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Rare disease

Recurrent atrial flutter and fibrillation in pregnancy

Charly J Sengheiser, Kevin C Channer

Department of Cardiology, Royal Hallamshire Hospital, Sheffield, UK

Correspondence to Dr Charly J Sengheiser, charlysengheiser@doctors.org.uk

Summary

The authors report the case of a woman who only developed atrial fibrillation during successive pregnancies. They believe that this case is unique.

CASE PRESENTATION

A 26 year-old-woman developed a regular tachycardia of 160 beats/min during labour at 41 weeks of gestation. She was given 6 mg adenosine intravenously which demonstrated flutter waves, confirming the diagnosis of atrial flutter with 2:1 A-V nodal block. Routine blood tests were normal including thyroid function tests and full blood count. She denied taking any over the counter medications or drugs of abuse. Oral digoxin (500 mcg) was given and this effected cardioversion to sinus rhythm at 90 beats/min. A caesarean section was then done delivering a healthy baby. She remained in sinus rhythm postoperatively and persistently. Digoxin treatment was discontinued before discharge. She was seen on follow-up by a cardiologist, who noted that she had no further palpitations and was well. An echocardiogram showed normal ventricular size and function, no significant valve lesion and normal atrial dimensions. Twenty-four hour ambulatory ECG revealed normal sinus rhythm throughout.

Three years later at 33 weeks into her second pregnancy she developed palpitations, light headedness and mild breathlessness. She was admitted as an emergency to hospital and an ECG showed atrial fibrillation with a ventricular response rate of 190 beats/min. The arrhythmia persisted for 9 h and although she was haemodynamically stable, it was decided to cardiovert her because of her symptoms and rate, and to avoid the use of drugs in pregnancy. She was anaesthetised, but spontaneously reverted to sinus rhythm before the shock was delivered. Overnight monitoring confirmed persistent sinus rhythm and she was discharged.

Two months later at 39 weeks gestation she was admitted with chest tightness, breathlessness and palpitations. An ECG revealed atrial fibrillation with a fast ventricular response rate of 190–200 beats/min. She was given 500 mcg digoxin but remained in atrial fibrillation. She was anaesthetised, cardioverted with a 100 joules synchronised DC shock and then a healthy female baby was delivered by caesarean section. Postoperatively she remained in sinus rhythm. She was discharged on bisoprolol 2.5 mg daily to reduce the likelihood of recurrence because she had had peri-partum atrial fibrillation. On follow-up 8 weeks later she remained in sinus rhythm.

DISCUSSION

Atrial fibrillation is rare in pregnancy in Western populations as the frequency of rheumatic heart disease has fallen. Historically, case series have shown that the haemodynamic changes of pregnancy can precipitate atrial fibrillation in older women with mitral valve disease,[1] and this may cause heart failure in pregnancy.

This situation is now only seen in the UK, in women who have been born in areas of high incidence of rheumatic fever (eg, the Indian subcontinent). In the UK, today, atrial fibrillation is more likely to occur in women with treated congenital heart disease which comprise the more common cohort of women with heart disease in pregnancy.[2] It can also occur because of thyrotoxicosis.[3]

Atrial fibrillation in the absence of structural heart disease is called 'lone atrial fibrillation', which is much more common in men. We have found only four other case reports in the literature of lone atrial fibrillation in pregnancy. All these cases occurred in older women towards the end of pregnancy (table 1). We believe that the case we present is unique in that atrial fibrillation complicated two consecutive pregnancies and did not occur when she was not pregnant. The literature shows that atrial fibrillation may be precipitated by drugs used in premature labour, especially terbutaline and nifedipine.[4–7]

The mechanism underpinning the development of atrial fibrillation is stretch of the atria; any condition that does this can cause the arrhythmia. The most common cause in population studies is hypertension.[8] The volume loading of pregnancy and the changes in blood pressure towards the end of pregnancy especially in older women with a less compliant circulation, may be the initiator for the arrhythmia in these case reports. However, our patient is much younger than those previously reported and developed the arrhythmia in successive pregnancies. This suggests that she has an underlying arrhythmic potential as seen in patients with atrial flutter where there appears to be an arrhythmic focus in the pulmonary veins.[9]

Atrial fibrillation is a benign arrhythmia and causes its haemodynamic effects because of the loss of atrio-ventricular synchrony. In late pregnancy, it is not well tolerated and DC cardioversion is a reasonable safe option. Prior anticoagulation is not necessary if cardioversion is

Table 1 Reported cases of atrial fibrillation in pregnancy

Author	Age (years)	Structural heart disease	Gestation	Management	Outcome
Cacciotti *et al*[14]	35	None	36 weeks	β-blocker	LSCS live birth
Gowda *et al*[15]	29	None	38 weeks	Spontaneous cardioversion	Normal
Walsh *et al*[11]	41	None	35 weeks	Flecainide	Normal
Kuczkowski[16]	26	None	During LSCS	Spontaneous	LSCS
Parasuraman *et al*[4]	39	None	33 weeks during treatment for threatened labour with nifedipine	DCCV	Normal
Arimie[5]	24	None	34 weeks during treatment for premature labour with terbutaline	Diltiazem, digoxin, esmolol, then DCCV	Normal
Carson *et al*[6]	30	None	35 weeks (twins) during treatment for premature labour with terbutaline	Digoxin, diltiazem, esmolol, procainamide	Normal twins
Lashgari *et al*[7]	20	None	31 weeks during treatment for premature labour with terbutaline	Diltiazem	(Baby not born at time of publication)

DCCV, direct current cardioversion; LSCS, lower segment caesarian section.

done within the first 48 h after onset in women with no structural heart disease.[10] In women with rheumatic mitral stenosis there is a substantial increased risk of thromboembolism and anticoagulation is mandated. Chemical cardioversion by drugs with class Ic antiarrhythmic properties, like flecainide, have been used in pregnancy[11] but experience is limited.

If, instead of cardioversion, a strategy of rate control is used then digoxin and β-adrenoceptor blockers are probably the best drugs with the widest experience of use. Digoxin has not been associated with teratogenic effects in man or animals.[12] β-blockers are used in pregnancy for hypertension control and are generally thought to be safe.[12] Anticoagulation should be guided by risk assessments (eg, CHA2DS2-VASC score),[13] as full anticoagulation by heparin or warfarin is associated with significant foetal and maternal morbidity.

Given this patient's arrhythmic potential resulting in gestational atrial arrythmias she would need cardiology follow-up during any future pregnancies.

Learning points

- Atrial fibrillation in pregnancy is rare in the Western world.
- It can be precipitated by drugs used in premature labour or thyrotoxicosis.
- In late pregnancy, atrial fibrillation is not well tolerated and should be controlled.
- DC shock is a safe option of cardioversion during pregnancy.
- Chemical cardioversion can be achieved with flecainide, rate can be controlled using digoxin or β-blockers.
- Anticoagulation is often not necessary.

Competing interests None.

Patient consent Obtained.

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