Asystole Following Neuromuscular Blockade Reversal in Cardiac Transplant Patients

The Editor,

Asystole has been described in eight patients following neuromuscular block reversal with neostigmine and glycopyrrolate.[1,2] Seven of these patients were heart transplant recipients, and with the exception of one, they were all 3–27 years posttransplant. The youngest was a 13-month-old female with a biopsy-confirmed rejection who experienced asystole following reversal 1-month posttransplant. The other patients were aged 54-68 year old. They all varied in their demographics. Clinical doses of neostigmine and glycopyrrolate had been administered in all these cases though it is unclear if they were administered concurrently. Six of these patients had experienced some transplant-related complication. Four of these six patients received a permanent pacemaker following the event, one of them had a coronary artery stent placed and the sixth was treated with steroids for humoral rejection. The seventh patient had no prior evidence of transplant-related complication and needed no further treatment following successful cardiopulmonary resuscitation (CPR).

Asystole has also been described in an 18-month-old male patient following a congenital glaucoma surgery.[3] This patient had no prior history of cardiac or systemic pathology and had been stable throughout the procedure. The oculocardiac reflex and parasympathetic dominance might have contributed to the increased susceptible to reversal agents in this infant. In all cases, CPR was initiated, and the patients recovered fully.

The anticholinesterase drug commonly used to reverse neuromuscular blockade is neostigmine. This drug prevents the hydrolysis of tonically released acetylcholine by parasympathetic neurons in the cardiac parasympathetic pathway before they reach the postsynaptic membrane receptors. Accumulation of acetylcholine at the neuromuscular junction ensures thereby competitively antagonizing any nondepolarizing relaxant. Neostigmine thus indirectly stimulates both nicotinic and muscarinic receptors.

The activation of muscarinic cholinergic receptors in the cardiac parasympathetic pathway by acetylcholine and the blocking of cholinesterase activity in the presence of acetylcholine from a reinnervated ganglion in heart transplant patients have been suggested as the cause of neostigmine-induced bradycardia.[4] Reinnervation and increased sensitivity to acetylcholine, patient's condition including sinus node disruption during cardiac surgery have also been implicated in previous studies.[1,2]

Heart transplant patients, especially those with transplant-related complications, appear to be at increased risk for asystole following reversal with neostigmine and glycopyrrolate. The course for this has not yet definitively been established. Edrophonium, a noncarbamate muscarinic antagonist, produces lest bradycardia and should be considered for reversal in these patients. Sugammadex has been reported to cause atrioventricular block in a healthy individual but appears safe in a heart transplant patient.[4,5,6] Another option is to administer either atropine or glycopyrrolate before administering a neostigmine. Patients in whom this phenomenon is observed should be offered further testing as this might indicate an underlying cardiac pathology. Close monitoring following administration of any reversal agent is strongly recommended, especially in cardiac transplant patients.

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Conflicts of interest

There are no conflicts of interest.

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