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LETTER TO THE EDITOR

# A case of atrioventricular block (Wenckebach type) induced by sugammadex

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To the Editor:

Summadex is a muscle relaxant antagonist that reverses muscle relaxation by binding to muscle relaxants. There is almost no known noteworthy adverse reaction to this drug, although there have been some reports of QT prolongation on electrocardiogram (ECG) induced by summadex [1–3]. We recently encountered a case who developed second-degree atrioventricular block (Wenckebach type) after having received sugammadex.

A 21-year-old female patient (height 165 cm, body weight 47 kg) had no noteworthy previous disease history, and preoperative tests revealed no abnormalities of the ECG, serum electrolytes, or other factors. Surgery for resection of nevus pigmentosus of the brachium and face was scheduled. Anesthesia was induced with propofol (100 mg) and rocuronium (40 mg) and maintained with oxygen (40 %), sevoflurane (1.5 %), and remifentanil (0.02 μg/kg/min). The surgeon gave locally 6 ml 1 % lidocaine mixed with 1:200,000 epinephrine to maintain a bloodless surgical field with local anesthesia. Circulation dynamics remained stable intraoperatively. The operation lasted for 60 min. At the completion of the operation, when consciousness was restored, sugammadex (200 mg) was administered. Before tracheal extubation, the patient showed ECG changes consistent with second-degree atrioventricular block (Wenckebach type) (Fig. 1). There were no changes of the heart rate, blood pressure, or $SpO_2$ after the administration of sugammadex as compared with preadministration values. When the course of atrioventricular block was followed, the block converted to first-degree atrioventricular block within 3 min and to normal sinus rhythm within a few more minutes.

In this young patient for whom no particular problem was detected in preoperative tests, second-degree atrioventricular block (Wenckebach type) developed after a dose of sugammadex.

Sugammadex binds to muscle relaxants, forming an inactive complex (inclusion compound), thereby effectively removing the muscle relaxant from the neuromuscular junction. Features of sugammadex that are advantageous over the conventional anticholinesterase agents include the lack of a ceiling effect, capability of the drug to reverse even deep muscle relaxation, the slight effects of acid–base balance, body temperature, or anesthetic agents on the drug's actions, rapid manifestation of action, lack of muscarinic receptor stimulation, and lack of paradoxical reduction of the muscle strength [1–4]. Known adverse reactions to sugammadex include nausea/vomiting, cough, and hypotension. Anaphylaxis and QT prolongation have been reported as serious adverse reactions to this drug [1–3]. QT prolongation is considered to show no association with the dose level of sugammadex [5].

Tachycardia and bradycardia, for example, often appear when the patient emerges from anesthesia following treatment with anticholinesterase agents or sugammadex [1–4]. However, it is rare for atrioventricular block to develop in a patient with no organic heart disease emerging from anesthesia.

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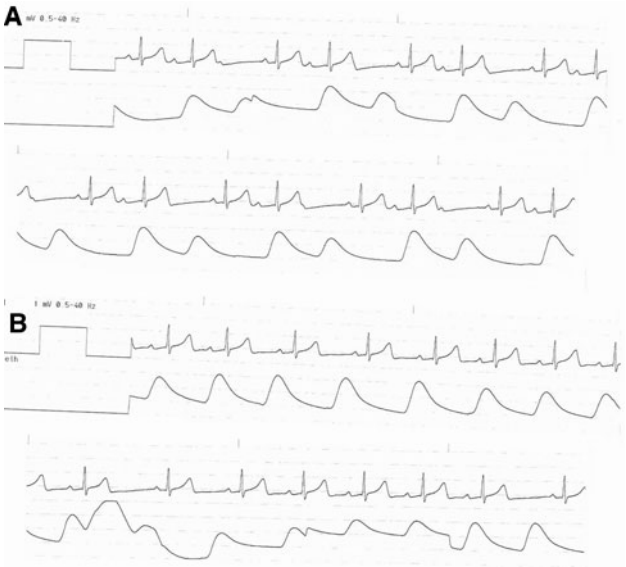


**Fig. 1** Changes in the ECG and the waves from pulse oximeter after sugammadex treatment. **a** Second-degree atrioventricular block immediately after a dose. **b** First-degree atrioventricular block 3 min after a dose

Although there was the possibility that atrioventricular block was caused by stimulating the parasympathetic nervous system in this case, we cannot rule out the possibility that treatment with sugammadex caused the atrioventricular block.

**Conflict of interest** None.

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