

Pharmacological Cardioversion by Intravenous Amiodarone for Primary Treatment of a Neonatal Atrial Flutter

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Abstract

Atrial flutter is an uncommon arrhythmia in newborn patients, but it can be fatal in intractable cases, which require emergent therapy with multiple antiarrhythmic therapies and an electrical cardioversion. Amiodarone is an antiarrhythmic drug commonly used to treat tachyarrhythmias of supraventricular tachycardia and atrial tachyarrhythmia in adults. The efficacy of amiodarone therapy for neonatal tachyarrhythmia including atrial flutter is currently unknown, but its estimated effectiveness is low with small experiences in neonates. We present a case of successful pharmacologic cardioversion, using intravenous amiodarone, of atrial flutter and did not require electrical cardioversion in a newborn. We suggest that intravenous amiodarone administration can be used to treat neonatal atrial flutter as effective pharmacological therapy in patient with hemodynamic stability.

Keywords: Amiodarone; Neonatal atrial flutter; Neonatal tachyarrhythmia

Introduction

Neonatal atrial flutter is a rare arrhythmia with a 3% incidence in newborns but a higher (30%) incidence in fetuses [1]. Most atrial flutter cases in neonates have a benign course, but some cases that occur along with congenital heart disease can result in a high risk of an intractable tachycardia attack with hemodynamically cardiac collapse. In a small number of neonates, atrial flutter appears spontaneously with or without congenital heart disease and results in unexpected complications such as cardiac ischemia, heart failure, hypotension, and cardiac arrest. The first choice of treatment is rate control by electrical cardioversion in hemodynamically unstable patients combined anticoagulation with warfarin. Recurrent atrial flutter can be unresponsive to electrical cardioversion, resulting in the need for additional antiarrhythmic drugs [2,3]. Amiodarone is an antiarrhythmic drug commonly used to treat tachyarrhythmias of supraventricular tachycardia and atrial fibrillation in adults, but unknown in newborns because of limited therapeutic experience of its efficacy and the low incidence of neonatal atrial flutter. Here, we report an atrial flutter with complete termination by intravenous amiodarone as primary therapy without using an electrical cardioversion and anticoagulation in a hemodynamically stable neonatal case.

Case Report

A male neonate was referred to the neonatal intensive care unit at Jeju National University Hospital for a tachypnea at birth. He was born with a birth weight of 4.2 kg at 38 weeks gestation by a repeated cesarean delivery. His mother had a history of hypothyroidism treated with synthroxine and type 2 diabetes mellitus treated with insulin during pregnancy. There was no perinatal history of a fetal arrhythmia or fetal hydrops. At admission, he had a mild chest retraction with tachypnea without cyanosis. The initial heart rate was 133 beats/min, with a respiratory rate of 48 beats/min, a body temperature of 36.4°C,

and pulse oximetry saturation of 98% with room air. The patient's blood glucose level was 65 mg/dl. Chest X-ray revealed no active lung lesions or cardiomegaly. Two-dimensional echocardiogram showed a patent foramen ovale. On the second day of life, tachycardia appeared abruptly with a heart rate up to 228 beats per minute, along with a blood pressure of 71/42 mmHg and tachypnea (respiratory rate of 70 breaths per minute). A 12-lead electrocardiogram was then performed (Figure 1).



Figure 1: An initial electrocardiogram showing narrow QRS tachycardia and heart rate of 227 beats per minute.

Intravenous adenosine administration (0.2 mg/kg) showing a saw-tooth pattern confirmed typical atrial flutter with differentiated a diagnosis of supraventricular tachycardia on the electrocardiogram (Figure 2). As primary therapy of rate control, intravenous amiodarone of loading dosage (5 mg/kg for 1 h) was once infused without using direct current electrical cardioversion, and this treatment successfully converted atrial flutter to a normal sinus rhythm without inducing complications such as hypotension (Figure 3). A follow-up electrocardiogram showed intermittent premature atrial complexes with aberrant conduction without recurrence of the atrial flutter (Figure 4). Intravenous amiodarone with maintenance dosage (5 ug/kg/min) was infused with combined oral propranolol (1mg/kg/dose, four times per a day) until premature beats have disappeared

completely. The patient was discharged with only oral propranolol on 17 days after birth, which was discontinued oral propranolol 2 months later. During followed 2 yrs, patient showed complete resolution of arrhythmias without recurrence of atrial flutter.



Figure 2: After adenosine administration, a typical saw-tooth atrial flutter shape (narrow arrow) appeared with a 2:1 atrioventricular conduction ratio.



Figure 3: Intravenous amiodarone infusion converted atrial flutter to a normal sinus rhythm.

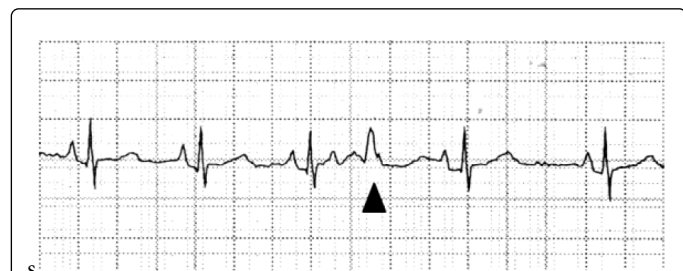


Figure 4: A follow-up electrocardiogram showed intermittent premature atrial complexes with aberrant conduction (arrowhead) without recurrence of atrial flutter.

Discussion

Atrial tachycardia, including atrial flutter, is often observed during the fetal stages but usually resolves after birth [1,4]. Diagnosis of atrial flutter is usually based on surface electrocardiogram results, and most patients are rapidly diagnosed. A characteristic electrocardiogram of atrial flutter shows rapid atrial rates of 250-600 beats/minute and atrioventricular conduction ratios of 2:1, 3:1 or 4:1 in neonates. Sometimes atrioventricular conduction ratio of 1:1 in atrial flutter makes its differentiated diagnosis of supraventricular tachycardia difficult in neonatal cases (Figure 2). Therefore, adenosine is helpful for easy diagnosis of atrial flutter because its atrioventricular conduction

blockage can induce a typical saw-tooth pattern of atrial flutter in also our case. The mechanism of atrial flutter consists of a reentrant in the right atrium circling the tricuspid valve annulus. Although most cases of atrial flutter are believed to be triggered by atrial stress secondary to congenital heart disease, surgery, or volume overload, the mechanism of most neonatal atrial flutter cases in normal hearts remains unknown [3]. In our case, his mother had hypothyroidism with synthroxine, but his mother and his thyroid function test were normal. It was likely to have a relation between patent foramen ovale on his echocardiogram and his atrial flutter by left to right shunt.

According to previous studies, mortality of newborns with atrial flutter is uncommon, and in children, its prognosis had a wide spectrum from asymptomatic outcome to sudden death [5,6]. Morbidity and mortality of neonates with atrial flutter depend on age at presentation, cardiac anatomy, ventricular function, prompt recognition of the arrhythmia, and initiation of adequate therapy [2]. Sustained tachyarrhythmia with atrial flutter in neonates presents heart failure with risk of sudden death. Direct current electrical cardioversion is generally prescribed as the first choice treatment in hemodynamically unstable patients [3-6]. Patients treated by electrical cardioversion should also undergo anticoagulation for risk of thromboembolism [3].

The efficacy of antiarrhythmic drugs on arrhythmia may be low, and the risks of side effects in neonates could be high. Nevertheless, antiarrhythmic drugs such as sotalol, propranolol, procainamide, and amiodarone have been used to treat intractable atrial flutter and prevent recurrence of arrhythmia in children and neonates [6-9]. Amiodarone is a class III antiarrhythmic agent which prolongs the refractory period of the atrioventricular node. This drug had been used commonly in supraventricular tachycardia and ventricular tachyarrhythmia in adults but rarely used in neonatal cases. It has been used in pediatric patients for nearly two decades with small experience. Etheridge et al evaluated the efficacy and safety of amiodarone in infants (average age 1 month) with supraventricular tachycardia. They assessed the safety and efficacy of amiodarone as primary therapy for supraventricular tachycardia in infancy. 50% of infants with amiodarone therapy had complete resolution. The remaining patients with amiodarone and propranolol were asymptomatic and 90% of them were in sinus rhythm [10]. A number of investigators have achieved results that amiodarone could play a role in the management of refractory arrhythmia in children and infants.

Conclusion

Our neonatal case was successfully treated by intravenous amiodarone and did not need using electrical cardioversion. Also, our case had no adverse effects of amiodarone such as hypotension, vomiting, thyroid function changes, and QT prolongation because of short therapeutic time [11]. In conclusion, we agree that babies with atrial flutter who are hemodynamically unstable should be treated by electrical cardioversion as a first-line therapy. However, amiodarone therapy without electrical conversion may be effective and safe therapeutic option for stable neonates with atrial flutter. We have limitations of experience in single medical center and additional studies are needed to fully determine the efficacy of amiodarone therapy for neonatal atrial flutter.

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