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CASE REPORT

Electrical cardioversion during pregnancy: safe or not?

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Abstract Two pregnant patients with a sustained symptomatic maternal supraventricular arrhythmia are presented. Both patients were treated with direct-current cardioversion. Electrical cardioversion during pregnancy is a rarely applied but highly effective procedure in the treatment of maternal cardiac arrhythmias and is assumed safe for both mother and child. However, once foetal viability is reached, monitoring of the foetal heart rate is advised and facilities for immediate caesarean section should be available.

Keywords Pregnancy · Cardioversion · Complications

Introduction

The occurrence of symptomatic maternal arrhythmias during pregnancy is a cause for concern for the well-being of both the mother and the foetus. As in the non-pregnant population, sustained symptomatic arrhythmias should be treated. Treatment depends upon the diagnosed or suspected specific arrhythmia. In case of supraventricular arrhythmias, electrical cardioversion (ECV) is applied when physical treatment such as sinus carotid massage or Valsalva manoeuvres and drug therapy fail or in life-threatening situations when the patient is haemodynamically unstable [1–8].

No large-scale studies concerning the safety of ECV in pregnancy are available. This article describes two cases and gives an overview of the reported efficacy and safety of ECV during pregnancy.

Cases

A 34-year-old woman without a relevant medical or obstetric history is pregnant of her third child. The course of the pregnancy is uncomplicated until a gestational age of 21+1 weeks, when she experiences progressive palpitations and feelings of agitation. Physical examination reveals an irregular pulse rate of 160 beats/min. Blood pressure (BP) is 127/86 mmHg, and there is no peripheral oedema. Auscultation of heart and lungs is normal. Anaemia and hyperthyroidism are excluded. The electrocardiogram (ECG) shows atrial fibrillation with a ventricular rate of 194 beats/min, and slight repolarisation disturbance in leads II, III, aVF and V3–V6. Echocardiography demonstrates no structural or functional abnormalities of the maternal heart. Treatment with a β-blocker is started to control ventricular rate. This does not result in conversion to sinus rhythm, and ECV (50 J) is successfully applied. The foetal condition is monitored by ultrasound, and no adverse effects are seen. The patient is discharged the same day without medication. At a gestational age of 40+1 weeks, she gives birth to a healthy boy of 3620 g. During 24 h of postpartum observation, no complications are noted. Paediatric evaluation shows no abnormalities.

The second patient is a 29-year-old primigravida with no relevant medical history and with antenatal care provided

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by a community midwife. At a gestational age of 34 weeks, she is referred to the hospital with palpitations. Maternal pulse rate is 160 beats/min, BP 118/79 mmHg, and SpO_2 98%. Anaemia and hyperthyroidism are excluded. Auscultation of heart and lungs reveals no abnormalities, and oedema of lower extremities is absent. The ECG shows a small complex tachycardia and a long r-p interval with a positive p wave in leads I and aVL and a negative p wave in leads II, III and aVF. Occasionally, 2:1 block is shown to the ventricle which excluded an atrioventricular re-entry tachycardia (AVRT); however, atrioventricular nodal re-entry tachycardia (AVNRT) and adenosine-sensitive atrial tachycardia cannot be ruled out. The diagnosis is most likely ectopic atrial tachycardia. Since adenosine and verapamil did not terminate the tachycardia, ECV is performed. ECV (100, 200 and 360 J) decreases the heart rate to some extent, but conversion to sinus rhythm does not occur. For additional rate control, digoxin and propranolol are given. Echocardiography shows normal systolic LV function and mild mitral and tricuspid insufficiency, which can be physiological in the third trimester of pregnancy. Foetal non-stress test and biophysical profile remain reactive throughout pharmacological treatment and ECV procedures. Maternal atrial tachycardia persists with variance in conduction, resulting in fluctuations of ventricular rate. Because of her declining clinical condition, echocardiography is repeated and reveals a decrease in left ventricular function probably due to tachycardiomyopathy. At a gestational age of 36+6 weeks, the foetal non-stress test shows deceleration of the foetal heart rate and a caesarean section is performed for foetal indications. A healthy baby girl of 3095 g is born. Postpartum drug-refractory symptomatic paroxysmal atrial tachycardia persists, and 18 months after delivery, a catheter ablation was performed. A subsequent pregnancy was uneventful.

Discussion

Cardiac arrhythmias are frequently diagnosed for the first time during pregnancy. An important risk factor for arrhythmias during pregnancy is the presence of organic heart disease. In most cases, there is no previous history of heart disease, and fortunately, arrhythmias that occur during pregnancy are almost always benign and no treatment is necessary. Sustained symptomatic arrhythmia, however, requires treatment. The incidence of maternal arrhythmias is 1.2 per 1000 pregnant women, of which 50% is asymptomatic. The incidence increases in the third trimester. During pregnancy, impressive haemodynamic, neurocardiogenic and autonomic changes occur, and it is supposed that the hyperdynamic circulation and the increased sensitivity of adrenergic receptors play a provocative role [1, 3, 5, 9]. Symptoms are dyspnoea, palpitations, dizziness and (pre) syncope [3, 5]. Most common arrhythmias in pregnancy are AVNRT, AVRT, atrial tachycardia and atrial fibrillation [3–6]. Atrial flutter and atrial fibrillation are often associated with hyperthyroidism.

Diagnosis can be hampered by the fact that symptoms of shortness of breath, palpitations, dizziness and presyncope frequently occur during normal pregnancy and increase with gestational age [3]. The presence of an arrhythmia is likely to cause an increase or even a new onset of these symptoms. As a result, the patient feels unwell. Clinical assessment and ECG investigation are mandatory for an accurate diagnosis of the arrhythmia [5]. Echocardiography is essential to exclude structural and functional heart disease.

Treatment of supraventricular tachycardias in pregnancy is the same as for the non-pregnant population. Initial treatment consists of stimulation of the vagus nerve by means of carotid massage or Valsalva manoeuvres. First-line pharmacological treatment in case of failure of physical procedures is with adenosine. Second choice is verapamil but only after the first trimester of pregnancy and only in acute circumstances [4–6, 9]. A low dose of β-blockers can be effective treatment for supraventricular extrasystoles or tachycardia**.** When drugs fail or in case of life-threatening symptoms as shock of pulmonary oedema, ECV is indicated [2–6, 9, 10].

We searched the English literature from 1965 and found 44 case reports that describe the use of ECV during pregnancy. There is considerable variation in specific arrhythmias for which ECV is applied and required energy varied from 50 to 400 J. Successful ECV after one or more attempts is reported in 41 pregnant women (93.2%). ECV in the non-pregnant population is reported to be successful in 42–92% [11, 12]. ECV success depends on the type and length of the arrhythmia, cardioversion method, voltage and type of energy. In three cases, a normal sinus rhythm is acquired only after additional drug treatment [13–15]. Two maternal deaths are reported shortly after cardioversion [16, 17]. In both cases, maternal death is attributed to the severity of underlying heart disease and no relation with cardioversion has been assumed.

There are limited data on perinatal outcome: 22 cases do not report pregnancy outcome [18–20], and 13 cases report an uncomplicated continuation of pregnancy resulting in term vaginal deliveries [2, 3, 13–15, 17, 21–24]. Three cases of a spontaneous preterm delivery are reported [8, 21, 25]. However, as in two cases where the delivery is 4 to 8 weeks after cardioversion, a direct relation with ECV is unlikely [8, 21]. In two cases, foetal distress directly after the cardioversion is noted, necessitating an immediate caesarean section at 37 and 28 weeks of gestational age, respectively [10, 26]. Both women are known with underlying cardiac disease. Both neonates are born healthy.

The first case shows a notably hypertonic uterus, while the current used was only 50 J. It was hypothesised that the hypertonic uterus resulted in foetal bradycardia, which could be a direct consequence of the cardioversion. Caution should be applied since the hyperaemic uterine muscle as well as the amnion fluid are excellent conductors of electricity [10, 27]. However, the uterus is usually not involved in the ECV trajectory, and only a minimum amount of current reaches the uterus. It is supposed that there is little effect on the foetal heart because of the high fibrillation threshold of a small heart [1, 8, 10, 20, 28, 29]. Possible explanations are placement of the pads over the apex beneath the left breast and/or the relatively large third trimester uterus.

These cases underline the importance of carrying out the procedure with facilities available for foetal monitoring and emergency caesarean section [17].

Conclusion

Provided that a multidisciplinary approach, continuous foetal heart rate monitoring and the possibility to perform a caesarean section are applied, it can be concluded that cardioversion is a safe and effective treatment for maternal tachycardia in pregnancy.

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