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Research Article

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Successful Reversal of Tachycardia Induced Cardiomyopathy After Flecainide Therapy in an Infant With Focal Atrial Tachycardia

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Abstract

Introduction: *Focal atrial tachycardia (FAT) is an uncommon but clinically important cause of persistent supraventricular tachycardia in infancy and may lead to tachycardia-induced cardiomyopathy (TIC) when rhythm control cannot be achieved. Early recognition and effective rhythm control are essential to prevent progressive myocardial dysfunction.*

Case Presentation: *We report a 4.5-month-old former preterm infant with structurally normal heart anatomy who presented with persistent narrow QRS atrial tachycardia detected on Holter monitoring. Initial treatment with propranolol followed by continuous intravenous amiodarone achieved partial rate control but failed to restore sinus rhythm. During follow-up, progressive myocardial dysfunction developed, with a decline in left ventricular ejection fraction from 66% to 58%, left ventricular dilatation, and worsening atrioventricular valve regurgitation. Because of persistent arrhythmia and evolving TIC, flecainide therapy was initiated in combination with propranolol. Sinus rhythm was rapidly restored after flecainide administration, and subsequent Holter monitoring confirmed rhythm stability without recurrent tachycardia. Echocardiographic follow-up demonstrated improvement in ventricular systolic function and marked regression of valvular insufficiency. Clinically, feeding intolerance, reduced activity, and overall functional status improved significantly.*

Conclusions: *This case highlights the importance of early rhythm control in infantile FAT and demonstrates that partial rate control alone may be insufficient to prevent ongoing myocardial injury. Prompt restoration of sinus rhythm may be crucial for reversing TIC and achieving complete functional recovery. In addition, limited regional availability of flecainide represented a significant therapeutic challenge during management.*

Keywords: Flecainide, Focal Atrial Tachycardia, Infant, Pediatric Arrhythmia, Supraventricular Tachycardia, Tachycardia-İnduced Cardiomyopathy

1. Introduction

Focal atrial tachycardia (FAT) accounts for a relatively small proportion of supraventricular tachycardias in infancy but represents an important cause of persistent tachyarrhythmia and tachycardia-induced cardiomyopathy (TIC). Unlike reentrant supraventricular tachycardias, FAT is generally caused by abnormal automaticity, making rhythm control more difficult and reducing responsiveness to conventional antiarrhythmic strategies aimed primarily at ventricular rate reduction.

Infants are particularly vulnerable to the hemodynamic consequences of sustained tachycardia because of limited myocardial reserve and immature ventricular compliance. Prolonged tachyarrhythmia may therefore rapidly lead to ventricular dysfunction, chamber dilatation, and secondary atrioventricular valve insufficiency. Importantly, early recognition and restoration of sinus rhythm may result in near-complete reversal of myocardial dysfunction.

We report a former preterm infant with drug-resistant FAT complicated by evolving TIC, in whom restoration of sinus rhythm with flecainide resulted in rapid echocardiographic and clinical recovery.

2. Case Report

A 4.5-month-old infant born at 33+4 weeks of gestation with a birth weight of 1480 g was referred to our pediatric cardiology department after atrial tachycardia was identified on ambulatory Holter monitoring performed during routine follow-up.

At admission, body weight was approximately 4.0–4.3 kg. Physical examination demonstrated persistent tachycardia without signs of severe hemodynamic compromise. Electrocardiography revealed narrow QRS tachycardia with variable R–R intervals (Figure 1). Initial Holter monitoring demonstrated a markedly fluctuating heart rate ranging from 100 to 210 beats per minute, with near-continuous non-sustained atrial tachycardia dominating the entire recording, indicating a high arrhythmic burden consistent with focal atrial tachycardia (Figure 2).

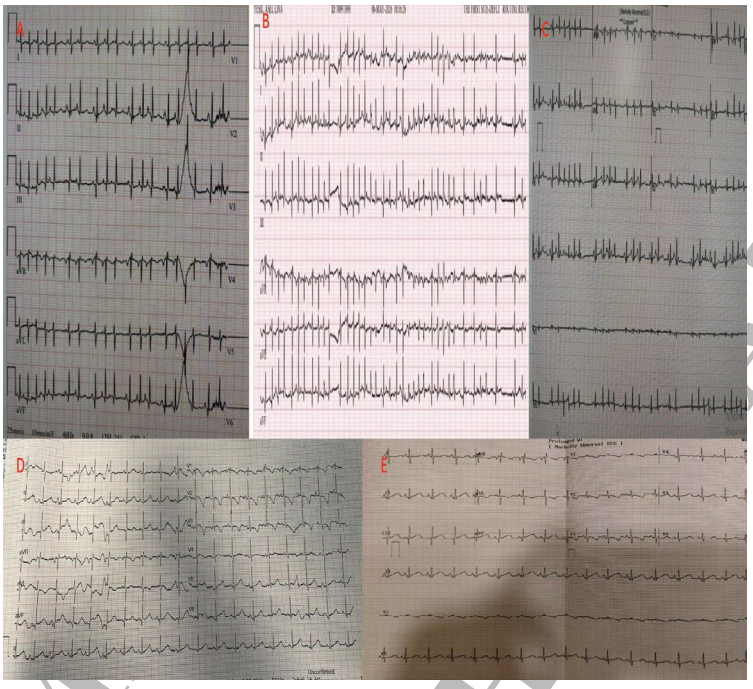

Figure 1: (A–C) Electrocardiographic recordings demonstrating narrow QRS tachycardia with irregular R–R intervals, consistent with atrial tachycardia. **(D–E)** Recordings obtained following flecainide therapy, showing stable sinus rhythm. Mild QTc prolongation is noted in panel (E), most likely related to prior amiodarone exposure

Initial transthoracic echocardiography demonstrated structurally normal cardiac anatomy except for a small secundum atrial septal defect (3 mm, left-to-right shunt). Left ventricular systolic function was initially preserved with an ejection fraction (EF) of 66%. Mild-to-moderate tricuspid regurgitation and trivial mitral regurgitation were present

Propranolol therapy was initiated; however, sustained atrial tachycardia persisted. Because of ongoing arrhythmia burden, the patient was transferred to the pediatric intensive care unit for close rhythm monitoring and antiarrhythmic treatment. Intravenous amiodarone was administered with a loading dose of 5 mg/kg followed by continuous infusion at 5–7 mg/kg/day. Although partial ventricular rate control was achieved, sinus rhythm could not be restored (Figure 2). Tachycardia persisted despite improvement in rate variability

Serial echocardiographic assessment demonstrated progressive deterioration of myocardial performance. EF decreased from 66% to 58%, accompanied by left ventricular dilatation and increasing atrioventricular valve regurgitation, particularly involving the tricuspid valve These findings were considered compatible with evolving tachycardia-induced cardiomyopathy.

Because persistent tachycardia continued despite amiodarone therapy, amiodarone was discontinued and oral flecainide therapy was initiated at 100 mg/m²/day divided into two doses in combination with propranolol Following initiation of flecainide, rapid restoration of sinus rhythm was achieved. Electrocardiography demonstrated stable sinus rhythm at approximately 107 beats/min (Figure 1). Repeat Holter monitoring revealed complete normalization with uninterrupted sinus rhythm throughout the entire recording and no evidence of recurrent atrial tachycardia (Figure 2). Notably, no QRS widening was observed under flecainide therapy, indicating good tolerability. During prior amiodarone infusion, transient QTc prolongation up to 0.50 seconds was observed; however, the infusion had already been discontinued within 24–36 hours. Serial electrocardiographic monitoring demonstrated normalization of QTc intervals, and QTc measurements were closely followed throughout the clinical course.

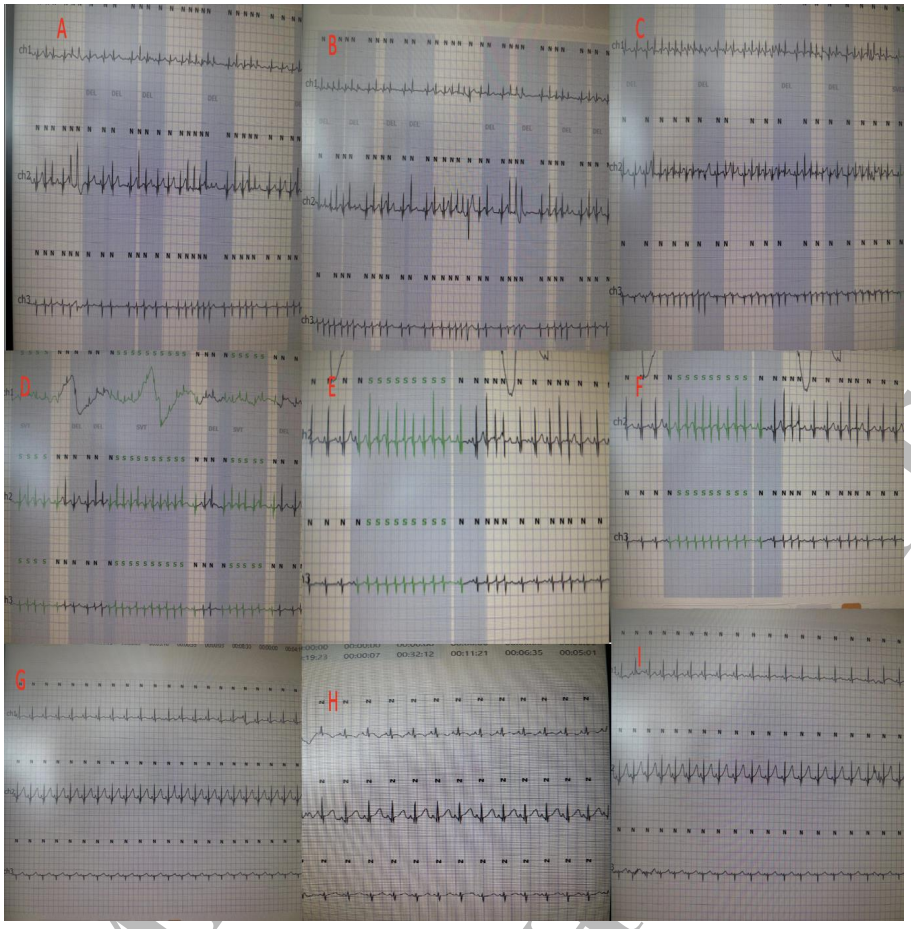

Figure 2: (A–F) Three-channel Holter recordings demonstrating a high arrhythmic burden with near-continuous non-sustained atrial tachycardia throughout the entire recording. The tracings show narrow QRS complexes with irregular R–R intervals, consistent with focal atrial tachycardia. (G–I) Follow-up Holter recordings obtained under combined flecainide and propranolol therapy, demonstrating complete normalization of rhythm with sustained sinus rhythm throughout the entire recording period

Subsequent echocardiographic examinations showed improvement in ventricular systolic performance together with regression of valvular insufficiency. Mitral regurgitation regressed to trivial levels, while tricuspid regurgitation improved to mild severity. Clinically, feeding tolerance, activity level, and overall well-being improved substantially, allowing transfer from intensive care to the pediatric cardiology ward.

At the time of discharge, the patient was clinically stable, with good feeding and normal activity for age. The patient was discharged on oral propranolol (2 mg/kg/day) and flecainide (100 mg/m²/day) therapy. Close outpatient follow-up was arranged, and the patient has been monitored regularly in our clinic without recurrence of symptoms. Electrophysiological study and possible catheter ablation were planned for a later period after adequate weight gain. An additional challenge during management was the limited availability of flecainide in our country, requiring acquisition through special arrangements before treatment initiation.

3. Discussion

This case demonstrates several clinically important aspects of infantile focal atrial tachycardia (FAT) and its associated myocardial consequences.

FAT represents a distinct subgroup of supraventricular tachycardias characterized predominantly by abnormal automaticity rather than reentrant mechanisms, which makes it inherently less responsive to conventional rate-controlling therapies [1-4]. Consequently, pharmacologic strategies primarily targeting atrioventricular nodal conduction may reduce ventricular response without eliminating the arrhythmogenic focus itself. In the present case, both propranolol and continuous amiodarone infusion resulted in partial rate control; however, sinus rhythm could not be restored, and myocardial dysfunction continued to progress. This clinical course is consistent with prior pediatric series demonstrating limited efficacy of rate control alone in automatic atrial tachycardias [1,2]. An important and often underappreciated concept highlighted by this case is that **partial rate control may conceal ongoing myocardial injury.** Despite apparent improvement in heart rate, serial echocardiographic evaluation revealed progressive deterioration in left ventricular systolic function, chamber remodeling, and increasing atrioventricular valve regurgitation. These findings

suggest that the cumulative burden of tachyarrhythmia, rather than ventricular rate alone, plays a critical role in the development of myocardial dysfunction. Similar observations have been reported in pediatric populations, where arrhythmia burden was identified as a key determinant of myocardial recovery [5,6].

Tachycardia-induced cardiomyopathy (TIC) is a well-recognized but potentially reversible cause of myocardial dysfunction resulting from sustained tachyarrhythmia. Experimental and clinical data have demonstrated that prolonged tachycardia leads to impaired calcium homeostasis, myocardial energy depletion, neurohormonal activation, and progressive ventricular remodeling, often accompanied by functional atrioventricular valve regurgitation [7]. Contemporary studies further emphasize that timely elimination of the arrhythmia substrate is the most critical factor for myocardial recovery, particularly in pediatric patients [5,6].

Infants are especially vulnerable to the deleterious effects of sustained tachycardia due to immature myocardial structure and limited compensatory reserve. In this context, even relatively short durations of persistent tachyarrhythmia may result in rapid deterioration of ventricular function. Current supraventricular tachycardia guidelines acknowledge that younger patients may develop ventricular dysfunction more rapidly and therefore may require more aggressive rhythm-control strategies [8,9].

Flecainide played a pivotal role in rhythm restoration in the present case. Its efficacy in automatic atrial tachycardias has been well documented, particularly in pediatric populations where first-line therapies fail [1,10,11]. In addition, contemporary guidelines and expert consensus statements support the use of class IC agents, including flecainide, in selected pediatric supraventricular tachyarrhythmias under appropriate monitoring conditions (8,9,12). In our patient, initiation of flecainide resulted in prompt restoration of sinus rhythm, which was followed by rapid clinical and echocardiographic improvement.

The marked recovery of left ventricular systolic function and regression of mitral and tricuspid regurgitation after restoration of sinus rhythm strongly support TIC as the underlying mechanism of myocardial dysfunction. This reversible pattern is clinically significant, as progressive valvular insufficiency in infants with FAT may otherwise be misinterpreted as primary structural pathology rather than secondary functional remodeling due to ventricular dilation.

Another important aspect of this case is the limited availability of flecainide in certain healthcare settings. In many regions, access to pediatric antiarrhythmic medications remains restricted, potentially delaying effective rhythm control and prolonging exposure to tachyarrhythmia. Although rarely addressed in the literature, this represents a significant real-world challenge that may directly impact clinical outcomes in pediatric arrhythmia management.

Taken together, this case underscores the importance of an **early rhythm-control strategy** in persistent infantile FAT. While rate control may provide temporary hemodynamic stabilization, it may not be sufficient to prevent progressive myocardial dysfunction. Restoration of sinus rhythm appears to be the key determinant of myocardial recovery, in line with current pediatric electrophysiology recommendations [8,9,12].

This report has some limitations. As a single case report, the findings cannot be generalized to all infants with focal atrial tachycardia. Nevertheless, it highlights an important clinical message regarding the potential inadequacy of rate control alone and the benefits of timely rhythm control in preventing tachycardia induced cardiomyopathy.

4. Conclusion

Persistent focal atrial tachycardia in infancy may rapidly lead to tachycardia-induced cardiomyopathy despite partial ventricular rate control. Early restoration of sinus rhythm can result in significant reversal of myocardial dysfunction and secondary valvular insufficiency. Flecainide may be highly effective in refractory infantile FAT; however, limited availability of this agent remains an important therapeutic challenge in certain regions.

Declarations

Ethical Approval

This case report was conducted in accordance with the ethical principles of the Declaration of Helsinki. According to institutional policy, formal ethical approval was not required for the publication of a single case report.

Consent to Participate

Written informed consent to participate was obtained from the patient's parents.

Consent to Publish

Written informed consent for publication of this case report and any accompanying images was obtained from the patient's parents.

Data Availability

The data supporting the findings of this case report are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare that they have no competing interests.

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References

1. Kang, K. T., Etheridge, S. P., Kantoch, M. J., Tisma-Dupanovic, S., Bradley, D. J., Balaji, S., ... & Sanatani, S. (2014). Current management of focal atrial tachycardia in children: a multicenter experience. Circulation: *Arrhythmia and Electrophysiology, 7*(4), 664-670.
2. Salerno, J. C., Kertesz, N. J., Friedman, R. A., & Fenrich, A. L. (2004). Clinical course of atrial ectopic tachycardia is age-dependent: results and treatment in children< 3 or≥ 3 years of

age. *Journal of the American College of Cardiology, 43*(3), 438-444.
3. GILLETTE, P. C., & GARSON, A. (1977). Electrophysiologic and pharmacologic characteristics of automatic ectopic atrial tachycardia. *Circulation, 56*(4), 571-575.
4. Sancaktar, A., Gömeç, T. B. Ö., & Ersayoğlu, İ. (2026). Successful Reversal of Tachycardia Induced Cardiomyopathy After Flecainide Therapy in an Infant With Focal Atrial Tachycardia.
5. Moore, J. P., Patel, P. A., Shannon, K. M., Albers, E. L., Salerno, J. C., Stein, M. A., .& Balaji, S. (2014). Predictors of myocardial recovery in pediatric tachycardia-induced cardiomyopathy. *Heart Rhythm, 11*(7), 1163-1169.
6. Moore, J. P., Shannon, K. M. (2020). Tachycardia-induced cardiomyopathy in children. *Curr Opin Cardiol. 35*:92–97.
7. Fenelon, G., Wijns, W., Andries, E., Brugada, P. (1996). Tachycardiomyopathy: mechanisms and clinical implications. *Pacing Clin Electrophysiol. 19*:95–106.
8. Brugada, J., Blom, N., Sarquella-Brugada, G. (2020). Supraventricular tachycardia in children: current management. *Eur Heart J. 41*:425–435.
9. Brugada, J., Katritsis, D. G., Arbelo, E. (2019). ESC guidelines for the management of patients with supraventricular tachycardia. *Eur Heart J. 41*:655–720.
10. Lindinger, A., Heisel, A., Von, Bernuth, G. (1998). Permanent junctional reciprocating tachycardia and automatic atrial tachycardia in infants: efficacy of flecainide therapy. *Eur Heart J. 19:*148–151.
11. Kugler, J. D., Danford, D. A. (2021). Pediatric arrhythmias and current treatment strategies. *Curr Cardiol Rep.* 23:1–9.
12. Saul, J. P., Kanter, R. J., Abrams, D. (2016). PACES/HRS expert consensus statement on the use of catheter ablation in children and patients with congenital heart disease. *Heart Rhythm*. 13:e251–e289.