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Case report

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Management of fetal supraventricular tachyarrhythmia – case report

Postupak kod dijagnosticirane fetalne supraventrikularne tahiaritmije – prikaz slučaja

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Summary

The conduction system of the fetal heart is defined by the16th week of gestation when it matures and normally produces a regular rhythm and rate between 110 and 160 beats per minute (bpm) for the remainder of the pregnancy. Deviations from these parameters are fetal arrhythmias. They are diagnosed in 2% of unselected pregnancies. They are mostly benign and transient but some of them are persistent and associated with structural defects or can cause heart failure, fetal hydrops and intrauterine death. Routine prenatal care includes screening for fetal arrhythmias in the second and third trimester with fetal ultrasound examinations which include a view of the four cardiac chambers and both ventricular outflow tracts. The fetal outcomes are improved upon appropriate antepartum diagnosis and care. Here we present a pregnancy and multidisciplinary management, prenatal evaluation and intervention with maternal transplacental treatment of a 28-year-old female, gravida II, para II, in 28+5 weeks of gestation with fetal arrhythmia, in tertiary university hospital. She had a history of previous caesarean section, in the 40th week of gestation due to an infection of the synus pylonidalis. We confirmed suspected fetal arrhythmia as supraventricular tachyarrhythmia without fetal hydrops, based on the ultrasound doppler M mode imaging, and started transplacental administration of antiarrhythmyc agent, digoxin. It has been considered the first line agent for treatment of fetal supraventricular tachycardia but higher maternal doses are required to maintain a therapeutic serum level. We converted fetal heartbeat into normal sinus rhythm after three days of administration of digoxin. We continued to monitor the fetus once a week with controlling levels of digoxin and electrolytes in maternal blood until the end of the pregnancy at 38+6 weeks of gestation.

Key words: fetal arrythmia, prenatal diagnosis, transplacental therapy

Sažetak

Provodni sistem fetalnog srca definiran je do 16. tjedna trudnoće, kada producira regularan ritam i frekvenciju od 110 do 160 otkucaja u minuti, do završetka trudnoće. Fetalne aritmije definiraju se kao odstupanja od ovih parametara. Dijagnosticiraju se u 2% svih trudnoća. Uglavnom su benigne i prolazne, ali neke od njih povezane su sa strukturalnim defektima, mogu izazvati dekompenzaciju srca, fetalni hidrops ili intrauterinu smrt. Rutinska prenatalna skrb uključuje ultrazvučni skrining za fetalne aritmije u drugom i trećem tromjesečju. Uključuje pregled četiri srčane komore, kao i izlazišta velikih krvnih žila. Ishod trudnoće je bolji što se prije postavi dijagnoza i započne s liječenjem. Ovdje predstavljamo trudnoću i mutidisciplinarni pristup u liječenju 28-godišnje trudnice, u njezinoj drugoj trudnoći u 28+5 tjednu gestacije s dijagnosticiranom fetalnom aritmijom, u tercijarnom centru. Prethodna trudnoća dovršena je carskim rezom u 40. tjednu trudnoće zbog infekcije sinus pilonidalisa. Po primitku u bolnicu, ultrazvučno doplerom u M modu je postavljena dijagnoza supraventrikularne tahiaritmije bez fetalnog hidropsa, te je nakon toga započeta transplacentarna terapija antiaritmikom digoxinom. Smatra se prvom linijom liječenja fetalnih supraventrikularnih tahikardija, uz neophodno davanje većih doza majci, kako bi se postigao

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terapijski učinak. Normalan sinus ritam je postignut nakon trećeg dana primjene digoksina. Nakon toga nastavilo se praćenje fetusa jednom tjedno, uz kontrolu serumske koncentracije digoksina i elektrolita u majčinoj krvi, sve do kraja trudnoće u 38+6 tjedana.

Ključne riječi: fetalna aritmija, prenatalna dijagnoza, transplacentalna terapija

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Introduction

Fetal arrhythmias are phenomena with rather complicated etiologies. There are debates regarding prenatal diagnosis and treatment of fetal arrhythmias. Understanding the human cardiogenesis is necessary to understand pathophisiology of fetal arrhythmia and congenital heart disease. The embryological development of the cardiovascular system begins with cardiac progenitor cells migration in the epiblast just lateral to the primitive streak. The cardiac loop is finished by approximately 22-28 days after the heart tube elongates.[1] At five weeks of gestation, there is a first sign of sinus node development.[2] The AV node is recognizable morphologically when the looping heart divides into atrial and ventricular components. At 16 weeks of gestation the conduction system is functionally developed.[3] The cardiac electrical conduction system consists of: Sinoatrial Node, Atrioventricular Node, His-Purkinje System which propagates electrical impulse through the myocardium to produce each heartbeat. Automaticity is a characteristic of the cardiac conduction system. Cardiac electrophysiology is extremely important since arrhythmias result from abnormalities in the generation and/or conduction of electrical impulses.[4] Clinically, fetal arrhythmias can be categorized into 3 types: premature contractions, tachyarrhythmias and bradyarrhythmias.[5] Premature contractions are most common. They can be divided by their origin: premature atrial contractions and premature ventricular contraction. It is difficult to distinguish premature atrial contraction from premature ventricular contractions. Prognosis is good in the near and long terms, and fetal growth and development are not affected. In general, isolated premature contractions do not require therapy and they resolve spontaneously before delivery.[6] Fetal tachycardia is defined as HR >180 bpm. It can be classified as sustained when the arrhythmia and present for more than 50% of the examination time or intermittent when periods of tachycardia alternate with predominately normal heart rate, and subdivided into sinus tachycardia, supraventricular tachycardia and ventricular tachycardia. The most common fetal tachycardias are paroxysmal supraventricular tachycardia either with 1:1 atrioventricular conduction or atrial flutter with variable, mostly 2:1 AV conduction.[4] Bradyarrhythmia is defined as persistent fetal HR of less than 110 bpm, and may be secondary to sinus bradycardia, blocked atrial bigeminy or high-grade atrioventricular block. Persistent sinus bradycardia below 100 bpm is rare and can be seen in fetal distress, hypoxia and acidosis. Another important cause of fetal bradycardia is also altered conduction of atrial impulses to the ventricles resulting in complete dissociation of the atria and ventricle.[7] Isolated complete atrioventricular block in the fetus in the absence of congenital heart disease is usually immune mediated in association with transplacental transfer of circulating antibodies to Ro (SSA), and La (SSB) antigens from the mother. The risk is 2-3% with recurrence risk of 14-17%.[8] Currently, fetal echocardiography in combination of M mode and Doppler tissue imaging is the most common used tool for diagnosis and follow up of fetal arrhythmias in clinical practice. In the presence of a suspected arrhythmia, the important features to be evaluated are: fetal heart rate, rhythm regularity and the relation, and time intervals of the atrial and ventricular contractions. In case of arrhythmia, M-mode cursor is usually placed across an atrium and ventricle, so that the relationship of atrial to ventricular contractions is recorded, while pulsed Doppler echography AV time interval assesses. Fetal magnetocardiography is also used, which is ECG analogous and a noninvasive technique for recording the electrical activity of the fetal heart, but it is still a very expensive method and has limited clinical applicability.[4] The prognosis of fetal arrhythmias depends on the type and severity of arrhythmia and the associated fetal conditions. Life threatening fetal arrhythmias include atrial flutter (AF), ventricular tachycardias and bradyarrhythmia.[5] Fetal demise occurs in cases of fetal congestive heart failure, hydrops fetalis or congenital heart disease. Benign fetal arrhythmias do not need any treatment before or after birth. Individualized and clinical treatment should be determined according to specific types.[9]

Case report

The patient has provided informed consent for publication of the case. A previously healthy pregnant 28-year-old woman (gravida two para two) was presented to our hospital at 29 weeks of gestation with

fetal arrhythmia. She had a history of previous caesarean section due to sinus pylonidalis. We confirmed fetal arrhythmia as supraventricular tachyarrhythmia, based on the ultrasound doppler M mode imaging. Fetal echocardiogram revealed structurally normal heart with no evidence of hydrops or ventricular dysfunction. Maternal serum electrolyte levels were normal. We prescribed oral metildigoxin loading dose for rapid loading of 1 to 2 mg, which had been given in three doses: 0,5 mg; 0,3 mg; 0,2 mg over 24 hours, followed by a metildigoxin blood level. Our target level was 1 to 2 ng/ml. After this target was achieved, we waited for a 48 to 72 hours period of observation to assess the fetal response to maximum maternal dose. We monitored daily the maternal electrocardiographic changes and other potential symptoms looking for low grade metildigoxin toxicity. Fetal ultrasound after 72 hours of administration of metildigoxin with levels of 1 ng/ml revealed the termination of tachycardia. She continued to take metildigoxin at a dose of 0.2 mg, 0.2 mg; 0.1 mg with a correction of the dose depending on the concentration in the blood. We controlled the levels of metildigoxin once a week together with the maternal echocardiography and electrolyte serum level. Follow up assessment at 32, 34 and 36 weeks showed normal heart rate and rhythm. The baby was delivered at 38+6 weeks of gestation by elective cesarean section at our perinatology center. It was male, 3490 g in weight and 51 cm long. Apgar was ten at the first minute. The transthoracic echocardiogram was performed and showed normal cardiac function and the absence of structural heart defects. The electrocardiogram showed no signs of arrhythmia in the next seven days, so the baby was discharged with mandatory cardiological control after one month. Periodic assessment during the follow-up showed sinus rhythm. Referring such patients to the tertiary centers with expertise in diagnosis and management is of essential importance.

Discussion

Supraventricular tachycardia is the most common form of fetal tachycardia.[9] It can be seen in maternal hyperpyrexia, use of stimulants, maternal thyrotoxicosis or fetal systemic disease such as anemia, fetal distress and rare infections. Factors which are included in the decision to treat tachycardia or not are: mechanism of tachycardia, fetal gestational age and the well-being, presence or absence of congenital heart disease. In general, three options are available: no treatment with close monitoring, transplacental drug therapy and finally delivery of fetus. Sustained fetal arrhythmias that predispose the occurrence of hydrops fetalis, cardiac dysfunction, or even fetal demise require early treatments and the effect of therapy depends on the type and etiology of fetal arrhythmia and fetal condition.[10] The conversion rate is high with the use of the first line antiarrhythmic agents via transplacental route. Individualized and clinical treatment should be determined according to the specific type. In general, the transplacental administration of antiarrhythmic agents includes digoxin which is widely accepted as the first line treatment. Sotalol, amiodaron and flecainide are second line drugs when digoxin fails to achieve conversion to sinus rhythm. Combined therapy is reserved for refractory fetal tachycardias.[11] Digoxin is prized for its safety and efficacy, but higher doses are required to maintain a therapeutic serum level, especially in the presence of hydrops fetalis.[12] Sotalol and flecainide should be used as a first line treatment for hydropic fetal tachyarrhythmias. Concerning the treatment of fetal tachycardias, digoxin monotherapy showed a lower rate of effectiveness than combined digoxin and sotalol/flecainide.[13] In conclusion, fetal arrhythmias have to be diagnosed on time and treated using the right antiarrhythmic drugs. Delayed treatment might result in permanent damage. Awareness and knowledge of proper management and treatment of fetal tachyarrhythmias should be kept in mind so prematurity and other complications could be avoided.

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