CASE REPORT

Fetal atrial flutter – case report and therapeutic discussion

Flutter *atrial fetal – relato de caso e discussão terapêutica*

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DOI: 10.5935/2238-3182.20150050

ABSTRACT

Fetal tachycardia occurs in 0.4 - 0.6% of all pregnancies. Out of the sustained fetal tachyarrhythmias, the atrial flutter is the second most common. It causes fetal heart failure when not properly treated, and early pregnancy interruption may be required. The diagnosis of tachyarrhythmias is mainly achieved by intra-uterus echocardiography. The first choice treatment is the maternal oral administration of digoxin. The association with other antiarrhythmics such as sotalol or amiodarone is needed if there is no response to digoxin or if the fetus evolves with dropsy. In this case report, the treatment began with digoxin and because there was no response, it was associated with propafenone. Sotalol or amiodarone were not used due to maternal bradycardia and hypotension.

Key words: Atrial Flutter; Propafenone; Digoxin; Obstetric Labor, Premature.

RESUMO

A taquicardia fetal ocorre em 0,4-0,6% de todas as gestações. Das taquiarritmias fetais sustentadas, o flutter atrial é a segunda mais comum. Quando não tratada corretamente, causa insuficiência cardíaca fetal e pode ser necessária a interrupção precoce da gestação. O diagnóstico das taquiarritmias é feito, principalmente, pela ecocardiografia intraútero. O tratamento de primeira escolha é a administração oral materna de digoxina. Caso não haja resposta adequada ou o feto evolua com hidropisia, é necessário associar outro antiarrítmico como sotalol ou amiodarona. Neste relato, iniciou-se com digoxina; e, em função da ausência de resposta, foi associada propafenona. Sotalol ou amiodarona não foram usados devido à bradicardia e à hipotensão materna.

Palavras-chave: Flutter *Atrial; Propafenona; Digoxina; Trabalho de Parto Prematuro.*

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INTRODUCTION

Fetal tachycardia was first recognized in 1930 by Hyman et al.[1] Its occurrence is in between 0.4 and 0.6% of pregnancies. The diagnosis and management of fetal arrhythmias have grown substantially over the past three decades. Sustained tachycardia may be responsible for relevant fetal clinical alterations such as dropsy and even death. Among them, the atrial flutter is the second most common, occurring in one-third to one-fifth of cases and, although infrequent, it is considered a serious disturbance in fetal heart rate.

This report describes an uneventful pregnancy until the 29th week when fetal atrial flutter was diagnosed with concomitant preterm labor. After the premature birth, there

Submitted: 2013/11/29
Approved: 2014/06/02

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was an evolution with a reversal of tachycardia and overall stabilization within 15 days of newborn life.

CASE REPORT

Male newborn (RN); mother with a history of kidney stones and urinary tract infection. At the 29th week of pregnancy was diagnosed with fetal atrial flutter and concomitant preterm labor, with uterine contractions and 50% cervix deletion confirmed by transvaginal ultrasound.

The option was the use of uterolytic nifedipine, at the attacking maintenance dose (40 mg/day) for 24 hours and progesterone (400 mg/day). Due to uterine contractions and progressive cervical alterations, we opted for the association of betamethasone (12 mg/day) for fetal lung maturity.

Fetal tachycardia was detected in the 29th week of pregnancy during clinical examination, through fetal auscultation with a sonar Doppler and confirmed in the fetal echocardiography by the device Cuson XP-10c ultrasonographic unit 3.5-MHz transducer. Atrial flutter was diagnosed with alternating conduction from 1:1 to 2:1, the atrial rate of 423 bpm, and ventricular alternation from 423 to 210 bpm. Cardiac morphological change was not detected; however, pericardial effusion and mild ascites were observed. The myocardial contractility was adequate, with a shortening fraction of 36% and a cardiothoracic index of 0.55.

Digoxin administration was started in the mother after maternal echocardiogram and ECG. The attack dose was administered on the first day (0.5 mg at 8/8 hours for 24 hours) and subsequently 0.75 mg per day (0.25 mg at 8/8 hours). Monitoring was carried out through serum digoxin dosing and maternal electrocardiogram, which revealed sinus bradycardia, nonspecific alterations in ventricular repolarization, and an overload of the right atrium.

Fetal Doppler echocardiography was performed after three days of digoxin use, which accused reversed flutter and fast periods (less than three seconds) of atrial tachycardia with spontaneous reversion. The fetus maintained discreet pericardial effusion, with ascites resolution.

At 30 weeks of gestation, sustained fetal tachycardia was observed (fetal heart rate between 257 and 261 bpm), with mild ascites, bilateral hydrocele and polyhydramnios, and amniotic fluid index (ILA) of 28.5 cm. The association of another antiarrhythmic drug was chosen because digoxin had already achieved serum levels above those permitted. The maternal bradycardia and hypotension were considered contraindications to the use of sotalol and amiodarone; therefore, propafenone, 300 mg, at 8/8 hours was administered. A significant improvement in maternal tolerance to the medication prescribed for both atrial flutter and inhibition and prevention of preterm birth was observed after 24 to 48 hours.

In the 31st week, repeated periods of fetal heart rate (FHR) around 220-240 bpm were still observed, as well as mild pericardial effusion and bilateral hydrocele, ILA = 19.5 cm. There was a complete reversal of fetal tachycardia after 10 days of propafenone use while maintaining FCF around 140 bpm (Figure 1).

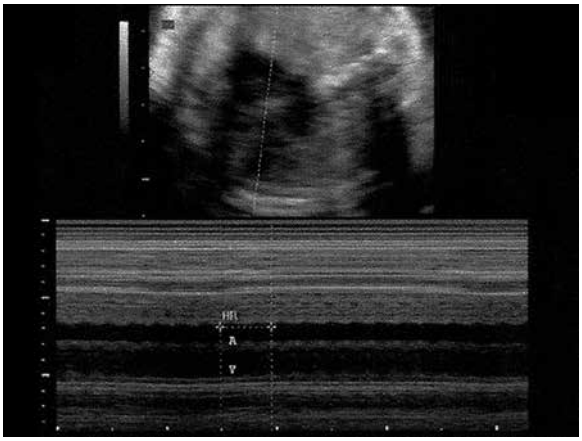

Figure 1 - Fetal echocardiogram. Atrial rate of 257 bpm, with rare supraventricular extrasystoles.
Source: Patient's ultrasound described in this case report.

The mother went into labor at 33 weeks and five days of gestation. She arrived at the maternity ward with cervical dilation of 6 cm, herniated amniotic sac, De Lee plan-3, and forehead presentation. Caesarean section was performed, which revealed clear amniotic fluid. The newborn was born with Apgar score 9 and 10 in the first and fifth minutes of life, respectively, with no need for resuscitation, weighing 1,990 grams. He was admitted to the Neonatal Intensive Care Unit in clinical and hemodynamic stability in the first 36 hours of life.

On the second day of life, he showed atrial tachycardia with a heart rate of 260 bpm, without an adequate response to the three administered adenosine bolus. An intravenous attack of amiodarone (5 mg/kg in one hour), followed by 5 mg/kg in 23 hours under continuous infusion was conducted. Amiodarone was maintained at the dose of 5 mg/kg per day.

The rate remained stable in the first week, however, with new episodes of tachyarrhythmia on the 10th day. Propafenone (8 mg/kg per day divided into three doses) was associated, with tachycardia reversion. The total stabilization occurred at 15 days of life. He was discharged at 20 days of age, in good conditions and using oral amiodarone (5 mg/kg/day) and propafenone (8 mg/kg/day).

Currently, at 18 months old, he is stable with no episodes of tachyarrhythmia. The use of propafenone (8 mg / kg / day) was maintained, and amiodarone is in progressive regression. Slow discontinuation of drugs and rigorous and continuous observation of his heart rate should be carried out.

DISCUSSION

The fetal atrial flutter can cause heart failure associated with pericardial and pleural effusion, ascites, and even fetal death. Therefore, because there is the possibility of treatment, it requires an early diagnosis and easy access to expert service. The accurate early diagnosis is crucial for the selection of prenatal and appropriate postnatal treatments.[2]

Most cases is primarily detected by auscultation of the fetal heart rate during follow-up prenatal consultations or during monitoring, through cardiotocography.[3] Cardiotocography, because its non-invasive characteristics and very efficient and immediate analysis of results, has a leading role in the biophysical assessment of fetal well-being in both antepartum period and during labor. However, despite these advantages, it is still a method of relative cost, which hinders its use. Thus, the clinical auscultation of fetal heart rate using the Doppler sonar is the most important initial test and should be routine in all pre-natal consultations.[4] In the presence of auscultation alterations, diagnostic confirmation is required with more specific methods.[3]

The best method for intrauterine diagnosis of fetal atrial flutter is the echocardiography, which allows the accurate description of the intracardiac anatomy, analysis of the chambers' sequence, and the recognition of malformations and function and cardiac rhythm disorders in the pre-natal period.[5,6] Thus, the data obtained on the echocardiography can help in a sure diagnosis and interfere with the therapeutic approach and fetal prognosis.

In this case, fetal atrial flutter and concomitant preterm labor were diagnosed at 29 weeks. However, we cannot state that the flutter had started only at that gestational age because it is likely that the preterm labor resulted from fetal decompensation determined by the fetal atrial flutter.

Therefore, tachyarrhythmia might already be present. After the diagnosis of atrial flutter, the factors that determined the therapeutic approach, in this case, were gestational age, episodes of tachycardia, and fetal maturity.

The approach adopted in relation to preterm labor at 29 weeks was negative tracking genitourinary infections and choosing nifedipine as an uterolytic, and progesterone and betamethasone, which postponed labor until 33 weeks.

The control of FCF was obtained only after the association of digoxin (from the 29th of gestation) and propafenone (from 30 weeks and four days). In a meta-analysis on the diagnosis, treatment, and outcome of fetal atrial flutter,[7] digoxin is reported as the first choice for treatment in 67.6% of cases; less than half of cases (45.1%) had cardioversion with isolated prescription of digoxin.

Clinical improvement was observed in the association of propafenone, which is assumed to be a second-line drug, with cardio conversion rates of 1:3. However, the few studies of its use in pregnant women report no adverse effects to the fetus and newborn, therefore, there is not enough information about the safety of its use during pregnancy and is considered as category C by the Food and Drug Administration.[8] Its prescription during pregnancy is only allowed if the benefits justify the potential risks to the fetus.

In this report, the RN started episodes of tachyarrhythmias after 36 hours of life, probably after renal excretion and decrease in the serum levels of drugs used by the mother during pregnancy. Only after the association of two antiarrhythmic, amiodarone and propafenone, the conversion to sinus rhythm was observed.

CONCLUSIONS

This report on fetal atrial flutter emphasizes the importance of auscultation properly performed prenatally to identify life-threatening tachyarrhythmias in the fetus. This examination, of simple implementation and low cost, enables an early diagnosis that is essential to prevent complications such as those identified in this study (ascites, pericardial effusion, and polyhydramnios). Unfortunately, a low quality of prenatal

care can compromise the antenatal diagnosis, even in situations where the simple correct fetal auscultation could indicate the need for further evaluation.

The management of fetal arrhythmias requires a quality hospital environment, allowing the safe conduct of invasive fetal procedures and vaginal or operative delivery if needed. Similarly, the presence of an appropriate and specialized multidisciplinary team is necessary to increase survival and quality of life for these patients.

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