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Case Report

Fetal and Neonatal Management of Atrial Flutter in Emergency Room: Adenosine, Amiodarone and Successful Direct Current Cardioversion

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ABSTRACT

We describe a case management of 35 weeks gestational age of pregnant woman referred to our emergency room for suspicious of premature ectopic beat. Our fetal echocardiogram showed normal heart anatomy, atrial flutter, fetal suffering. Cesarean delivery was done quickly and newborn showed, enlarging of right heart chambers, mitral and tricuspid valve insufficiency, fast supraventricular arrhythmia and adenosine ev was administrated for diagnosis. After adenosine clear atrial flutter waves were seen with atrial rate greater than 500 bpm with variable atrio ventricular conduction and successful therapy with amiodarone and synchronized cardioversion was performed.

Keywords: Fetal and neonatal atrial flutter; Amiodarone; Direct current cardioversion; Adenosine; Echocardiography

INTRODUCTION

We report a fetal arrhythmia accidently detected at 35 weeks of gestational age (GA) in a pregnant woman, who didn't assume any drugs and report any comorbidities, referred to our hospital for dilatation of right cardiac sections. Fetal echocardiography showed a tachyarrhythmia, occasionally detected and not reported before, with atrial heart rate (HR) of 500 beats per minute (bpm), and ventricular HR of 210 bpm measured at M-mode with simultaneous atrio-ventricular (AV) recording (Figure 1), moderate dilatated right heart sections, moderate tricuspid regurgitation (TR) and trivial mitral regurgitation (MR), but no hydrops. As near term pregnancy, our team of gynaecologists, neonatologists and pediatric cardiologists decided for urgent caesarean delivery in order to treat the newborn in intensive care unit (ICU). The ECG tracing at birth was marked by a fast supraventricular tachyarrhythmia (SVT), after a first inefficacy attempt to restore sinus rhythm (SR) through diving reflex, we decided to administer adenosine bolus (green arrow Figure 2A) at dosage of 0,1 mg/kg (weight's newborn 3 kg), during iv infusion ECG showed the atrial flutter (AFL) waves, so-called "sawtooth" (black arrows Figure 2A).

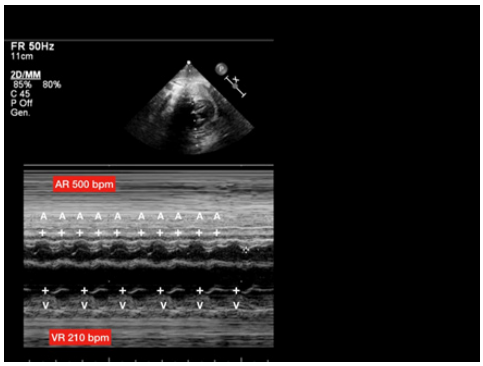

Figure 1: Fetal echocardiography M-mode shows atrial flutter 2:1 AV conduction with Atrial Rate (AR) of 500 bpm and Ventricular Rate (VR) of 210 bpm with ventricular conduction of 1:1.

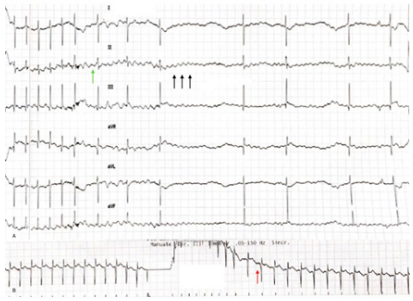

Figure 2: (A) Limb leads ECG trace shows atrial flutter with variable AV conduction after adenosine bolus (green arrow) with typical "sawtooth" waves (black arrows) (B) Syncronized cardioversion (black arrow) with 1 J/kg restored Sinus Rythm (SR) (red arrow).

CASE REPORT

Then, SR was successful restored by direct current cardioversion (DCC) with 1 J/kg (Figure 2B) and then antiarrhythmic therapy was started with iv amiodarone at dosage of 5 mg/kg in 1 hour and 15 mg/kg/min in the first 24 h. Despite maintenance antiarrhythmic therapy, after one day, during iv amiodarone infusion, the patient had another episode of AFL treated with DCC. We didn't use transesophageal overdrive stimulation to treat arrhythymia.

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Few hours later, echocardiogram confirmed normal heart anatomy, the fetal diagnosis of moderate TR, trivial MR and moderate right section dilatation (Figure 3A), right ventricular pressure of 70 mmHg.

Patient was discharged in amiodarone therapy per os and low dosage of furosemide per os to improve TR and right side dilatation of the heart. No neurological pathology was detected immediately after birth and at the follow up. Regular follow up was done with 24 h ECG Holter monitoring that demonstrated the maintenance of SR. After two months, amiodarone was stopped as SR was maintained and also for its possible side effects; echocardiogram showed normal right chamber dimensions (Figure 3B).

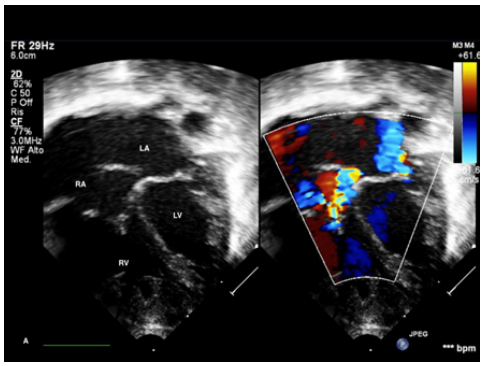

Figure 3A: Four chamber views shows right atrial and ventricle dilatation, levoversione of atrial septum and Color Doppler shows moderate tricuspid and trivial mitral regurgitation.

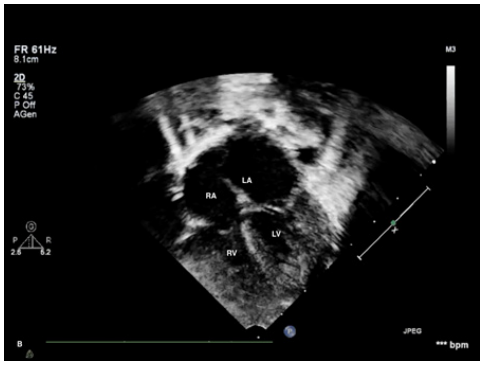

Figure 3B: Four chamber view shows after two months normal right chamber dimension. LA: Left Atrium; LV: Left Ventricle; RA: Right Atrium; RV: Right Ventricle.

DISCUSSION

AFL is a rare arrhythmia encountered in neonatal period, caused often by the re-entry circuits sited in the right atrium. It mainly concerns children affecting from congenital heart diseases, but may also develop in patients with normal heart anatomy, mainly in newborns or in foetuses, in whom may induce non-immune fetal hydrops and heart failure. In a more severe form, it may be potentially lethal. A perinatal mortality of approximetely 8 % is reported, therefore it's crucial to know diagnosis and management of fetal and perinatal arrhythmias [1].

The characteristic feature on the ECG is represented by "saw-tooth" flutter waves, seen in leads II, III, aVF, V1, with an atrial rate beteween 300 and 500 beats/min with vary degree of AV block and a ventricular rate of 160 and 240 bpm. AV node is not involved in the arrhythmia's circuit and then its block induced by adenosine cannot terminate AFL, but unmasks the flutter wave causing AV block.

AFL is the second fetal tachy-arrhythmias for incidence. AFL is observed almost exclusively in the third trimester, which is probably related to the large atrial size achieved at 27–30 weeks of GA, with high vulnerability to atrial extrasystoles. Accessory AV pathways and reentrant SVT are a common association in 70% of fetuses. This fetal arrhythmia may be associated with myocarditis, immune-mediated heart block/carditis or congenital heart disease such as Ebstein anomaly [2,3].

The end point of intrauterine treatment is the restoration of sinus rhythm. The first line inutero antiarrhythmic treatment recommended is sotalol in case of fetal hydrops. which has been effective in converting 50%–80% of fetuses with AFL without mortality. Digoxin or amiodarone may be considered as the second choice. Procainamide is contraindicated.

In hydropic fetuses with treatment-refractory AFL, premature delivery and postnatal conversion to sinus rhythm should be considered at gestational age >34 week [4].

At birth, the recommended treatment for a newborn, both stable and unstable, is either DCC or transoesophageal atrial overdrive to restore sinus rhythm. It is important to be prepared with backup external pacing after conversion because sinus node suppression may occur from in utero drug therapy. DCC appears to be the most effective treatment of AFL, although in a stable newborn antiarrhythmic drugs can be tried with a long time of efficacy in restoring SR [4].

Since our patient was hemodinamically stable and AFL didn't last for a long-time, anticoagulant therapy wasn't required.

In our case we used adenosine as diagnostic tool to patent AFL. As the hemodynamically stability, high atrial rate and initial signs of heart failure at echocardiogram, we decided not perform overdrive stimulation through transesophageal atrial study and we restored SR directly with DCC. We performed two DCC during amiodarone infusion and used furosemide iv to improve AV valve regurgitation. Although pharmacological prophylaxis beyond the neonatal period is unnecessary, we decided to prolong amiodarone therapy until two months because of AFL relapse in ICU. In case of AFL, amiodarone and DCC appears to be most effective in restoring SR if AFL is incessant. Different studies reported high success of DCC at returning AFL into SR; Lisowski et al. reported 45 cases of fetal and neonatal AFL, nine of twelve patients in AFL at births received DCC as initial successfull treatment [5].

The cardiac conduction system is originally derived from the cardiac neural crest, in which certain gene expressions are likely to control myocardiocyte differentiation into a pacemaking cell phenotype. It has been described that genes guiding the developmental and functional status of the cardiac conduction system may be responsible for the arrhythmogenicity. A specifically channellophaty of the hyperpolarization-activated cyclic nucleotide-gated channel 4 (HCN4) is involved in the mechanisms of the arrhythmogenicity of AFL [6].

Possible risk factors for fetal or neonatal AFL are macrosomia or be born to diabetic mothers than the general population, but both conditions wasn't present in our case. Important factors for clinical course are beginning of the signs, duration of arrhythmia and degree of ventricle response to AFL [7]. In our case no macrosomia signs or maternal diabete were present and AFL didn't last for long

time and no fetal hydrops arose.

Infants with AFL generally have an excellent prognosis once in sinus rhythm with a low risk of recurrence, and long-term antiarrhythmic therapy is unlikely to be necessary.

The brain ultrasound assessment during the first months of life is also recommended to exclude hypoxic changes in the brain [8].

We can state that AF is a serious and life-threatening rhythm disorder both in the neonatal and fetal period, specifically for the risk of hydrops, neurological damage and intra uterine death.

Treatment of fetal AFL should be carried out at a reference center. The management of fetal arrhythmia should be carried out in a tertiary center in cooperation with the medical team: obstetricians, fetal cardiologists and neonatologists, in order to monitor during treatment both the fetus and the pregnant. Our case is important because no fetal antiarrhythmic treatment was done without intrauterine death. Use of multiple fetal scan is mandatory to avoid rhythm problems at birth.

CONCLUSION

Fetal arrhythmias are potential life-threating conditions and can lead to intrauterine death. The prognoses are very different depending on the type and severity of fetal arrhythmias and the associated fetal conditions such as congenital heart defects, cardiac dysfunction, and fetal hydrops. Responses of fetal arrhythmias to individual treatments and clinical schemes are heterogeneous, and the prognoses are poor particularly under such conditions. Thus, fetal rhythm monitoring is necessary for preventing the progression of severe types of fetal arrhythmias. The relapse of AFL at birth represents a troublesome issue and requires intensive-care admission of these fragile patients. Treatment therefore is required, primarily aiming at reaching an adequate ventricular rate and preferably conversion to sinus rhythm. This case underlines the importance of fetal scan and rhythm control during fetal life in order to avoid emergency treatment at birth. Our case is important for fast management of fetal arrhythmia occasionally detected in the third trimester and potentially lethal without maternal anthyarrhytmic treatment.

LEARNING POINTS

1. Fetal AFL could be lethal and the use of DCC and amiodarone is safe and effective

2. AFL diagnosis in the third trimester needs fast management

3. DCC and amiodarone iv have to be used in AFL relapse

4. Adenosine remains a mandatory drug for diagnosis of SVT in newborn

5. Fetal atrial flutter need tertiary centre and on same department because could represent emergency.

DISCLOSURE STATEMENT

No potential conflict of interest was reported by the authors.

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