Case Report
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Junctional rhythm with severe hypotension following infiltration of lidocaine containing epinephrine during dental surgery

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We experienced an unusual case of accelerated junctional rhythm with severe hypotension after infiltration of lidocaine containing epinephrine during dental surgery under general anesthesia. The patient's electrocardiogram exhibited retrograde P-waves following the QRS complex, which could be misinterpreted as ST-segment depression. As a temporary measure, administration of ephedrine restored the patient's blood pressure to normal levels. The importance of this case lies in its demonstration of an unexpected and serious side effect of commonly used epinephrine infiltration. This case also highlights the need for accurate interpretation of the electrocardiogram and comprehensive understanding of best practices for patient management.

Keywords: Arrhythmia; Electrocardiography; Epinephrine; Hypotension.

INTRODUCTION

A junctional rhythm is characterized by QRS complexes and is morphologically identical to sinus rhythm, without the preceding sinus P-waves [1]. Junctional rhythm is slower than the expected sinus rate. An accelerated junctional rhythm usually has a rate between 60 and 100 beats/min and can result from a variety of conditions, including digoxin toxicity, open cardiac surgery, acute myocardial infarction, or isoproterenol infusion [2]. Volatile anesthetics can affect the sinoatrial (SA) node and may be associated with the development of junctional rhythm during surgery [3,4]. Regional infiltration of local anesthetics that contain epinephrine is widely used for pain control and reducing blood loss during surgery; however, systemic side effects of this procedure—such as hypertension, tachycardia, and other arrhythmias—have been documented in the literature [5,6]. In this report, we present our experience with a patient who developed intraoperative junctional rhythm and severe hypotension following regional injection of a local anesthetic that contained epinephrine.

CASE REPORT

A 69-year-old woman (height 159 cm; weight 55 kg) was scheduled for mandibular dentigerous cystectomy under general anesthesia. She was diagnosed with

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hypertension and had been taking an angiotensin receptor blocker for 2 years, with no other home medications. The patient's preoperative electrocardiogram revealed sinus bradycardia with a heart rate of 55 beats/min and no associated clinical symptoms such as dizziness or syncope. No abnormalities were observed on preoperative chest radiography and routine laboratory examinations. The day before surgery, her mean blood pressure and pulse rate were 136/71 mmHg and 54 beats/min, respectively. The patient received no premedication, and standard monitoring was performed. An electrocardiogram before the induction of anesthesia showed sinus rhythm with a heart rate of 58 beats/min. Her blood pressure was 140/78 mmHg and oxygen saturation was 99% on pulse oximetry. General anesthesia was induced with a target-controlled intravenous infusion of propofol and remifentanil. Nasotracheal intubation was performed after administration of intravenous rocuronium (40 mg). General anesthesia was maintained with propofol (with a target effect-site concentration of 3–4 μg/mL) and remifentanil (with a target effect-site concentration of 3.5–4.5 ng/mL) to achieve a bispectral index of 40–55. Ventilation was controlled to achieve an end-tidal CO_2 partial pressure of 35–45 mmHg with a mixture of O_2 and air. After induction of anesthesia, the electrocardiogram showed a sinus rhythm with a heart rate of 61 beats/min, and the patient's noninvasive blood pressure was 111/63 mmHg.

At 20 min after the induction of anesthesia, we administered a local infiltration of 8 mL of 1% lidocaine (10 mg/mL) combined with 1:100,000 epinephrine (10 μg/mL) before the surgical incision. The inferior alveolar nerve block was performed first, and submucosal infiltration was administered to the depth of the mucobuccal fold and around the lesion. All injections were administered after aspiration and completed in around 3–4 min. At 5 min after the infiltration of lidocaine containing epinephrine, the electrocardiogram revealed an accelerated junctional rhythm, with a heart rate of 70 beats/min (Fig. 1A). The patient's noninvasive systolic blood pressure was 50 mmHg immediately after we noticed the changes in the electrocardiogram. Two, 8-mg intravenous injections of ephedrine were administered. The systolic blood pressure increased to 138 mmHg and remained around 110 mmHg when measured at intervals of 2–3 min for 10 min. The electrocardiogram showed a normal sinus rhythm around 6 min after the onset of the junctional rhythm (Fig. 1B). There were no significant changes in other parameters—such as oxygen saturation, end-tidal CO_2 partial pressure, and peak airway pressure—while the patient exhibited arrhythmias and hypotension.

There were no further significant blood pressure changes or arrhythmias until the end of the surgery, which was 55 min in duration. Tracheal extubation was performed when the patient could respond to verbal commands, and she exhibited adequate spontaneous ventilation after administration of sugammadex. She blood pressure was normal and there were no abnormal electrocardiogram findings during her stay in the postanesthesia care unit. The patient's postoperative course was uneventful, and she was discharged the day after the surgery.

DISCUSSION

Recent evidence suggests that junctional rhythms may be attributed to various mechanisms, all of which involve triggered activity and abnormal automaticity [7]. Kunimatsu [8] and Kishimoto et al. [9] have reported cases of intraoperative junctional rhythms, and epinephrine was suggested to cause imbalances in the pacemaker activities of the sinus and atrioventricular (AV) nodes. Lee et al. [10] have reported a junctional rhythm that occurred during an electrophysiology laboratory that was apparently caused by isoproterenol-enhanced AV junctional automaticity.

In the case reported here, an accelerated junctional rhythm was observed after regional infiltration of lidocaine that contained epinephrine, and the patient suffered from severe hypotension. The dose of lidocaine

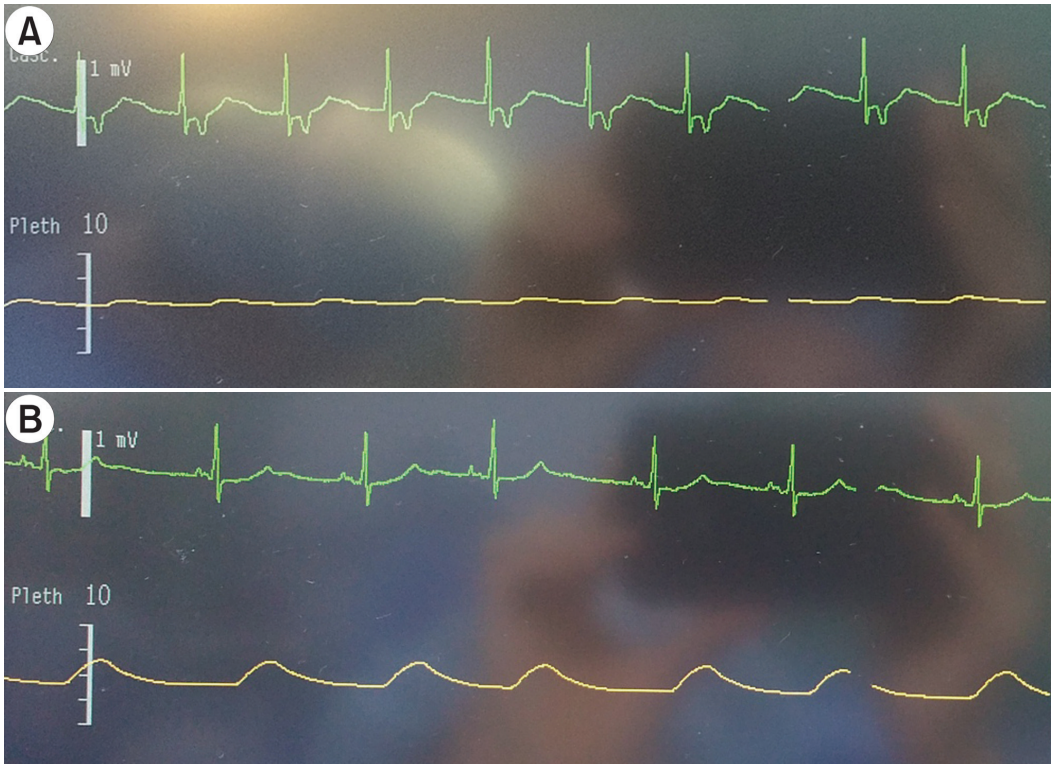

Fig. 1. (A) The electrocardiogram revealing an accelerated junctional rhythm with a heart rate of 70 beats/min and characterized by retrograde P-waves following the QRS complex. These could be misinterpreted as ST-segment depression. Abruptly reduced amplitude of plethysmographic waveforms is observed before onset of severe hypotension. (B) Normalization of sinus rhythm with a heart rate of 59 beats/min after administration of corrective measures.

and epinephrine corresponded to 1.5 mg/kg and 1.5 μg/kg, respectively. We believe that the AV nodal pacemaker cells accelerated due to elevated sympathetic activity following the epinephrine injection. This caused an accelerated junctional rhythm. A junctional rhythm is regarded as a benign arrhythmia that usually does not require treatment; however, the lack of atrial kick can decrease systemic arterial blood pressure [1,11]. In other words, there may be greater reductions in blood pressure when atrial contraction does not contribute to ventricular filling, since the impulse arrives in the ventricles earlier than in the atrium [11]. This phenomenon can manifest as retrograde P waves that follow the QRS complex, as shown in our patient's electrocardiogram. Furthermore, our patient's electrocardiogram could be misinterpreted as exhibiting ST-segment depression due to inverse P-waves. A junctional rhythm should be rapidly identified, based on electrocardiography, so that proper management can be performed.

When the junctional rhythm is accompanied by hypotension, the administration of sympathomimetics such as ephedrine may stimulate the SA node to "take over" the pacemaker's activities. Nevertheless, this should be considered a temporary measure to increase blood pressure as it may only increase the rate of the junctional rhythm in some cases [11]. If this happens, transesophageal atrial pacing may be performed to restore atrial contraction and improve preload [4]. The careful administration of a beta-adrenergic blocker may restore the electrical dominance of the SA node under general anesthesia [4,12]. However, administration of drugs that can exacerbate bradycardia is recommended when the rate of the junctional rhythm is greater than 60 beats/min [4]. Lee et al. [10] reported that adenosine and verapamil suppressed isoproterenol-induced accelerated junctional rhythm by altering junctional automaticity.

The systemic side effects associated with regional infiltration of epinephrine-containing local anesthetics

include epinephrine-induced sympathetic stimulation [5,6]. Epinephrine binds to all adrenergic receptors; however, the aim of local infiltration is vasoconstriction with a direct effect on alpha-adrenergic receptors [5]. Yang et al. [13] have reported blood pressure reduction caused by a low-dose epinephrine infiltration and described the mechanism as a beta 2-adrenergic effect. Lidocaine, which is commonly used for local infiltration, may cause central nervous system toxicity (such as agitation and seizures) and cardiovascular symptoms (such as atrioventricular block and reduced myocardial contractility) when the recommended dose is exceeded or if a large amount of intravenous lidocaine is administered rapidly [14]. Enlund et al. [15] have indicated that lidocaine reduces blood pressure after the local infiltration of lidocaine–epinephrine.

An accelerated junctional rhythm can be associated with myocardial infarction, heart surgery, digitalis intoxication, or sympathetic activation [2]. Therefore, it should be possible to exclude other factors in cases of intraoperative junctional rhythm. The use of volatile inhalation agents for general anesthesia may be associated with junctional rhythm [4]; however, total intravenous anesthesia with propofol and remifentanil was used in this case. Therefore, there do not appear to be any direct causes other than local drug infiltration.

In summary, we present a case of an accelerated junctional rhythm with severe hypotension following infiltration of lidocaine that contained epinephrine under general anesthesia. The patient's electrocardiogram showed inverted P-waves following the QRS complex, which could be misinterpreted as ST-segment depression. The patient's severe hypotension required emergent intervention. After administration of ephedrine, her blood pressure returned to normal levels. The regional infiltration of epinephrine-containing local anesthetics is widely used during surgery; therefore, awareness and knowledge regarding the occurrence and management of junctional rhythms are necessary for safe patient care.

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