Cardiac arrhythmias in pregnancy: clinical and therapeutic considerations

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Abstract

Pregnancy can precipitate cardiac arrhythmias not previously present in seemingly well individuals. Risk of arrhythmias is relatively higher during labor and delivery. Potential factors that can promote arrhythmias in pregnancy and during labor and delivery include the direct cardiac electrophysiological effects of hormones, changes in autonomic tone, hemodynamic perturbations, hypokalemia of pregnancy, and underlying heart disease. Paroxysmal supraventricular and ventricular tachycardia may cause hemodynamic compromise with consequences to the fetus. Management of arrhythmias in pregnant women is similar to that in non-pregnant but a special consideration must be given to avoid adverse fetal effects. No drug therapy is usually needed for the management of supraventricular or ventricular premature beats, but potential stimulants, such as smoking, caffeine, and alcohol should be eliminated. In paroxysmal supraventricular tachycardia, vagal stimulation maneuvers should be tried first. Adenosine or a cardioselective beta-blocker could be used if vagal maneuvers are ineffective. Alternatively, verapamil or diltiazem may be given. In pregnant women with atrial fibrillation, the goal of treatment is conversion to sinus rhythm or to control ventricular rate by a cardioselective beta-adrenergic blocker drug or digoxin. Ventricular arrhythmias may occur in the pregnant women with cardiomyopathy, congenital heart disease, valvular heart disease, or mitral valve prolapse. Termination of ventricular arrhythmias can usually be achieved by intravenous lidocaine or procainamide or by electrical cardioversion. Amiodarone is not safe for the fetus. Beta-blocker therapy must be continued during pregnancy and postpartum period in women with long QT syndrome and torsade de pointes.

Keywords: Arrhythmias; Pregnancy; Electrocardiogram; Antiarrhythmic drugs; Treatment

1. Introduction

Pregnancy may increase the incidence of various arrhythmias, complicate their invasive evaluation, and raise special considerations for their treatment. A greater number of women with known or potential heart disease, especially those with adult congenital heart disease are becoming pregnant [1,2]. Although there is an increased recognition of cardiac arrhythmias in pregnancy, partly owing to the frequent visits to physicians as a part of the antenatal care, pregnancy can precipitate cardiac arrhythmias not previously present in seemingly well individuals. Arrhythmias can be seen denovo or can be exacerbated by pregnancy, and can pose risks for both mother and the fetus. Risk of arrhythmias is relatively higher during labor and delivery. In an analysis where electrocardiograms of pregnant women during labor and delivery were reviewed, it was found that almost all of them had resting abnormalities, which included...
premature atrial, ventricular or nodal complexes, sinoatrial arrest, wandering atrial pacemaker, sinus tachycardia, and paroxysmal ventricular tachycardia [4]. The factors that can promote potential for arrhythmia during pregnancy and labor and delivery include the direct cardiac electrophysiological effects of hormones, changes in autonomic tone, hemodynamic perturbations, mild hypokalemia of pregnancy, and underlying heart disease [1,3]. Atrial and ventricular premature beats are frequently present during pregnancy and are usually benign. Supraventricular tachycardia and malignant ventricular tachyarrhythmias occur less frequently. Most arrhythmias in young women are not associated with underlying structural heart disease and their management in pregnant women usually does not differ much from that in the non-pregnant women.

2. Electrocardiographic changes in pregnancy

Caution should be exercised when interpreting the electrocardiographic abnormalities in pregnant women, and must account for the normal physiological changes that occur in pregnancy. There is an increase in resting heart rate about 10 beats/min during pregnancy [5]. This may result in decreased PR, QRS, and QT intervals, but usually there is no change in the amplitude of the P wave, QRS complex, and T wave [6]. The electrical axis shift can occur, more commonly leftward, due to rotation of the heart secondary to the enlargement of the gravid uterus [7]. Premature atrial and ventricular depolarizations are common during pregnancy.

3. Tachyarrhythmias in pregnancy

In 1956, Mendelson [8] was the first to report occurrence of supraventricular tachycardia during pregnancy. There are several studies with conflicting results regarding the prevalence and severity of paroxysmal supraventricular tachycardia in pregnancy [8–12]. Many studies have suggested a high probability of the new onset of paroxysmal supraventricular tachycardia during pregnancy, but, conversely, a larger study cited a low probability that a first paroxysmal supraventricular tachycardia episode will occur during pregnancy [8–12]. Tawam et al. [9] found an increased risk of both new onset (34%) and exacerbation (29%) of supraventricular tachycardia during pregnancy. Although the supraventricular tachycardias often occurs during reproductive years, the mechanisms responsible for the pregnancy-induced onset or exacerbation of supraventricular tachycardias are not clear, hyperdynamic state of pregnancy may be a major contributory factor. Wolff–Parkinson–White syndrome may first manifest during pregnancy, or the frequency of tachyarrhythmia may increase in women who have previously diagnosed Wolff–Parkinson–White syndrome. Widhorn et al. [10] described three patients with stable supraventricular tachycardia and Wolff–Parkinson–White syndrome who experienced a marked increase in the frequency of supraventricular tachycardia episodes during pregnancy. Lee et al. [11] observed that the relative risk of new onset of paroxysmal supraventricular tachycardia during pregnancy was higher for those with accessory pathways than for those with atrioventricular nodal reentry tachycardia. Contrarily, Siu et al. [12] followed a group of 25 women through pregnancy who had prior history of supraventricular tachycardia, half with Wolff–Parkinson–White syndrome, and found that three of the 12 with Wolff–Parkinson–White syndrome and six of the 13 without the syndrome had episodes of supraventricular tachycardia during pregnancy.

Atrial fibrillation and atrial flutter are rare in pregnancy and, when encountered, appear secondary to congenital or valvular heart disease, or underlying metabolic disturbances such as thyrotoxicosis and electrolyte perturbations. Because of an increased risk of thromboembolism and the potential detrimental effects of fast ventricular rates on the fetus, it is important to treat these arrhythmias early with conversion to sinus rhythm or to control ventricular rates. No specific maintenance therapy for rhythm control is essential. If atrial fibrillation is associated with mitral stenosis, pulmonary edema may develop rapidly, especially in late pregnancy, if the ventricular rate is rapid. For rate control, beta-blockers alone or in combination with digoxin are preferred drugs in such cases.

Ventricular tachycardia is usually uncommon in healthy women without underlying organic heart disease. Although occurrence of these arrhythmias
should raise a suspicion of underlying cardiovascular disease, there are reports of new onset ventricular tachycardia during pregnancy in absence of structural heart disease [13,14]. Physical and psychological stresses are the stimuli for precipitation of ventricular tachycardia in a majority of pregnant women without structural heart disease. Most of the reported cases of ventricular tachycardia without structural heart disease during pregnancy were of monomorphic left ventricular type, and have responded well to beta-blocker therapy [13,14]. Holter monitoring and electrophysiological testing performed after delivery have failed to induce arrhythmia in most of such cases. In patients who present with new onset ventricular tachycardia in the last few weeks of pregnancy or within 6 months of the delivery, a possibility of peripartum cardiomyopathy should be considered.

Pregnancy in patients with congenital long QT syndrome may pose a difficult problem. Rashba et al. [15] analyzed 111 pregnant women affected with long QT syndrome and found a significant increase in the risk of cardiac events in the postpartum period, but not during pregnancy. Increased heart rate secondary to pregnancy may bear a protective effect on QT interval. As heart rate decreases after delivery, there is potential for increase in QT interval, which coupled with the lack of sleep and increased stress of caring the newborn could potentially increase the incidence of torsade de pointes in the postpartum period. Beta-blocker therapy has been shown to decrease the risk of torsade de pointes related cardiac events (death, aborted cardiac arrest, or syncope) in patients with long QT syndrome [16,17], and, therefore, must be continued during pregnancy and postpartum period in women with long QT syndrome.

### 4. Bradyarrhythmias in pregnancy

Compared to the tachyarrhythmias, bradyarrhythmias are uncommon in pregnancy, and when occur, are usually well tolerated. In women with symptomatic bradycardia or heart block, a pacemaker can be implanted, if necessary, at any stage of pregnancy using echocardiographic guidance. A rate adaptive pacemaker could be a preferred choice, although pregnant women with fixed-rate mode permanent pacemakers have been able to tolerate pregnancy generally well, as cardiac output increases by an augmented stroke volume in them [18].

### 5. Antiarrhythmic therapy in pregnancy

No antiarrhythmic drug is entirely safe during pregnancy. In addition, the risks of antiarrhythmic drugs are further increased due to alterations in the drug absorption and metabolism associated with pregnancy. Drug therapy should be avoided during the first trimester if possible and drugs with the longest record of safety should be used as the first-line therapy [19]. The potential fetal effects of antiarrhythmic drugs (Table 1) and the risks of diagnostic procedures such as electrophysiological testing should be considered while managing arrhythmias during pregnancy. At the same time, it is essential to avoid hemodynamically significant arrhythmias to prevent possible fetal harm during associated maternal hypotension [20,21]. The best option would be to avoid chronic antiarrhythmic drug therapy during pregnancy, except in patients with severe arrhythmias, and to avoid invasive procedures.

<table>
<thead>
<tr>
<th>Antiarhythmic Drug</th>
<th>Reported Fetal Adverse Effects</th>
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<tbody>
<tr>
<td>Adenosine</td>
<td>None reported except for one case of fetal bradycardia</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Hypothyroidism, growth retardation, prematurity</td>
</tr>
<tr>
<td>Beta-blockers, sotalol</td>
<td>Growth retardation, bradycardia, hyperbilirubinemia, hypoglycemia, uterine contractions</td>
</tr>
<tr>
<td>Digin</td>
<td>Low birth weight</td>
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<tr>
<td>Diltiazem</td>
<td>None reported</td>
</tr>
<tr>
<td>Disopyramide</td>
<td>Uterine contraction</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Central nervous system depression</td>
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<tr>
<td>Mexiletine</td>
<td>Bradycardia, low birth weight, low Apgar score</td>
</tr>
<tr>
<td>Procainamide</td>
<td>None reported</td>
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<tr>
<td>Phenytoin</td>
<td>Mental and growth retardation</td>
</tr>
<tr>
<td>Quinidine</td>
<td>Thrombocytopenia, eighth cranial nerve damage</td>
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<tr>
<td>Varapamil</td>
<td>Heart block, hypotension</td>
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requiring pelvic fluoroscopy. Thus the goal of therapy is to protect the patient and fetus through delivery, after which definitive therapy could be instituted.

Electrical cardioversion is necessary in all the patients who are hemodynamically unstable. On the other hand, in stable patients with supraventricular tachycardia, a vagal maneuver should be tried first to terminate reentrant tachycardias involving atrioventricular node. For short-term management, when the vagal maneuvers fail, intravenous adenosine is the first-choice drug in hemodynamically stable patients and may safely terminate the supraventricular tachycardia [22]. There has been only one report of fetal bradycardia secondary to adenosine used [23]. Digoxin, beta-blockers, or calcium channel blockers could also be used for termination of supraventricular tachycardia. Although these drugs do cross placenta, they do not appear to cause substantial harm to fetus.

In hemodynamically stable patients with ventricular tachycardia, initial therapy with lidocaine or procainamide should be considered. Amiodarone can cause fetal hypothyroidism, growth retardation, and prematurity. If prophylactic therapy is needed, cardioselective beta-blockers are considered as the first choice. In cases where beta-blocker therapy is ineffective, sotalol could be an effective therapeutic alternative [24]. In women with long QT syndrome, beta-blocker therapy must be continued during pregnancy and the postpartum period. In pregnant women with aborted sudden death an implantable cardioverter-defibrillator is indicated. Natale et al. [25] evaluated outcome of pregnancy in women with permanent cardioverter defibrillator implanted. There was no increased risk of major implantable cardioverter defibrillator related complications during pregnancy. Authors suggested that women with implanted cardioverter-defibrillator could consider pregnancy unless otherwise contraindicated by the underlying heart disease.

6. Anticoagulation in pregnancy

All forms of chronic anticoagulation may result in bleeding between the placenta and uterus and subsequent pregnancy loss. If given in high doses for long periods, heparin may cause osteoporosis and warfarin may be associated with embryopathy and abnormalities of central nervous system. Pregnant women who require anticoagulation could receive heparin in the first trimester and in the terminal stages of pregnancy. Warfarin is relatively safe during the remainder periods of pregnancy until just before delivery when it has to be discontinued.

7. Conclusions

Cardiac arrhythmias in pregnancy can be due to multiple factors including the stress of pregnancy on cardiovascular system and an increased sympathetic tone and sensitivity, both of which can exacerbates the preexisting underlying arrhythmias or cause de novo occurrence of arrhythmias. Treatment of rhythm disturbances may pose a risk to fetus. Therefore, the use of antiarrhythmic therapy must be well justified and care should be exercised in selection of antiarrhythmic drugs because no antiarrhythmic drug is absolutely safe in pregnancy.

References