Conservative management of fetal bigeminy arrhythmia leading to persistent bradycardia

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Summary

A case of persistent fetal bradycardia first detected at 32 weeks of gestational age is described. The diagnosis of atrial ectopic beats was made by means of M-mode echocardiography. Since the fetus was normally grown and showed no signs of distress, the vaginal route of delivery was allowed, also because continuous M-mode echocardiographic monitoring was possible during labour.

Fetal arrhythmia; Echocardiography; Fetal monitoring

Introduction

Atrial ectopic beats are often observed in the fetus. They are usually transient and do not need any treatment. If they are frequent, weekly fetal heart rate monitoring is suggested because they may trigger supraventricular tachycardia. When atrial ectopic beats are blocked and in bigeminy sequence, they cause a bradycardia which must be differentiated from more ominous causes of bradycardia, that is, fetal distress or complete atrio-ventricular block which is associated with cardiac malformations in 20–50% of cases.

Until a few years ago the differential diagnosis could be made only by fetal ECG [1]. Now M-mode echocardiography is used [2].

We describe a case of persistent bradycardia due to blocked atrial ectopic beats in which the correct diagnosis and monitoring by means of M-mode echo-cardiography allowed a conservative management of pregnancy and labour.
Case report

A 30-year-old primigravida was referred to our ultrasound unit because of persistent bradycardia (60–90 bpm) first detected at 32 weeks gestational age. The fetus was normally developed and there was a normal amount of amniotic fluid. Two-dimensional echocardiography showed normal heart structures. M-mode echocardiography showed that the arrhythmia was due to blocked atrial ectopic beats (Fig. 1). A sinus rhythm was observed only for a few minutes in 30 min of continuous monitoring. We decided that no treatment was needed and kept on monitoring the fetus once a week to assess cardiac rhythm and fetal growth.

The arrhythmia persisted, with episodes of sinus rhythm that never lasted for more than 15 min. Normally reactive cardiotograms were recorded both during bradycardia and during regular rhythm (Fig. 2). Doppler ultrasound assessments from fetal aorta and umbilical arteries showed abnormally high PI values during bradycardia and normal values during sinus rhythm.

At 38 weeks gestational age the woman went into labour after spontaneous rupture of the membranes. To differentiate bradycardia due to ectopic beats from possible hypoxic bradycardia, we used M-mode echocardiography monitoring. Sinus rhythm at 130–140 bpm was alternating with bradycardia at 70–90 bpm. Echocardiography during the bradycardic periods showed that they were due to ectopic beats. Bradycardia was never observed during sinus rhythm.

Therefore vaginal delivery was allowed and a 3280 g male, with Apgar scores of 9 at 1 and 5 min was born. ECG continuous monitoring during the first week of life

![Fig. 1. M-mode recording through an atrium and an atrio-ventricular valve. There is an atrial contraction (a) followed by the ventricular contraction, as indicated by the closure of the atrio-ventricular valve (arrow); the next atrial contraction (a’) is not followed by the ventricular contraction; in fact, the atrio-ventricular valve remains open (open arrow). The atrial ectopic beats are in bigeminy sequence.](image-url)
showed persistence of the arrhythmia (Fig. 3). No treatment was undertaken and in the next weeks the episodes of bradycardia became shorter and fewer, until they almost completely disappeared at the age of 2 months.

Discussion

This case shows that a fetus can bear high degrees of bradycardia and therefore, when a reliable diagnosis of benign rhythm disturbance is made, it is correct to have a conservative management of pregnancy.

If the arrhythmia is persistent, the pregnancy must be regarded as 'at risk' and intensive biophysical monitoring must be activated. A nonstress test seems to be the best way of assessing fetal well being, since reactivity is maintained during bradycardia. Doppler flow measurements, on the contrary, could be misleading; in fact the PI value in the umbilical arteries is inversely related to the fetal heart rate frequency and is abnormally high irrespectively of the cause of the bradycardia. Therefore in these cases it cannot be assumed as an indicator of the fetal condition. Normal fetal growth and absence of signs of heart failure (ascites and/or hydrops) must be assessed by two-dimensional imaging.
Fig. 3.
Fig. 3 (opposite page). ECG at birth. (a) Standard peripheral leads. It shows sinus P waves (P) normally conducted to the ventricles and premature blocked P'. Note that in lead 3 and aVF P and P' have an opposite polarity, suggesting a different origin of the impulse. However, as shown by positivity in lead 2 and negativity in aVR the site of the ectopic impulse formation is probably very close to the sinus node. (b) precordial leads. In leads V4, V5, V6 a regular sinus rhythm is present at a sinus cycle length of 430 ms. These P waves (P) are normally conducted to the ventricles (beats 1, 2 and 3). The fourth atrial wave (P') is premature with a P-P' interval of 320 ms; the following P'-P interval is 530 ms. The sum of P-P' and P'-P is therefore 850 ms. P' is a premature atrial beat with a fully compensatory pause, due to interferences at S-A junction between the retrograde atrial premature impulse and the normal sinus discharge.

Usually this kind of arrhythmia disappears before the end of pregnancy. In this case the bradycardia due to blocked atrial ectopic beats lasted until term. Monitoring of the fetal heart with M-mode echocardiography during labour showed to be a good guide to vaginal delivery.

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References