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Refractory Rapid Atrial Fibrillation in Pregnancy: A Case Report

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Abstract

Atrial fibrillation (AF) is a rare cardiac arrhythmia during pregnancy. While it can occur at any stage, it most commonly develops in late pregnancy due to the significant hemodynamic and autonomic changes that evolve as pregnancy approaches term. AF typically presents with a sudden onset of heart palpitations and shortness of breath. Although these symptoms may resemble those seen in non-pregnant women, during pregnancy, they may compromise cardiac output and placental perfusion, increase the risk of thromboembolic complications, and evoke a more cautious treatment response because of concerns for foetal safety. This report presents a case of refractory, rapid AF at 38 weeks' gestation in a woman with well-controlled gestational hypertension. Despite pharmacological intervention, the arrhythmia persisted, necessitating early delivery. The patient reverted to sinus rhythm immediately postpartum, highlighting the significant impact of pregnancy physiology on both normal and abnormal cardiac function. This case highlights maternal cardiovascular adaptation during pregnancy, the effect of AF, and the rationale for delivery as a treatment option when medical therapy is unsuccessful.

Keywords: Atrial fibrillation; Pregnancy; Cardiac Rhythm Disorders; Cardioversion; Cardiac Function

Introduction

Pregnancy causes significant cardiovascular remodeling to support uteroplacental blood flow. Cardiac output increases by up to 45–50%, primarily due to increased stroke volume and heart rate, while systemic and pulmonary vascular resistance decrease [1]. Plasma volume expansion, heightened adrenergic tone, atrial stretch, and increased hormonal sensitivity all contribute to conditions that enhance arrhythmic vulnerability. Palpitations are common during pregnancy and are usually harmless, caused by episodes of sinus tachycardia, ectopic beats, anemia, or increased sympathetic activity. It is vital to distinguish these from serious arrhythmias because atrial fibrillation, though uncommon, can significantly affect blood flow and foetal support [2]. When AF occurs, the added stress of pregnancy may exacerbate its impact: rapid ventricular rates reduce diastolic filling time, lower stroke volume, and can impair oxygen delivery to both mother and foetus. This report describes a rare case of new-onset, idiopathic AF in late pregnancy that was refractory to intensive medical management but resolved immediately following delivery. The case underscores the complex interplay between pregnancy physiology and cardiac rhythm, and highlights delivery as a decisive therapeutic intervention when medical management is inadequate.

Case Report

A 33-year-old woman, G2P1, at 38 weeks' gestation, presented with sudden onset of palpitations, shortness of breath, and significant tachycardia. Her prenatal course was complicated by gestational hypertension, which was treated effectively with low-dose labetalol three times daily. She had no history of cardiac dysfunction, arrhythmia, or other cardiovascular risk factors. Her previous pregnancy resulted in an emergency lower-segment cesarean section for fetal distress during labor. She was planning an elective cesarean at term for current confinement. Upon arrival, her heart rate was between 140 and 160 bpm, with stable blood pressure and oxygen saturation. An electrocardiogram confirmed atrial fibrillation. The foetal cardiotocograph was reassuring. She was transferred to the intensive care unit for continuous cardiac telemetry and combined maternal–foetal monitoring. Additional investigations revealed a structurally normal heart on

echocardiography with preserved systolic function. Laboratory tests showed normal electrolytes, thyroid function, inflammatory markers, and hemoglobin levels. There was no evidence of infection, thyroid dysfunction, pulmonary embolism, or any other reversible trigger. Foetal growth and movements were normal.

A stepwise medical approach was implemented to control heart rate. The patient received digoxin at a daily dose of 250 mcg, which is within the upper therapeutic range, and her labetalol was titrated from 200 mg to 800 mg three times daily. Intravenous magnesium sulfate was administered to help with rate control, and low-molecular-weight heparin was started for thromboprophylaxis. Despite this treatment, her ventricular rate remained between 130 and 150 bpm over the next three days. Although she was hemodynamically stable, she continued to experience symptoms. Due to persistent tachyarrhythmia at term, limited options for further pharmacological escalation, and increasing suspicion that pregnancy physiology was maintaining the arrhythmia, a multidisciplinary team consisting of intensive care, cardiology, obstetrics, and anesthesia reviewed the case. Electrical cardioversion was considered but rejected, given the opportunity for near-term delivery. A decision was made to proceed with a lower-segment caesarean section. Carbetocin 100 mcg IV was administered as the oxytocic agent, estimated blood loss was 200 mL, and a healthy neonate was delivered without complication. Notably, within minutes of fetal delivery and before completing the procedure, the patient spontaneously reverted to normal sinus rhythm. The postoperative recovery was uncomplicated. The patient remained in stable sinus rhythm without further need for antiarrhythmic therapy. She recovered well and established breastfeeding normally. Her antihypertensive medications were transitioned to enalapril and nifedipine, and low-molecular-weight heparin was continued for six weeks. Holter monitoring and cardiology follow-up were arranged following discharge to check for underlying myocardial or conduction abnormalities to help stratify long-term cardiac risk.

Discussion

Atrial fibrillation is the most common sustained arrhythmia in adults, but it remains rare in pregnancy, especially among women without structural heart disease. Estimates suggest an incidence of about 27 cases per 100,000 pregnancies [3]. AF can develop at any point during pregnancy or postpartum, with the third trimester being the highest-risk period [2]. Several maternal factors are linked to its occurrence, including valvular heart disease, congenital defects, and cardiomyopathies [4]. Szekely et al. reported AF in up to 8% of women with rheumatic heart disease, usually associated with pre-pregnancy symptoms [5]. In addition to structural disease, increased risk is associated with age, obesity, hypertension, diabetes, African/American descent, electrolyte imbalance, pre-excitation syndromes, stimulant use, pulmonary embolism, and a history of pre-existent symptoms. Even without underlying cardiac disease, normal pregnancy causes physiological stresses that increase arrhythmic risk. Rising estrogen levels boost adrenergic receptor sensitivity, while shifts in autonomic balance increase sympathetic tone. At the same time, circulating plasma volume expands, cardiac output rises, and atrial chambers stretch under increased preload [6]. Systemic vascular resistance decreases, and mild hypokalemia may develop, further impacting myocardial electrical stability. These changes combine to create a pro-arrhythmic environment in which atrial stretch, elevated catecholamines, and hormonal effects on ion channels lower the threshold for AF onset and support its persistence.

The report noted a history of gestational hypertension. Hypertensive disorders of pregnancy—most notably pre-eclampsia—demonstrate the evolution of acute cardiovascular stress through endothelial dysfunction, increased vascular resistance, and impaired myocardial relaxation, all of which might lower the threshold for arrhythmias in susceptible women [7]. While pre-eclampsia is most strongly linked to cardiovascular disease, even well-controlled gestational hypertension can evoke triggers for AF. In this case, the combination of pregnancy-related volume expansion and hypertension-related myocardial stress may have lowered her arrhythmic threshold and helped sustain rapid AF. AF in pregnancy often presents with palpitations and dyspnea, symptoms that may overlap with normal pregnancy physiology but are usually more pronounced [2]. When AF occurs with a rapid ventricular response, hemodynamic instability is more likely. Shortened diastole reduces ventricular filling and cardiac output—changes that are especially critical when the placenta acts as a low-resistance, high-flow organ. Even modest reductions in maternal cardiac output can impair uteroplacental perfusion and cause foetal distress [1]. For the mother, persistent tachyarrhythmia raises the risk of heart failure, symptomatic hypotension, thromboembolism, stroke, and recurrent arrhythmias. The hypercoagulable state of pregnancy further increases the potential risk of thromboembolic events.

Management

Rate control

Managing AF in pregnancy requires a personalized approach that balances maternal stabilization with foetal safety, best achieved through a coordinated multidisciplinary team involving obstetrics, cardiology, intensive care, anesthesia, and neonatology. In haemodynamically stable women, rate control remains the mainstay of treatment, with the primary objective of increasing diastolic filling time and maintaining cardiac output [8].

Digoxin, beta-blockers, and calcium-channel blockers are the most commonly used agents, although each has important pregnancy-specific considerations. Specifically, digoxin and calcium-channel blockers must be used cautiously in women with pre-excitation syndromes, as changes in atrioventricular conduction can paradoxically increase the ventricular response [9]. Digoxin crosses the placenta easily but is generally considered

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safe at recommended doses, although digoxin-like immunoreactive substances in late pregnancy may cause falsely elevated serum levels on assay [10]. Among beta-blockers, cardioselective agents like metoprolol are preferred because they have a lower risk of peripheral vasodilation, uterine relaxation, and fetal hypoglycemia [6].

Cardioversion

A fall in maternal blood pressure or the development of fetal bradycardia requires urgent cardioversion. Generally, cardioversion should be performed within 48 hours of AF onset to minimize the risk of thromboembolic complications. Pharmacological cardioversion is used selectively during pregnancy, as many antiarrhythmic drugs pose foetal risks, including congenital malformations and fetal bradycardia [11]. Quinidine has traditionally been the agent of choice for hemodynamically stable women, though its use is limited by potential side effects such as QT prolongation, preterm labour, neonatal thrombocytopenia, and eighth cranial nerve toxicity [1]. Other agents—procainamide, flecainide, and propafenone—have been used with relative safety but have less extensive clinical experience. Amiodarone is generally avoided because of its association with fetal hypothyroidism, hyperthyroidism, and goiter [12]. Women with persistent AF who do not respond adequately to medical therapy may be suitable for electrical cardioversion, which is considered safe at all stages of pregnancy. It can be performed under sedation with rapid-acting agents such as propofol, which has an established safety profile during pregnancy. Only a small fraction of the electrical current reaches the uterus, and the foetus has a high threshold for fibrillation1. Continuous fetal monitoring during and after the procedure is recommended to detect any transient fetal dysrhythmias.

Thromboprophylaxis

Thromboprophylaxis is recommended for all pregnant women with AF. The preferred anticoagulants during pregnancy are heparin-based agents because they do not cross the placenta. LMWH has a better safety profile, with fewer side effects such as bleeding, thrombocytopenia, and osteoporosis [1].

Delivery

For women at term with refractory AF despite maximum medical therapy, delivery becomes a rational and effective treatment option. In this case report, the absence of reversible triggers indicates that pregnancy physiology was the primary factor maintaining the arrhythmia. Spontaneous cardioversion after delivery is well documented in the literature. [13] In this case, the patient's rapid return to sinus rhythm immediately after childbirth highlights the sudden hemodynamic changes that occur, including decreased preload, lower circulating volume, and reduced sympathetic tone—all of which help restore atrial electrophysiological stability. Recurrence of AF in subsequent pregnancies is considered rare in the absence of structural heart disease, although data are limited.

Conclusion

Atrial fibrillation in pregnancy is rare but clinically significant because the unique hemodynamics and hormonal changes of late gestation can both trigger and sustain refractory arrhythmias despite standard treatments. Increases in plasma volume, atrial stretch, and sympathetic activation during pregnancy create a physiological environment that lowers the threshold for AF and can diminish the effectiveness of traditional rate-control strategies. Although medications such as digoxin, beta-blockers, and calcium channel blockers remain the cornerstone of care, they may not always achieve adequate rate control. In this setting, particularly when the pregnancy is near or at term, delivery becomes a reasonable and potentially definitive treatment option. The immediate return to normal sinus rhythm after birth in this and other recorded incidences highlights how pregnancy physiology can sustain and potentiate abnormal cardiac function. Optimal outcomes depend on early recognition, thorough maternal–fetal assessment, and coordinated multidisciplinary management involving obstetrics, cardiology, anesthesia, and intensive care. This case demonstrates that, in selected women with refractory AF at term, delivery should be considered not only as an obstetric decision but as a vital part of arrhythmia management.

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