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Foetal arrhythmia – what every obstetrician should know

Zaburzenia rytmu serca płodu – co każdy położnik wiedzieć powinien

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Słowa kluczowe

echokardiografia płodowa, zaburzenia rytmu serca płodów, kardiotokografia, blok przedsionkowo-komorowy u płodu, częstoskurcz nadkomorowy u płodu, skurcze dodatkowe u płodu, przeciwciała anty-Ro

S u m m a r y

Heart rhythm abnormalities are easy to visualise but more difficult to precisely diagnose in the prenatal period. Arrhythmia is not an indication for premature delivery, however, foetuses should be monitored using ultrasound, not cardiotocography.

Foetal arrhythmia is classified as follows: 1. Premature contractions (80-85%): a) premature atrial complexes PAC, b) premature ventricular complexes PVC – very rare. 2. Tachyarrhythmia: a) sinus tachycardia (10-15%), b) supraventricular tachycardia (SVT), c) atrial flutter/fibrillation, d) ventricular tachycardia – sporadic. 3. Bradyarrhythmia (5-10%): a) sinus bradycardia – obstetric causes should be excluded, b) blocked atrial contractions, c) non-sinus atrial rhythm, d) atrioventricular blocks.

Premature contractions are the most common and mild form of arrhythmia. In most cases, they do not require treatment and resolve spontaneously in the perinatal period. Atrial bigeminy can mimic a complete atrioventricular block. Foetuses with supraventricular tachycardia require transplacental treatment. The method of treatment will depend on the type of tachycardia, which can be diagnosed by a prenatal cardiologist. Conversion into the sinus rhythm can be achieved in more than 90% of cases. Premature delivery without consultation in the reference foetal cardiology center is a mistake. Complete heart block (CHB) without structural heart defect is an intrauterine acquired condition due to foetal electrical conduction system damage, most often caused by SS-A and SS-B antibodies. Women with positive antibodies should be included in a special surveillance program to monitor atrioventricular (AV) conduction time in foetuses from 16 weeks gestation. Foetuses with complete heart block should be born at term, as prematurity significantly affects their further development. Complete heart block with structural heart defect and foetal heart failure is a lethal disorder. Close cooperation between obstetrician and foetal cardiologists enables optimal treatment outcomes in foetuses and neonates with prenatally diagnosed arrhythmia.

S t r e s z c z e n i e

Zaburzenia rytmu serca są łatwe do zobaczenia, ale trudniejsze do precyzyjnego zdiagnozowania w okresie prenatalnym. Arytmie nie są wskazaniem do wcześniejszego zakończenia ciąży, płody powinny być monitorowane badaniem ultrasonograficznym, a nie kardiotokograficznym.

Arytmie dzielimy na: 1. Nieregularny rytm serca, czyli skurcze dodatkowe (80-85%): a) adkomorowe PAC (ang. *premature atrial contractions*), b) komorowe PVC (ang. *premature ventricular contractions*) – sporadycznie. 2. Tachyarytmie: a) tachykardia zatokowa (10-15%), b) częstoskurcz nadkomorowy, c) trzepotanie/migotanie przedsionków, d) częstoskurcz komorowy – niezmiernie rzadko. 3. Bradyarytmie (5-10%): a) bradykardia zatokowa – należy wykluczyć przyczyny położnicze, b) bradykardia spowodowana zablokowanymi skurczami dodatkowymi, c) rytm pozazatokowy – przedsionkowy, d) bloki przedsionkowo-komorowe.

Skurcze dodatkowe są najczęstszą, łagodną postacią arytmii. Zazwyczaj nie wymagają leczenia i ustępują w okresie okołoporodowym. Bigeminia przedsionkowa może imitować całkowity blok przedsionkowo-komorowy.

Płody z częstoskurczem nadkomorowym wymagają leczenia przezłożyskowego. Sposób leczenia uzależniony jest od rodzaju częstoskurczu, co może zdiagnozować kardiolog prenatalny. Konwersję do rytmu zatokowego można uzyskać w ponad 90% przypadków. Przedwczesne rozwiązanie ciąży bez konsultacji w ośrodku referencyjnym kardiologii prenatalnej powinno być uznane za błąd.

Conflict of interest
Konflikt interesów

None
Brak konfliktu interesów

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Całkowity blok przedsionkowo-komorowy bez wady strukturalnej jest chorobą nabytą wewnątrzmacicznie z powodu uszkodzenia układu bodźcowo-przewodzącego płodu najczęściej przez przeciwciała SS-A i SS-B. Pacjentki z dodatnimi przeciwciałami muszą być objęte specjalnym programem nadzoru kontroli czasu przewodzenia przedsionkowo-komorowego u płodów od 16. tygodnia w każdej ciąży. Płody z blokiem całkowitym powinny rodzić się w terminie porodu, gdyż wcześniactwo w sposób istotny pogarsza ich dalszy rozwój.

Całkowity blok przedsionkowo-komorowy z wadą serca i niewydolnością krążenia płodu jest wadą letalną.

Ścisła współpraca położniczo-kardiologiczna zapewnia optymalne wyniki leczenia w przypadkach arytmii u płodów.

INTRODUCTION

For many years, cardiotocography (CTG) has been a gold standard in foetal monitoring. A close cooperation between obstetricians and cardiologists has shown that CTG is not "a gold standard" for the assessment of foetal rhythm abnormalities or foetal condition during arrhythmia. CTG involves a simultaneous registration and graphical presentation of heart rate, uterine contractions and foetal movements. The results are presented graphically in the form of curves on a scale paper. In the case of foetal arrhythmia, CTG recordings show 'averaged' heart rhythm. This gives rise to completely incorrect readings in premature contractions, which do not pose a threat to foetal development and should not be an indication for premature delivery. The CTG recording is illegible for slow (below 60 bpm) or fast (> 200 bpm) heart rate (1). This information is very important as foetal arrhythmia diagnosis and foetal condition assessment are only possible based on ultrasonography and echocardiography.

Foetal rhythm disturbances are the second most common prenatal cardiovascular pathology. They occur in 1-3% of foetuses and are an indication for echocardiography in about 15% of cases in our centre. Foetal rhythm abnormalities are classified into three large groups: irregular heartbeat, tachycardia and bradycardia (2).

In 2004, The Polish National Registry for Foetal Cardiac Pathology (available at www.orpkp.pl) was launched. Since the programme is voluntary, it contains incomplete data. Table 1 shows the number of life-threatening foetal arrhythmias diagnosed and registered on www.orpkp.pl between 2004 and 2015.

Tab. 1. The most common type of foetal arrhythmia in Poland and in refernce center for fetal cardiology in Warsaw

Type of arrhythmia	Poland	Warsaw Centre
Supraventricular tachycardia	157	82 (52%)
Atrial flutter	22	12 (55%)
Atrial fibrillation	5	3 (60%)
Paroxysmal atrial tachycardia	11	7 (64%)
Complete block	95	55 (58%)

DIAGNOSIS

An ultrasound examination allows for the diagnosis of foetal arrhythmia, while echocardiography determines its type.

The first step is to investigate whether the foetal heart rhythm is regular or not. Decreases in foetal heart rate, including a complete few-second arrest, which are a physiological phenomenon unrelated to pathology of the electrical conduction system, but to its immaturity, are often observed up to 25 weeks gestation. This is not an indication for echocardiography. Embrio heart rate should be assessed using M-mode ultrasound from the first trimester as decreased or increased heart rate may be the first symptom of foetal disease. Foetal heart rate in numbers is absolutely necessary between 11.0-13.6 weeks of gestation. Physicians using the FMF software need to enter the heart rate values in order to calculate the risk of chromosomal aberrations. Unfortunately, sometimes foetal heart rate of 140 is automatically entered into the report during the first trimester scanning, which is a considerable mistake as such slow heart rate is not normal in this period of foetal development as opposed to the later stages of pregnancy. Heart rate of 140 bpm is automatically entered in the first trimester ultrasound, which is abnormal in a healthy foetus as it indicates bradycardia. In the case of doubts about heart rate in the mid-pregnancy, the number of 140 bpm in the first trimester may indicate problems occurring already in this period and if not documented in an image, interpretation of this result is impossible.

Foetal Cardiovascular Profile Score was introduced into prenatal cardiology by J. Huhta (fig. 1) (3). Using foetal ultrasound, Doppler and echocardiography, foetal cardiovascular system can be precisely evaluated. The scale allows foetal monitoring as well as prediction of neonatal condition. A newborn with prenatal score below 5 is known to have little chance of survival, especially if born prematurely. Implementation of appropriate transplacental therapy enables effective treatment.

Since different ECG waves correspond to certain mechanical cardiac cycles, it is possible to identify the type of foetal rhythm disturbances. In M-mode with a four-chamber view, the rate of atrial and ventricular contractions should be calculated – under normal conditions each atrial contraction is followed by ventricular contraction and the heart rate is between 120 and 160 bpm (fig. 2a).

Doppler ultrasound evaluates the atrioventricular (AV) conduction time measured from A-wave (atri-

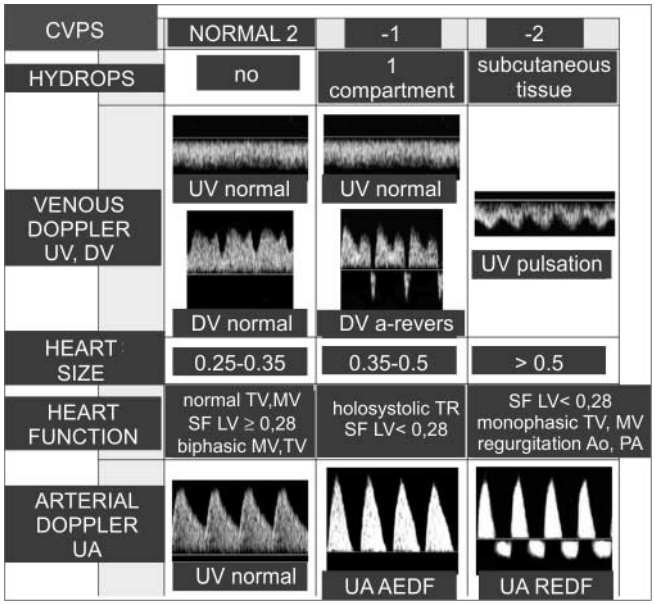

Fig. 1. Huhta's cardiovascular scale. A healthy foetus is given a score of 10. One or two points are taken for every diagnosed abnormality – in accordance with the table

al contraction) of the mitral valve to aortic ejection. It corresponds to the "mechanical" PR interval on the ECG (fig. 2b) and is up to 150 ms in sinus rhythm.

CLASSIFICATION OF CARDIAC RHYTHM ABNORMALITIES

Foetal cardiac rhythm abnormalities:

1. Irregular heartbeat, i.e. premature contractions (80-85%):
   a) premature atrial contractions,
   b) premature ventricular contractions – sporadic.
2. Tachyarrhythmias:
   a) sinus tachycardia (10-15%),
   b) supraventricular tachycardia,
   c) atrial flutter/fibrillation,
   d) ventricular tachycardia – very rare.
3. Bradyarrhythmias (5-10%):
   a) sinus bradycardia – obstetric causes should be excluded,
   b) bradycardia secondary to blocked premature contractions,
   c) non-sinus atrial rhythm,
   d) atrioventricular blocks.

PREMATURE CONTRACTIONS

Premature contractions are a mild form of arrhythmia not requiring treatment. The long, floppy septum primum, which is called formen ovale aneurysm, hits the posterior wall of the left atrium during the cardiac cycle and is the mechanical cause of premature atrial contractions. Premature contractions may be conducted – when each premature contraction is followed by ventricular contraction, or non-conducted, when atrial contraction is not followed by ventricular contraction. Atrial bigeminy, when every other atrial contraction is blocked, results in a slow ventricular rhythm, usually

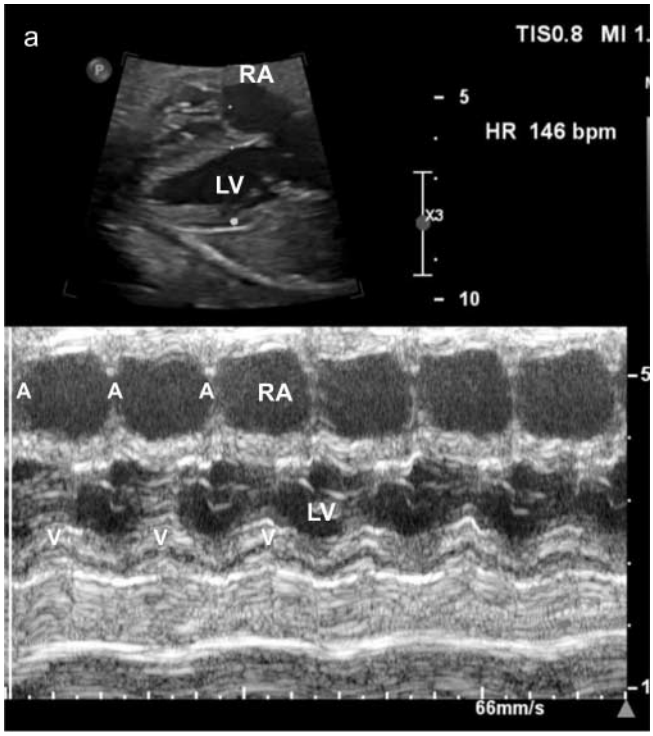

Fig. 2a. M-mode in a foetus with sinus rhythm of 146 bmp. A section through the right atrium (RA) and the left ventricle (LV)

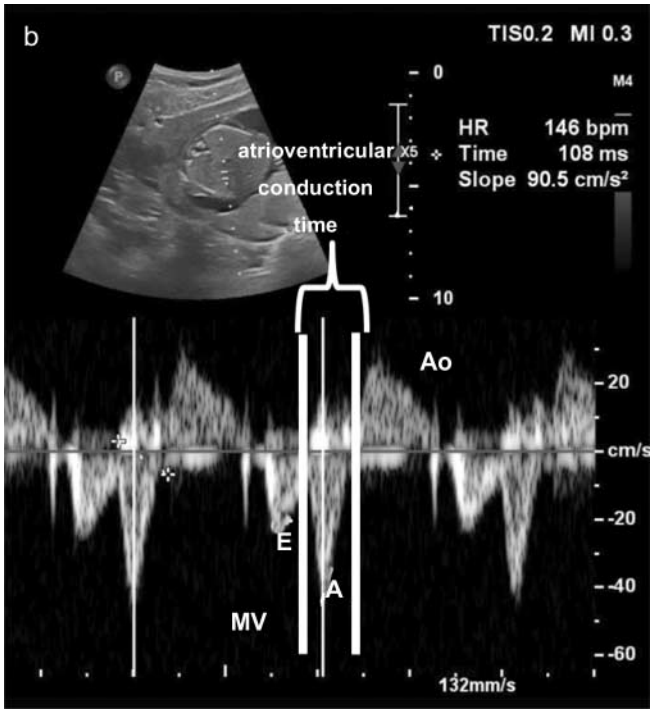

Fig. 2b. Doppler ultrasound – a gate inserted between the left ventricular inflow and outflow tracts, registering mitral valve (MV) and aortic (Ao) flow with atrioventricular (AV) conduction time measurement

about 70-90 bpm, is the greatest concern for an obstetrician. This type of foetal heart rhythm should be differentiated from a complete heart block (CHB). Blocked atrial contractions resolve spontaneously and do not require treatment (fig. 3a-3c). In contrary to CHB, atrial and ventricular contractions are quite regular, with blocked every other atrial contraction. In CHB, the atrial and ventricular

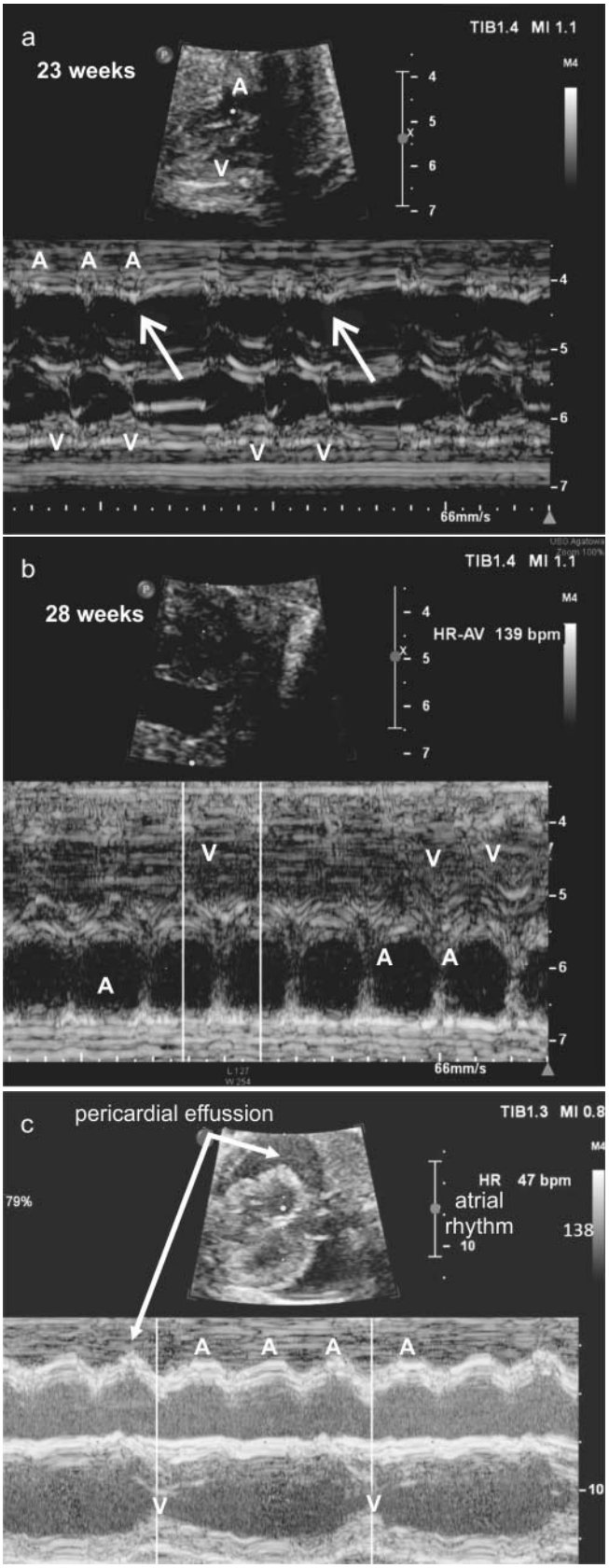

Fig. 3a-c. The use of M-mode in the diagnostics of rhythm and conduction disturbances:
a. M-mode in a foetus with blocked atrial premature contractions (bold arrows) causing foetal bradycardia of 80-90 bpm. A – atrial contractions, V – ventricular contractions
b. The same foetus at 28 weeks gestation; regular atrial and ventricular contractions, no treatment
c. A foetus at 29 weeks gestation with complete heart block and significant pericardial effussion indicating heart failure

rhythms are independent of one another.

In about 10-15% of cases premature atrial contarctions can cause more dangerous arrhyhmia, like supraventricular tachycardia. Isolated extrasystoles, even when multiple, do not require transplacental treatment or any other therapy. They are not an indication for premature delivery, but for transplacental treatment.

Premature contractions significantly affect CTG recording. As mentioned at the beginning, cardiotocography is not a good diagnostic method for heart rhythm abnormalities as it does not record all contractions. In arrhythmia, the recording is not characteristic for life-threatening foetal decelerations, but periods of "strange" slow heart rhythm occur (fig. 4a). This type of CTG trace is an indication for ultrasound examination. The foetal heart should be immediately investigated; irregular heart rhythm indicates that the abnormal CTG is due to mild arrhythmia, which is not an indication for treatment or premature delivery (fig. 4b). Of course, heart rate and size should be measured; evaluation of peripheral blood flow and the volume of amniotic fluid is indicated. Also, heart anatomy should be assessed as premature contractions sometimes occur in foetuses with structural defects. This type of CTG trace is not an indication for pregnancy termination. In the case of doubts, the patient should be consulted at a prenatal cardiology center.

TACHYARRHYTHMIAS

In foetal sinus tachycardia, the heart rate ranges between 160 and 200 bpm. It is likely to occur in intrauterine infection, hyperthyroidism, or foetal distress. Foetal anaemia does not induce tachycardia.

Maternal cultures and laboratory tests for infection should be performed. Prenatal diagnosis of hyperthyroidism is very difficult unless foetal thyroid goitre is visible. Thyroid function should be assessed in a newborn, who may require hyperthyroidism and tachycardia treatment. In each case, foetal echocardiography as well as neonatal electrocardiography and echocardiography should be performed.

Supraventricular tachycardia (SVT) is a heart rate above 200 bpm with 1:1 atrioventricular conduction (fig. 5a). Doppler ultrasound allows for an approximate determination of the type of supraventricular tachycardia, followed by the implementation of appropriate transplacental treatment. Untreated foetal SVT may result in heart failure, hypoxia and CNS damage.

Ultrasound usually shows foetal heart enlargement, atrial enlargement in particular. The ventricular function is significantly impaired. Since the end-diastolic ventricular pressure increases, the inflow into the ventricles occurs only during atrial contraction. This results in monophasic filling of the ventricles, only during atrial contraction, not during end-diastole. Increased end ventricular and atrial pressure leads to rise of the venous pressure. Abdominal effusion occurs, followed by pericardial, and pleural effusion, and subcutaneoun oedema in the most severe cases.

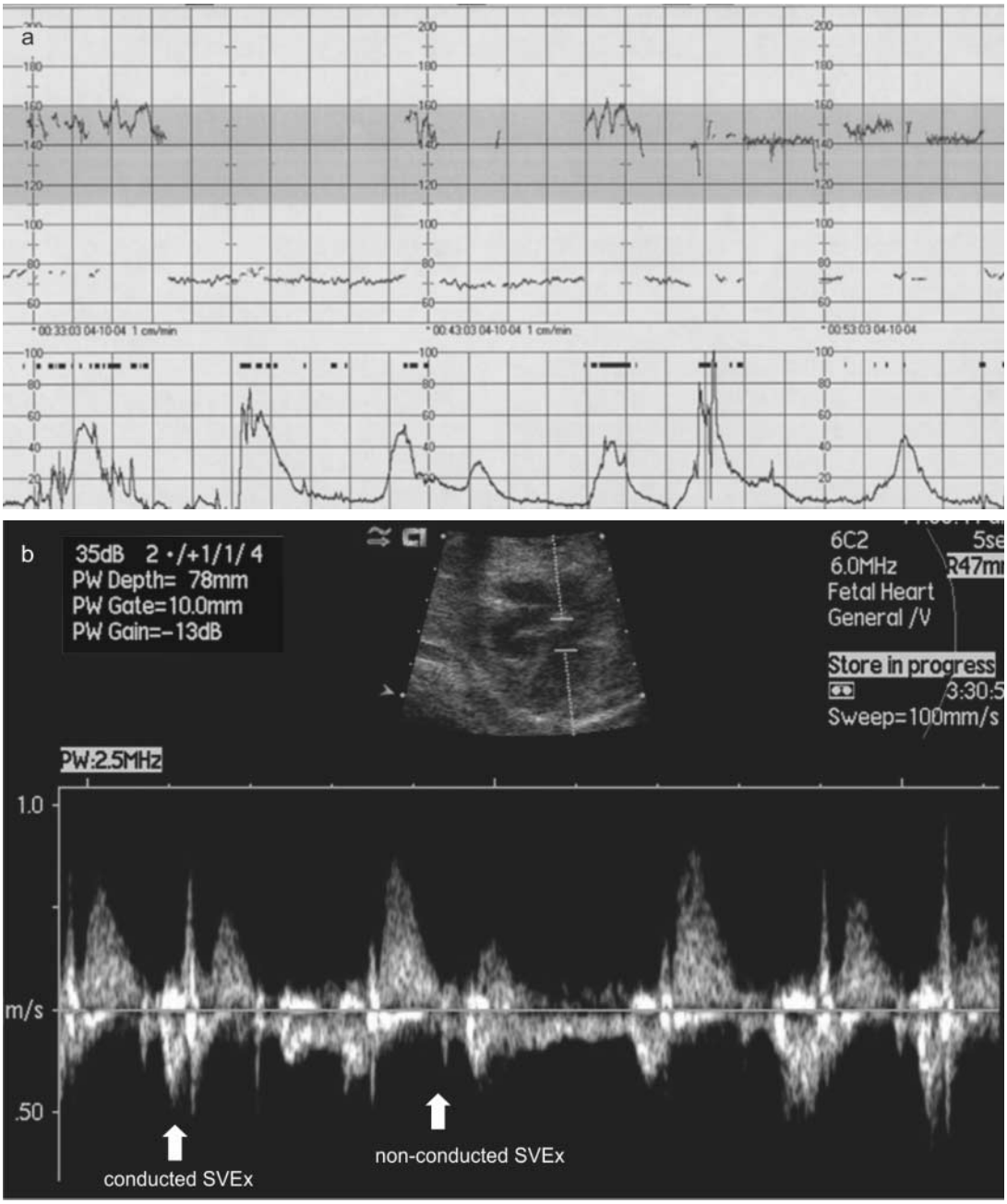

Fig. 4a, b. A correlation between CTG and ECHO in a foetus with supraventricular extrasystoles – non-threatening cardiac rhythm disturbances significantly affect CTG, which is not a good diagnostic method in arrhythmias:
a. CTG reading in a 37-week foetus with multiple conducted and non-conducted premature contractions
b. Doppler ultrasound in the same foetus performed immediately after CTG. Premature conducted and blocked atrial contractions can be seen. Normal biometrics, amniotic fluid volume, peripheral flows and cardiovascular anatomy. These are mild arrhythmias not requiring treatment or other medical interventions

Doppler ultrasound shows abnormal peripheral venous flows (fig. 6a, b) with normal blood flow in the umbilical artery and, frequently, with reduced resistance in the middle cerebral artery (fig. 6a, c), which is a manifestation of foetal circulatory compensation aimed at an adequate supply of oxygen to the CNS. Fortunately, normal placental function in SVT allows for an implementation of effective therapy.

Premature delivery in cases with foetal tachycardia is a mistake.

The treatment should be implemented by a prenatal cardiologist, who is familiar with the strategies of transplacental treatment, is able to assess the foetal condition and decide on the date of pregnancy termination. The foetus receives transplacental treatment, while the healthy mother is administered medications. Additional tests and adverse effects monitoring should be also performed in the mother. The drug doses are higher than those administered in adult patients as they only partially pass through the placenta and reach the foetus. The signs in ECG recordings should be well-known. In the case of digoxin, ECG changes may suggest myocardial hypoxia, especially in automatic interpretation.

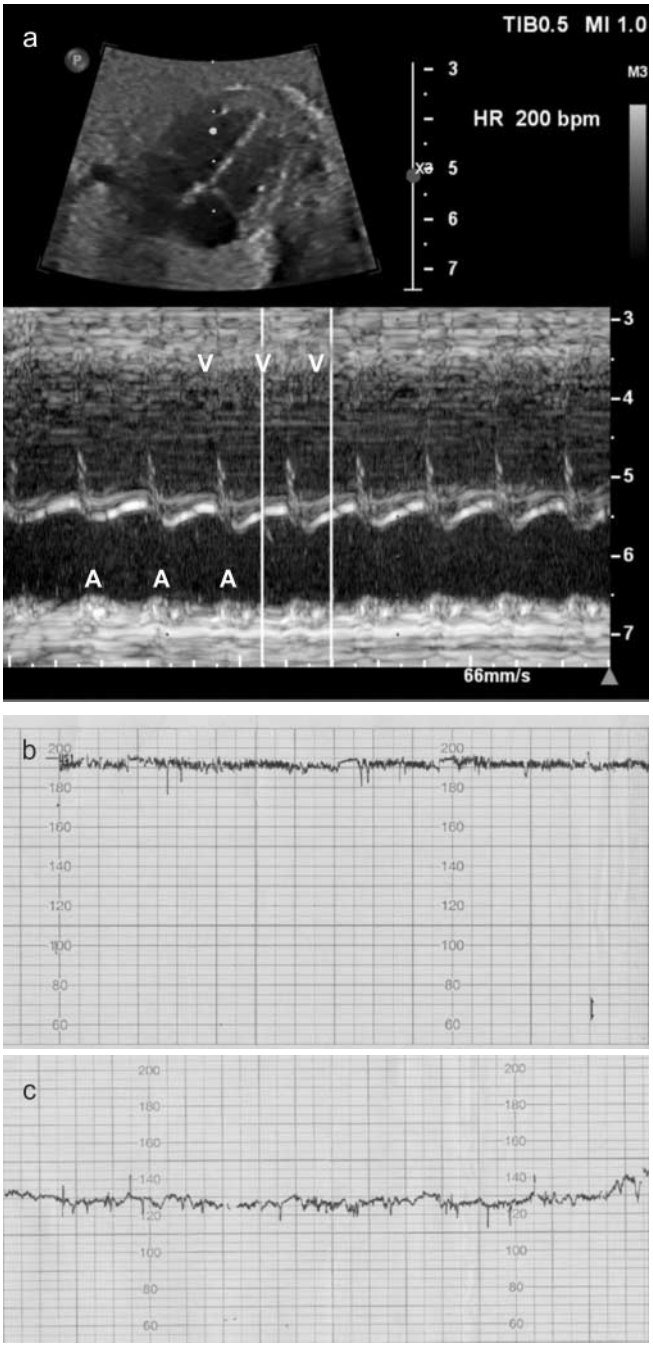

Fig. 5a-c. Foetal supraventricular tachycardia – ultrasound and CTG images taken during treatment and after conversion into sinus rhythm:
a. M-mode in a foetus with supraventricular tachycardia of 200 bpm.
b. CTG during treatment in a foetus with supraventricular tachycardia – slower tachycardia rate
c. CTG after conversion into sinus rhythm ranging between 120 and 140 bpm. CTG variability is often reduced in this period due to electrical conduction system blockage induced by anti-arrhythmic therapy. Sometimes, the reading is completely "silent", which is normal after the inhibition of tachycardia or atrial flutter

In prenatal cardiology, the therapeutic method depends on the time of ventricular-atrial conduction as well as the presence or absence of foetal oedema. So far, no international strategy of antiarrhythmic therapy for foetuses with SVT has been established (4). In our centre, which boasts the most extensive experience in the treatment of foetal arrhythmia in Poland, digoxin and amiodarone are the most common therapy. Adenosine and amiodarone are administered directly into the umbilical vein in treatment-resistant arrhythmia, however, in very rare cases. It is easier to achieve conversion into sinus rhythm in foetuses without oedema.

Despite the fact that CTG is not the best method to monitor foetal arrhythmia, it is still widely used in obstetric departments. CTG interpretation in foetuses with arrhythmias and those receiving treatment should be completely different. Reduced rate of tachycardia in CTG reading is the first sign of the activity of antiarrhythmic agents (fig. 5b). After conversion into sinus rhythm, CTG (fig. 5c) trace shows slower foetal heart rhythm and is less reactive than and less variable than in healthy foetuses, which is due to foetal electrical conduction system block induced by antiarrhythmic agents.

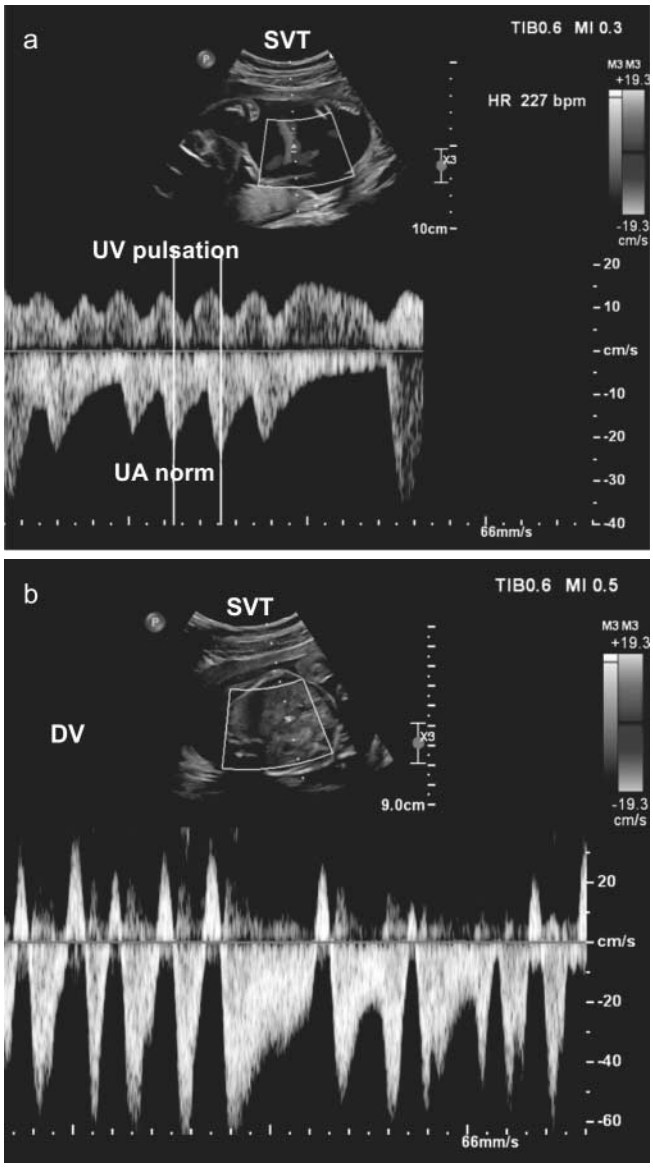

Fig. 6a, b. Supraventricular tachycardia in a foetus receiving anti-arrhythmic treatment – peripheral flows:
a. Umbilical arterial and venous flow in a foetus with SVT – a clear high venous pulsation, normal arterial flow
b. A reversed venous a-wave in a foetus with tachycardia, periodical inhibition of atrioventricular conduction can be seen

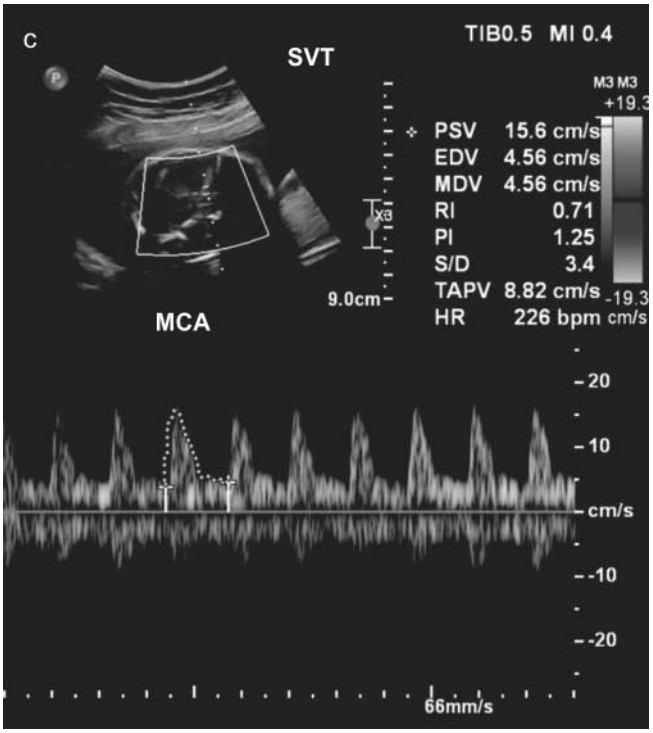

Fig. 6. Supraventricular tachycardia in a foetus receiving anti-arrhythmic treatment – peripheral flows:
c. MCA flow with slightly reduced PI in a foetus with SVT

From a total of 97 SVT foetuses treated in Warsaw between 2002 and 2015, prenatal conversion into sinus rhythm was achieved in 90% of cases. The longest duration between therapy onset and sinus rhythm achievement was 6 weeks; the boy is now 12 years old and develops properly. At the age of 6 years, he required accessory-pathway ablation.

Atrial flutter is a heart rhythm with atrial frequency of more than 300 bpm (heart rate of more than 500 bpm is atrial fibrillation, which is rare in the prenatal period) with atrioventricular block (usually 2:1, but also 3:1 or 4:1). The higher the degree of atrioventricular block, the lower the ventricular rate, but the rate is hemodynamically inefficient due to the too rapid atrial rate. Foetal diagnosis is mainly based on M-mode ultrasound showing higher atrial rate compared to ventricular rate (fig. 2b). Atrial flutter is slightly more common than SVT in foetuses with congenital heart defects.

The prenatal treatment of AF is difficult and involves the administration of digoxin and amiodarone. Digoxins are not used after 35 weeks of pregnancy, when electrical cardioversion may be necessary in the newborn.

BRADYARRHYTHMIAS

Bradycardia with 1:1 conduction is a heart rhythm below 110 bpm. First of all, obstetric causes of bradycardia, i.e. placental insufficiency, should be excluded. In the case of normal amniotic fluid volume and peripheral blood flows, accurate foetal cardiac diagnosis should be performed.

Bradycardia with 1:1 conduction is the most common sinus bradycardia, which may be caused by congenital hypothyroidism in the foetus, long QT syndrome (undetectable in the prenatal period) and, sometimes, maternal medications that decrease the heart rate. Congenital sinus bradycardia without other pathologies is very rare. If no foetal symptoms indicating heart failure are observed, no treatment is needed.

Bradycardia with 1:1 conduction with a rate of 90-110 bpm may be also caused by the absence of sinus node which commonly occurred in foetuses with left atrial isomerism. They usually have atrial, nodal or junctional rhythm, which is always slower than sinus rhythm. The most common echocardiographic abnormality is an interruption of the inferior vena cava with azygos continuation, regardless of the intracardiac anatomy. Foetuses with 1:1 conduction bradycardia and suspicion of atrial rhythm have efficient cardiovascular system and do not require treatment.

Atrioventricular blocks

First-Degree Atrioventricular Block (AV I°)

It can be diagnosed based on atrioventricular conduction time measurement (fig. 2b). IF AV conduction time exceeds 150 ms, first-degree atrioventricular block may be suspected. The heart rate in foetuses with first-degree atrioventricular block is usually normal.

The assessment of atrioventricular conduction time is important in SS-A and SS-B positive women as this is the only way to monitor foetal electrical conduction system.

Second-Degree Atrioventricular Block (AV II°)

This condition can also be diagnosed based on atrioventricular conduction time measurement.

In Mobitz I block, gradual AV prolongation until the lack of intraventricular conduction, i.e. no flow into the aorta, occurs after the flow through the mitral valve.

In Mobitz II block, the duration of atrioventricular conduction is stable, but after one or more atrial contractions, i.e. the inflow through the mitral valve, no intraventricular conduction occurs. Foetal bradycardia is identified in these cases. This type of AV block is most common in the foetuses of Ro positive mothers.

Third-Degree Atrioventricular Block (AV III°)

The condition is diagnosed when atrial and ventricular contractions are independent of each other. Ventricular rate usually ranges between 50 and 70 bpm, atrial rate is between 100 and 140 bpm. Third-degree atrioventricular block occurs in foetuses with both normal cardiovascular structure and structural heart defects.

Third-Degree Atrioventricular Block with normal cardiovascular anatomy

Maternal anti-Ro (SS-A) and anti-La (SS-B) antibodies, which pass through the placenta and cause foetal electrical conduction system damage, are the most

common cause of this condition. These antibodies induce inflammatory reaction within the AV node and the myocardium, leading to conduction impairment. The antibodies are present in women with systemic connective tissue diseases (lupus erythematosus, Sjögren's syndrome), but they are commonly detected in the mother after a complete AV block is diagnosed in the foetus. This is an acquired intrauterine disease (fig. 7a, b). Permanent foetal atrioventricular block is incurable (5). Polish recommendations for the management of pregnant women with systemic connective tissue diseases have been issued (6).

It is a known fact that antibodies begin to pass the placenta at about 16 weeks of pregnancy. From this time point up to 24 weeks gestation, the time of AV conduction should be monitored every 7 days, followed by echocardiography every 4 weeks. Foetal cardiovascular efficiency (fig. 3c), heart size, ventricular contractility and the presence of fluids in the body cavities should be evaluated. Transplacental treatment is not needed in majority of cases. If signs of heart failure occur, a rescue treatment with intramuscular or oral betamethasone or dexamethasone is implemented. Beta-agonists can be used to increase the foetal heart rhythm in the case of decreased heart rate below 55 bpm. Both, monitoring and treatment should be provided in a centre with experience in the the assessment of foetuses with complete AV block. A total of 43 foetuses with complete AV block in anti-Ro positive mothers were monitored in the Warsaw Centre between 2002 and 2014, and then treated at the Cardiology Clinic in the Child's Child Health Centre.

The group of foetuses with CHB without structural heart defect whose mothers are SS-A and SS-B negative is much smaller. These children usually have primary myocardial disease. There were two foetuses with noncompaction myocardium in our material. Premature delivery is not recommended in CHB cases. The chances of successful treatment increase in term newborns. The available literature indicates that the most severe complications in children are due to prematurity, and not the underlying disease (7).

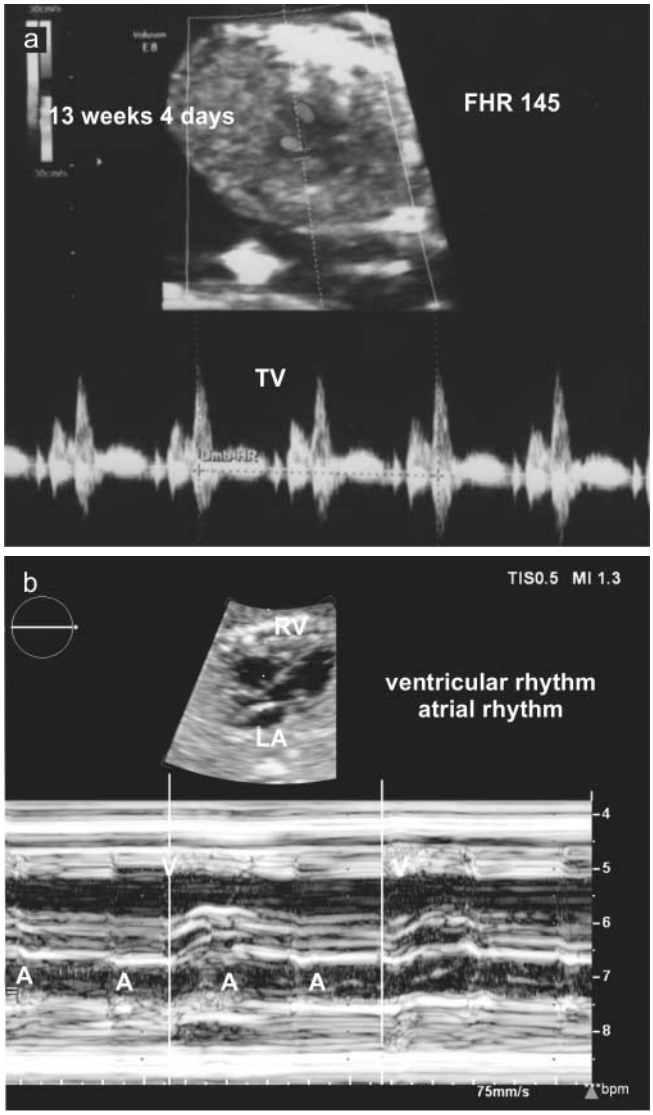

Fig. 7a, b. Heart images of a foetus whose mother has anti-Ro antibodies detected after the diagnosis of complete heart block at 24 weeks gestation:
a. Heart rate of 145 bpm in a foetus at 13 weeks gestation – the lower limit of normal (the test was performed outside our centre)
b. M-mode in the same foetus at 26 weeks gestation – complete heart block with cardiovascular efficiency

Third-Degree Atrioventricular Block with congenital heart defect

During embryonic development, damage of the electrical conduction system may occur in some cases. These mostly include complex congenital defects: congenitally corrected transposition of the great arteries and visceral heterotaxy syndrome – left-isomerism with complex intracardiac pathology. As opposed to intrauterinely acquired AV block due to positive anti-Ro antibodies, the AV block in foetuses with congenital heart defect can be diagnosed already in the first trimester of pregnancy due to the absence of proper atrioventricular conduction. The coexistence of complex congenital defect and complete AV block very often results in severe intrauterine heart failure with generalised oedema of the foetus and, consequently, foetal or early postnatal death. Basing on our experience, such conditions are lethal and perinatal pallaitive care should be provided for those families who decided to carry on the pregnancy. Between 2002 and 2015, complete AV block with congenital heart defect was diagnosed in 26 foetuses with only one survived child with isolated corrected transposition of the great arteries, with no heart failure.

CONCLUSIONS

Heart rhythm abnormalities are easy to visualise but more difficult to precisely diagnose in the prenatal period. A close cooperation between obstetricians and cardiologists is needed to ensure the best possible care for pregnant women and their children with heart rhythm disturbances. Arrhythmias are not

an indication for premature delivery. Foetuses with arrhythmia should be monitored by ultrasound and echocardiography, not cardiotocography.

Premature contractions are a mild form of arrhythmia. In majority of cases, they do not require treatment and resolve in the perinatal period. A formane ovale aneurysm is a common mechanical cause of this type of arrhythmia.

Foetuses with supraventricular tachycardia require transplacental treatment; conversion into the sinus rhythm can be achieved in more than 90% of cases. Premature delivery without reference foetal cardiology center consultation is a mistake. Complete atrioventricular block without structural heart defects is an intrauterine acquired condition due to SS-A and SS-B antibodies, which induce damage of the foetal conduction system. Foetuses with complete block should be born at term as prematurity adversely affects further development. Complete atrioventricular block with foetal heart defect and cardiovascular failure is lethal disorder and perinatal palliative care should be provided.

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