INVITED REVIEW

Management of tachyarrhythmia during pregnancy

Gebelik sırasında taşiaritmi tedavisi

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Summary– Maternal tachyarrhythmia is a common complication during pregnancy due to hormonal changes that enhance pre-existing arrhythmias or induce new arrhythmias in the presence of congenital heart defects in pregnant females. Presence of tachyarrhythmia during pregnancy poses risk to the mother and fetus, calling for proper treatment with medications. Use of antiarrhythmic drugs in cases of maternal tachyarrhythmia must give due consideration of potential teratogenic side effects. Utilization of antiarrhythmic drugs during pregnancy has been well studied; some result in minimal fetal harm or none at all. New techniques, such as cardiac ablation, have also been implemented with minimal or no radiation exposure to the fetus or mother. Pregnant women with tachyarrhythmia have been successfully treated with little to no impact on the developing fetus as result of increasing experience with antiarrhythmic drugs and progress of new procedural techniques.

Özet– Annede taşiaritmi, hamile kadınlarda önceden var olan aritmileri arttıran veya doğuştan kalp defektleri varlığında yeni aritmileri başlatan hormonal değişikliklere bağlı olarak sık görülen bir komplikasyondur. Gebelik sırasında taşiaritminin varlığı anne ve fetüs için risk oluşturmakta ve ilaçlarla uygun tedaviyi gerektirmektedir. Teratojenik yan etkilerini de göz önüne alarak maternal taşiaritmide antiaritmik ilaçların kullanılması düşünülmelidir. Gebelik sırasında antiaritmik ilaçların kullanılması iyice incelenmiş olup bazıları minimal fötal hasara yol açacak bazıları hiç zararlı olmayacaktır. Kardiyak ablasyon gibi yeni teknikler de fetüs veya anneyi minimal (veya hiç) radyasyona maruz bırakmadan uygulanmıştır. Antiaritmik ilaçlarla giderek artan deneyim ve yeni prosedürel tekniklerdeki ilerleme sayesinde gebe kadındaki taşiaritmiler başarıyla ve gelişmekte olan fetüse çok az (veya hiç) etkiyle tedavi edilmektedir.

Taking antiarrhythmic drugs during pregnancy can pose different risks to the fetus, depending on the class of antiarrhythmic drug and which trimester the mother is in. The greatest teratogenic risk to the fetus is during first trimester of pregnancy, whereas second and third trimesters pose more risk for fetal growth, development, and possible induction of arrhythmia in the fetus.[1,2] Teratogenic risk during first trimester alludes to the fact that organogenesis occurs during this stage of pregnancy. Therefore, medical treatment of arrhythmia is only advised in situations of severe symptoms or increased risk of adverse consequences to the mother or fetus.[1] There are many physiological changes during pregnancy that can affect absorption, bioavailability, and elimination of anti-arrhythmic drugs, making it difficult to maintain blood therapeutic drug levels.[1,2] These changes include cardiac output and blood volume, decreased serum protein concentration, increased renal perfusion, enhanced liver metabolism, impairment of gastric secretion and motility, and hormonal stimulation of liver enzymes.[1,2] Due to possible adverse effects of drugs on the mother or fetus, there is a US Food and Drug Administration classification system to define the level of risk of the drug if taken during pregnancy. The aim of this review article is to describe the classification system and several case studies along

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with most common treatment strategies for managing different types of arrhythmia in pregnancy.

Arrhythmias in Pregnancy

Premature atrial complexes and premature ventricular complexes

Premature atrial complexes (PACs) and premature ventricular complexes (PVCs) are very common during pregnancy. Oftentimes they are anxiety-provoking in patients who have not experienced them before. However, in healthy pregnant women with structurally normal hearts they are very benign. Pregnant women who are asymptomatic call for reassurance that these complexes will cause no harm. It is important to educate on stimulants, such as caffeine and alcohol, which increase frequency of PACs and PVCs. In pregnant patients with severe symptoms or symptoms interfering with their daily lives, beta blockers, such as metoprolol and propanolol, can be given for symptomatic management.[3]

Supraventricular tachycardia

Supraventricular tachycardia (SVT) is defined as narrow complex tachycardia with heart rate greater than 100 bpm and mechanism of development at the level of the atrioventricular (AV) node. SVT is the most common arrhythmia present in women of reproductive age, with a prevalence of 24 per 100 000 hospital admissions.[4,5] Risk of developing SVT increases during pregnancy, labor, and delivery.[4] Furthermore, exacerbation of previously diagnosed SVT occurs in approximately 20% of pregnant females.[2,5] Among subtypes of SVT, AV nodal reentrant tachycardia (AVNRT) and atrioventricular reciprocating tachycardia are the most common forms, with atrial tachycardia being the least frequent cause of SVT in pregnant females.[4,5] It is important to diagnose and recognize SVT because it can pose harm to the mother and fetus, such as hemodynamic instability and fetal hypoperfusion, resulting in emergent caesarean section when the fetus is pre-term.[4] There is a relationship between gestational paroxysmal SVT and septal cardiac defects. Therefore, in pregnant women, it is very important to recognize and treat SVT.

Atrioventricular nodal reentrant tachycardia and Wolff-Parkinson-White syndrome

When treating pregnant females with an SVT, always consider severity of the symptoms and physiological effect of the arrhythmia. If the patient is asymptomatic or the symptoms are mild and there is no hemodynamic compromise, then reassurance alone is advised.[2]

Abbreviations:

AV	*Atrioventricular*
AVNRT	*AV nodal reentrant tachycardia*
DC	*Direct current*
IV	*Intravenous*
LVOT	*Left ventricular outflow tract*
PAC	*Premature atrial complex*
PVC	*Premature ventricular complex*
RVOT	*Right ventricular outflow tract*
SVT	*Supraventricular tachycardia*
VT	*Ventricular tachycardia*
WPW	*Wolff-Parkinson-White syndrome*

Initial management in pregnant patients for acute conversion in the setting of SVT is vagal nerve stimulation by way of carotid massage.[1,2,4,6,7] Various vagal maneuvers can also be performed. Examples include exhibiting gag reflex, the Valsalva maneuver, immersing the patient's face in ice-cold water, or coughing. If vagal nerve stimulation fails, proceed to medical management. In pregnant patients, first line medical treatment in AVNRT should be intravenous (IV) adenosine (category C). It is effective in terminating paroxysmal SVT in approximately 84% to 90% of maternal cases.[1,4–7] Standard dose of adenosine is rapid IV administration of 6 mg, followed by 12 mg dose if the first dose fails to convert the patient to normal sinus rhythm after 1 to 2 minutes.[4,8] Many studies have demonstrated variations of second dose, ranging between 12 and 18 mg if the initial dose of 6 mg fails to convert the patient to normal sinus rhythm.[9–11] Second dose of adenosine has been safely administered in doses up to 24 mg IV during pregnancy.[10] Increasing dose to 15 mg in 1 study led to fetal bradycardia in 1 patient, which reverted back to normal within 10 minutes of administering bolus.[9] Adenosine has been most studied during second and third trimesters, and is generally accepted as safe for use. Adenosine use in first trimester is still not advised due to lack of sufficient studies indicating safe administration and good outcome.[2] Beta blockers, such as IV propranolol (category C) and IV metoprolol (category C), have been extensively studied for use during pregnancy and are considered safe to use in the absence of adenosine.[1,2,5–8,10] Although digoxin (category C) is considered to be one of the safest drugs to use during pregnancy 10, there are few data showing effectiveness on converting the patient to normal sinus rhythm when used alone. Therefore, combined use with beta blocker is advised.[5] In pregnant patients with AVNRT, 10 mg of IV verapamil (category C) over 3 minutes 11, or

5 mg of IV verapamil given over 5 minutes has been shown to successfully convert pregnant patients back to normal sinus rhythm.[12] However, verapamil is considered third line agent in treatment of SVT during pregnancy due to potential adverse effects on the fetus, such as bradycardia, heart block, hypotension, and sometimes fetal demise.[5,6,9–11]

In pregnant patients with AVNRT and Wolff-Parkinson-White syndrome (WPW), long-term prophylactic treatment should only be considered in cases of hemodynamic instability and in patients with symptoms affecting everyday life.[7] For AVNRT, metoprolol, propranolol, and digoxin have been extensively studied as the safest drugs to use for prophylaxis during pregnancy and are considered first line therapy. Beta blockers and verapamil are not recommended for use in patients with WPW. Instead, flecainide (category C), quinidine (category C), and procainamide (category C) can be used to aid in blocking accessory pathway conduction.[1]

If refractory to the aforementioned treatments, fetal distress may ensue. Hence, it is imperative to continue step-wise approach to treatment with direct current (DC) cardioversion. DC cardioversion poses low risk to the mother and fetus, possibly due to lack of shock to uterine area.[10] Yilmaz et al. discussed a case where cardioversion with 100 J successfully converted a patient back to normal sinus rhythm. When 100 J fails to convert the patient back to sinus rhythm, energy of up to 400 J has been used during pregnancy at all stages with no adverse effects on the mother or fetus.[13]

There have been some studies that show good outcomes in the fetus after catheter ablation.[14] However, the procedure is traditionally inadvisable due to radiation exposure to the developing fetus.[7,15] Luckily, new techniques of ablation have been attempted and been successful with minimal use of fluoroscopy, and sometimes without radiation exposure at all.[14,15] Szumowski et al. presented 9 cases with mean fluoroscopy time of 42±37 seconds that resulted in successful ablation. In order to better protect the fetus, external shielding of the abdomen is recommended during procedure. One case study by Clark et al. resulted in successful delivery and no recurrence after catheter ablation had been performed in a pregnant woman with SVT. Ablation was guided by EnSite NavX (St. Jude Medical, Inc./Abbott Laboratories, Lake Bluff, IL, USA) cardiac mapping system with no use of fluoroscopy.[15] Despite the fact there are new methods of mapping and ablation that can be done with minimal to no use of fluoroscopy, ablation should only be considered as a last resort in pregnant females that have hemodynamic instability that is resistant to conservative management.[14]

Atrial tachycardia

Atrial tachycardia during pregnancy has not been very well studied because it is relatively rare. It often leads to tachycardia-induced cardiomyopathy in pregnant women, and treatment is usually indicated.[16] For these patients, antiarrhythmic therapy is first line therapy. Oftentimes, arrhythmia is difficult to treat with medical therapy due to refractory nature of these arrhythmias. In these cases, catheter ablation should be considered.

Pregnant patients with atrial tachycardia should first undergo vagal maneuvers, similar to SVT, prior to initiating antiarrhythmic therapy. If maneuvers fail, first line antiarrhythmic therapy in acute treatment of atrial tachycardia in pregnancy, as with other SVT subtypes, is IV adenosine.[1,5] Adenosine is successful approximately 30% of the time in pregnant women with atrial tachycardia.[7] If adenosine is not available, flecainide can be used for conversion to sinus rhythm. Another option to consider is sotalol, although caveat is 3% to 5% chance of developing torsade de pointes and has been associated with fetal loss in some cases.[11] In pregnant patients, amiodarone should be used as last resort antiarrhythmic agent due to its teratogenic effects on the fetus.[17]

Majority of pregnant women with atrial tachycardia have demonstrated persistent tachycardia despite use of antiarrhythmic treatment that eventually led to need for catheter ablation. Due to risk of radiation exposure to the fetus, catheter ablation was often avoided when the patient needed the procedure. However, as mentioned before, there have been multiple cases showing minimal to no radiation exposure using new techniques involving 3-dimensional mapping systems.[14,16,18–21]

When rate control is necessary in pregnant women, beta blockers, digoxin, and verapamil are all category C drugs that have been shown in previous studies to be relatively safe for the fetus.[22,23]

DC cardioversion has rarely been proven successful, and has no role in standard treatment of atrial tachycardia.[7,19,22]

Atrial fibrillation and atrial flutter

It is uncommon to see pregnant females presenting with new onset atrial fibrillation or atrial flutter. When present, it is most often in the setting of structural heart disease (specifically rheumatic valvular disease or dilated left atrium), congestive heart failure, hypothyroidism, or women with previous history or documentation of atrial fibrillation or flutter. Hence, good history and appropriate tests in these patients are warranted.[7,24] Immediate treatment is imperative due to negative effects of atrial fibrillation and atrial flutter on prenatal care of the mother and fetus. Rapid ventricular response during either arrhythmia may lead to hemodynamic compromise in both the mother and fetus.[25]

In pregnant patients who are hemodynamically unstable, consider electric cardioversion (50–100 J for atrial fibrillation and 25–50 J for atrial flutter).[1,7] In hemodynamically stable patients, both quinidine (category C) and procainamide (category C) are considered safe agents.[1] Ibutilide and flecainide have shown success, but there is limited data to support their efficacy.[7,24–26] Newer Class III intravenous agent ibutilide (category C) has 1.7% possibility of inducing torsade de pointes.[27] With ibutilide, there have been some cases showing successful termination of acute atrial fibrillation with no adverse consequences or reactions in the fetus.[24,28] In pregnant patients with atrial flutter or fibrillation, .25 mg to 1 mg dose of IV ibutilide can be administered over 10 minutes to convert the patient to sinus rhythm. This can be repeated after 30 minutes if conversion to normal sinus rhythm is not achieved.[24,25] There is less experience with IV propafenone; therefore, this drug should only be considered for cardioversion as last resort when no other therapy has been successful.[7] Similar to treatment of SVT and atrial tachycardia, amiodarone should only be used in emergent situations when all other agents and techniques have failed to convert the patient to normal sinus rhythm.

In pregnant patients who do not convert to normal sinus rhythm with rhythm controlling methods, rate-controlling agents must be administered to prevent hemodynamic instability in both the patient and fetus.1 Beta blockers alone, specifically metoprolol or propranolol, or beta blockers in combination with digoxin, are considered first line treatments for both acute and chronic treatment of rate control.[6,29]

There is increased risk for venous thromboembolism in pregnant patients with new-onset atrial fibrillation and even higher risk in those with hypertension, congestive heart failure, prior cerebral vascular event, or rheumatic heart disease.[3] Therefore, proper anticoagulation should be administered throughout the pregnancy. Type of anticoagulation depends on specific risk factors and presence of structural heart disease. For higher risk patients, thromboembolic risk is assessed with CHADS2 and CHA2DS2-VASc tools.[26,30] If the patient has combined CHADS2 and CHA2DS2-VASc score greater than 4, or a score of 2 or greater for either CHADS2 or CHA2DS2-VASc, there is increased risk for thromboembolism, and anticoagulation agents are recommended based on gestational age. Warfarin (category X) should almost always be avoided in pregnancy due to its potential to cross the placenta and cause spontaneous abortion, fetal hemorrhage, mental retardation, and birth malformations.[3] If warfarin is needed in high risk patients with atrial fibrillation, period of time starting at 13 weeks gestational age in second trimester until 4 weeks prior to due date is the only window in which it is considered acceptable.[3,6,26] Maintaining internationalized normalized ratio from 2.0 to 3.0 is the goal when using warfarin. Increasing dose of warfarin over 5 mg increases risk of fetal malformation from 3.6% to 8%.7 Unlike warfarin, unfractionated heparin cannot cross the placenta and continuous infusion of unfractionated heparin based on weight is preferred during first trimester and as of 4 weeks prior to delivery.[3,6,7,26,31] Novel anticoagulants, including rivaroxaban, dabigatran, and apixaban, have not been extensively used in pregnancy and are not recommended for use at this time. All anticoagulation therapy should be stopped at onset of labor.[3,6,7]

Ventricular tachycardia, ventricular fibrillation, torsade de pointes

Ventricular tachycardia (VT) is rare, with prevalence of 2 in 100,000 hospital admissions.[5] Pregnant women with structural heart disease, including cardiomyopathy, congenital heart disease, valvular heart disease, and mitral valve prolapse, have increased risk of

developing VT during pregnancy.[1,6,7] It is important to evaluate pregnant patients who develop VT for structural defects, inherited arrhythmogenic disorders, and cardiovascular disease, because prevalence is 4.5 to 15.0 per 1000 pregnancies.[5–7] Presence of structural heart defects and other risk factors increase morbidity and mortality of patients with VT. Hence, proper history, physical examination, and echocardiography are important tests to rule out structural defects.[11,32] If a patient presents with new onset VT, it is important to evaluate for peripartum cardiomyopathy with echocardiography.[6,7,33] Even though VT is most often seen in patients with structural heart disease, there are data suggesting new onset idiopathic VT in healthy pregnant patients.[34] Treatment will vary significantly in high-risk patients compared with patients who have idiopathic new onset VT. Patients with new onset idiopathic VT have much lower chance of developing life threatening arrhythmias.[1]

New onset idiopathic VT without structural heart defects

New onset of VT in young, healthy pregnant females is usually idiopathic VT arising from the right ventricular outflow tract (RVOT) or the inferior left-sided ventricular septum. Idiopathic VT in young patients usually presents with hemodynamic stability and much lower risk for sudden cardiac death.[1]

Pregnant women are frequently symptomatic; however, some studies have shown up to 35% of patients with sustained VT during pregnancy experience nothing more than mild lightheadedness denying cerebral symptoms.[35] As mentioned previously, patients without associated heart disease are at lower risk for morbidity and mortality, and in some cases may require routine follow-up and continued reassurance.[36,37]

If arrhythmia is symptomatic and requires prophylactic therapy, initiate antiarrhythmic medications, such as verapamil and beta blockers.[6,33,38] Decision as to which medication to use depends on whether arrhythmia is RVOT or left ventricular outflow tract (LVOT) VT. For RVOT VT with left bundle branch block morphology, preferred treatment is beta blockers, such as metoprolol.[33,39] Beta blockers are relatively safe during pregnancy. However, there is risk maternal hypotension may result in fetal hypo-perfusion.[1] If beta blocker therapy fails, second line management includes sotalol or flecainide.[5] In rare instance that young woman presents with idiopathic LVOT VT with right bundle branch block morphology, verapamil is first line treatment due to its ability to block calcium ion influx.[11,33,40,41] In case of drug-refractory idiopathic VT during pregnancy causing hemodynamic instability or in case of recurrent idiopathic VT occurring postpartum, radiofrequency ablation is advisable.[7,39] Idiopathic LVOT can be treated with 2.5 mg IV verapamil or managed with oral verapamil throughout pregnancy. Ablation should be attempted postpartum.[40,41] Interestingly, the hormone progesterone can be used to induce arrhythmia. With the assumption of mimicking physiological state of pregnancy, utilization of progesterone can induce VT, which would allow for successful mapping and ablation.[41]

It is recommended to check for sustained presence of arrhythmia after delivery with electrocardiogram (ECG) or Holter ECG event monitor. In most cases of low risk patients, arrhythmia resolves spontaneously after delivery.[6]

New onset VT with structural defects or previous history of VT

In pregnant patients with VT who have structural defects and previous history of VT, risk of sudden cardiac death is higher and management differs from those without structural defects. Treatment depends on degree of hemodynamic instability and whether or not long-term treatment is indicated.

In pregnant patients who are hemodynamically stable, literature differs on appropriate first line therapy. In some studies, first line treatment is 50 to 100 mg of either IV procainamide or ajmaline (Class Ia) over 5 minutes.[7,11] Procainamide (category C) has been used frequently in pregnancy and is considered relatively safe. Ajmaline, on the other hand, has not been used extensively during pregnancy and therefore, no FDA category has been assigned to this drug. Risk to the mother and fetus is unclear. Therefore, ajmaline should only be used in emergency situations.[42] Other studies suggest lidocaine as first line treatment.[5,6] However, there have been a few instances in which lidocaine had toxic effects on the fetus in prolonged labor, fetal acidosis, or when the mother showed signs of liver or heart failure.[10] Therefore, in instances of prolonged labor, fetal acidosis, or ma-

ternal liver failure, avoid lidocaine.[10] Quinidine is second line therapy if procainamide, lidocaine, and ajmaline are not available.[5,43]

Pregnant patients with unstable VT should be cardioverted with 50 to 100 J. If first shock is ineffective, increase energy to 100 to 360 J.[1,5,7,10,11] Cardioversion is safe and has been shown to have little effect, if any, on the mother and fetus.[7,11] Due to radiation exposure, procedure time, and anesthetic use, ablation should only be used as last resort in emergent situations when all other therapies have been unsuccessful.[5] Recent studies have shown successful conversion with minimal fetal exposure to radiation; therefore, this option may become more readily available and acceptable in the future.[32,44–46]

For pregnant patients needing chronic prophylactic management of VT, beta-blocking agents, such as metoprolol or propranolol, are preferred.[5–7,29,33,44] Propafenone and flecainide have been shown to be relatively safe and effective for prophylactic therapy in patients without structural heart disease,[10,43,47,48] but all Class Ic agents should be avoided in patients with structural heart disease.[5,7] One study showed that sotalol can also be used for prophylaxis,[5] although it is not considered first line choice due to 3% to 5% risk of developing torsade de pointes.[5,11] Quinidine, mexiletine, and procainamide are also possible treatments to consider when first line therapy fails.[5,43] In cases where the patient is refractory to prophylaxis, consider insertion of implantable cardiac defibrillator in order to maintain hemodynamic stability in the mother during pregnancy.[5,7,10,11,49]

Conclusion

Even though there are limited data on treatment of arrhythmias during pregnancy, there is sufficient evidence to successfully treat women with various types of arrhythmia without adverse effects on the fetus. Most effective drugs with safest profiles should be considered first line treatment. For most cases, treatment is only indicated when there are symptoms affecting quality of daily life in the mother or when there is concern about hemodynamic instability in the mother and resulting negative impacts on the fetus. Generally, antiarrhythmic drugs are safer to use in second and third trimester than first trimester. This is primarily due to occurrence of organogenesis in first trimester. When DC cardioversion is needed for any specific arrhythmia, it is considered safe in pregnant patients with no effect on the fetus. In instances where catheter ablation must be performed, there are now new techniques utilizing electrophysiological mapping that employ minimal to no radiation. Implantation of cardiac defibrillator is recommended mainly in pregnant patients with VT or VF occurring in setting of structurally abnormal heart that is refractory to all other management strategies. Research in the future will continue to provide more proof and insight into preferred management of arrhythmias in pregnant patients.

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Keywords: Anti-arrhythmic agent; atrial tachycardia; pregnancy; premature atrial complex; premature ventricular complex; supraventricular tachycardia; tachyarrhythmia; ventricular tachycardia.

Anahtar sözcükler: Antiaritmik ilaç; atriyal taşikardi; gebelik; prematüre atriyal kompleks; prematüre ventriküler kompleks; supraventriküler taşikardi; taşiaritmi; ventriküler taşikardi.