Case Report

Blocked Atrial Bi/Trigeminy In Utero Evolving in Supraventricular Tachycardia after Birth

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1. Introduction

Fetal sinus bradycardia was defined before as a heart rate <100 bpm, but in 2009 [1] this threshold was revised to <110 bpm by the American College of Obstetrics and Gynecology in response to population data. Transient episodes of fetal heart rate of less than 110 bpm are usually benign and typically result from increased vagal stimulation in the fetus commonly associated with abdominal pressure by the ultrasound probe. Causes of sustained fetal bradycardia include sinus bradycardia, blocked atrial bigeminy/trigeminy, high-degree atrioventricular block, and long QT syndrome. Persistent atrial bigeminy and trigeminy with blocked premature beats may lower the average heart rate of the fetus to 70–100 bpm. This benign form of fetal bradycardia is the result of blocked premature atrial contractions occurring after one or two sinusual beats (resp., an atrial bigeminy or trigeminy) [2] that are not conducted to the ventricle and consequently nor to the aorta, probably due to the refractoriness of the A-V tissue. Subsequently a sinus pause is observed and a consequently fetal heart rate is low. The diagnosis can be reached by identifying the relationship between atrial and ventricular contraction using simultaneous recording in M-mode fetal echocardiography.

Generally, this type of bradycardia is intermittent, does not require treatment, resolves spontaneously with advancing gestation or after birth, and is not usually associated with cardiac failure [3]. It occasionally may present as sustained bradycardia, which makes it difficult to differentiate from A-V block. However, blocked atrial bigeminy can be distinguished from sinus bradycardia or atrioventricular block by examination of the Doppler flow pattern in the inferior vena cava or hepatic veins, which shows flow reversal [4, 5].

Nevertheless, data regarding the prevalence, mechanisms, and the long-term outcome of fetuses with bradycardia are still limited.

2. Case Report

We present herein the case of a 34-year-old Caucasian patient G1P0 that was referred to our department for “blocked atrial...
bigeminy with pseudobradycardia” detected elsewhere at 33
of weeks of gestation. Furthermore, a placenta previa was
diagnosed.

The patient was scanned at our department at 37 weeks
of gestation. An echocardiography showed during all the
examination an arrhythmia that in M-mode resulted in being
a blocked atrial trigeminy with a mean fetal heart rate (FHR)
of 100 bpm (Figures 1 and 2).

A female neonate of 2770 gr and Apgar score 9/10 at
1/5 minutes, respectively, was born by cesarean section at
38 weeks because of the placenta previa.

An ECG performed at birth revealed blocked supraventricu-
lar extrasystoles (Figure 3). Instead, successively at card-
diomonitoring, no extrasystoles were registered.

An ECG the day after did not register any extrasystole.

On day 3, an ECG showed no atrial extrasystoles and a
normal QTc interval at upper limits. An echocardiography
performed the same day showed a small patent FO with
moderate left-to-right shunt and a trivial tricuspid regurgita-
tion with an indirect estimate of pulmonary artery pressure
of 35 mmHg.

To our surprise, on day 11, ECG revealed supraventricu-
lar trigeminy and episodes of paroxysmal supraventricular
tachycardia (Figure 4). A treatment with Lanoxin syrup
0.25 mL twice a day was started. A control on day 17 showed a
paroxysmal supraventricular tachycardia interrupted by some
sinusual beats (Figure 5). Digoxinemia level was at 1.4 ng/mL,
and therapy with Sotalol hydrochloride 2 mg/kg twice a day
was started.

On day 18, an episode of PSVT that needed a “diving
reflex” maneuver was registered. The same day, a cardiomoni-
toring showed extrasystoles and episodes of bradycardia
with a heart rate of 80 bpm.

A 24 hr ECG/Holter monitoring on day 20 recorded a
sinusual rhythm with a mean heart rate at lower limits for age,
some blocked supraventricular extrasystoles, and 3 isolated
and monomorphic ventricular extrasystoles. During sleep,
some episodes of 2nd-degree AV block Mobitz type I and
Mobitz type II were recorded. Interventricular conduction
was regular. There were no significant alterations of the
ventricular repolarization.

3. Discussion

Although most cases of fetal bradycardia verified during
obstetrical ultrasound examination are due to vagal stimula-
tion because of fetal compression by the transducer, attention
should be paid in case of sustained bradycardias, trying to
evaluate simultaneously atrial and ventricular contraction
for evidence of nonconducted ectopic atrial beats causing
low ventricular contraction rate. Furthermore, these benign
arrhythmias can present with bradycardia during the FHR
monitoring leading erroneously to emergent preterm deliv-
er. Consequently, in cases when an atypical bradycardia
is registered at FHR monitoring, a fetal echocardiography
should be done to determine if it is the case of blocked atrial
bigeminy or trigeminy, because these arrhythmias do not represent an obstetrical emergency.

However, the literature shows that although once considered an entirely benign arrhythmia, fetal ectopy is now thought to be a manifestation of a number of diseases. Ectopy should not be dismissed as benign without fetal assessment, especially if risk factors such as a family history of sudden death, prior fetal loss, or maternal pregnancy complications exist [4]. Whether ectopy represents spontaneous automaticity of the atrium or reentry is unclear. When coupling of the ectopic beat to the prior QRS is fixed, as opposed to variable, it is likely to be related to a reentrant atrioventricular pathway; in this setting, the risk of supraventricular tachycardia is about 0.5% for simple ectopy (isolated, bigeminy, or trigeminy) [3, 6] and up to 6% for complex ectopy (atrial couplets or triplets). This increased risk of supraventricular tachycardia extends into the neonatal period, although most ectopy resolves by 1 month of age.

In our case, the fetus presented with bradycardia due to atrial trigeminy and three subsequent postnatal ECGs until day 3 showed no evidence of atrial extrasystoles, confirming the well-known frequent regression of this kind of benign arrhythmia. But on day 11 recurrence of supraventricular trigeminy and development of episodes of paroxystic supraventricular tachycardia were observed.

Although our case represents a single evidence, it denotes the risk of development of more serious arrhythmia in postnatal life in cases of fetal blocked atrial ectopic beats. This seems to occur when the underlying mechanism is an accessory AV conduction pathway that appears occult even at basal ECG.

On the basis of this observation, we recommend that fetuses with complex atrial ectopic beats should be closely monitored before and after birth for evidence of new arrhythmias. Since time period for monitoring is unknown, parents should be advised to verify baby’s heart beat during feeding quietness, or sleep and refer both low and high frequencies.

References