

Atrial Flutter: A Rare Rhythm Disorder in Neonates

Vivel Zareth Arrieta Diaz*, Orlando Andrés Velandia González, Yojandrys Janeth Nájera Jaramillo and Yoselin Yuceth Rocha Jaramillo

Medicine Program, Rafael Núñez University Corporation, Cartagena de Indias, Colombia

***Corresponding Author:** Vivel Zareth Arrieta Diaz, Medicine Program, Rafael Núñez University Corporation, Cartagena de Indias, Colombia.

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Tachycardia in the neonatal period is defined by a sustained heart rate greater than 180 beats per minute (BPM). Asymptomatic and temporary heart rhythm disturbances occur constantly in the fetal and neonatal period; fetal tachyarrhythmias occur in 1 to 2% of all gestations and fetal atrial flutter is the second most common tachyarrhythmia among those diagnosed prenatally after supraventricular tachycardia and constitutes about 30% of all neonatal cardiac arrhythmias, being generally of idiopathic origin [1,2].

Atrial flutter (AF) is a supraventricular tachyarrhythmia characterized by rapid, disorganized atrial activation between 400 and 700 bpm, with subsequent deterioration of atrial mechanical function, in addition to having an atrial/ventricular rate ratio of 2: 1 or greater; likewise, it is an uncommon rhythm disorder, in which central venous pressure is elevated and cardiac output is decreased, which can cause fetal hemodynamic compromise, even leading to neonatal heart failure (20%) and in a more severe form, unexplained hydrops fetalis. The latter occurs in approximately half of the cases of sustained tachyarrhythmias and causes fetal death in 9% of untreated patients; in all cases, the exclusion of underlying cardiac lesion is mandatory. It should be taken into account that, given that most newborns present a normal cardiac anatomy, being that about 80% of patients are asymptomatic, and making the etiology of AFN is not well defined. However, with early prenatal diagnosis and appropriate treatment, most cases of neonatal atrial flutter show a good prognosis [1-4].

This arrhythmia is caused by myocardial immaturity and high right atrial pressures in the perinatal period; these are factors that favor the appearance of atrial reentry, which generates flutter in the fetus or neonate. The diagnosis of AF by electrocardiogram is simple, due to its characteristic sawtooth flutter waves, which are best seen in leads II, III and aVF. In the absence of structural heart disease, atrial fibrillation is seen almost exclusively in infants during the third trimester or at birth. It should be taken into account that it is also commonly presented in patients with congenital heart defects, making it necessary to rule out pathologies such as transposition of the great vessels, complex cyanotic heart defect, atrial septal defect, Ebstein's anomaly, pulmonary stenosis and tricuspid valve diseases; atrial dilatation being a common finding among them [2-5].

In the prenatal diagnosis of arrhythmias, fetal echocardiography is very useful because it allows the type of arrhythmia to be studied and the hemodynamic repercussion to be evaluated, which most frequently demonstrates 2:1 or variable atrioventricular conduction. The development of hydrops fetalis has been related to a lower gestational age at diagnosis and to the incessant nature of the arrhythmia, mainly in fetuses with atrial flutter with high ventricular response [2,5].

The goal of treatment of AFN is to restore sinus rhythm or slow the heart rate to avoid ventricular dysfunction; the type of treatment is going to depend on gestational age, fetal involvement, maternal status, and potential risk to both [1,5].

There are different approaches to the treatment of a fetus with tachycardia, such as watchful waiting, induction of labor in term pregnancies and administration of postnatal treatment, intrauterine treatment administered to the mother and direct fetal intrauterine treatment [1]. Expectant management is usually reserved for cases of nonsustained tachycardia without hemodynamic repercussions, as spontaneous reversal has been reported in 25% of cases. Intrauterine treatment will depend on the presentation of the tachyarrhythmia, whether it is sustained or not, and its hemodynamic repercussions, with varying degrees of heart failure, ventricular dysfunction and even hydrops fetalis [4]. During gestation, maternal therapy with antiarrhythmic drugs is the most commonly used treatment, until the 1990s amiodarone and quinidine therapy was the most widely used, currently digoxin is the drug used as the first line in fetal atrial flutter in most cases, due to its safety, with no association with fetal mortality or severe maternal toxicity, with a success rate of 59%; maternal treatment with digoxin has shown good results in AF in fetuses without hydrops; however, other antiarrhythmics, such as propranolol, amiodarone, or flecainide, are also useful. In cases with hydrops, the tendency is to administer sotalol associated with digoxin in doses of 80 mg every 12 hours, due to the limitation of digoxin to cross the placental barrier, increasing the effectiveness of the treatment to 93%. Maternal adverse events related to antiarrhythmics are more common than expected, including gastrointestinal symptoms in addition to rhythm disturbances that can affect up to 78% of patients, being more frequent in cases managed with combined therapies. However, most of these adverse events are mild and a dose reduction allows treatment to continue [1,2,4].

The most commonly used postnatal treatment, both stable and unstable, and with the highest rate of effectiveness in atrial flutter to date is synchronized electrical cardioversion, with success in 87% of cases, or transesophageal atrial overdrive pacing. Current energy recommendations for synchronized cardioversion of stable supraventricular tachycardia in children are 0.5 to 1 J/kg for the first attempt and 1 to 2 J/kg for subsequent attempts if the first is unsuccessful [1,2]. Antiarrhythmic drugs can be tried in a stable newborn. Although, it takes some time to restore sinus rhythm, the recommended drugs are digoxin with the addition of flecainide or amiodarone in case of no therapeutic effect; but it should be kept in mind that as neonatal atrial flutter may be resistant to conventional pharmacotherapy, the possibility of electrical cardioversion at any time should be kept in mind [3].

As for recurrences, they may recur within 2 weeks of starting treatment, even if well controlled when managed prenatally; maintenance of postnatal treatment will not be necessary when AF returns spontaneously to sinus rhythm or responds readily to electrical conversion. Recurrences usually occur within 24 hours of the initial episode, in patients with associated arrhythmias and those with associated structural abnormalities; these patients usually receive beta-blockers or digoxin for 6 to 12 months, although recurrences are usually uncommon [2]. In the absence of structural heart disease, if treated early, AF has a low risk of recurrence, with an excellent prognosis and no need for chronic antiarrhythmic therapy [4].

In conclusion, atrial flutter is a rare type of fetal and neonatal arrhythmia. The duration of the arrhythmia conditions the clinical presentation, since the longer the duration, the fetus may present hydrops and the neonate heart failure. Maternal therapy with antiarrhythmic drugs is the most commonly used treatment during gestation in FAN, and the most commonly used postnatal treatment is synchronized electrical cardioversion; early treatment leads to an excellent prognosis, making prophylaxis beyond the infant period unnecessary, since neonatal recurrence is rare.

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