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A Review Article on Tachyarrhythmias in Pregnancy

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ABSTRACT

Tachyarrhythmias, classified as an irregular heartbeat greater than 100 beats per minute, are pervasive during pregnancy and can increase the risk of cardiac morbidity and hospitalizations. Preexisting arrhythmias are exacerbated by pregnancy's hormonal and autonomic changes, whereas de novo arrhythmias are predisposed to by the increased cardiac output, heart rate, and ventricular stretching required to accommodate the increased cardiac output. The absence of randomized controlled studies evaluating the safety and efficacy of anti-arrhythmic drugs and anticoagulants makes the therapy of arrhythmias in this sensitive demographic group seem to be difficult. This review emphasizes the pathophysiologic mechanisms and incidence of tachyarrhythmias in pregnancy, highlighting the evidence-based therapies directed at ameliorating the effects of arrhythmias in pregnancy. We searched journal articles published between 1990 and December 2021 on PubMed, Embase and Google Scholar to identify studies evaluating the incidence and management of tachyarrhythmias in pregnancy. The goal of this review is to discuss the incidence of

tachyarrhythmias and current evidence for treatment in pregnancy to optimize maternal and fetal outcomes.

Keywords: Arrhythmias; Tachyarrhythmias in pregnancy; Management of arrhythmias; Anti-arrhythmic medications; Cardiovascular complications of pregnancy

Tweetable Statement

Hormonal & autonomic changes in pregnancy can cause de novo tachyarrhythmias or exacerbate preexisting ones. This review evaluates existing evidence to optimize treatment to improve maternal & fetal outcomes.

INTRODUCTION

During pregnancy, hormonal and autonomic changes occur to accommodate an increase in cardiac output to meet the metabolic demands of the growing fetus. Cardiac arrhythmias pose a serious threat to the health of both the mother and fetus[1]. Pregnancy may trigger exacerbations of pre-existing arrhythmias. Women with established tachyarrhythmias, congenital heart defects, or channelopathies have the highest risk for the development of arrhythmias when they become pregnant[2,3]. Although, they can also develop *de novo* or occur in women without apparent heart diseases. Tachyarrhythmias, including both supraventricular and ventricular tachycardias, is the most common cardiac complications observed during pregnancy[1].

We searched PubMed, Embase and Google Scholar to identify studies evaluating the incidence and management of tachyarrhythmias in pregnancy. Search terms included “tachyarrhythmias,” “atrial fibrillation,” “supraventricular tachycardia,” “atrial flutter,” “ventricular tachycardia” “ventricular fibrillation”, “ventricular arrhythmias”, “pregnancy”, “pregnant women”, “beta blocker”, “calcium channel blocker”, “digoxin”, “Anti-arrhythmic medicines”, “flecainide”, “propafenone”, “lidocaine”, “amiodarone” “anticoagulation”, “heparin”, “warfarin”, “oral anticoagulants”, “cardioversion”, “catheter ablation” and “ICD”. The search was limited to studies published in English in peer-reviewed journals between 1990 and December 2021.

Early retrospective studies by Li et al[4] demonstrated that the most commonly encountered arrhythmias were sinus tachycardia (ST), sinus bradycardia (SB), and sinus arrhythmia with a frequency of 104 in 100,000 pregnancy-related admissions followed by supraventricular tachycardia (SVT) with a prevalence of 24 in 100,000 hospital admissions. Approximately 20% of patients with paroxysmal SVT may become symptomatic during pregnancy. Atrial fibrillation (AF), ventricular tachycardia (VT), and ventricular fibrillation (VF) were relatively rare with a prevalence of 2/100,000. The frequency of ST, SB, and sinus arrhythmia may be an under estimation of the true incidence given these arrhythmias are usually seen in an outpatient setting. SVT is considered the most frequent sustained arrhythmia in pregnancy. AF and ventricular arrhythmias are described with varying frequencies[4,5].

Mechanism of arrhythmias in pregnancy

The precise mechanism of increased arrhythmia burden during pregnancy is unclear, but it is likely because of a combination of hemodynamic, hormonal, and autonomic changes (Figure 1). During pregnancy, blood volume increases by 35-40 % and is accompanied by an increase in heart rate and a simultaneous decrease in vascular resistance. This leads to an increase in cardiac output of 30–50 %, starting within the first weeks of pregnancy and with the largest increase occurring in the first 16 weeks[6-8]. This increase in blood volume has a physiological structural impact on the heart. The ventricles and atria dilate and the left ventricular mass increases[6,9]. These effects are even greater in twin/multiple pregnancies[6,10]. Mechanical stretch of the ventricles and atria can facilitate arrhythmias by depolarization of the membrane potential, premature depolarizations and dispersion in refractoriness[11,12]. Furthermore, the increase in heart rate during pregnancy may act as a trigger in susceptible patients[13]. There are also indications that estrogen and progesterone may contribute to altered cardiac repolarization facilitating arrhythmias[14]. Estrogen has been shown to increase the number of adrenergic receptors in the myocardium and adrenergic responsiveness seems to be increased in pregnancy[15,16].

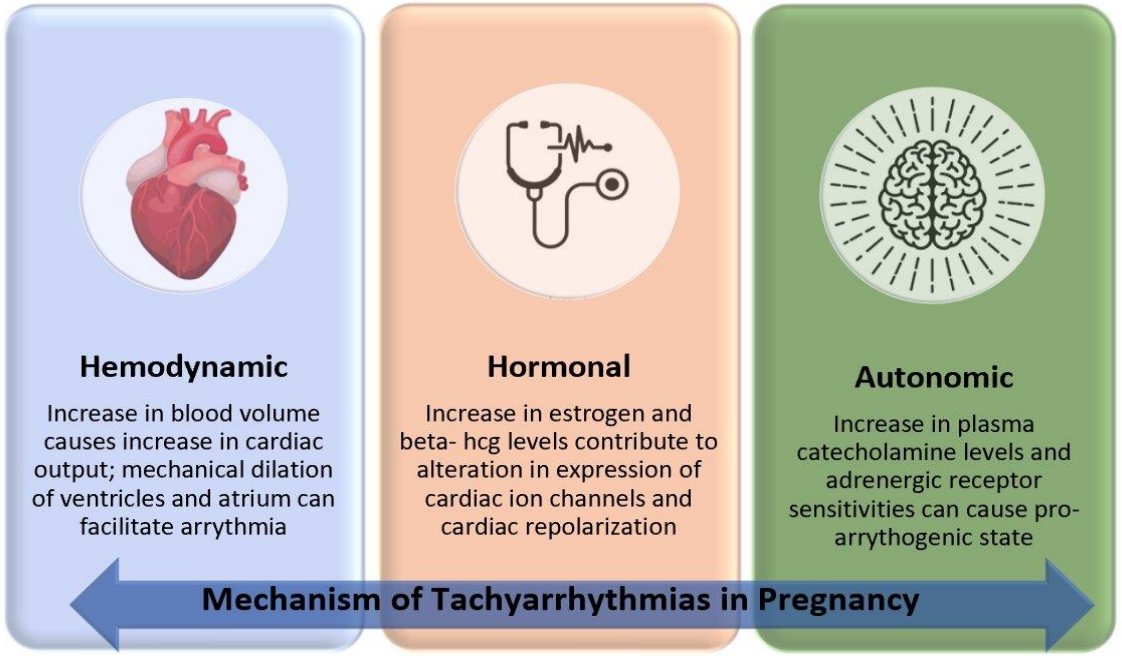

Figure 1: Pathophysiology of Tachyarrhythmias in Pregnancy

Incidence of various arrhythmias

Supraventricular premature beats are the most frequently observed arrhythmias during pregnancy followed by paroxysmal SVT; incidences are 33 and 24 per 100,000 pregnancies respectively[4]. Tawan et al[17] reported the incidence of *de novo* paroxysmal SVTs during pregnancy as13 out of 38 (34%). This is in contrast to a study by Lee et al[18] wherein *de novo* paroxysmal SVT developed in only 3.9 % of the pregnancies. However, the recurrence rate was high, observed in 55 out of 65 (85%) pregnant women with a history of paroxysmal SVT[18]. Atrioventricular nodal reentrant tachycardia (AVNRT) and atrioventricular reentry tachycardia (AVRT) are the most common paroxysmal SVTs both in pregnant and non-pregnant women with structurally normal hearts and usually do not cause hemodynamic deterioration.

Focal atrial tachycardia (FAT) occurs rarely during pregnancy and has mainly been described in case reports[20]. AF or AFL develops infrequently during pregnancy; a combined incidence of 2 per 100,000 pregnancies has been reported[4], however, the recurrent rate is high in patients with known AF/AFL, about 52 % in Silversides et al[20] data.

Recent studies suggest a change in the frequencies of specific arrhythmias. Using data from the Agency for Healthcare Research and Quality database, Vaidya et al[21] explored temporal trends in the frequency and outcomes of arrhythmias in pregnancy in over 57 million pregnancy-related hospitalizations from 2000-2012. The overall frequency of arrhythmias are as follows: any type of arrhythmia (68/100,00); AF 27 per 100,000; AFL 4 per 100,000; SVT 22 per 100,000; VF2 per 100,000 and ventricular tachycardia (VT) 16 per 100,000. The number of pregnancy-related hospitalizations for arrhythmias increased by 58% primarily due to AF and VT, while SVT rates remained stable over time. Arrhythmias were more common in black women compared to white women (116 vs 73/100,000), women with lowest vs highest income quartile (82 vs 66 per 100,000), and women 41 to 50 years of age.

Arrhythmias in pregnancy is associated with an elevated odds ratio for mortality. Among all comorbidities, arrhythmias were present most frequently in women with congestive heart failure (4654 per 100,000). Pregnancy-related hospitalizations with arrhythmias had a greater frequency of in-hospital death (5.9%) and maternal or fetal complications (36.5%)[21]. Contrary to previous studies, AF has emerged as the most frequent arrhythmia in pregnancy which could be due to an increase in maternal age (the increase in pregnancy rates in women in their 30s and 40s and continuing decline in women in their 20s) and increase in risk factors such as hypertension, diabetes mellitus, obesity, and congenital heart disease (CHD). The presence of any type of arrhythmia is associated with adverse maternal and fetal outcomes, including death[21].

In women with CHD, arrhythmias have been described in varying frequencies. Opotowsky et al[5] reported SVT in 16.4 of 100 000 and AF in 14.5 of 100 000 pregnancies whereas the incidence of SVT was 4.5 % in Drenthen et al[2] study and ROPAC (Registry on Pregnancy and Cardiac Disease) study reported a much lower incidence of about 1.3% of AF/AFL in pregnancy. Although the overall incidence of AF/AFL in pregnant women with heart disease was relatively rare, women with mitral valve disease were at higher risk (2.5%) compared with women with other cardiac lesions[22]. Arrhythmias in pregnant patients with CHD were associated with

multiple factors, including the presence of cyanotic heart disease (corrected/uncorrected) and the usage of cardiac medication before gestation[2]. Interim analysis of ROPAC reported a 100 times higher maternal mortality rate in women with heart disease compared to the general pregnant population[23].

The multicenter CARPREG II study[24] analyzed 1938 pregnancies with heart disease that progressed beyond 20 weeks gestation. The researchers found that adverse maternal cardiac events occurred in 307pregnancies (16%). Maternal cardiac death or cardiac arrest was rare and occurred in 11 pregnancies (0.6). The study also found that 64% of cardiac events occurred during the antepartum period (most likely in the second trimester), 4%during labor and delivery, and 32% in the postpartum months following discharge. These findings suggest that the hemodynamic and hormonal changes of pregnancy likely have different impacts in the presence of an arrhythmic substrate versus a structural cardiac abnormality[24].

The above findings were reconfirmed in the Stergiopoulos analysis[25], which used the same data set and time frame (2003-2012) as Vaidya et al. Stergiopoulos reported a 24.7% increase in pregnant women delivering with heart disease primarily due to an increase in the number of mothers with CHD and pulmonary hypertension. Valvular heart disease was also observed but not an increasing trend. There was an 18.8% increase in major adverse cardiac events primarily due to arrhythmias, close to the 16% observed in CARPREG II. In pregnant women with cardiomyopathy, arrhythmia was the second most common adverse cardiac outcome after heart failure.

The incidence of VT and VF in pregnancy, fortunately, appears to be low, as described by Li et al[4] study, where the incidence of VT and AF was only 2 per 100,000 pregnancies. In women with structurally normal hearts, monomorphic VT may develop during pregnancy, for example as a result of coronary artery disease or left ventricular dysfunction[26,27]. However, VTs occur more often in patients with underlying acquired (coronary artery disease, valvular heart disease, peripartum cardiomyopathy) or inherited (CHD or channelopathies) heart disease. VT recurrences have been described in 27% of women with heart disease[20]. Potentially life-threatening ventricular tachyarrhythmia (VTA) is rare during normal pregnancy (2 per 100,000 pregnancies)[28] but may be associated with maternal hemodynamic compromise causing adverse consequences for both mother and fetus[29].

Ertiken et al studied pregnancy outcomes after VTA in patients with cardiovascular disease using ROPAC data. The study involved a large prospective international registry of 2966 pregnancies with heart disease and reported the incidence of VTA during pregnancy to be 1.4%, mainly occurring in the third trimester. NYHA class>1 before pregnancy was an independent predictor of VTA and was associated with a marked increase in neonatal death rate, preterm birth rate, low birth weight rate, and poor Apgar score. The incidence of VTA in pregnant patients with CHD was 1.2%[30].

Niwa et al[31] reported a 14% prevalence of non-sustained VTA with the highest incidence in patients with previous surgical correction of Tetralogy of Fallot (TOF)[31,32]. Cardiomyopathies (CMP) predispose to the development of VTA in pregnancy. The incidence of VTA was 7.4% in the ROPAC study in patients with CMP and 3% in dilated CMP in the Grewal et al study[33]. In hypertrophic CMP, the incidence of arrhythmias including VTA was not increased[34], however, in

hypertrophic CMP patients with an implantable cardioverter, VTA is a common complication with an observed incidence of 22%[35]. Arrhythmias are common in patients with peripartum cardiomyopathy (PPCM). The reported incidence of ventricular arrhythmias is variable. A large inpatient database study reported that of 9841 hospitalizations for PPCM, 18.7% had an arrhythmia, with VT occurring in 4.2% and cardiac arrest in 2.2%[36]. Much smaller series have reported rates between 20 and 25%[37,38]. AF also occurs commonly and was observed in 3.1 to 11.9% of patients with PPCM in various studies[37,39,40].

Management

The literature on therapeutic options for the management of arrhythmias in pregnancy is generally limited to single case reports or small series favoring the use of older antiarrhythmic agents because of more abundant reports on the safe use of these drugs[41]. The lack of randomized trials and systematic data on the efficacy and safety of anti-arrhythmic drugs (AADs) renders the management of tachyarrhythmias difficult in pregnancy. The majority of AADs are Food and Drug Administration category C, meaning that risk to the fetus cannot be ruled out. Thus, a primary concern when administering AADs is the potential risk to the fetus[42]. The goal of AAD therapy in general is to reduce ectopic activity or modify critically impaired conduction. The ideal AAD has a greater effect on the arrhythmogenic substrate than on normal depolarizing tissues, decreases mortality, and has no side effects[1]. Pharmacological therapy of a pregnant woman is imperative in case of hemodynamic instability and/or diminished placenta-uterine blood flow. Maintenance of adequate therapeutic drug levels in pregnant women is challenged by increased intravascular volume, reduction of plasma protein concentrations, increased renal blood flow, and hepatic metabolism. Gastrointestinal absorption is also altered by changes in gastric secretion and intestinal motility[43,44].

Although all medications have potential side effects for both the mother and the fetus at any stage of pregnancy, if possible, drugs should be avoided in the first trimester when the risk of congenital malformations is greatest[41]. The lowest recommended dose should be used initially, accompanied by regular monitoring of clinical response[41] (Table 1). During the second and third trimesters, the effects of AADs on fetal growth and development, fetal arrhythmias, and uterine contractility become a concern[43].

Table 1: Management of Supraventricular Tachyarrhythmias in Pregnancy

Tachyarrhythmia Type	Recommendations	Factors
All forms of sustained Tachycardia **	**Acute Management** Emergency Direct Current (DC) cardioversion to restore sinus rhythm. If the tachycardia is hemodynamically well tolerated, attempt drug therapy if drug treatment fails, perform DC cardioversion **Long Term Management** procainamide* should be administered (less negative inotropic) verapamil should be withheld. (Ineffective in most ventricular tachycardia forms, has negative inotropic effects which may be significant during sustained tachycardia.	Caffeine, smoking, alcohol, thyroid dysfunction, heart disease, congenital heart defect, physical emotional stress
Atrial fibrillation (AF), Atrial Flutter (AFL), atrial Tachycardia (AT)	**Acute Management** Attempt termination of AF episodes to avoid the need for anticoagulants, coumarines are teratogenic and should be avoided during the first trimester +; Replace with heparine * (quinidine or procainamide) * DC cardioversion; adenosine **Long Term Management** Verapamil, Beta-blockers or Digoxin Radiofrequency (RF) ablation prior to pregnancy + (in patients with frequent AFL) RF ablation for AF applies to a specific patient group	Alcohol, thyroid dysfunction
Supraventricular and Ventricular Ectopic beats	**Acute Management** Management by reassurance **Long Term Management** beta-blockers may be effective	Caffeine, smoking, alcohol
Supraventricular Tachycardia utilizing the Atrioventricular node	**Acute Management** Vagal Maneuvers If these measures fail, adenosine is the first choice **Long Term Management** Verapamil, Beta-blockers or Digoxin class I antiarrhythmic drugs; Procainamide and Quinidine * flecainide and propafenone avoided + RF ablation prior to pregnancy may be considered +	Medications, smoking, alcohol, emotional or physical stress

* Long history of safe use should be selected in pregnant patients; + avoided in pregnant patients; – not recommended.

** hemodynamically not well tolerated (resulting in fetal hypoperfusion); AF = atrial fibrillation; AFL= Atrial Flutter; AT = atrial tachycardia; DC = Direct current; RF= Radio frequency.

AVNRT in pregnant patients should initially be managed with the avoidance of precipitating factors and use of vagal maneuvers in the supine position to terminate acute episodes of arrhythmia. In hemodynamically stable patients unresponsive to vagal maneuvers, adenosine is the drug of choice as it is safe and terminates ≈90% of paroxysmal SVT[45]. The initial dose for adenosine is 6mg rapid bolus intravenously. If ineffective, 2 additional infusions of 12 mg may be administered. Higher doses of adenosine may be necessary in some cases; safe administration of up to 24 mg has been reported[44].

If adenosine is ineffective, intravenous (IV) metoprolol or propranolol should be used. IV verapamil has been used effectively for the acute treatment of SVT in pregnant women; however, there is a higher risk of maternal hypotension than with adenosine[46]. Reports on diltiazem use for acute SVT termination in pregnancy are more limited than for verapamil, yet similar effects are expected[47]. IV procainamide has been used safely to treat a variety of maternal and fetal supraventricular and ventricular arrhythmias and can be effective when used for acute conversion[47,48]. Procainamide is generally best avoided as long-term therapy because it can cause a lupus-like syndrome unless other options are contraindicated or ineffective. Although IV amiodarone has been administered safely during pregnancy, multiple adverse effects on the fetus have also been reported[49]. An important concern is the possibility of fetal hypothyroidism, reported in approximately 17% of cases[47,49]. Short-term IV infusions have less concern for side effects given that most toxicities are related to cumulative drug dose.

For patients with frequent symptomatic episodes, drugs such asmetoprolol, propranolol, and digoxin are considered safe first-line agents for chronic oral prophylaxis given their long record of safety. Caution is advised, however, given that therapy with beta-blockers has been associated with intrauterine growth restriction[47,50]. This effect appears to be more pronounced with atenolol, especially in mothers who received atenolol earlier in gestational age and who were treated for a longer duration[51]. Flecainide and propafenone have been used effectively to treat a variety of maternal and fetal tachycardias, yet remain reserved for patients without underlying structural heart disease or ischemic heart disease[52].Women presenting with new AF/AFL, or hemodynamically unstable episodes of AF or AFL should be treated with electric cardioversion. It can also be considered electively for drug-refractory arrhythmias. Before electrical or chemical cardioversion, it is important to consider precipitating factors and the need for pre-cardioversion anticoagulation. When required, cardioversion can be performed during pregnancy[22] and does not compromise blood flow to the fetus[53]. In addition, because only a small amount of energy reaches the fetus, the risk of inducing fetal arrhythmias is small[43,44]. In the later stages of pregnancy, there is a theoretical risk of initiating preterm labor. There are case reports of emergency cesarean delivery because of fetal arrhythmias after cardioversion: fetal monitoring is advised[54].

Flecainide and ibutilide have been used safely in case reports for pharmacological conversion of AF in pregnancy, but there is no broad experience with chemical cardioversion in this setting[55-57]. Digoxin and Sotalol may be harmful when used for pharmacological cardioversion of AF and are not recommended[58]. Quinidine,

procainamide, disopyramide and dofetilide should not be started in the hospital for conversion of AF to sinus rhythm[58]. Regarding anticoagulation, the ACC/AHA Guidelines for the management of patients with AF recommend administering antithrombotic therapy (anticoagulant or aspirin) (Table 2) throughout pregnancy to all patients with AF (except those with lone AF)[58]. In a retrospective study of pregnant patients with AF, the use of aspirin and anticoagulants was low at 2.5% and 3.3%, respectively[59]. While a majority of pregnant women are young and have a low-risk assessment by the $CHADS_2VASc_2$ score (1 derived from female gender) as in the prior study, risk assessment tools for thromboembolic risk have not been validated in pregnancy and the additive risk of pregnancy to the total thromboembolic risk is unclear[29].

Table 2: Use of Anticoagulants for Atrial Fibrillation/Flutter in Pregnancy

Anticoagulation therapy	Indication	Dosage	Complications
Aspirin	• Non-valvular atrial fibrillation with low CHADSVASC score • Lone atrial fibrillation	• Low dose (81 mg/day) aspirin, during second and third trimester only. Aspirin use during first trimester remains uncertain	• Bleeding • Congenital defects if used in first trimester
Low molecular weight heparin Unfractionated heparin	• Higher thrombotic risk atrial fibrillation	• Early pregnancy body weight: Enoxaparin 1 mg/kg body weight BID. Recommended use first trimester and last month of pregnancy	• Bleeding • Heparin induced thrombocytopenia • Osteoporosis
Warfarin	• Mechanical valvular disease • High thrombotic risk atrial fibrillation	• Dosage adjusted to goal INR 2-3 • Recommended use during second and third trimester of pregnancy	• Bleeding • Embryopathy
Direct Oral Anticoagulants (Apixaban, Rivaroxaban, Dabigatran, Edoxaban, Betrixaban)	• Has not been well studied in pregnant women, hence not recommended in pregnant women		

Pregnant women with mechanical valves and AF should discontinue warfarin between 6 and 12weeks gestation and then receive a continuous infusion of IV unfractionated heparin dose adjusted or low molecular weight heparin (LMWH) subcutaneous[60], especially in the first trimester and the last month of pregnancy[55,61]. Heparin is also recommended for patients with persistent episodes in whom electric cardioversion is planned[42]. Warfarin may be used in the second and third

trimesters. There is modest data about the safety or use of newer oral anticoagulants such as dabigatran or rivaroxaban or IV direct thrombin inhibitors in pregnancy except for case reports of the use of IV agents in patients with heparin-induced thrombocytopenia[62]. Therefore, the routine use of these agents is not recommended in pregnancy. Management of anticoagulation at the time of delivery is complex and must be carefully coordinated by the health care team[22]. For recurrent arrhythmias or those that result in significant maternal decompensation, treatment with catheter ablation can be an option. The risk of radiation exposure to the fetus is a concern with catheter ablation in pregnant patients because high-dose ionizing radiation has been linked to excess malignancy and congenital malformations[63]. However, the fetal radiation dose for most common cardiovascular interventions is not likely to exceed the 50-mGy negligible-risk threshold dose for excess malignancy[64].

One study that used phantoms to simulate pregnancy estimated a low lifetime risk of malignancies from radiation exposure to the concept us during a typical ablation procedure. Furthermore, with current technologies such as electro anatomic mapping systems, catheter ablation procedures using minimal or even zero fluoroscopy have been described in pregnant women[65]. Thus, if a catheter ablation procedure is required in a pregnant woman, radiation-reduction technologies should be used[63]. The procedure should be avoided in the first trimester when the teratogenic risk is greatest and delayed until the second trimester and performed under echocardiographic guidance. Of note, shielding the fetus by covering the mother with a lead apron does not eliminate radiation to the fetus because most of the radiation to the fetus comes from scattered particle emission. In pregnant patients with structural heart disease, the underlying cardiac condition should drive treatment of VT. For the acute management of VT, electric cardioversion should be performed in the setting of hemodynamic instability. In hemodynamically tolerated VT, pharmacological cardioversion with lidocaine should be tried first. Procainamide or quinidine may then be used if lidocaine is ineffective.

Chronic AAD therapy is often warranted in patients with VT (Table 3) and structural heart disease given the risk of hemodynamic compromise and sudden death. Sotalol may be considered if β-blockers are ineffective[42]. Mexiletine and quinidine are reasonable alternative agents, but class IC agents should not be used because they are associated with increased mortality in nonpregnant patients with structural heart disease[64]. For pregnant women high risk for sudden cardiac death or with unstable ventricular arrythmias, ICD placement should be considered. Implantation of ICDs is considered safe in pregnancy and ICD shocks have not been associated with adverse events in fetuses[66].

Table 3: Management of Ventricular Arrhythmias in Pregnancy

Arrhythmias	Treatment	Pharmacotherapy in Pregnancy	Side effects/Caution

Ventricular Tachycardia	*In pregnant patients with structural heart disease, treatment of VT should be tailored to underlying cardiac condition		
	Acute Lidocaine (1st line)	Food and Drug Administration Class B	• Bradycardia • CNS adverse effects
	Procainamide Quinidine (2nd line) + Cardioversion in hemodynamically unstable patients	Food and Drug Administration Class C	• Drug induced lupus (Procainamide) • Torsade de pointes (Procainamide, Quinidine) • Thrombocytopenia (Quinidine) • Ototoxicity (Quinidine) •
	Chronic Beta-Blockers Non-dihydropyridine calcium channel Blockers Sotalol Dofetilide	Food and Drug Administration Class C	Beta-blockers • Intrauterine growth restriction • Preterm growth • Neonatal hypoglycemia, bradycardia, and hypotension Sotalol/Dofetilide • QT prolongation • Torsade de pointes Calcium channel blockers • Hypotension • Tocolysis

Pre-pregnancy counseling should be provided for women with congenital long-QT syndrome, an inheritable condition that can pose malignant tachyarrhythmia. Studies have shown the reduction of long-QT syndrome-related events during pregnancy due to the hypoestrogenic state, but an exponentially increased risk in the nine-month postpartum period. It is important to incorporate routine ECG for evaluation of corrected QT as well as monitoring of electrolytes (magnesium and potassium) to avoid

potentially prolonging QT interval. Beta-blockers should be continued throughout pregnancy and especially postpartum regardless of symptoms for prevention of ventricular arrhythmias and sudden cardiac death. In a case-control study, women with LQT1 who did not receive β-blockers during pregnancy were at increased risk of cardiac arrest or syncope. Non-selective beta blockers are preferred. QT-prolonging medications including oxytocin or ondansetron should be avoided[67].

CONCLUSION

The most prevalent cardiac concerns during pregnancy are arrhythmias. Hormonal, autonomic, and hemodynamic changes during pregnancy might worsen preexisting arrhythmias or cause denovo arrhythmias. Supraventricular arrhythmias are more prevalent while ventricular tachycardia occurs in women with CHD. Arrhythmia treatment in pregnancy is difficult owing to changes in blood volume, hepatic and renal metabolism, and plasma binding proteins. There is also a considerable teratogenic risk associated with anti-arrhythmic drugs. The majority of AADs fall under FDA class C. Beta-blockers are typically well tolerated, however, atenolol may cause intrauterine fetal growth restriction. Electrical cardioversion for hemodynamically unstable and new AF/AFL is generally well tolerated and the risk of inducing fetal arrhythmias is small. Thromboembolic risk assessment tools such as the CHADVASC score are not well studied in pregnancy, therefore guidelines recommend starting anticoagulation in all patients with AF. While warfarin can be safely used during the second and third trimesters, it should be stopped during the first due to teratogenic risk. Heparin and LMWH should be used during the first trimester and labor. Newer oral anticoagulants haven't been well-studied in randomized clinical trials and are not recommended for pregnant women. Catheter ablation for recurrent/treatment-resistant arrhythmias is safe if the radiation dosage does not exceed 50-mGy. Ablation with zero fluoroscopy has been reported in pregnant women to avoid fetal events. ICD implantation and shocks are safe during pregnancy and have not been linked to fetal harm.

CONFLICT OF INTEREST

All the authors report no conflict of interest.

SUBMISSION DECLARATION

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AUTHORSHIP

All authors had access to the data and a role in writing the manuscript.

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