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Unusual presentation of supraventricular tachycardia degenerating into ventricular fibrillation during pregnancy: Aortocaval compression the probable culprit

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ABSTRACT

Cardiac arrhythmias are common and often benign in pregnancy. However, haemodynamic instability can occur when tachyarrhythmias are accompanied by aortocaval compression, which can lead to loss of cardiac output. We present an atypical case of a pregnant woman with a supraventricular tachyarrhythmia, which degenerated into ventricular fibrillation arrest while supine due to aortocaval compression. Inducible atypical atrioventricular nodal re-entry tachycardia was subsequently detected on electrophysiological study and presumed to be the most likely initial supraventricular tachyarrhythmia.

1. Learning objective

While most arrhythmias are not life-threatening in pregnancy, accompanying aortocaval compression while supine can cause haemodynamic instability. Aortocaval compression syndrome is a phenomenon in mid-to-late pregnancy, which can potentially compromise cardiac preload, blood pressure and uteroplacental perfusion. Our case highlights the potential for ventricular fibrillation arrest to be induced by loss of cardiac output from aortocaval compression with a concomitant supraventricular tachycardia.

2. Case report

A 30-year-old woman who was 24 weeks' pregnant presented to the emergency department with rapid onset of regular palpitations, associated with 40-minutes of pre-syncope. Her heart rate was 200 beats/minute and blood pressure 108/73 mmHg. She went into cardiac arrest within 60-seconds of being placed supine, before an electrocardiogram (ECG) of the tachyarrhythmia was obtained. Immediate cardiopulmonary resuscitation (CPR) was performed. The first documented rhythm was ventricular fibrillation (VF; Fig. 1A), and subsequent direct current cardioversion at 200-Joules restored sinus rhythm. She was then placed in left lateral position to relieve aortocaval compression (ACC).

Personal and family history were unremarkable for cardiac disease and sudden cardiac death (SCD). There was no history of smoking, alcohol or recreational drug use, nor palpitations, syncope or angina. Resting ECG was normal (Fig. 1B), while serum electrolytes, troponin, D-dimer and thyroid screen were unremarkable. Transthoracic echocardiogram (TTE) and cardiac magnetic resonance (CMR) imaging revealed bicuspid aortic valve with mild regurgitation, but normal ventricular function and chamber sizes. Coronary angiography was unremarkable, excluding spontaneous coronary artery dissection. Inpatient telemetry was unremarkable.

Adenosine challenge with 12-lead ECG showed atrioventricular block, without evidence of anterograde accessory atrioventricular conduction. At that time, electrophysiological study (EPS) was not

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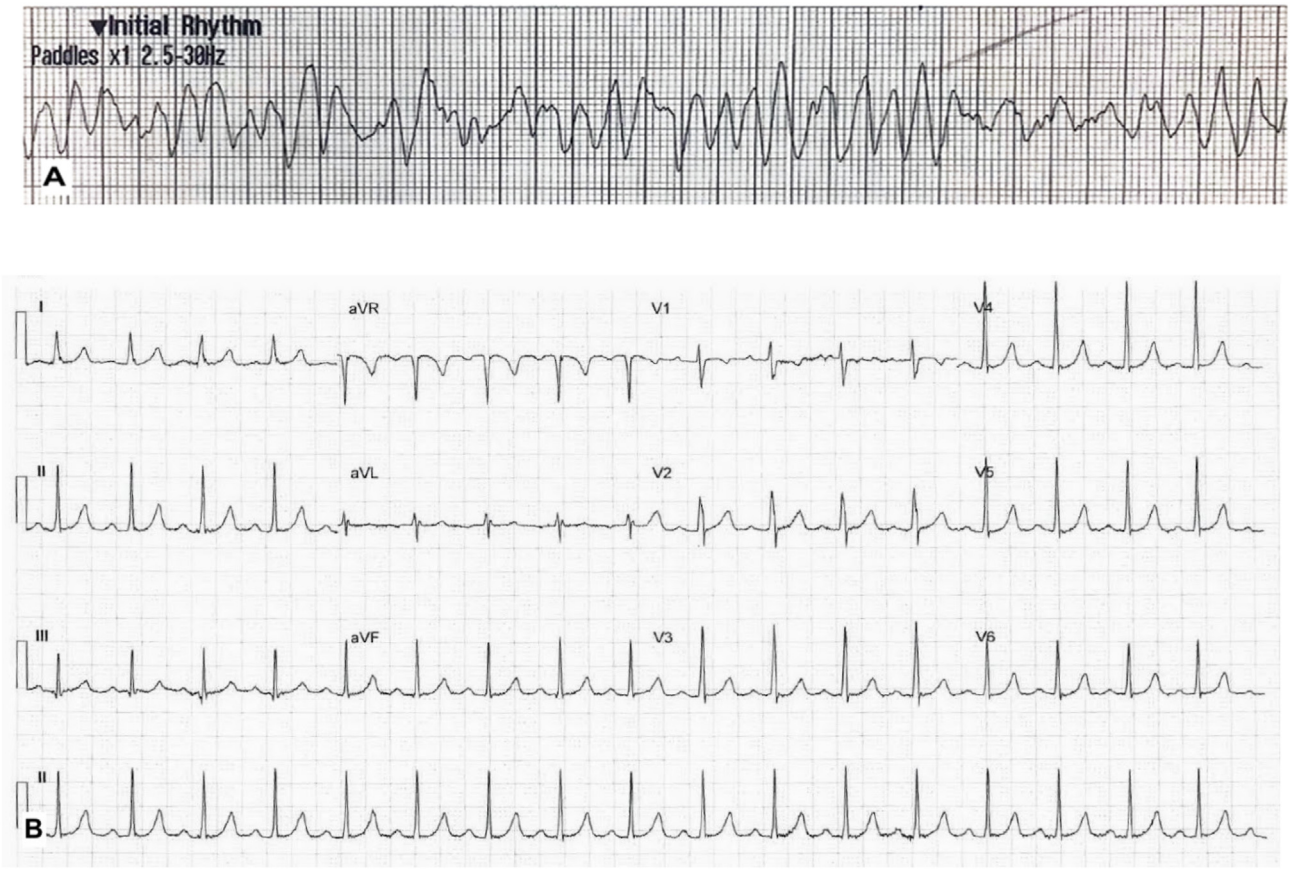

Fig. 1. A: Initial rhythm strip showing ventricular fibrillation. **B**: Resting electrocardiogram showing sinus tachycardia, normal axis and normal QTc interval.

performed given risks of maternal-fetal fluoroscopic radiation exposure. She underwent subcutaneous-implantable cardioverter defibrillator (S-ICD) implantation for secondary prevention of SCD. Both patient and fetus received ongoing review by cardiology and obstetric teams. Fetal wellbeing ultrasound scans, serial TTE and routine S-ICD interrogation were unremarkable throughout admission, and she eventually underwent an uneventful caesarean delivery.

At 12-months post-partum, EPS was performed to investigate for substrate of the observed malignant ventricular arrhythmia (Fig. 2A). Dual atrioventricular nodal pathway physiology was confirmed, without evidence of accessory atrioventricular conduction. Ventricular programmed extrastimulation with up to four extras was used, attempting to induce ventricular tachycardia in sedated state (S1 = 400, S2–S4 to refractoriness or 200ms, S5 to refractoriness). No ventricular tachycardia (VT) was induced, down to following coupling intervals: 400/250/230/200/180 (Fig. 2B). Burst atrial pacing in the presence of isoprenaline readily induced atypical atrioventricular nodal re-entry tachycardia (AVNRT; Fig. 3A and B). EPS and radiofrequency ablation were considered during initial admission, potentially obviating need for S-ICD implantation. However, risks of foetal radiation exposure swayed us against this approach. Furthermore, without clear documentation of AVNRT prior to VF and recognising the otherwise presumed benign course of this arrhythmia, slow pathway ablation then would not have removed need for S-ICD.

No further sustained ventricular arrythmias have occurred, nor S-ICD therapy has been needed. The patient underwent successful ablation of atypical AVNRT pathway, remaining clinically well with regular follow-up in our cardiology clinic. The S-ICD has not been explanted since the ablation, but this remains a consideration after informed discussions with the patient.

3. Discussion

Supraventricular tachycardias (SVTs) are the most common sustained arrhythmias in pregnancy, with the most frequently encountered form in women without structural heart disease being AVNRT [1]. Pregnancy is associated with haemodynamic, autonomic and hormonal changes, contributing to increased incidence of arrhythmias [2]. Sustained ventricular arrhythmias are rare in pregnancy, often associated with structural heart disease or history of arrhythmias [3]. While our patient had bicuspid aortic valve, TTE and CMR revealed normal cardiac function, excluding outflow tract obstruction or aortic stenosis (AS). Moreover, an otherwise structurally normal heart on serial imaging, and lack of VT inducibility during EPS make VT unlikely cause for her initial palpitations. Importantly, no anti-arrhythmic medications had been given to our patient after the cardiac arrest or before the EPS procedure. PPCM was excluded given normal ventricular sizes and function, and lack of clinical evidence of heart failure. Channelopathies were unlikely given consistently normal ECGs, while lack of high burden of symptomatic premature ventricular contractions make ectopy-induced VF unlikely.

Earlier reports have described SVT degenerating into polymorphic VT in structurally normal hearts, with dual AV nodal pathways identified on subsequent EP studies as seen in our case. While the exact mechanism underlying these associations is unclear, one hypothesis is that an embryological remnant of the developing ventricular conduction system, termed “dead-end

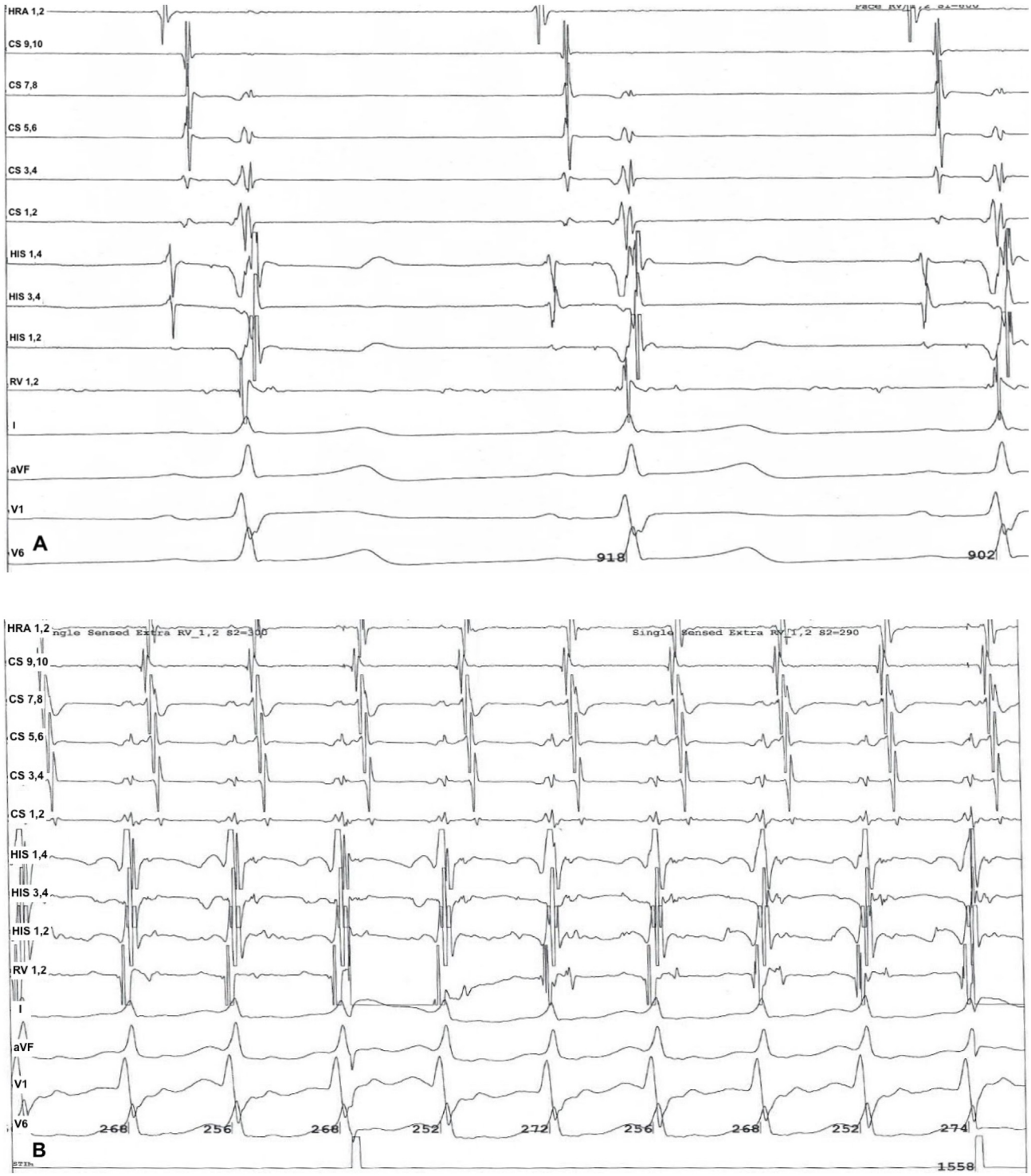

Fig. 2. A: Intra-cardiac electrocardiogram at 98mm/sec recordings while in sinus rhythm. **B**: Intra-cardiac electrocardiogram at 98mm/sec recordings while in AVNRT (AVNRT: atrioventricular nodal re-entry tachycardia; CS: coronary sinus lead [numbered]; HIS: Bundle of His lead [numbered]; HRA: high right atrium lead; RV: right ventricular lead).

tract", may connect AV nodal region and focus for idiopathic VT. This "dead-end tract" may have arrhythmic potential, associated with occurrence of idiopathic ventricular arrhythmias [4].

In this case, SVT degenerating into VF arrest may also have been contributed to by loss of preload from ACC. A potential explanation for our patient's presentation was symptomatic atypical AVNRT, later inducible during post-partum EPS, initially manifesting as sustained regular palpitations before first medical contact. Subsequent ACC while supine could have compromised venous return and cardiac output, thus precipitating VF arrest.

ACC syndrome is a well-documented phenomenon in pregnancy, present from around 20 weeks of gestation onwards. It was first reported in 1942, with reports theorising that the gravid uterus compresses inferior vena cava (IVC) and abdominal aorta while

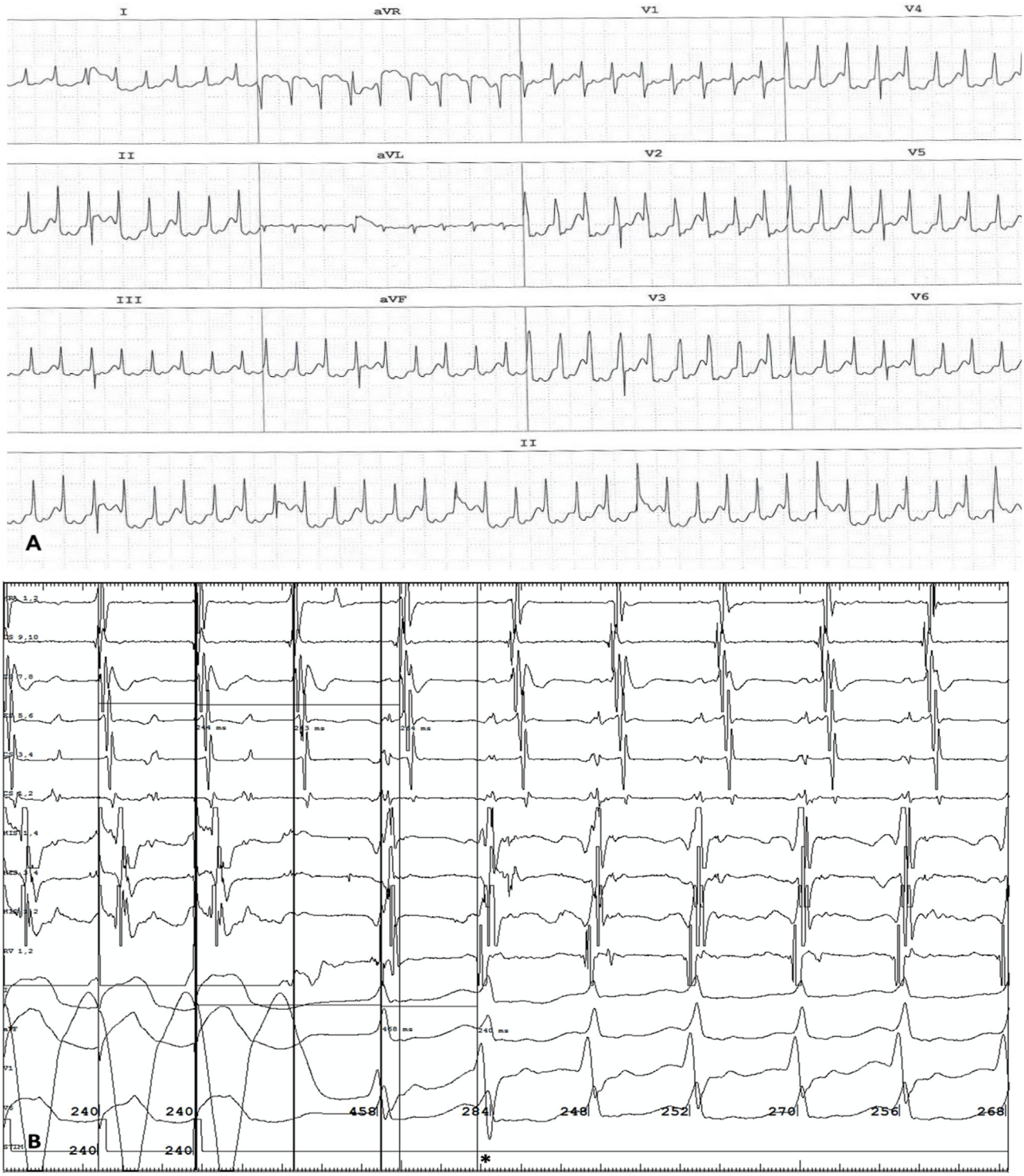

Fig. 3. A: 12-lead electrocardiogram of induced narrow-complex tachycardia consistent with atypical AVNRT. **B**: Intra-cardiac electrocardiogram at 98mm/sec recordings while in AVNRT. Tachycardia response to continuous atrial advancement by right ventricular apical overdrive pacing at 240 ms: the post pacing interval (PPI) is 458 ms, while the tachycardia cycle length (TCL) at this time is around 250–270 ms as per the third return beat onwards (note that the second return beat after ventricular pacing (*) is likely pre-empted by right ventricular mechanically induced ectopy as per timing (240 ms), reversal of His 1,4 ventricular electrogram vector and QRS fusion (most notable in V1)); HRA: high right atrium, CS: coronary sinus [numbered]; HIS: His recording channel [numbered]; RV: right ventricular apex.).

supine [5]. Significant reduction of venous return from lower extremities back to maternal circulation may then reduce cardiac output [6]. In an early study by Kerr et al., dye injection through bilateral femoral vein catheterisation in supine term pregnant women revealed complete obstruction of IVC in 80% of cases (10/12 cases) [7]. Subsequently, Higuchi et al. similarly demonstrated, through magnetic resonance imaging in term women, that near complete IVC compression occurred while supine [8].

Prior studies have not reported VF and cardiac arrest precipitated by ACC in patients with structurally normal hearts. However,

Chen et al. demonstrated ACC and loss of consciousness in a 35-year-old woman at 36 weeks' gestation with subvalvular AS [9]. Specifically, loss of consciousness, bradycardia and hypotension occurred from left-tilt to supine position during caesarean section. The proposed underlying mechanisms were ACC syndrome reducing venous return while supine and left ventricular outflow obstruction from subvalvular AS [9].

ACC alone may not necessarily cause significant haemodynamic instability, as compensatory reflex increases in systemic vascular resistance and heart rate may maintain blood pressure [10]. Furthermore, compensatory increases in collateral venous flow through paravertebral and azygos systems may augment preload [6]. Susceptibility to haemodynamic compromise from ACC may depend on factors, including heart rate, baseline ventricular function, and pre-existing cardiac conditions [9,10]. In this case, our patient likely had preceding atypical AVNRT rapidly sustained at 200 beats/min, subsequently inducible on postpartum EPS, which likely overwhelmed compensatory mechanisms, contributing to loss of preload, cardiac output and deterioration into VF arrest when combined with ACC.

Standard supine CPR in late pregnancy may be less effective, as ACC may impede venous return and forward blood flow during chest compressions. Relief of IVC obstruction through manoeuvres, including manual left uterine displacement, have therefore been recommended by Society for Obstetric Anaesthesia and Perinatology and American Heart Association [3,6]. Another therapeutic manoeuvre, left lateral tilt manoeuvre at $\geq 30^{\circ}$, may relieve IVC obstruction, but interfere with chest compression during CPR [5].

ACC in mid-to-late pregnancy may reduce cardiac output, blood pressure and uteroplacental perfusion. Our case describes cardiac arrest from loss of preload due to ACC in a pregnant lady, whom presented with palpitations likely from atypical AVNRT. Further reports are needed to corroborate AVNRT deteriorating into VF arrest associated with ACC, alongside determine optimal methods for relieving ACC in pregnancy. Ultimately, given potential for ACC to cause haemodynamic instability, maternal-fetal morbidity and mortality, awareness of this phenomenon remains important in pregnancy.

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