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Atrial Flutter in a Newborn: A Case Report

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ABSTRACT

Introduction: Arrhythmias in the neonatal period are not uncommon and may occur in neonates with a normal heart or in those with structural heart disease. The incidence of neonatal arrhythmia is reported to be 1%–5% in all neonates. Atrial flutter (AFL), a rhythm disturbance typically caused by a reentry mechanism, accounts for nearly 32% of neonatal arrhythmias. In most cases, the condition can be reverted to sinus rhythm through antiarrhythmic therapy, transesophageal overdrive pacing, or synchronized electrical cardioversion.

Case Presentation: We describe a late preterm male infant, born at 34 weeks of gestation, who required two NICU admissions. The first was immediately after delivery due to prematurity, polycythemia, and hyperbilirubinemia. The second occurred at 24 days of life for irritability and poor feeding. On examination, he exhibited tachypnea and tachycardia with an irregular rhythm, reaching a heart rate of 205 bpm. Electrocardiography (ECG) revealed a supraventricular tachyarrhythmia, while echocardiography confirmed normal cardiac structure. The patient was initially treated with adenosine, suspecting supraventricular tachycardia; however, post-treatment ECG demonstrated atrial flutter with 3:1 atrioventricular conduction. Synchronized electrical cardioversion was then performed, successfully restoring sinus rhythm at 124 bpm. The infant was later diagnosed with a Klebsiella urinary tract infection, treated appropriately, and discharged in stable condition.

Conclusions: Atrial flutter represents a significant neonatal arrhythmia associated with rapid ventricular rates. If not promptly managed, it may lead to cardiac failure. In cases resistant to medication, electrical cardioversion is often required to achieve rhythm normalization and prevent complications. Sepsis—including urinary tract infections (which was evident in our case)—may precipitate atrial flutter by increasing sympathetic activation, inflammatory cytokine release, and myocardial irritability.

Keywords: *Atrial Flutter, Neonate, Antiarrhythmic Therapy, Synchronized Cardioversion*

Introduction

Cardiac rhythm disturbances occur in about 1% of neonates and 1–3% of pregnancies during the third trimester. Among neonatal arrhythmias—sinus tachycardia, atrioventricular re-entrant tachycardia,

and atrial flutter (AFL)—the latter accounts for nearly one-third of cases, though it is uncommon beyond infancy. AFL arises from a re-entry mechanism and is identified on ECG by characteristic saw-tooth flutter waves, best seen in leads II, III, and aVF, with atrial rates of 280–450 bpm and typically 2:1 atrioventricular conduction (Southall *et al*., 1980)

Case Presentation

A late preterm male infant, born at 34 weeks C.S delivery, did not require resuscitation at birth, was initially admitted to the neonatal intensive care unit (NICU) for management of prematurity, polycythemia, and hyperbilirubinemia. Baby was treated for 5 days and was discharged home in healthy condition. At 24 days of age, the baby was readmitted due to irritability and poor feeding interval between the two admissions baby was asymptomatic. On arrival, he presented with tachypnea and tachycardia, baby was admitted considering possibility of late onset sepsis. Sepsis markers were withdrawn including blood and urine culture. The heart rate was 196 bpm and ECG showed non sinus tachycardia (supraventricular tachyarrhythmia), oxygen saturation ranged from 98–99%, and blood pressure measured 80/48 mmHg. Clinical examination confirmed tachypnea (60 breaths/min) and tachycardia (205 bpm), with no cardiac murmur. The liver edge was palpable 2 cm below the right costal margin, soft and non-tender. The infant received adenosine 200 µg/kg, after which the ECG revealed atrial flutter with 3:1 atrioventricular conduction. Following consultation with a pediatric cardiologist, synchronized direct current (DC) cardioversion was performed, which successfully terminated the arrhythmia and restored sinus rhythm (Fig. 1-2).

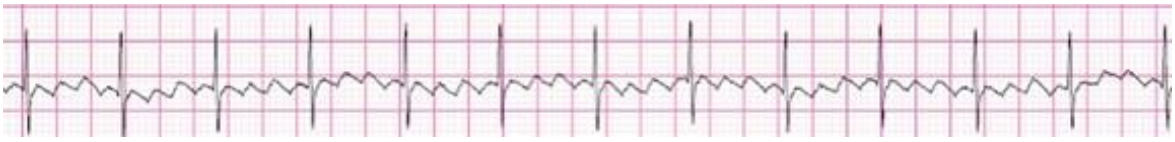

Figure 1: ECG showing atrial flutter.

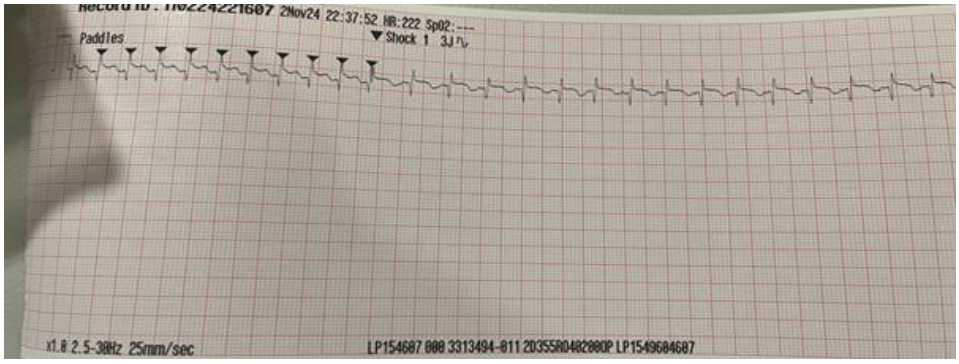

Figure 2: ECG showing effect of synchronized cardioversion.

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No recurrences were observed during course of hospitalization, and no maintenance antiarrhythmic medication was prescribed. A sepsis evaluation revealed a positive urine culture for Klebsiella. Baby was treated with intravenous antibiotics for seven days. A functional echocardiogram showed normal cardiac structure and function. The baby had a stable clinical course and was discharged home in good condition.

Discussion

In the absence of structural cardiac abnormalities, atrial flutter is typically identified during the late stages of pregnancy or the early neonatal period (Kundak *et al*., 2013). The incidence appears to be equal in both sexes. AF in neonates is often well tolerated, and complications such as hydrops fetalis or fetal demise are rare. When fetal arrhythmia occurs before 36 weeks of gestation, maternal administration of antiarrhythmic agents such as digoxin or dexamethasone can be effective (Jaeggi and Öhman, 2016). In this case, the infant was born to a mother with gestational diabetes at 34 weeks' gestation. The initial NICU stay was uncomplicated, suggesting that atrial flutter developed postnatally, and therefore, maternal therapy was not required. Previous studies have shown an association between maternal diabetes, macrosomia, and atrial arrhythmias in fetuses and neonates (Spearman and Williams, 2014). Additionally, late-onset sepsis, as observed in this case, may have acted as a triggering factor. Atrial flutter is characterized by a macro-reentrant circuit within the atrial myocardium, with variable conduction to the ventricles, independent of the atrioventricular node. Because cardiac output depends directly on ventricular rate, excessive ventricular activation can result in reduced stroke volume and potential acute heart failure. Furthermore, stagnant blood flow due to rapid atrial contractions can lead to thrombus formation and subsequent embolism. The goal of acute therapy is to terminate the tachyarrhythmia or, if rhythm control is not achieved, to reduce the ventricular rate to maintain hemodynamic stability (Camm and Garratt, 1991). When AFL persists, transesophageal overdrive pacing can be employed to reestablish sinus rhythm. In this case, synchronized DC cardioversion successfully restored normal rhythm. Prophylactic anticoagulation was not initiated due to normal cardiac contractility and atrial size (Fenrich *et al*., 1995). The baby was discharged home in healthy condition without any maintenance anti-arrhythmic agents as ECHO was not suggestive of any structural heart disease. The recurrence rate of AFL is very low (<10%) and hence risks of chronic anti-arrhythmic medications (negative inotropy, pro-arrhythmia, electrolyte disturbances) outweigh potential benefits. and once sinus rhythm is maintained for 24–48 hours, long-term therapy is unnecessary (Price *et al*., 2002). Maintenance antiarrhythmics are reserved for the minority with recurrent flutter, heart failure, or identifiable substrates (e.g., accessory pathways)., where selected drugs such as digoxin or class III agents can reduce further episodes (Oeffl *et al*., 2023).

Conclusions

Atrial flutter is one of the most frequent high-ventricular-rate rhythm disorders in the neonatal period. Most cases can be effectively converted to sinus rhythm through synchronized cardioversion or transesophageal pacing. If left untreated, AF can result in heart failure; thus, prompt recognition and rhythm restoration are essential, especially in cases unresponsive to antiarrhythmic drugs, to prevent hemodynamic compromise and other complications.

Conflict of Interest: The authors declare no conflicts of interest.

Legal Consent: Consent from the family was requested, family approved

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