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Case Report

Wolff-Parkinson-White syndrome in pregnancy: a case report

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ABSTRACT

Wolff-Parkinson-White (WPW) syndrome is a rare congenital cardiac pre-ex citation syndrome encountered during pregnancy. However, WPW under this physiological process might be responsible for life threatening arrthymias leading to dangerous outcomes for both mother and the fetus. It is a ventricular pre-excitation syndrome of anterograde activation of the ventricle resulting from aberrant conduction pathway as well as the AV node. Very small percent of patients with WPW syndrome that is less than 1% are at risk of sudden cardiac death. Most of the antiarrthymic drugs are a threat to the fetus therefore careful choice of drug has to be made. Digoxin is the drug that is contraindicated in adults with WPW syndrome and some drugs like verapamil increase danger of ventricular fibrillation and therefore use of drugs are a topic of concern. We present a case of 25-year-old primi gravida 31 weeks 5 days woman was admitted with oligohydramnios. Patient known case of WPW syndrome not on treatment showing ECG changes of short PR interval and delta wave slurring along the QRS complex with heart rate of 98 beats/ min and echo demonstrated mild tricuspid regurgitation (TR) and mild pulmonary arterial hypertension (PAH). Patient was taken for emergency LSCS in view of Doppler changes (CPR less than 1) and delivered an alive preterm baby.

Keywords: WPW, Tachyarrthymias, Antiarrthymic drugs

INTRODUCTION

In the early 1900, Frank Wilson and Alfred Wedd explained first ECG patterns that later got the name of WPW pattern. In 1930, Wolff-Parkinson and White described the classical pre-excitation syndrome based on a case series of 11 patients who underwent paroxysmal tachycardia associated with ECG changes of sinus rhythm with short PR interval with wide QRS complex.[3] The electrocardiographic changes of preexcitation were first seen in correlation with an anatomic proof of an anomalous conducting tissue or bypass tracts in 1943. Characteristic ECG changes, history of palpitations, dyspnea, anxiety, angina pain and fatigue help in the diagnosis of WPW syndrome.4 The general prevalence of WPW has been estimated to be between 0.1 to 0.3% of population and the incidence of patient that progresses to arrthymias is around 1 to 2% per year. The risk of sudden death is rare but less than 1% but is real. WPW can serve as a damage to both maternal and fetal prognosis. Appropriate maternal diagnosis with proper management are important to enhance maternal and fetal outcomes.

CASE REPORT

A 25-year-old primigravida woman (31 weeks 5 days) was admitted with oligohydramnios. Paitent known case of WPW syndrome diagnosed 6 months back with ECG changes of short PR interval and delta wave slurring along the QRS complex and patient not on treatment and echo report with mild TR, Mild PAH. She had complaints of burning micturition on and off for 3 days at the time of admission. Patient had fever on and off for 3 days. She didn't have abdominal pain, bleeding or leaking PV. Perceiving fetal movements well. Paitent general examination showed satisfactory general condition: afebrile, heart rate of 88 bpm, regular rhythm, blood pressure of 120/70 mmHg, S1 and S2 heard, Clear chest, Abdomen was, approximately 30 weeks size, fetal parts

felt, fetal heart rate localized with 145 bpm.

Interval growth scan was done and it was corresponding to dates with placenta being anterior, liquor reduced, with normal doppler study. Urine culture was sent. Patient was started on IV fluids and L arginine sachet and astymin forte in view of oligohydramnios.

Soon after the admission ECG and echo was taken. ECG showed changes of short PR interval and delta wave slurring across the QRS complex with heart rate of 98 beats/ min. Echo was done showing mild TR and mild PAH. Cardiology opinion was obtained with stable cardiac status and thus advised planning surgery under mild cardiac risk. After 2 days of admission repeat antenatal growth scan with Doppler was done showing CPR ratio less than 1 and was planned for emergency ISCS in view of severe oligohydramnios and doppler changes. Patient delivered a live preterm baby boy with 1.4 kg and Apgar 8\10 and 9\10. Baby was kept in NICU for 1 month. Postoperatively patient vitals were monitored and were stable with BP-120/80 mmHg, PR-78/min, cardiology opinion was obtained and no cardiac intervention was advised. Postnatally urine culture showed growth showing *Klebsiella pneumoniae*, which was sensitive to linezolid and she was started on tab linezolid 600 mg BD. Patient with stable vitals was discharged after one month as the baby was admitted for weight gain.

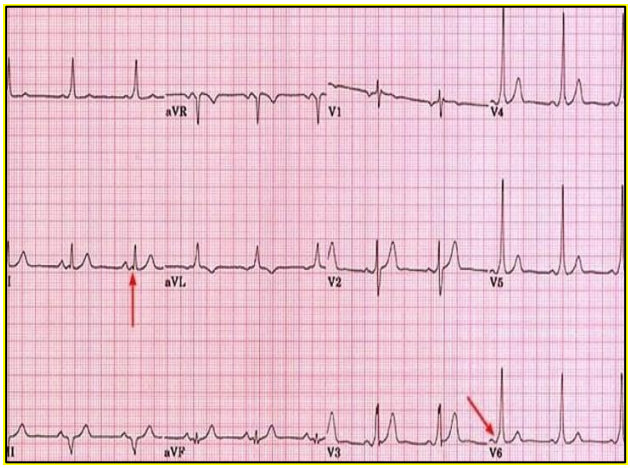

Figure 1: ECG report.

DISCUSSION

Wolf Parkinson's white syndrome was first explained by Kent in 1893 and is now known for more than a century. It explains how the normal conduction pathway is bypassed due to the presence of anterograde accessory pathway. Thus the presence of this muscular connection system of heart predispose patients to ventricular preexcitation resulting in widening of initial part of QRS complex (delta wave),this results in creation of re entry circuit between the normal and accessory pathway which leads to antidromic or orthodromic atrioventricular nodal reentrant tachycardia resulting to recurrent paroxysmal episodes of supraventricular tachycardia.[6] In pregnant patients with WPW syndrome, atrioventricular reciprocating tachycardia can lead to hemodynamic compromise that needs immediate treatment.7 The general incidence of WPW syndrome is 0.1-0.3% and the risk of sudden death due to a malignant arrhythmia is around 1% to 2% per year in these patients.[8] This syndrome may result in malignant arrthymias which might result in sudden death. Symptomatic patients have an estimated risk reported to be approx. 0.2% per year or 3 to 4% over lifetime. The exact incidence of WPW syndrome during pregnancy is not known; however, some reports have indicated that pregnancy may facilitate the onset of tachyarrhythmias in patients with previous asymptomatic pre-excitation.9,[10] Several mechanisms are proposed to explain this increased incidence of tachyarrhythmias during pregnancy in the presence of WPW syndrome. Maternal blood volume rises by an average of 40% above non-pregnant levels. The high plasma catecholamine concentrations, increased adrenergic receptor sensitivity and high-end diastolic volumes associated with pregnancy all increase the chances of arrthymias. An increase in heart rate in those patients may induce unidirectional block in the re-entrant pathway and trigger reciprocating atrioventricular tachycardia.[11] Adding on to these, stress, anxiety, and fear about fetal health and integrity may activate the sympathetic nervous system with potential arrhythmogenic effect.[12] Half of the patients are asymptomatic before pregnancy. But pregnancy exposes them to the risk of developing palpitations, dyspnea, dizziness. During the course of pregnancy importance should be given to any sort of new onset symptoms such as palpitations, shortness of breath, syncope seizure dizziness confusion and excessive intolerance. As it is difficult to difference between normal common pregnancy symptoms and small degree of cardiac problem that needs further evaluation.

Easiest diagnosis can be made through electrocardiogram. Treatment choice depends on tolerance of arrthymias, presence of underlying heart disease, effect of antiarrthymic drug. Tachycardias in WPW can be responsible for hemodynamic instability with danger of sudden death for both baby and mother.8

Pre-conceptional counselling is done for patients to discuss regarding the fetal risks for cardiac anomalies. The risk for cardiac anomalies is 2 to 5 times higher if the mother is affected. Pre-conceptional counselling allows for proper maternal cardiac evaluation, to optimise her condition, modify medications for treatment in pregnancy, and deal with the other comorbidities that may have a effect on pregnancy.[9]

However certain measures such as lifestyle, dietary modifications and advice on left lateral decubitus should be considered. Decision regarding the mode of delivery depends mainly on obstetric indications or the associated comorbidities.[14,15]

Mostly all antiarrthymic drug should be considered fetotoxic and one should asses the risk benefit ratio before starting treatment. It is better if AAD are avoided during the first trimester of pregnancy.

Adenosine and verapamil both can prolong refractory period of AV node and successfully terminate 90%of acute attacks.

In hemodynamically stable patients' vagal manoeuvre can be tried if failed, intravenous adenosine can be given. Adenosine has good efficacy and safety profile which has been shown by a number of studies, but needs close monitoring of fetal cardiac activity due to the risk of bradycardia.[9]

On verapamil there is limited data, but teratogenic or maternal side effects are not noted.

If this also unsuccessful then intravenous flecanide can be used to terminate tachycardia.[10]

In any stage of pregnancy electric shock is recommended in case of hemodynamic instability or sustained supraventricular arrthymia.[12] But fetal monitoring is compulsory though electric cardioversion is safe.If cardioversion is postponed due to some reason then drug therapy should be used for 24 hours to see whether the drug therapy is effective. But this just increases the duration of maternal tachycardia and adverse effects of AAD.

Prophylactic treatment for arrthymia includes beta-blockers, flecanide in case of intolerance or uncontrollable symptoms. Catheter ablation is indicated only in highly selected cases or uncontrolled and poorly tolerated arrhythmia.[16,17] The ablation should be performed in an experienced centre. It is preferable to postpone the ablation to the second trimester after the organogenesis is completed. Dose for radiation less than 50 mGy, there is no evidence of risk of congenital malformation or abortion, nevertheless fluoroscopy should be as brief as possible with use of a protective X-ray shield on the abdomen to minimize the risk of foetal radiation, or use a zero-fluoroscopy technique as better alternative to conventional fluoroscopy. It is advised to use the electro-anatomical mapping system which is reported to significantly decrease the overall radiation dose of the ablation procedure.[17]

Our patient had showed mild TR and mild PAH and cardiology opinion was obtained. LSCS was done under mild cardiac risk for obstetric indication. Patients delivered a live preterm boy baby with 1.4 kg and Apgar 8\10 and 9\10. Baby was kept in NICU for 1 month for weight gain. Postnatally cardiology opinion was obtained for our patient and no cardiac intervention was needed. Though our patient did not need any intervention but patients who are symptomatic or might later show onset of tachyarrthymias needs multidisciplinary approach.

CONCLUSION

WPW syndrome in pregnancy is considered serious and a rare condition that should be evaluated and diagnosis should not be missed as it can be life-threatening to both the fetus and the mother, so close monitoring should be done. Paitents who are asymptomatic or mild symptoms with structurally normal heart can be reassured and antiarrthymics are reserved for intolerable symptoms. The aim of treatment is to terminate complex arrthymias, prevent recurrence and control the ventricular pace. Regarding the use of correct antiarrthymics that should be based on paitent induvial characteristics, type of arrthymias and properties of medications. Direct current cardio version is acceptable in all stages of pregnancy treatment with proper fetal monitoring. The severity of WPW syndrome during pregnancy needs accurate diagnosis, adequate management on the basis of multidisciplinary coordination between obstetricians, cardiologists, and neonatologists.

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