

## VECURONIUM AND CARDIAC ARREST

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### ABSTRACT

Vecuronium is widely used to provide skeletal muscle relaxation in mechanically ventilated Intensive Care Unit patients. The absence of significant cardiovascular side-effects is one of its stated advantages. There have been isolated reports of Brady-dysrhythmias including sinus arrest and atrioventricular block when vecuronium was administered with other agents especially opioids. We report a case of cardiac arrest following administration of vecuronium alone in a mechanically ventilated child in the Intensive Care Unit.

**Keywords:** Vecuronium, Sinus arrest, Atrioventricular block.

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### INTRODUCTION

Vecuronium bromide, a non-depolarizing neuromuscular blocker is frequently cited as a good option for muscular relaxation since it is free from cardiovascular activity [1]. However, several case reports have described severe bradycardia [2] and even cardiac arrest [3] after vecuronium administration. Many of these cases [4, 5] were associated with opioid administration. Since vecuronium has virtually no blocking activity on cardiac, vagus or autonomic ganglion, there is a tendency for bradycardia to occur in association with the use of opioid analgesia. Subsequent study has indicated that vecuronium alone does not cause bradycardia. Considering the various cardiovascular side-effects especially cardiac arrest, this drug should be used with utmost caution particularly when being used along with opioid analgesics.

### CASE REPORT

This case report was prepared after taking an informed consent of the father. A 10 y old female child was admitted to Pediatric Intensive Care Unit with 1 d history of fever and altered sensorium. On examination, the child was afebrile, irritable but conscious, responsive to the command and hemodynamically stable. There was neither history of cardiovascular or other medical problems nor any drug allergy. Investigation after admission revealed the following: - hemoglobin 9.2 gm/dl, total leukocyte count 7700/mm<sup>3</sup>, neutrophil 51%, total platelet count-4.29 lac., Serum calcium-7.9 mg/dl. Serum electrolytes, renal function test and liver function test were normal. Immunochromatographic Test for malarial parasite was negative. Child deteriorated the next day. Glasgow Coma Scale (GCS) decreased to 7-8. A cerebrospinal fluid study done was normal. MRI brain was also normal. Arterial Blood Gas showed respiratory alkalosis with compensated metabolic acidosis. She was intubated for low GCS with midazolam 0.1 mg/kg. No opioid or any other anaesthetic agent was given during the procedure. Post intubation, the patient was hemodynamically stable and maintaining saturation on 40% FiO<sub>2</sub>. She was fighting against ventilator. Therefore, vecuronium 1 mg/kg was given to paralyze her and prevent patient-ventilator dyssynchrony. She developed sudden profound bradycardia which was rapidly followed by cardiac arrest. Prompt institution of cardiopulmonary resuscitation was done. She remained asystolic, did not respond to any resuscitation and expired.

### DISCUSSION

Vecuronium is an aminosteroid neuromuscular blocker developed by Savant, Durant, Bowman and Marshall. It is an intermediately acting muscle relaxant outstandingly noted for its absolute hemodynamic stability when administered in clinical doses. Nevertheless, vecuronium can induce bradycardia and some cases of cardiac arrest after its use were found in the literature [3, 5]. The reason for this reduction in heart rate is poorly understood.

Cozanitis [6] and Miller suggest that vecuronium per se is devoid of intrinsic bradycardic-activity. They postulate that because of lack of vagolytic activity of this neuromuscular agent any vagal mediated bradycardia is caused by other drugs (fentanyl) used simultaneously or by surgical stimuli. The combination of vecuronium with high doses of opioid produces negative chronotropic and inotropic effects resulting in a decrease in heart rate, cardiac output and blood pressure. In contrast, Inoue and colleagues [7] as well as Salmenpera and co-workers [8] suggested that vecuronium possesses an intrinsic bradycardic activity. Vagolytic pre-anesthetic medication apparently protects from this undesirable effect [9].

Sinus node exit block following administration of vecuronium has been reported by Yeaton. [10] Vecuronium related atrioventricular block has been reported by Macrae. A possible correlation between cardiac arrest and vecuronium bromide has been reported by physicians to Food & Drug Administration (FDA) and Fact Med between January 2004 and October 2012. A total of 1884 vecuronium bromide drug adverse event reactions (AERS) were reported with the FDA during this period, of which 162 patients had cardiac arrest. This amounted to 8.59% of the total AERS. Drug Informer, an official FDA website reported 114 cases of AERS related to vecuronium between 2004 and 2014. Of these, 20 patients died due to cardiac arrest which amounted to 17.24%.

### CONCLUSION

Considering the fact that vecuronium related cardiovascular complications especially cardiac arrest are being increasingly reported in the recent past, this drug can no longer be cited as devoid of cardiovascular effects. It is essential to be aware and recognize this potential complication in order to prevent possible death related to the vecuronium-induced cardiac arrest. Use of anticholinergic agent as a part of premedication or prior to induction of anesthesia should be seriously considered.

### CONFLICT OF INTERESTS

Declared none

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