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CASE REPORTS

Incessant atrial tachycardia accelerated by pregnancy

J Colin Doig, Janet M McComb, Douglas S Reid

Abstract

A 24 year old patient presented with incessant atrial tachycardia during the course of a twin pregnancy. Medical treatment slowed the ventricular response without restoring sinus rhythm. During labour the tachycardia spontaneously reverted to sinus rhythm. Subsequently the same arrhythmia was documented with a slower ventricular response than during pregnancy.

Case report

At 21 weeks' gestation of a twin pregnancy a 24 year old patient was transferred for management of incessant supraventricular tachycardia because of concerns about the effect on placental function. There was a three month history of palpitation and chest discomfort, dizziness, and exertional dyspnoea that had begun eight weeks into the pregnancy. There was no history of viral illness and she had been otherwise healthy with no previous history of palpitation. At admission the heart rate was 200 beats/min and systolic blood pressure was 90 mm Hg. Her electrocardiogram showed a narrow QRS complex tachycardia with upright P waves in the inferior limb leads and V1, with a frontal P wave axis of +90° (fig 1A). The PR interval was 140 ms. Occasionally nonconducted P waves of identical configuration and timing were seen during the tachycardia (fig 1B). The rate of the arrhythmia showed considerable diurnal variability ranging from 135 to 210 beats/min. Isolated ventricular extrasystoles did not reset the tachycardia. There was QRS alternans at ventricular rates faster than 190 beats/min. Haematological studies, urea and electrolytes, and liver function tests were normal. A free thyroxine plasma concentration of 164 nmol/l (normal range 60–140) and thyroid stimulating hormone concentration of 1·4 IU/l (normal range 0·15–3·2) were consistent with pregnancy. Transthoracic echocardiography and chest *x* ray were normal. Fetal echocardiography showed normal hearts and rates.

The arrhythmia did not respond to carotid sinus massage, Valsalva manoeuvres or intravenous verapamil. Digoxin was used without benefit and cardioversion did not restore sinus rhythm or affect the ventricular response rate. Metoprolol and digoxin in combination slowed the ventricular rate to 185 beats/min but the diurnal fluctuation in PP and RR intervals continued. Flecainide was therefore introduced in place of metoprolol and digoxin. This slowed the atrial rate to 115 beats/min and allowed transient episodes of sinus rhythm at 75 beats/min to appear with a frontal P wave axis of +10°. The PR interval was 200 ms with no evidence of pre-excitation (fig 2). This was considered a satisfactory interim result and the patient was discharged on oral flecainide 100 mg three times daily. During the next month the QRS duration increased from 80 ms to 100 ms and the heart rate was never consistently below 130 beats/min on Holter monitoring. Because of this inadequate rate control, flecainide was stopped and amiodarone was introduced at a dose of 800 mg a day for one week and maintained at 400 mg daily. This slowed the heart rate to 110 beats/min. On this treatment, she felt better, but was considerably restricted in her exercise capacity by palpitation, fatigue, and dyspnoea. There was no clinical evidence of heart failure or compromised fetal development.

At 31 weeks' gestation two healthy infants were delivered vaginally by forceps. Sinus rhythm returned spontaneously and the mother's heart rate fell below 100 beats/min during the first stage of labour. After delivery she reported that palpitation had ceased and she remained in sinus rhythm when seen in outpatients two weeks after hospital discharge. Amiodarone was stopped at this time and her electrocardiogram was similar to that in fig 2. The diagnosis was therefore assumed to be pregnancy related atrial tachycardia and this was supported by the prominent left atrial component to the P wave in sinus rhythm. She was re-referred, however, six months after delivery with non-cardiac symptoms at which time an electrocardiogram showed return of atrial tachycardia with a ventricular rate of 115 beats/min. She was on no antiarrhythmic treatment.

Discussion

Pregnancy is frequently associated with sinus tachycardia and increased ectopic activity has

Regional Cardiothoracic Centre, Freeman Hospital, Newcastle upon Tyne
J C Doig
J M McComb
D S Reid

Correspondence to
Dr J Colin Doig, University Department of Cardiology, Regional Cardiothoracic Centre, Freeman Hospital, Newcastle upon Tyne, NE7 7DN.

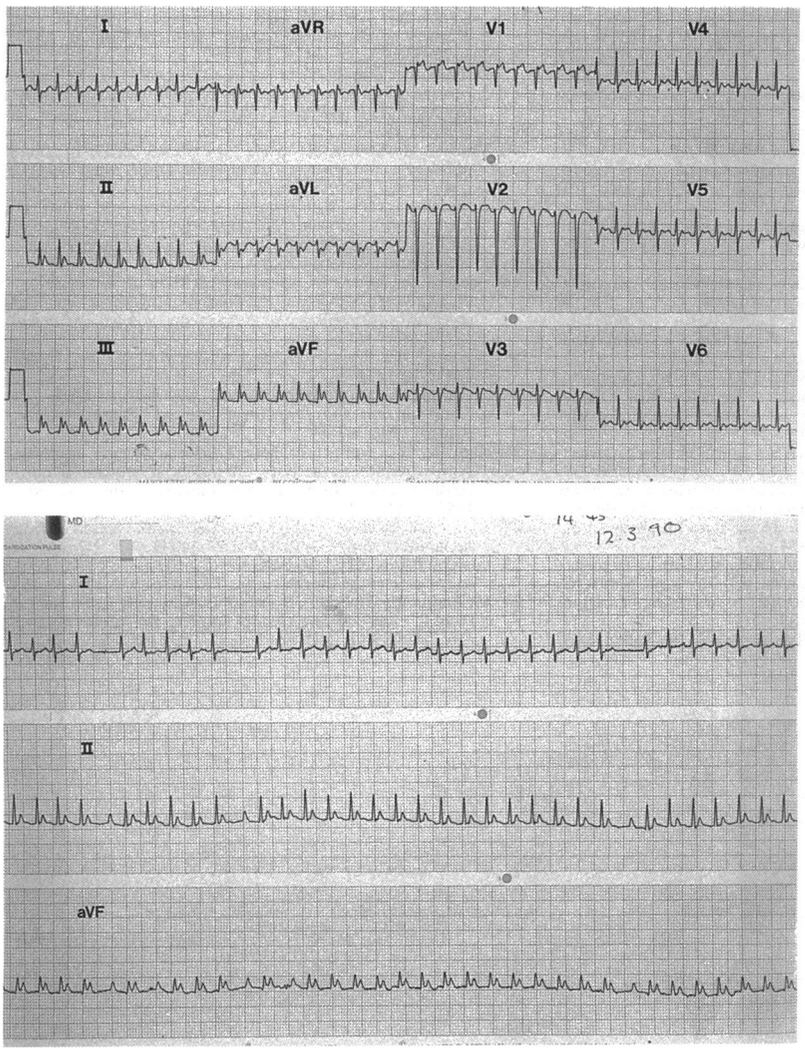

Figure 1 (A) Electrocardiogram showing narrow QRS tachycardia. (B) Rhythm strip showing non-conducted P waves of identical configuration and timing during tachycardia.

been noted both in the atrium and the ventricle throughout pregnancy.[1 2] Paroxysmal supraventricular tachycardia has been seen[1 3 4] and usually occurs in a patient with a history of supraventricular arrhythmias before pregnancy or in a patient with pre-existing rheumatic heart disease.[5] Such arrhythmias usually occur in the third trimester[6] but the true incidence is unknown.[7] Persistent atrial tachycardia such as was present in this patient is uncommon and has rarely been reported during pregnancy.[8 9] It has not previously been seen reverting to sinus rhythm during labour. An automatic atrial tachycardia more typically occurs in childhood[10 11] or complicating heart disease[12] or metabolic upset including digitalis toxicity.[13] In this case, recurrence of the same tachycardia some months later suggests that the patient had a potential for atrial tachycardia unrelated to pregnancy, but which first presented at an accelerated rate during pregnancy.

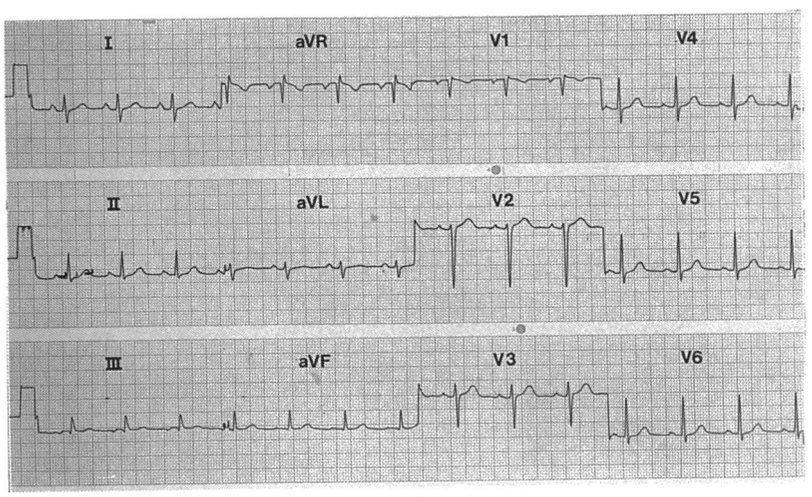

Figure 2 Electrocardiogram after return to sinus rhythm.

The mechanism of the tachycardia in this patient was probably enhanced sympathetic reactivity of an ectopic atrial focus. This accords with the increased plasma catecholamine concentrations and the increased adrenergic receptor sensitivity during pregnancy.[4 14] Increased atrial stretch due to plasma volume enlargement might also be implicated. The possible influence of the high concentrations of oestrogen and progesterone in pregnancy is unknown. An enhanced response to prostaglandins has been reported in pregnancy,[15] and in susceptible patients this could perhaps induce an atrial tachycardia. Thyrotoxicosis could also facilitate atrial arrhythmias; but there was no clinical or biochemical evidence of thyroid overactivity. Why the arrhythmia stopped during labour is not clear.

Because the persistent atrial tachycardia might have caused congestive cardiac failure in our patient[16–18] or have affected placental function and subsequent fetal development[19] we tried to reduce the ventricular rate response when sinus rhythm could not be restored. The management of such arrhythmias in pregnancy is difficult. Verapamil, digoxin, or cardioselective β blockade are appropriate and usually well tolerated.[20 21] Flecainide seems to be safe and cardioversion is not contraindicated.[22] Amiodarone is regarded as an agent of last resort because of concerns about neonatal goitre and its presence in breast milk.[23 24] An alternative treatment is to insert a temporary pacing electrode to terminate the arrhythmia or convert it to atrial fibrillation. Though appropriate radiation protection could be used, pacing was not thought likely to be successful in our patient and we thought that atrial fibrillation might further compromise placental function.

This rare case highlights the risk that inpatients with the potential for atrial tachycardia the condition can be precipitated and accelerated by pregnancy. The arrhythmia was persistent and led to unrealised concerns about placental function but it reverted to sinus rhythm early during labour. Treatment of a persistent tachycardia in pregnancy may be potentially toxic to the mother or fetus or both. Despite a twin pregnancy and incessant supraventricular tachycardia our patient delivered two healthy infants.

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