

Editorial Note on Cardiac Neonatal Atrial Flutter

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EDITORIAL

Twenty-five neonates (16 boys and 9 girls) who had atrial flutter were identified. Diagnosis was made on or before the first day of life in 18 (72%). Heart failure was present in 9 patients, and hydrops fetalis was present in another 5. Atrial and ventricular rates did not differ between symptomatic and asymptomatic patients. Atrioventricular conduction was variable in 16 patients, and documented 1:1 conduction occurred in 5. Digoxin was the initial drug therapy given to 21 patients, with 7 reverting to sinus rhythm. Electrical cardioversion (pacing or synchronized shock) was attempted in 13 of the 14 cases in which digoxin was not successful and was attempted as the first treatment in 3 cases. Sustained sinus rhythm was achieved in 9. Two infants died of complications from prematurity but without having been successfully converted to sinus rhythm. No patient had atrial flutter during long-term follow-up (median 23 months). Neonatal atrial flutter has significant morbidity but an excellent long-term prognosis.

Cardiac arrhythmias are an important aspect of fetal and neonatal medicine. Premature complexes of atrial or ventricular origin are the main cause of an irregular heart rhythm. The finding is typically unrelated to an identifiable cause and no treatment is required. Tachyarrhythmia most commonly relates to supraventricular reentrant tachycardia, atrial flutter, and sinus tachycardia. Several antiarrhythmic agents are available for the perinatal treatment of tachyarrhythmias. Enduring bradycardia may result from sinus node dysfunction, complete heart block and nonconducted atrial bigeminy as the main arrhythmia mechanisms. The management and outcome of bradycardia depend on the underlying mechanism.

Atrial flutter (AFL) is known to be a seldom type of fetal and neonatal arrhythmia. Although it could end in severe morbidities such as hydrops fetalis or even death, with early prenatal diagnosis and prompt therapeutic approaches the majority of AFL cases show good prognosis. Neonatal AFL might be resistant to first step therapies. Therefore, secondary agents like flecainide, amiodarone, sotalol and cardioversion, if required, could be influential in perinatal tachyarrhythmia. In addition, close follow-up even after discharge is very important to keep all follow-up appointments. Herein, we present three cases of fetal/neonatal AFL in light of the literature and discuss the characteristics, diagnosis and treatment options.

A pregnant female presented at 37 weeks of gestation in labor. The fetus was noted to be tachycardic on fetal monitor. Postnatally, the male neonate was still noted to be tachycardic with heart rates in the low 200 bpm range. EKG was consistent with tachycardia, but rhythm diagnosis was not definitively made. Echocardiogram with M-mode analysis clearly demonstrated 2:1 atrial to ventricular contraction pattern consistent with atrial flutter. The neonate was subsequently transferred to a tertiary pediatric hospital where the diagnosis of atrial flutter was confirmed.

We present a neonatal case with intractable atrial flutter that did not respond to digitalization and electrical cardioversion. Intravenous flecainide administration completely resolved the atrial flutter. Proarrhythmic effects were not induced by flecainide administration. Although the efficacy of flecainide for atrial flutter during the infantile or childhood period is low, intravenous flecainide is worth consideration as a treatment for atrial flutter, even in intractable cases as described here, during the neonatal period.

The clinical features and treatment of atrial flutter in eight infants (four male and four female) less than 2 months of age are presented. Atrial flutter was noted during the first week of life in six of the infants and between 6 and 8 weeks of life in the other two infants. Four of the eight infants had associated structural or functional cardiovascular disease, and in three infants a central venous pressure catheter was present in the atrium at the time atrial flutter was diagnosed. Classic flutter waves were apparent on 12-lead ECGs in only two infants. In six infants, flutter waves were not obvious on standard ECGs, but transesophageal electrogram recordings demonstrated the presence of atrial flutter with second degree atrioventricular block.

The atrial cycle length during flutter ranged from 135 to 180 ms (mean 149 ms; mean atrial rate 403 beats per minute); there was a 2:1 ventricular response to atrial flutter. Successful termination of atrial flutter was accomplished using three modes of electrical cardioversion in seven of the eight infants: direct current cardioversion in one, transvenous atrial pacing in one, and transesophageal atrial pacing in five. One asymptomatic infant converted to normal sinus rhythm 24 hours following digoxin administration. One infant had multiple atrial flutter recurrences and required chronic procainamide therapy. In seven of the eight

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infants, no recurrences have been noted in 6 months to 3 1/2 years of follow-up. These results demonstrate that atrial flutter

may be difficult to diagnose in infants with tachycardia unless transesophageal electrogram recording is utilized for evaluation.