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## **Cesarean Section In a Patient With Wolff-Parkinson-White Syndrome Under Spinal Anesthesia**

### **Spinal Anestezi Altında Wolff-Parkinson-White Sendromlu Hastada Sezeryan Ameliyatı**

#### **ABSTRACT**

Maternal arrhythmias during pregnancy may be life-threatening. Several studies demonstrate that pregnancy predisposes maternal arrhythmias in asymptomatic patients with Wolff-Parkinson-White syndrome. The presence of an accessory pathway may responsible for Wolff-Parkinson-White syndrome. Regional anesthesia induced sympathetic blockade causes a prompt decrease in atrial filling pressure and this process results in increased dysrhythmia. Autonomic, hemodynamic and hormonal changes may predispose to arrhythmias in pregnancy. Hormonal changes especially abrupt changes in serum levels of estrogens are thought to be responsible in the development of arrhythmias during pregnancy. This case report describes a pregnant woman with Wolff-Parkinson-White syndrome under spinal anesthesia for cesarean delivery surgery.

**Key words:** Anesthesia, Spinal; Wolff-Parkinson-White Syndrome

#### **ÖZET**

Gebelikte olan maternal aritmiler ya amı tehdit edebilmektedir. Bir çok çalı mada, Wolff-Parkinson-White sendromlu asemptomatik hastalarda gebeli in maternal aritmilere yatkınlı ı arttırdı ı bildirilmi tir. Wolff-Parkinson-White sendromundan aksesuar yolların var olu u sorumlu olabilir. Rejional anestezi sırasındaki sempatik blokaj atrial dolum basıncında hızlı azalmaya ve bu da artan disritmilere neden olur. Gebelerdeki otonomik, hemodinamik ve hormonal de i iklikler aritmeye yol açabilir. Hormonal de i iklikler, özellikle östrojenin serum seviyesindeki ani de i ikliklerin gebelik sırasındaki aritmilerin geli mesinden sorumlu olabilece i dü ünülmektedir. Bu olgu sunumunda Wolff-Parkinson-White sendromlu gebe bir bayanda spinal anestezi altında sezeryan ameliyatını tartı tık.

**Anahtar Kelimeler:** Anestezi, Spinal; Wolff-Parkinson-White Sendromu.

#### **INTRODUCTION**

Several reports have indicated that pregnancy predisposes asymptomatic patients with Wolff-Parkinson-White (WPW) syndrome (1). The presence of an accessory pathway may responsible for WPW syndrome in patients. Circus movement tachycardia and atrial fibrillation may occur. Once an arrhythmia occurs high ventricular rates depending on the characteristics of the accessory pathway follow. Ventricular fibrillation (VF) may occur during high ventricular rates. WPW syndromes related with a low risk of sudden death and a good prognosis in asymptomatic patients (2,3). Here we report a 32-yr-old woman with WPW syndrome during spinal anaesthesia for Cesarean delivery (C/D).

#### **CASE REPORT**

A 32-year-old woman of 35 weeks' gestation (166 cm in height and 82 kg in weight) with WPW syndrome required caesarean section (C/D) under spinal anesthesia. She has been complaint with occasionally chest pain and tachycardia. She was diagnosed as asthma and WPW syndrome preoperatively. She has been taking 150 mg propafenone (rytmonorm®) per orally twice a day for atrial fibrillation. The results of preoperative examinations were normal, except for the findings of the sinus tachycardia (105 beat.min-1) with preexcitation, inferolateral delta wave, shortening of PR and QR intervals on a 12-lead electrocardiogram (ECG). Premedication was not given. Preoperative 1000 ml ringer laktat (RL) was administered. Systolic blood pressure, diastolic blood pressure and heart rate on arrival at the operating room were 125 mmHg, 75 mmHg and 108 beat.min-1, respectively. Spinal anaesthesia was applied uneventfully and 7,5 mg of 0.5% levobupivacaine and 10 µg fentanyl was injected at the level of L3-4 interspace and sensory analgesia reached the T4 level. Systolic blood pressure fall down 80 mmHg about

10 minutes later after anaesthesia and we applied 5 mg ephedrine intravenously (iv). Following ephedrine administration, we saw long QRS interval after than 3 ventricular arrhythmia occurred. We applied 1 mg midazolam iv. The baby was born uneventfully and oxytocin and midazolom 1mg iv. repeated. Blood loss was approximately 650 ml. When the operation was finished she was transferred to gynecology and obstetric units. She had no further complications in the postoperative period.

## DISCUSSION

Maternal arrhythmias during pregnancy may be life-threatening both for the mother and fetus. The incidence of WPW syndrome has been reported to be 0.1 and 2.5 per 1000 in asymptomatic person. Incidences of supraventricular arrhythmias are increased during pregnancy with WPW. The suspected triggering factors are pregnancy related increases in plasma volume, hypovolaemia resulted from peripartum hemorrhage or prolonged labour, increased sensitivity to adrenergic stimuli by oestrogens, stress and anxiety (4).

Unknown WPW syndrome only with a retrograde, but not an anterograde-conducting accessory pathway, related with as many as 30% of cases of accessory pathway. Unknown WPW syndrome is concealed because it has a different course from classical WPW syndrome. A lot of studies have been conducted to patients with known WPW syndrome during anaesthesia. Incidence of tachyarrhythmia connected with anaesthesia is as high as 28% (5). Sympathetic blockade due to regional anaesthesia causes a prompt decrease in atrial filling pressure and this process results in increased dysrhythmia (4). There is limited experience of control of SVT during regional anaesthesia. In this case we experienced a hypotensive period and ventricular arrhythmia following spinal anaesthesia like other previously reported cases (2,4).

It is well known that autonomic, hemodynamic and hormonal changes may predispose to arrhythmias in pregnancy; however treatment should be planned for those with haemodynamic outcomes. A multitude of vasoactive agents has been used for the acute management of SVT in WPW in pregnancy. The optimal agent choice depends on both patients clinical course and anaesthetic choice which will be used for labour. In the past few decades pharmacological treatment choices include digoxin,  $\beta$ -agonists (methoxamine and phenylephrine),  $\beta$ -antagonists (propranolol and more recently esmolol), cholinomimetic agents (neostigmine), calcium channel blockers (verapamil), and the rapid-acting vasodilator adenosine (6,7).

Hormonal changes especially abrupt changes in serum levels of estrogens are thought to be responsible in the development of arrhythmias during pregnancy. Although the blood levels and the metabolism of catecholamines do not change significantly during pregnancy (8). Estrogens increase the excitability of uterine muscle fiber. However, some authors have concluded that estrogens increase both the number of  $\beta$ -adrenergic receptors and adrenergic sensitivity in susceptible tissues (myometrium, platelet) (9,10). Additionally  $\alpha$  adrenergic receptors in hypothalamus are increased. Therefore someone can postulate that the increased adrenergic sensitivity of hormone receptors and increased number of neurotransmitters in various tissues may potentially play a role in the genesis of arrhythmias by changing the refractory periods and conduction velocity in the reentrant circuit (11). These changes lead to various degrees of stress and anxiety which can result in arrhythmogenic effects due to stimulated sympathetic nervous system (12).

In conclusion, in the pregnant population mostly due to hormonal and other either physiological or non physiological factors it seems that there is an increased possibility for supraventricular tachyarrhythmias. Therefore there is an increasing need for further

prospective randomized studies clearly document this increased arrhythmia susceptibility during pregnancy and the preferred therapeutic strategies in these patients.

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