



Ventricular Tachycardia after Submucosal Infiltration of Lignocaine 2% with Adrenaline in Endoscopic Pituitary Surgery Under Desflurane Anesthesia

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Abstract

Keywords

- ▶ prolonged QTc
- ▶ desflurane
- ▶ adrenaline
- ▶ infiltration
- ▶ transnasal surgery

Infiltrative analgesia of the nasal cavity using a local anesthetic combined with a potent vasopressor like adrenaline is a well-accepted method for transnasal endoscopic approach in neurosurgeries under general anesthesia. The main purpose of vasoconstrictors in transnasal surgery is to cause blanching of mucosa and reducing bleeding. Although it can reduce the systemic absorption and potential systemic toxicity of a local anesthetic, there can be systemic absorption of the vasoconstrictor itself. Desflurane can further potentiate the arrhythmogenicity of lignocaine with adrenaline by causing QTc prolongation. We report a case of transient ventricular tachycardia in a patient following nasal infiltration with lignocaine adrenaline under desflurane anesthesia.

Introduction

With the advent of minimally invasive neurosurgeries, the emphasis is on anesthesia techniques with quick recovery, early ambulation, and minimal residual effects. Short-acting inhalational and intravenous (IV) agents have been used successfully for this purpose, but desflurane offers the clinical advantage of rapid induction and recovery from anesthesia when compared to others.¹ This is desirable in patients undergoing endoscopic pituitary surgery that warrants quick recovery, minimal residual effects, and allows for early neurological assessment.^{1,2}

Endoscopic transnasal transsphenoidal pituitary surgery routinely involves instillation of adrenaline with or without local anesthetics for nasal passage preparation in order to produce asanguineous surgical field by reducing bleeding, to

anesthetize the nasal mucosa to reduce pain and mitigate the cardiovascular responses mediated through trigeminal nerve.^{3,4} However, adrenaline infiltration in itself can lead to severe hemodynamic changes and lethal arrhythmias due to systemic absorption from the highly vascular nasal mucosa or sometimes due to inadvertent intravascular infiltration.⁵ Lignocaine with adrenaline combination is most widely used by surgeons and anesthesiologists; the addition of lignocaine to adrenaline has a protective effect against cardiovascular complications due to the myocardial stabilizing property of lignocaine. The maximum infiltration dose of adrenaline is 5 to 10 µg/kg. However, this may be altered with simultaneous use of inhaled anesthetics.⁶ The inhalational agents have suppressive action on the myocardial conduction system and predispose to arrhythmias that are further accentuated by adrenaline.⁷ Desflurane has shown to cause

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cardiac adverse effects by increasing intracardiac catecholamine release and neurocirculatory activation.⁸ Desflurane causes increase interlead variability of QT interval, predisposing to arrhythmias. The relationship to sympathetic activation is unknown.⁹ Desflurane causes prolongation of the QTc interval in patients without cardiovascular diseases.¹⁰ We report a case of transient ventricular tachycardia in a patient following nasal infiltration with lignocaine adrenaline under desflurane anesthesia.

Case Description

A 46-year-old female presented to the hospital 1 week prior to the planned surgery with complaints of diminished vision of 2 months and generalized headache of 1 month. She had no known comorbidities and her general physical examination were unremarkable. Pre-operative blood investigation showed a high serum prolactin of 37.48 ng/mL, decreased serum cortisol of 0.17 µg/dl and normal renal function test, hemogram and electrolytes. Electrocardiogram (ECG) showed a heart rate (HR) of 80 beats per minute (bpm) with normal sinus rhythm (►Fig. 1A). Echocardiography was

normal with good systolic and diastolic function. Brain imaging was consistent with diagnosis of pituitary macroadenoma. She was planned for endoscopic transnasal transphenoidal tumor decompression. She was accepted under American Society of Anesthesiologists (ASA) physical status 2. On the day of surgery, her nasal cavity was prepared using xylometazoline nasal drops and she received IV 16 mg of dexamethasone. In the operating room, standard ASA monitors (5-lead ECG, noninvasive blood pressure, pulse oximetry, airway gas monitor) and entropy were connected and baseline parameters were recorded. A 18G IV line was started and connected to 0.9% normal saline. She was induced with IV injection of 140 µg fentanyl, 250 mg thiopentone, and 8 mg vecuronium. After ensuring adequate muscle relaxation, airway was secured with 7.5 mm outer diameter flexometallic tube that was fixed at 19 cm at the angle of the mouth. A throat pack was inserted. Anesthesia was maintained with desflurane and nitrous oxide in oxygen targeting entropy 40 to 50 and minimum alveolar concentration 1 to 1.2. Prior to surgical incision, nasal septal mucosa of patients was infiltrated with 5 mL of 2% lignocaine with 1 in 200000 adrenaline using 23-gauge needle and a nasal speculum. Within

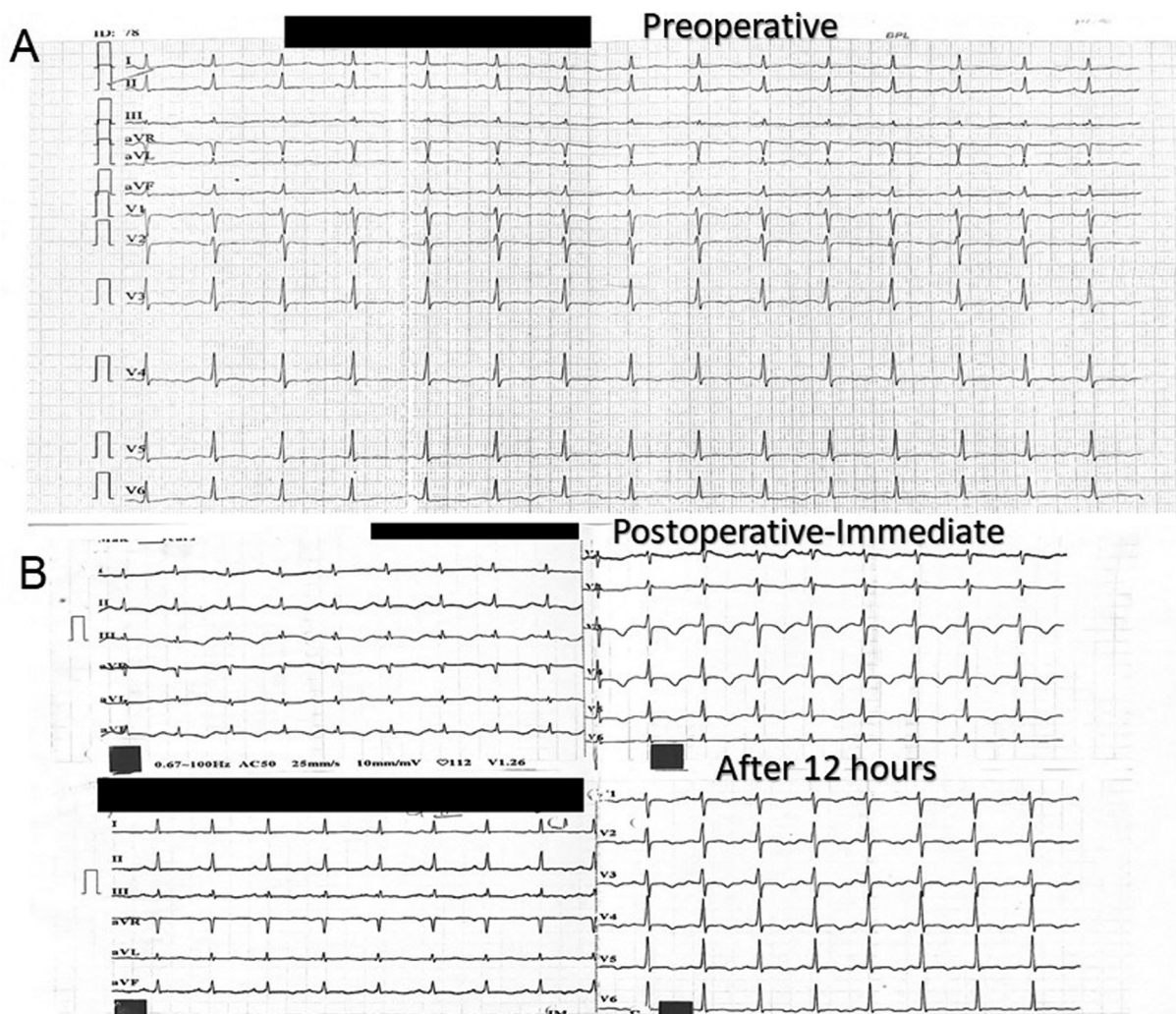


Fig. 1 (A) Preoperative electrocardiogram (ECG). (B) Immediate postoperative ECG.

60 seconds of the injection, the HR increased from 84 bpm to 200 to 210 bpm progressing to polymorphic ventricular tachycardia with no recordable blood pressure. The procedure was stopped and the patient was administered 100% O₂. The arrhythmias were transient and lasted for a period of about 60 seconds. IV lignocaine (120 mg) was injected that resulted in normal sinus rhythm with a HR of 67 bpm, noninvasive blood pressure of 215/109 mm Hg, oxygen saturation of 100%, end-tidal carbon dioxide of 35 mm Hg, and a state entropy of 42. Defibrillator was kept ready for the management of further recurrence of ventricular arrhythmias. The inhalational agent was changed to sevoflurane in oxygen and air. For the control of blood pressure, IV labetalol boluses of 5 mg at 5-minute interval were used twice. The HR was maintained around 80 to 90 bpm with sinus rhythm and blood pressure was 145 to 155/90 to 100 mm Hg. There was no further hemodynamic disturbances and the surgery proceeded without any further events. The patient was reversed and successfully extubated at the end of the procedure. She was shifted to the postanesthesia care unit and a 12-lead ECG was done that showed sinus tachycardia with QTc prolongation (460 ms vs. baseline QTc of 350 ms) and T-wave inversion in lead II, III, aVF, V3-V5 (► Fig. 1B). Arterial blood gas and electrolytes (sodium, potassium, magnesium, calcium) were all within normal limits. Troponin I was negative and two-dimensional echocardiogram revealed a normal cardiac function. She was started on treatment for hypertension and by third postoperative day her ECG changes reverted back to her preoperative status (► Fig. 2). She underwent reexploration and repair of the floor of anterior cranial fossa for cerebrospinal fluid rhinorrhea under anesthesia after 7 days, which was uneventful. She made a good clinical recovery with no further events in the hospital and was discharged 3 days after her second surgery.

Discussion

Infiltration with local anesthetic mixed with adrenaline is a common practice in endoscopic surgeries to provide better

visualization and reduce blood loss and toxic effects of local anesthetic.¹ The nasal mucosa has propensity to absorb injected adrenaline and cause rapid rise in plasma concentrations to more than 10 times its normal plasma levels.²

Infiltration under general anesthesia itself poses increased risk of inadvertent injection. In conscious patients, there is a tendency to use smaller needles and slower injection speed to reduce pain. In contrast, surgeons tend to use larger needles and inject faster in patients under general anesthesia. This can cause more rapid rise in plasma levels of adrenaline if inadvertent intravascular delivery occurs.³

Local anesthetics with adrenaline have the propensity to induce arrhythmia when used along with inhalational agents. This is due to the depressant activity on myocardium and interaction with alpha1 and beta adrenoreceptors. Previous studies have reported adrenaline-induced arrhythmias for surgeries involving the head and neck region under general anesthesia using sevoflurane or desflurane, even though these agents are less likely to cause an arrhythmia compared with other inhalational anesthetics like halothane. This is thought to be attributed to rapid absorption of adrenaline due to the presence of abundant blood flow in this region.⁵ Desflurane has been shown to be more arrhythmogenic compared to sevoflurane and the mechanism is mainly by its ability to prolong the QTc interval. In our patient, with a normal baseline QTc, a prolonged QTc developed due to the effect of desflurane that predisposed to the development of ventricular tachycardia with adrenaline infiltration.

Clinical evidence suggests that it is safe to use adrenaline up to a dose of 3 µg/kg without any significant cardiac effects. However, in the presence of additional risk factors like cardiovascular disease and hypertension, the dose needs to be reduced. The effect of other drugs also needs consideration. Occasionally, there may be hypersensitivity even in reduced doses.^{6,7}

The prompt detection and early treatment of adrenaline induced cardiovascular events are important in preventing

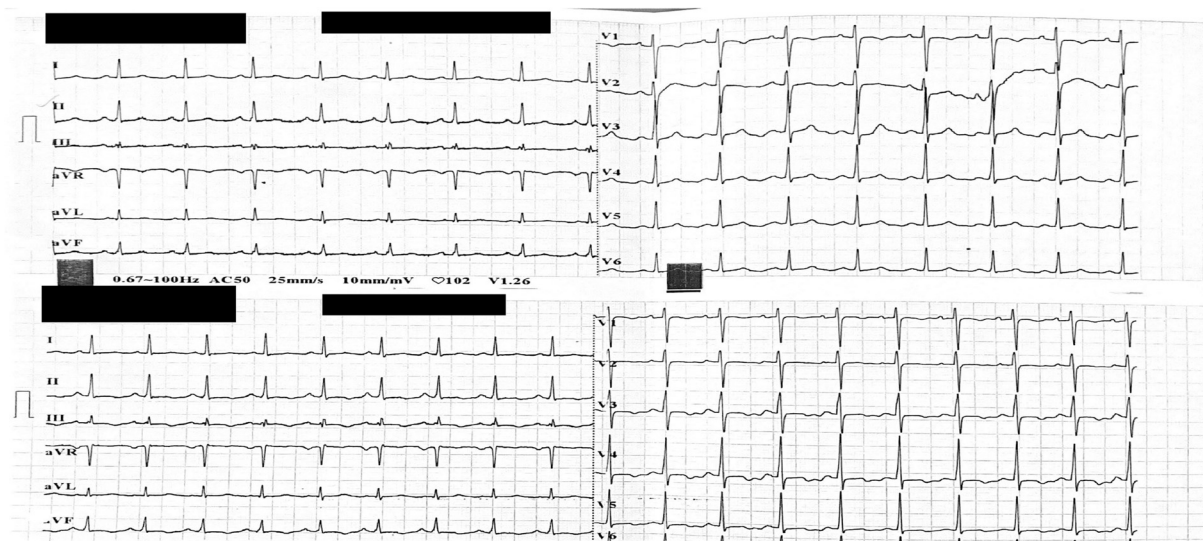


Fig. 2 Postoperative day 3 electrocardiogram.

adverse outcome. In addition, it is important to identify and eliminate contributing factors like desflurane in our case to prevent recurrence in the intraoperative period.

Conclusion

The arrhythmogenic potential of infiltrative local anesthetic with adrenaline can be potentiated by desflurane-induced QTc prolongation in patients undergoing transnasal surgery. Early detection and treatment can improve outcomes.

Conflict of Interest

None declared.

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