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CASE REPORT
Electrophysiology

Lessons from timing of ablation therapy for multi-drug refractory gestational atrial tachycardia with abruptio placentae: a case report

Joseph You [1]*, Eric Pagan[2], and Raman Mitra[2]

[1]Department of Internal Medicine, North Shore University Hospital, 300 Community Dr, Manhasset, NY 11030, USA; and [2]Department of Cardiology, Zucker School of Medicine at Hofstra/Northwell, North Shore University Hospital, Manhasset, NY, USA

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Background Supraventricular tachycardia poses a clinical challenge during pregnancy, particularly if refractory to antiarrhythmic medications. Performing catheter ablation during pregnancy necessitates careful risk benefit analysis for both the mother and foetus, especially with left-sided ablations that may require post-procedural systemic anticoagulation.

Case summary We describe a case of a 31-year-old pregnant woman with refractory atrial tachycardia which failed a multi-antiarrhythmic drug regimen and ultimately developed abruptio placentae, requiring a carefully staged ablation approach for definitive treatment.

Discussion This case highlights the importance of taking into consideration the risks of post-procedural anticoagulation in the event of clinical complications in pregnancy such as abruptio placentae and coordinating carefully with gynaecologists to optimize maternal and foetal outcomes. Here, careful risk stratification was paramount to successfully navigate through the management of her atrial tachycardia while ensuring foetal viability.

Keywords Atrial tachycardia • Supraventricular arrhythmias • Atrial flutter • Pregnancy • Ablation

ESC Curriculum 5.5 Supraventricular tachycardia • 9.8 Pregnancy with cardiac symptoms or disease

Learning points

- To highlight the importance of ablation timing with consideration to anticoagulation requirements in pregnant women with refractory supraventricular tachycardia.
- Underscore the importance of a multidisciplinary approach to gestational supraventricular tachycardia therapy with careful risk-benefit approaches for the mother and foetus.

Introduction

Clinical management of drug refractory, symptomatic gestational supraventricular tachycardia may be difficult, and a multidisciplinary approach is crucial to determine appropriate therapy. It is unclear whether pregnancy increases the risk of supraventricular tachycardia (SVT), though studies have shown that patients with pre-existing SVT may experience exacerbations during pregnancy.[1] For those who fail

* Corresponding author. Tel: 516-582-5266, Fax: 516-562-0100, Email: jyou2@northwell.edu
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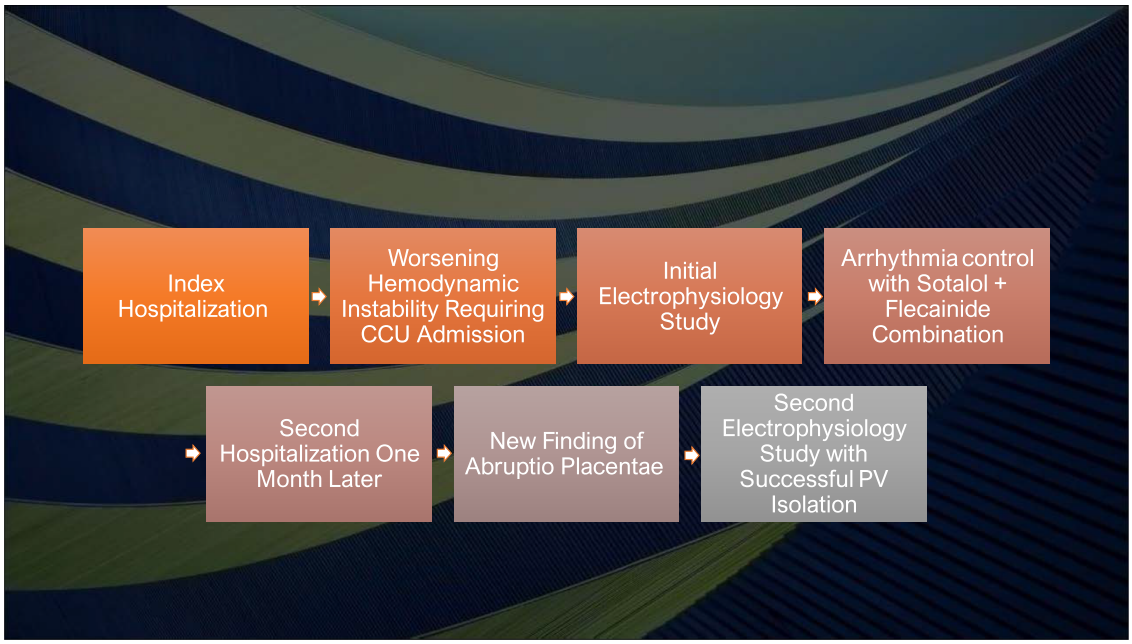
pharmacological intervention, catheter ablation remains an option, but candidacy must be evaluated on a case-by-case basis, including the risks of radiation exposure, thromboembolism, post procedure anticoagulation, vascular complications, and foetal compromise.

Timeline



Case presentation

A 31-year-old G3P2 female at 25 weeks of gestation presented with chest palpitations and light-headedness. Physical exam demonstrated tachycardia with borderline hypotension, without evidence of new murmurs on initial auscultation. Her medical history was notable for atrial tachycardia (AT) during two previous pregnancies managed with metoprolol and flecainide, though ultimately requiring induction of labour due to impending foetal compromise at 34 weeks. She underwent two failed catheter ablation procedures at outside institutions several months after delivery of her second child; however, the arrhythmia was not inducible during the first study, and the second was aborted prior to ablation due to haemopericardium requiring emergent pericardiocentesis. An implantable loop recorder (ILR) was placed at that time. The patient was reluctant to undergo another ablation and was treated with sotalol 40 mg twice a day by her primary cardiologist. During her third pregnancy, higher doses were not prescribed due to a low baseline blood pressure. This admission was prompted by chest palpitations and dizziness associated with an SVT at 160 beats per minute while on her home maintenance dose of sotalol.

Her blood work-up on index hospitalization did not demonstrate any electrolyte or thyroid abnormalities to potentially explain her arrhythmia exacerbation. Electrocardiogram revealed a narrow complex, long R to P wave interval tachycardia with ventricular rates of 162 bpm, and QTc 408 ms (*Figure 1*). Transthoracic echocardiogram showed grossly normal systolic ventricular function without any evidence of valvular pathology.

Intravenous boluses of metoprolol, adenosine, and diltiazem were administered without conversion to sinus rhythm; however, transient atrioventricular block was observed without affecting the atrial rate, consistent with the previous suspected diagnosis of AT. Sotalol was discontinued and she was switched to oral flecainide acetate and metoprolol tartrate, with the doses up titrated to 100 mg BID, respectively. Despite escalation of medical therapy, she continued to have multiple breakthrough episodes with concomitant palpitations, requiring diltiazem infusion at 10 mg/min. Her course was further complicated by worsening hypotension and risk of foetal bradycardia, requiring admission to the coronary care unit for vasopressor support with intravenous phenylephrine and foetal monitoring.

After coordinated discussions with electrophysiology and maternal foetal medicine, a decision was made to undergo a fluoroscopic-free catheter ablation with electroanatomic mapping to minimize radiation exposure. Due to the patient's anxiety, the procedure required general anaesthesia and continuous peri-operative foetal heart rate monitoring was performed under the supervision of maternal foetal medicine. Despite aggressive stimulation with quadruple stimuli and isoproterenol infusion, the procedure was unsuccessful at inducing the culprit clinical AT. A right atrial flutter was reproducibly induced and successfully ablated.

Empiric isolation of the pulmonary veins (PVs) was considered, but not pursued for several reasons. The lack of inducibility precluded accurate mapping of the site of origin, with no well-defined endpoint for ablation. Empiric PV isolation would require at least 4 weeks of post-ablation oral anticoagulation which could cause more harm during pregnancy, particularly if the site of origin was outside the PVs. Unfortunately, the AT recurred 2 h after the procedure, and the decision was made to up-titrate pharmacological therapy, with a combination of sotalol 120 mg and flecainide 100 mg twice daily. This suppressed the

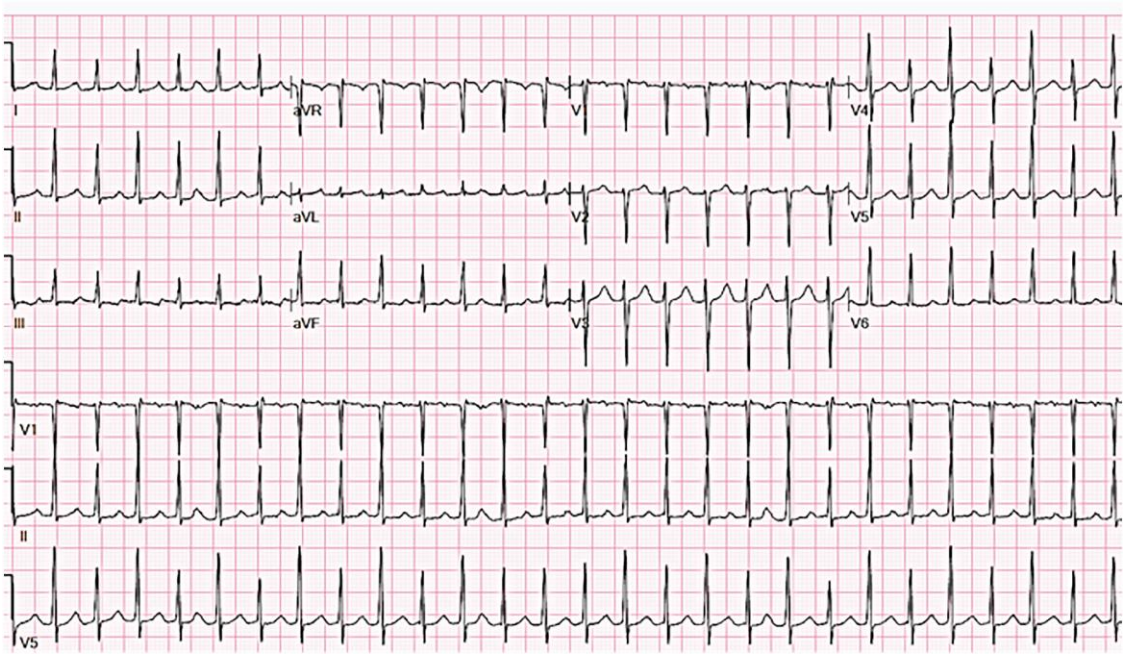


Figure 1 Initial electrocardiogram on index hospitalization demonstrating narrow complex supraventricular tachycardia with heart rate of 162 beats per minute, QT 248 ms/QTc 408 ms (Bazett calculation)/345 (Fridericia calculation).

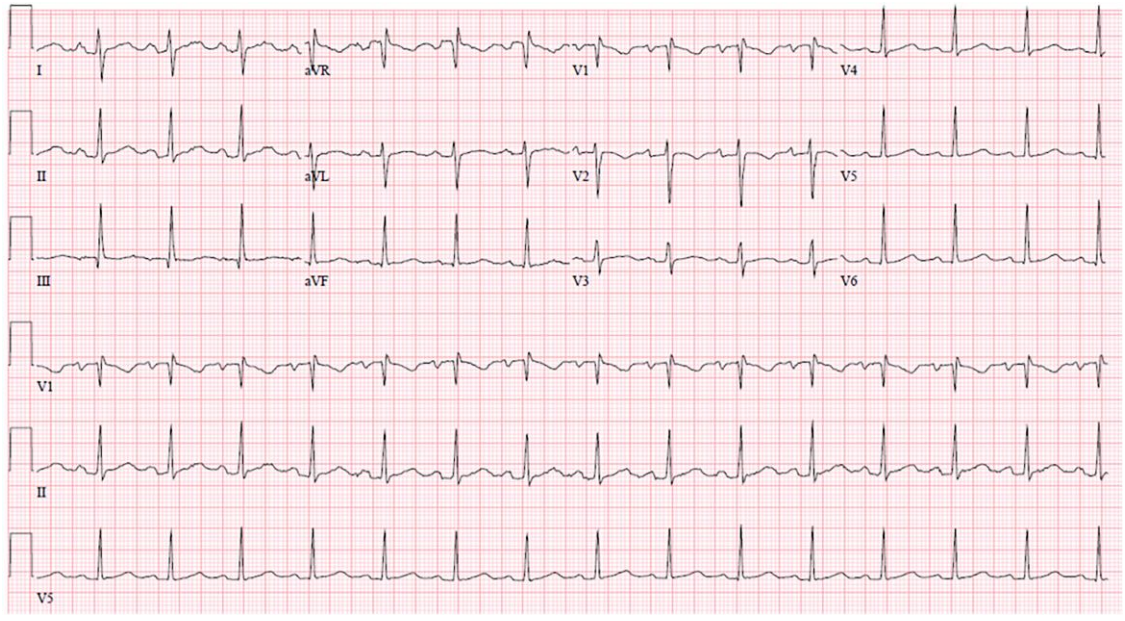


Figure 2 Electrocardiogram demonstrating normal sinus rhythm after initiation of pharmacologic therapy with sotalol and flecainide therapy. QTc 487 ms.

arrhythmia, and she was monitored for an additional 72 h as an in-patient with an acceptable QTc of 487 ms and sinus rate (*Figure 2*), following which she was discharged. She returned to the emergency department 6 weeks later at 31 weeks' gestation with another episode of symptomatic AT, shortly followed by a new onset of abruptio placentae, requiring an emergent, yet successful caesarean section.

Four days postpartum, she continued to have intermittent, non-sustained episodes of AT. The procedural bleeding risks and post-

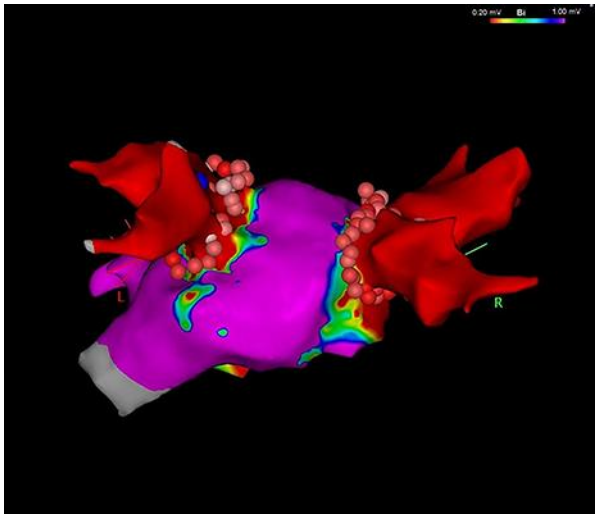


Figure 3 Posterior left atrium with normal voltage noted after successful pulmonary vein isolation.

ablation anticoagulation were discussed with obstetrics who deemed her to be of acceptable risk for initiation of oral anticoagulation. She underwent a repeat fluoroscopy-free electrophysiology study and ablation under general anaesthesia. This time, burst pacing from the coronary sinus at 300–400 ms on isoproterenol induced the left AT which matched her clinical arrhythmia. Based on high density mapping with a PENTARAY® catheter (Biosense Webster Irvine, CA), triggered activity was found primarily in the left superior PV: however, was also present in the right superior and left inferior PVs. She therefore underwent isolation of all four PVs with non-inducibility of any arrhythmia post ablation with and without isoproterenol infusion (*Figure 3*). Twenty-four months post-ablation, she has been symptom free and has had no SVT on the ILR. Both she and the baby are healthy with no complications from either the pharmacotherapy or ablation procedures.

Discussion

Currently, there are no explicit guidelines for antiarrhythmic drug (AAD) use during pregnancy and is not recommended except in severely symptomatic cases. There is a lack of randomized trials with little to no systematic data on the efficacy and safety of AAD use in pregnancy.[2] Pharmacotherapy side effects such as QTc prolongation, exacerbation of tachyarrhythmias, hypotension, risk of premature labour and differential effects to both mother and foetus require consideration. Studies have also demonstrated foetal complications in gestational arrhythmias medically treated with AADs, including but not limited to, respiratory distress syndrome, growth restriction, and foetal congenital heart disease.[3] Additionally, the risk of placental abruption is higher based on a retrospective study of 143 women during pregnancy, of whom 25% had arrhythmias with two cases of abruption placentae in the arrhythmia cohort, but none in those without arrhythmia.[4]

In our patient, the culprit AT could not be induced despite aggressive stimulation; therefore, empiric PV isolation was not performed without clear identification of the site of origin which could have been outside the PVs. Short-term risk of anticoagulation in the post-ablative period must be considered during the management of gestational arrhythmias due to effects on the foetus as well as bleeding complications such as abruptio placentae, which could be fatal to the mother and foetus.[5] The decision not to proceed with empiric PV ablation proved fortuitous given the subsequent placental rupture, which would have been further complicated on anticoagulation therapy. Also, early post-PV isolation irritability might still require pharmacological therapy, which may be detrimental.

In this case, due to the inability to suppress the arrhythmia with both flecainide and metoprolol, a combination of flecainide with sotalol was initiated in the hospital. This combination required careful monitoring of sinus rates, QRS duration, QT intervals as well as foetal haemodynamic stability. Although flecainide is a Class 1C agent with weak QT prolonging effects, the primary effect is sodium channel blockade, primarily manifesting as mild prolongation of the QRS at therapeutic doses. Prolonged flecainide use has been associated with cholestasis of pregnancy and decreased foetal heart rate variability.[6] Sotalol is a Class III agent with beta blocking effects resulting in QT prolongation and sinus slowing; therefore, it was considered safe to try the two in combination with careful monitoring and has been previously reported.[7]

Several case reports document successful zero-fluoroscopic ablations with the use of electroanatomic mapping or intracardiac echocardiography. The major benefit of this approach is the reduction of radiation exposure to both the mother and child which should be minimized particularly in early gestation during organogenesis, though the threshold exposure for foetal abnormalities varies.[8] This is further supported by studies that have demonstrated the safety of zero vs. minimal fluoroscopy ablation even for PV isolation.[9]

Though the reported frequency of gestational SVT related hospitalizations is approximately 22 per 100,000, catheter ablation should ideally be considered before pregnancy in patients with a history of symptomatic tachyarrhythmia; however, in the European Society of Cardiology guidelines, catheter ablation for SVT during pregnancy has a Class IIa indication if done at centres experienced in performing the procedure without fluoroscopy.[10] In the United States, ablation during pregnancy is considered a 'last resort' and carries a IIb indication if done with manoeuvres to minimize fluoroscopy.[11] In complex situations where SVT is refractory to medical therapy or the mother is not a candidate for ablation, the ultimate goal is to maintain maternal and foetal safety with consideration of a timely caesarean section with shared decision-making among cardiology, maternal foetal medicine, and obstetrics to ensure safe outcomes in this vulnerable demographic.

Lead author biography

Dr Joseph You completed his medical school training at the State University of New York Downstate. He is now completing his internal medicine residency training at Northwell Northshore and Long Island Jewish Hospital in New York with plans for cardiology fellowship in 2023. He is a member of the European Society of Cardiology with prior publications in the field of general cardiology and COVID-19 associated cardiovascular sequelae.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports*.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: Informed consent from the patient was approved prior to the submission of this publication in accordance with COPE guidelines.

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