A CASE OF PAROXYSMAL VENTRICULAR TACHYCARDIA DURING PREGNANCY

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We report a case of a 37-year-old woman who had paroxysmal ventricular tachycardia (VT) during early pregnancy. She had severe hyperemesis, palpitation at 6 weeks of gestation and many episodes of paroxysmal VT, but no apparent organic heart disease. At that time she had a transient increase of thyroid hormone levels. With bed rest and without medication, her symptoms and episodes of VT disappeared in accordance with the improvement of hyperemesis and thyrotoxicosis. She demonstrated a rare course of arrhythmias in which the deterioration of VT was observed at transient thyrotoxicosis and hyperemesis.

The management of pregnant women with serious cardiac arrhythmias is an important problem concerning the matter of the continuation of pregnancy and the medication. However, there are few reports regarding the course of arrhythmias during pregnancy. In this report we describe a pregnant woman whose paroxysmal ventricular arrhythmia became worse during thyrotoxicosis with severe hyperemesis.

CASE REPORT

A 37-year-old primagravida woman visited the obstetrics and gynecology clinic complaining of severe hyperemesis and palpitation on November 6, 1986, at 6 weeks of gestation. She was admitted to our hospital 2 weeks later because of her ventricular arrhythmia. There was no past history of rheumatic fever, myocarditis or congestive heart failure. Family history revealed negative for a sudden cardiac death.

Physical examination revealed blood pressure of 100/60 mmHg and an irregular pulse rate of 90 beats/min without heart murmur or click. A small thyroid goiter was palpated. The electrocardiogram revealed a structurally normal heart with good wall motion. There were no findings of mitral valve prolapse. An electrocardiogram showed a pattern of paroxysmal ventricular tachycardia (VT), characterized by wide QRS complexes with left bundle branch block morphology and atrioventricular dissociation (Fig. 1); at sinus rhythm there were no significant abnormalities in QRS complex or ST-T segment. Ambulatory ECG monitoring revealed runs of paroxysmal VT (825 episodes/day) and frequent ventricular premature complexes (VPCs) (15, 615/day). The longest run of VT (48 seconds) contained 178 beats at a rate of 225 beats/min (Fig. 2). Episodes of paroxysmal VT were often seen during mild to moderate daily physical activities. The patient did not complain of palpitation, faintness or syncope during the episode of VT, but she noticed palpitation during bigeminy or trigeminy cycles. The electrolytes test revealed sodium of 137 mEq/L, potassium of 3.6 mEq/L and chloride of 106 mEq/L. The thyroid function tests revealed T₃ of 140

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Fig. 1. 12-Lead electrocardiogram showing paroxysmal ventricular tachycardia with left bundle branch block morphology.

In accordance with the gradual improvement of hyperemesis, thyroid hormone levels decreased and became normal at 13 weeks of gestation. Coincidentally the complaint of palpitation subsided and ambulatory ECG monitorings performed at 17 and 20 weeks of gestation revealed no VT with several VPCs (760/day and 1,293/day, respectively). She was discharged from the hospital at 21 weeks of gestation and then had careful follow up at the outpatient clinic. She was admitted to the Department of Obstetrics and Gynecology for delivery at 38 weeks of gestation. Ambulatory ECG monitoring 2 weeks before delivery revealed no VT with several VPCs (1,469/day). At 40 weeks of gestation, she had

a normal spontaneous delivery of a 3,247 g female whose Apgar score was 8. ECG monitoring and ambulatory ECG monitoring revealed no episodes of paroxysmal VT during labor; however, runs of paroxysmal VT were observed several seconds after delivery and during ligation of perineal laceration, although there was no significant bleeding (Fig. 3).

After delivery she was treated with 300 mg daily doses of mexiletine with resolution of the symptoms. Thyroid function tests performed 1 and 6 months after the delivery were within normal range. Ambulatory ECG monitoring performed 3 months after delivery under mexiletine therapy revealed several episodes of VT of only 3 beats each. She has no symptoms in daily life. Total follow-up period is 24 months.

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DISCUSSION

Since McMillan and Ballet\(^2\) reported a case of a pregnant woman with VT in 1931, there have been seven\(^2-^8\) published reports involving 15 cases of VT during pregnancy. Excluding 3 cases whose clinical courses were unclear, of the 12 remaining cases, one had more VT in the first trimester, 9 cases had more VT in the second and 2 cases had more in the third trimester. The arrhythmia became worse with progression of the pregnancy in 2 cases. Drugs therapy was
administered in 7 cases. Such drugs as quinidine, procaaineamide, disopyramide, lidocaine and metoprolol were used successfully without causing deleterious effects to either mother or fetus. In these reports, the course of the VT varied in each case and there was no clear pattern. No case of transient deterioration of VT during severe hyperemesis has appeared in the literature.

It has been reported that VPC or VT with left bundle branch block morphology in patients without organic heart disease implies a benign prognosis. The following factors are reported as inducing idiopathic VT: emotional upsets, fear, exercise, excessive coffee consumption, heavy smoking, alcohol, trauma, changes of posture, hypokalemia, imbalance in the autonomic nervous system, etc. Female patients require caution because their ventricular arrhythmias might be worsened during pregnancy. We followed our patient carefully without medication and monitored the arrhythmia strictly by repeated ambulatory ECG monitoring. We determined that it was possible to continue her pregnancy because of the absence of organic heart disease, the short duration of her VT, little hemodynamic effect during VT and the improvement of arrhythmia at the end of first trimester. She had an uneventful pregnancy and had a safe spontaneous delivery.

It has been well documented that hyperthyroidism occurs during hyperemesis gravidarum. Basedow's disease was not relevant to our case because the thyrotoxicosis was seen only during severe hyperemesis, and because there was no increase in activity either of TBI or TSH in the clinical course. We considered it likely that the thyrotoxicosis was caused by severe hyperemesis. Although thyrotoxicosis has been recognized as a major cause of atrial fibrillation, ventricular arrhythmias have rarely been associated with thyrotoxicosis. In the hyperthyroid state, however, besides the direct effects of thyroid hormone on the heart, enhanced sensitivity to the cardiac effects of catecholamines occurs. Fairhurst reported a single case of ventricular tachycardia associated with thyrotoxicosis in which the author believed that the thyroid hormone increased the sensitivity of the cardiac muscle to adrenalin. In our case, thyrotoxicosis might have had some role in the worsening of the ventricular arrhythmia, probably through the mechanism of catecholamine sensitivity.

No episodes of paroxysmal VT occurred during labor, but they were observed just after delivery. According to the literature, sudden hemodynamic changes have been observed just after delivery. Considering the fact that no episode of VT was observed during labor, the hemodynamic changes might be the cause of the post-delivery episode of VT in our case. We presented this case because our patient demonstrated a rare course of arrhythmia in which transient deterioration of VT was observed during thyrotoxicosis and severe hyperemesis.

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