A Case of Wenckebach-type Atrioventricular Block Caused by Administration of Indigo Carmine

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We report a case of first-degree atrioventricular (A-V) block progressing to second-degree (Wenckebach-type) A-V block after administration of indigo carmine in a patient undergoing hysterectomy under general anesthesia. We believe that the onset of Wenckebach-type A-V block may have been induced by one or more of three factors: 1) preoperative first-degree A-V block, 2) the anesthetics used (propofol and remifentanil), and 3) administration of indigo carmine.

Key words: First-degree A-V block, Indigo carmine, Wenckebach-type A-V block

INTRODUCTION

Wenckebach-type atrioventricular (A-V) block has been reported to occur during spinal anesthesia [1–3] for pain relief [4]. To our knowledge, however, no studies to date have reported Wenckebach-type A-V block occurring with administration of indigo carmine during general anesthesia. Here, we report the progression of first-degree A-V block to Wenckebach-type A-V block after administration of indigo carmine during general anesthesia.

CASE REPORT

The patient was a 41-year-old woman 161 cm in height and 70 kg in weight. She had been scheduled for simple hysterectomy due to a diagnosis of uterine myomas.

A preoperative electrocardiogram (ECG) revealed first-degree A-V block (PR interval: 0.244 sec) (Fig. 1). Transechocardiography indicated favorable wall motion, and the ejection fraction was approximately 69% by modified Simpson method. The results of chest X-ray and other laboratory tests were within the normal range. The patient had no indication of potential problems and she was not on any medication. An earlier medical examination at the company at which she was employed had found evidence of arrhythmia, but this was ruled out after she was required to undergo Holter ECG at a nearby hospital. Premedication comprised intravenous drip infusion of 3 mg midazolam, 20 mg famotidine and 0.5 mg atropine sulfate at 30 min before entering the operating theater. Blood pressure was 130/70 mmHg and pulse rate was 85/min on admission to the operating theater. First-degree AV block was revealed on ECG. An epidural catheter was inserted between vertebrae L1 and L2, and a test dose of 1 ml of 1% lidocaine injected. Anesthesia was induced with intravenous infusion of 3 μg/ml propofol (Target Control Infusion), 0.5 γ remifentanil, and 8 mg vecuronium followed by intubation. No changes were observed in the patient's ECG readings after tracheal intubation. Anesthesia was maintained by air (5 L/min), oxygen (1 L/min), and continuous intravenous infusion of propofol (1.0 to 2.0 μg/ml) and remifentanil (0.2 to 0.25 γ) along with suitable administration of vecuronium. Anesthetic depth was adjusted to a Bispectral Index (BIS) value ranging from 40 to 60. Depth of sedation was monitored with a BIS monitor. No changes were observed in her ECG readings at the time of initial incision. At 60 min following incision, continuous epidural block was started at 4 ml/hr (92 ml of 0.2% ropivacaine + 8 ml fentanyl = 100 ml) for postoperative analgesia. Although blood pressure subsequently showed a gradual decrease, this was counteracted by an increase in infusion volume with commencement of auto-transfusion of ephedrine hydrochloride. At 120 min following the initial incision, indigo carmine was administered to determine whether any ureteral injury had been incurred intraoperatively. Shortly after administration of indigo carmine, the PQ interval began to lengthen gradually and the QRS wave form dropped out on the ECG monitor (Fig. 2, upper ~middle), leading to a diagnosis of Wenckebach type A-V block. Although the patient's status was stable, a follow-up was conducted. No improvement was observed in the Wenckebach type A-V block for the next 5 min, so 0.5 mg atropine sulfate was administered. The Wenckebach type A-V block subsequently recovered to first-degree A-V block (Fig. 2, low). Surgery was completed without further incident, and the patient was extubated. The anesthetic record can be seen in Fig. 3.

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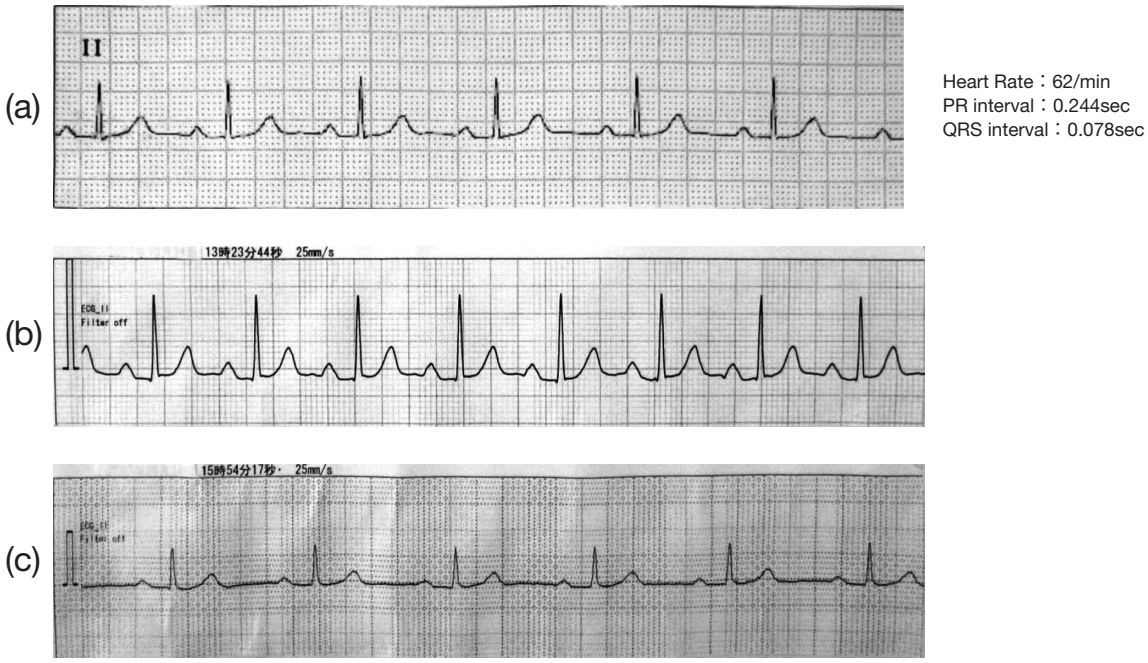

Fig. 1 (a) ECG of pre-operative : The ECG shows first degree A-V block (PR interval: 0.244Sec)
(b) ECG of at entering to operating room (c) ECG of indigo carmine pre-administarion
Wenckebach type AV block did not develop even in (a) (b) (c) ECG

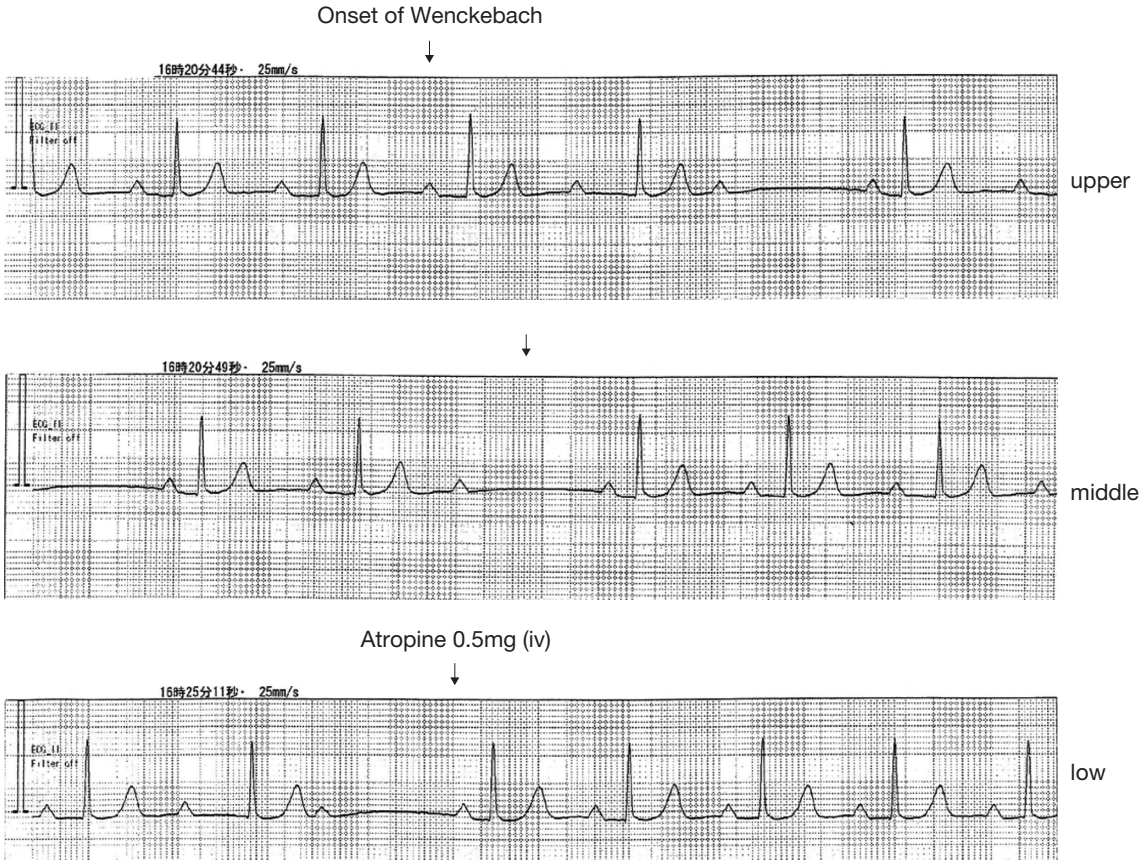

Fig. 2 Upper : The ECG shows onset of second degree A-V block
(the PQ interval began to lengthen gradually and the QRS wave form dropped out)
Middle : The arrow (↓) shows QRS wave form dropped out on the ECG
Low : The ECG shows Wenckebach type A-V block recovered to first-degree A-V block after administration of indigo carmine.

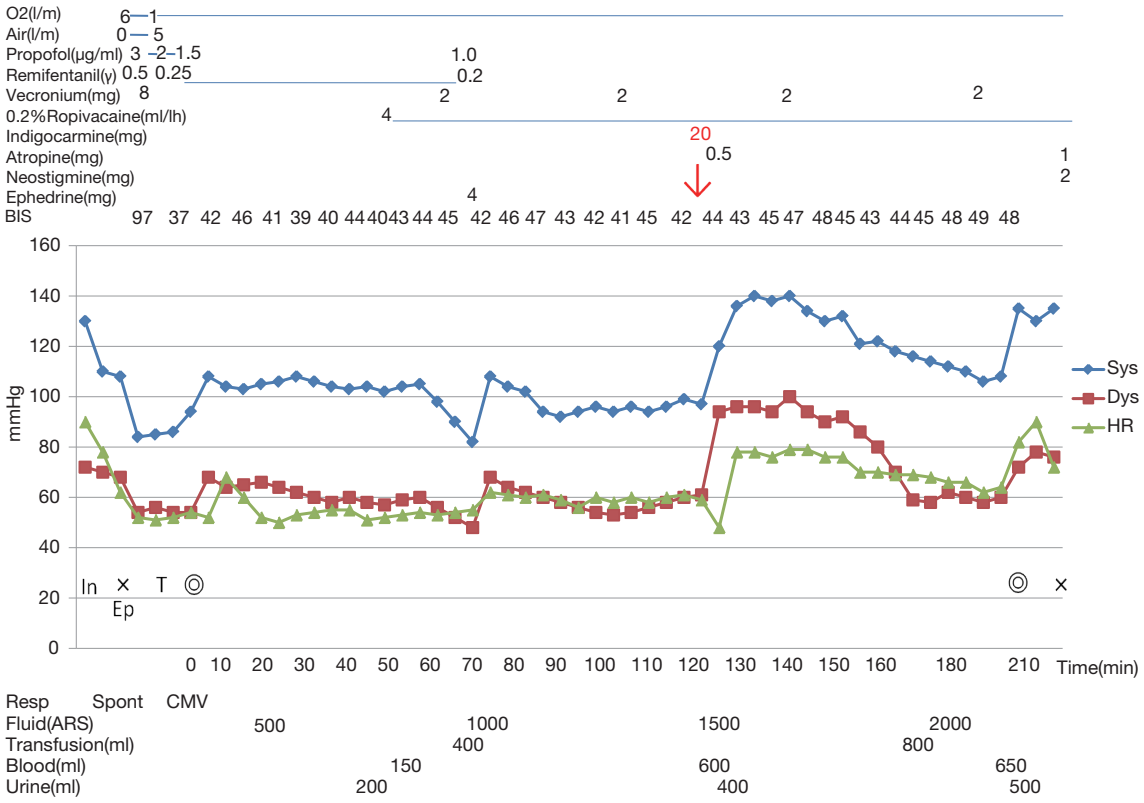

Fig. 3 Red arrow(↓) shows elevation of blood pressure and decrease of heart rate after administration of indigo carmine on the anesthetic record

DISCUSSION

Wenckebach-type A-V block can be inferred from a gradual prolonging of the PR interval and disappearance of the QRS waveform on ECG. The possible causes of this type of block include inferior wall myocardiac infarction, drug administration (digitalis, β blockers, Ca antagonists, or muscle relaxant antagonists), and increased vagus nerve activity [5]. We believe that the onset of Wenckebach-type A-V block in this case may have been caused by one or more of three factors: 1) preoperative first-degree A-V block, 2) continuous intravenous administration of two kinds of anesthetic drug (propofol and remifentanil), and 3) administration of indigo carmine. If the PR interval is greater than 0.24 sec, the cause of the block is most likely a conduction delay at the A-V node. In the present case, the PR interval was 0.244 sec, suggesting parasympathetic activity as the cause of the block. We also considered the anesthetics used as the cause of the AV block. Propofol is the most common anesthetic agent used in conjunction with other drugs. Prolongation of atrioventricular conduction is also sometimes observed as a side effect [7]of propofol. Remifentanil is an ultra-short-acting narcotic analgesic. Severe bradycardia (heart rate: 40/sec) was observed in 9% of patients receiving a combination of propofol and remifentanil [8]. We believe that co-administration of these two drugs tends to disturb atrioventricular conduction. Blood pressure was reduced by commencing epidural anesthesia in the present case. However, administration of fluids and vasopressors increased heart rate and recovered blood pressure. Stability of heart rate suggests that epidural anesthesia has little effect on the occurrence of Wenckebach-type A-V block. Indigo carmine dye is used to measure renal function [9]. Peeling the bladder and uterus in hysterectomy sometimes causes damage to the bladder. Subsequent leakage from the bladder may be determined by intravenous administration of indigo carmine. Administration of indigo carmine during anesthesia has been reported to affect the cardiovascular system, causing a number of problems, including hypotension [10], severe bradycardia [11], and supraventricular premature contraction [12]. However, reports of indigo carmine causing A-V block are rare [13]. Indigo carmine causes a number of side effects such as bradycardia and increased blood pressure. This increase in blood pressure is due to an increase in peripheral vascular resistance by α stimulation, triggering parasympathetic nerve activity, which causes bradycardia reflexively [14–15]. Changes in blood pressure and heart rate before and after administration of indigo carmine were observed in 20 patients. Following administration of indigo carmine, a significant elevation was observed in systolic blood pressure (Fig. 4, left) and a decrease in heart rate (Fig. 4, right). This suggests that indigo carmine acts predominantly on the parasympathetic nervous system. The evidence in the present case suggests that Wenckebach-type AV block occurred predominantly as a result of the reaction of the vagus nerve system to the combination of indigo carmine and the anesthetic drugs used. Therefore, anesthesia management should be conducted bearing in mind the possibility that a disturbance may occur in atrioventricular conduction with this combination.

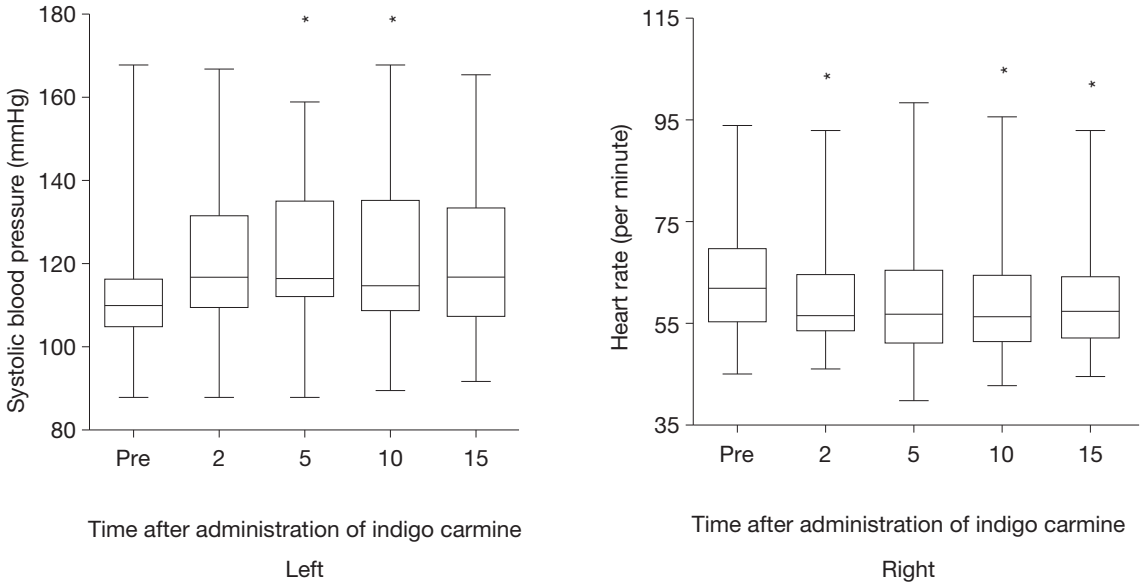

Fig. 4 (left). Systolic blood pressure was significant elevated after administration of indigo carmine. (right). Heart rate was significant decreased after administration of indigo carmine.

REFERENCES

1) Yokoyama K, Ehara R: A case of Atrioventricular block induced by atropine during spinal anesthesia. J. Clin. Anesth. (Jpn) 1987; 11; 2: 273–274.
2) Shimai N, Yokoyama K: Atrioventricular block (Wenckebach type) induced by atropine during 5% pethidine spinal anesthesia. Masui (Jpn) 1988; 38; 5: 684–686.
3) Ebata T, Karasawa H, Satoh T: A case of second-degree AV block (AV Wenckebach block) induced by atropine during spinal anesthesia. J. Clin. Anesth (Jpn) 1998; 22; 1: 103.
4) Hadano M, Hujiwara Y, Masuda N, Ooshiro K, Muranaka K, Ooshita S: A case of Wenckebach type AV block induced by post-traumatic pain. J. clin. Anesth (Jpn) 1990; 14; 8: 1201–1202.
5) Dennis L, K, Euquene B, Stephen H, Dan L J. Larry J, Anthony S F, : Harrison's, Principles of Internal Medicine: 15th edition macgrawhill, 1287.
6) Tanaka Y, Murata K, Sera A, Horibe M, Izumi H, Tsuchiya T: A case of first degree AV block migrated to second AV block after reverse of non-deporalizing muscle relaxitant drug. Masui (Jpn) 1990; 40; 4: 616–621.
7) Alphin RS, Martens JR, Dennis DM: Frequency-dependent effects of propofol on atrioventriocular nodal conduction in guinea pig isolated heart. Anesthesiology 1995; 83; 382–394.
8) Iwakiri Y: Solution to the special situation, adverse events, Remifentanil anesthesia that can be practiced from today. Nagata O reviews, Shinko-koheki published. Tokyo. 2007; 13–27.
9) C. C. Wu, Arthur J: The vasopressor effect of indigo carmine. Henry Ford Hosp. Med. J 1969; 17; 2: 131–134.
10) Scott G, Lennox H, Fatima V, Linda B: Life-threatenibg reaction to indigo carmine-A sulfa allergy? Int Urogynecol J 2005; 16: 418–419.
11) Satoh K, Sakamoto N, Shinohe Y, Satoh M, Joh S: Indigo carmine induced bradycardia in a patient during general anesthesia. Anesth Analg 2001; 92: 276–277.
12) Fujii T, Yoshinuma H, Kobayashi O: Indigo carmine induced polymorphic supraventricular bigeminy. Acta Anesth Scand 2009; 53; 3: 417–418.
13) Ion A: Atrioventricular block induced by indigo carmine. Can J Anesth 2008; 55; 10: 717–718.
14) William K, Karijadi W, Toshio A, John B: Cardiovascular and respiratory effects of indigo carmine. The J of Urology 1968; 775–778.
15) Thien N, Tapan D, Bulent K: Reaction to indigo carmine. The J of Urology 1976; 132–133.