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CLINICAL REPORT

Conversion of atrial flutter to sinus rhythm during landiolol infusion

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Abstract A 73-year-old male patient with a past history of hypertension and atrial premature contraction underwent endoscopic restoration of the left bubonocele. Sinus rhythm was confirmed by preoperative electrocardiography, but paroxysmal atrial flutter developed when abdominoscopy was started. Continuous administration of landiolol hydrochloride at a dose of 0.005 mg kg^{-1} min^{-1} after a loading dose of 0.04 mg kg^{-1} min^{-1} for 1 min resulted in control of heart rate without a decrease in blood pressure. Atrial flutter was converted to sinus rhythm 3 min after the start of administration. Landiolol hydrochloride was administered continuously until the morning of the following day and sinus rhythm was maintained postoperatively.

Keywords Atrial flutter · Landiolol hydrochloride · Tachyarrhythmia

Introduction

Atrial flutter is associated with diseases such as hypertension, ischemic heart disease, pericarditis, atrial septal defect, and hyperthyroidism, and may be induced by surgical stress, pregnancy, hypoxemia, and electrolyte abnormalities, even in cases with no specific past history [1, 2]. Here, we report a case of atrial flutter during endoscopic restoration of a bubonocele, in which a decrease in heart rate, conversion to sinus rhythm, and perioperative management were achieved with the administration of landiolol hydrochloride. To our knowledge, this is the first report of the use of landiolol for rhythm control in atrial flutter.

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Case

The patient was a 73-year-old male of height 159 cm and weight 61.0 kg who visited a department of surgery for the chief complaint of bulging in the left inguinal region. He was diagnosed with left bubonocele and scheduled to undergo endoscopic restoration of the left bubonocele. His past history included hypertension, hyperlipidemia and atrial premature contraction (APC), and his surgical history included appendectomy when he was 10 years old. Preoperative blood and biochemical tests showed increases of AST to 45 U l^{-1}, ALT to 47 U l^{-1}, and T-Bil to 1.7 mg dl^{-1}. An electrocardiogram indicated sinus bradycardia (heart rate of 55 beats min^{-1}) and isolated APC. Echocardiography showed no abnormal findings in heart wall motion and cardiac valves, and the left ventricular ejection fraction was 68%. Amlodipine besilate, valsartan, pilsicainide hydrochloride, fluvastatin sodium, and rebamipide were administered at this time.

The patient received oral administration of amlodipine besilate (5 mg) and pilsicainide hydrochloride (50 mg) in the early morning of the day of surgery, but no preanesthetic medication was given. Upon entry to the operating room, blood pressure and heart rate were 148/101 mmHg and 92 beats min^{-1}, respectively, and electrocardiography showed sinus rhythm and APC that developed 5–6 times per minute. Total intravenous anesthesia was performed using target-controlled infusion (TCI) of propofol. Anesthesia was

induced with propofol, remifentanil, and vecuronium at 3 μg ml^{-1}, 0.25 μg kg^{-1} min^{-1}, and 6 mg, respectively. TCI of propofol was then performed at a dose of 2–3 μg ml^{-1}, remifentanil was administered at 0.1–0.3 μg kg^{-1} min^{-1}, and vecuronium (2 mg) was given as needed to maintain anesthesia. APC was eliminated after inducing anesthesia.

Upon commencement of the surgery, blood pressure and heart rate were stable at 110/70 mmHg and 60 beats min^{-1}, respectively. However, when aeroperitoneum with carbon dioxide was commenced about 5 min after the start of surgery, a flutter wave (F-wave) was observed after frequent onset of APC and atrial flutter developed in association with ventricular conduction at a ratio of 1:4, with a blood pressure of 90/63 mmHg and a heart rate of 87 beats min^{-1} (Fig. 1a). No change was observed after the anesthetic depth was adjusted with propofol (2.5–3 μg ml^{-1}) and remifentanil (0.2–0.3 μg kg^{-1} min^{-1}) for 5 min, and blood pressure gradually deteriorated. Thus, intravenous administration of landiolol hydrochloride was initiated at a dose of 0.04 mg kg^{-1} min^{-1} for 1 min and then maintained at 0.005 mg kg^{-1} min^{-1}. As a result, heart rate began to decrease 3 min after the start of administration (55 beats min^{-1}) and sinus rhythm was confirmed by electrocardiography (Fig. 1b). There was no decrease in blood pressure, and anesthesia was subsequently maintained by administration of landiolol hydrochloride at 0.005–0.01 mg kg^{-1} min^{-1} to ensure a heart rate of 60–70 beats min^{-1}.

No APC or atrial flutter was observed for the remainder of the surgery. Awakening and extubation were performed immediately after surgery. The operation time and anesthesia time were 1 h 50 min and 2 h 40 min, respectively; the infusion volume of crystalloid fluid and the urine volume were 800 and 210 ml, respectively; and the hemorrhage level was low. After surgery, administration of landiolol hydrochloride was continued until the following day at 0.0025 mg kg^{-1} min^{-1} and no fast atrial flutter developed.

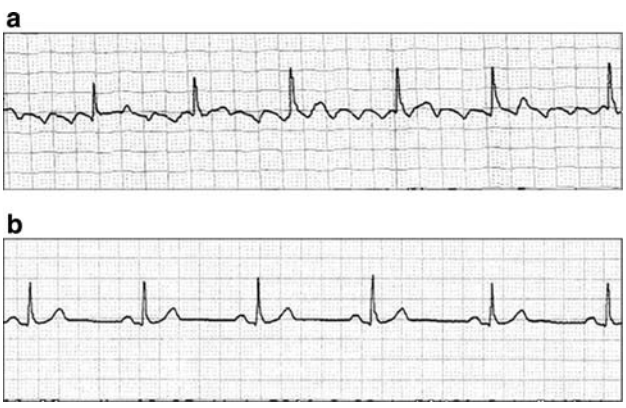

Fig. 1 Intraoperative monitor electrocardiograms (II induction): **a** at commencement of landiolol hydrochloride administration, and; **b** after landiolol hydrochloride administration

Discussion

Atrial flutter is associated with many diseases, but the common mechanism of development is thought to be dysfunction of atrial reentry caused by excessive secretion of catecholamines induced by sympathetic stimulation [1, 2]. Our patient had sinus rhythm but also a past history of hypertension and APC, and we believe that atrial flutter developed due to stimulation by abdominoscopy during surgery.

Rhythm control for conversion to sinus rhythm and control of heart rate are performed for treatment of patients with atrial flutter, as in treatment for atrial fibrillation [1]. Na-channel blockers are typically used for rhythm control, and β-blockers, Ca-channel blockers, and digoxin are used for rate control [3]. Atrial flutter was evident in the electrocardiogram in our case, but the pulse rate only increased to 80 and did not reach a level that required rapid lowering of the pulse. However, because atrial flutter persisted, we anticipated a fall in blood pressure and considered treatment to restore sinus rhythm. Since sympathetic nerve strain was thought to be a cause of the arrhythmia, β-blockers were the initial choice of drug. Due to their cardiodepressant properties, β-blockers may not be appropriate in a case with reduced cardiac function, but our patient showed no evidence of severe heart failure. Furthermore, administration of Na-channel blockers may decrease atrial deflection and lead to a risk of further tachycardia with 1:1 atrioventricular conduction [3].

Based on this background, the use of landiolol hydrochloride seemed appropriate as a β-blocker that does not cause a significant fall in blood pressure or cardiac function [4, 5]. Landiolol is a short-acting β1-selective blocker characterized by a short half-life (4 min) and good stability in continuous administration [4]. The drug has been used for treatment of perioperative tachyarrhythmia in Japan [5–8], and successful control of heart rate and conversion to sinus rhythm in patients with atrial fibrillation has also been achieved with landiolol [8]. A similar clinical course occurred in our patient with atrial flutter, with sinus rhythm obtained when arrhythmia was controlled by administration of landiolol. We note that esmolol [9, 10] is widely used in Europe and the US and is considered to be effective for defibrillation, and this may be one of the characteristics of β-blockers. However, it has also been suggested that landiolol is not effective for defibrillation in patients with atrial flutter-fibrillation [11], and that the effectiveness may depend on the type of atrial flutter-fibrillation; that is,

chronic or acute phase. This issue requires examination in future studies, but we suggest that the success rate of conversion to sinus rhythm may be high in cases with comparatively early phase atrial flutter, as in the current case.

The 2007 ACC/AHA guidelines for perioperative evaluation of patients undergoing noncardiac surgery recommend the use of β-blockers for patients with angina pectoris or hypertension, those with supraventricular tachycardia such as atrial flutter-fibrillation in which heart rate cannot be controlled, and those with a high risk of myocardial ischemia who are undergoing vascular surgery [12]. In addition, past studies have shown that β-blockers improve the long-term and lifespan prognosis after noncardiac surgery [13–15], and therefore the use of β-blockers in the perioperative period is likely to attract further attention.

It is possible that the conversion to sinus rhythm in our patient was caused by spontaneous recovery or other effects, and not by landiolol. Oral amlodipine besilate and pilsicainide hydrochloride were administered before surgery, but atrial flutter developed during surgery and was converted to sinus rhythm immediately after the administration of landiolol hydrochloride. Therefore, it is very likely that conversion was induced by landiolol. Landiolol also induced an immediate decrease in heart rate and conversion to sinus rhythm occurred without a change in blood pressure. Redevelopment of atrial flutter may also have been prevented and stable hemodynamics maintained by continuous administration of low-dose landiolol after conversion to sinus rhythm. Based on these findings, landiolol hydrochloride appears to be effective and safe for perioperative management and rhythm control in cases of atrial flutter.

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