intraoperative sinus arrest, severe atropine-resistant bradycardia, and complete heart block have now been reported in patients receiving amiodarone during anaesthesia.^{8,9} Nodal rhythm and/or complete heart block developed intraoperatively in ten of 15 patients reported by Liberman.¹⁰ In the presence of acute amiodarone-induced complete heart block, local anaesthetics with type I antiarrhythmic properties could suppress the escape rhythm from lower pacemakers and result in cardiac arrest.

Spinal anaesthesia is a particularly attractive regional anaesthetic technique in the patient receiving amiodarone because it avoids exposure to high systemic levels of local anaesthetic. Although serum levels are only slightly higher after the epidural injection of a low dose (100 mg) of lidocaine than after an equivalent dose is given into the subarachnoid space,¹¹ much larger volumes of local anaesthetic are generally necessary for epidural anaesthesia. This eventually yields serum levels, for either lidocaine or bupivacaine, that are four- to ten-fold higher after epidural than after spinal anaesthesia.^{12,13} There is no evidence that serum concentrations attained after spinal anaesthesia are cardioactive.

The decrease in mean arterial pressure and calculated SVR in this patient were attributed to sympathetic blockade superimposed upon the baseline amiodarone-induced noncompetitive α adrenergic block. A phenylephrine infusion was necessary to return SVR to baseline

and to maintain the pulmonary capillary wedge pressure between 9–10 cm H₂O. One might expect cardiac output to increase in the presence of constant left-sided filling pressures after the spinal-induced afterload reduction. Amiodarone-induced reduction in cardiac systolic reserve possibly prevented an increase in cardiac output in this patient. Laboratory investigation in dogs suggests that epinephrine may be the most effective agent to reverse the myocardial depressive effect of amiodarone, but this remains unproven in the clinical setting.¹⁴

Amiodarone possesses a multitude of electrophysiological actions and produces a noncompetitive adrenergic blockade, greatly reducing the effectiveness of sympathetic agents at both α and β receptors.¹⁵ Testing the responsiveness of α adrenergic agents before administering anaesthesia to patients on amiodarone has been suggested.¹⁶ Establishing the responsiveness to β adrenergic agents may also be beneficial, as has been suggested for patients on chronic β blocker therapy.¹⁷ As amiodarone may also directly affect intracardiac conduction, ventricular pacing must also be necessary.

The recent development of smaller gauge spinal needles and catheters suggests that a rebirth of the use of continuous spinal anaesthesia may occur. Hurley and Lambert recently reviewed the advantages and disadvantages of continuous spinal anaesthesia, using their new 32-gauge microcatheter.¹⁸ In patients with severe cardiovascular disease, a subarachnoid catheter is especially advantageous compared with a single-dose spinal technique. Small incremental doses of drug can be slowly titrated to a desired anaesthetic level and balanced against the coincident haemodynamic changes, for which vasoactive drugs can be instituted if necessary.

In conclusion, a case is presented in which the use of continuous spinal anaesthesia provided a safe, controllable alternative to general anaesthesia in a patient receiving amiodarone therapy. The advantages of using continuous spinal anaesthesia in the subgroup of patients with a history of severe cardiovascular problems having surgical procedures of the abdomen or lower extremities are discussed.

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