INTRODUCTION

Transient right ventricular hypertrophy (RVH) is a rare anomaly that is seen during the neonatal period (1-2). Metabolic diseases, maternal diabetes, non-steroidal anti-inflammatory drug (NSAID) treatment during pregnancy, and dexamethasone therapy in premature newborns for bronchopulmonary dysplasia may cause transient RVH in the newborn period (1-4). The peculiarity of our case is the complete echocardiographic regression of right ventricular hypertrophy and the lack of relationship between transient RVH and any secondary causes described above.

We have reported this case because of the rarity of transient RVH and we wanted to attract attention about the importance of pregnant nutrition.

CASE REPORT

A female neonate was born at 39 weeks gestation by cesarean section with general anesthesia because of perinatal distress. Maternal exposure to indomethacin or other NSAI medications and gestational diabetes were not noted during pregnancy. In addition, there was no family history of hypertrophic cardiomyopathy. Birth weight was 3300 g with high Apgar scores. Oxygen saturation as well as pulmonary and cardiac findings (except sinus tachycardia) were all normal. An electrocardiogram revealed sinus tachycardia and RVH. Chest radiogram was normal. Echocardiographic investigation which was performed on third day of life, showed severe RVH (wall thickness of 11 mm in diastole) with a small right ventricular cavity (1.85 cm² in diastole). Myocardial hypertrophy was seen at the anterior wall.
Transient Isolated Right Ventricular Hypertrophy in a Neonate

and apex of the RV and also at right side of the interventricular septum (Figure 1). Color Doppler echocardiography revealed only mild tricuspid regurgitation with a normal right ventricular pressure (~25 mmHg). There was no blood flow through ductus arteriosus (DA), so it was diagnosed as closed. The remainder echocardiographic findings were all normal. Complete regression of the right ventricular hypertrophy and normalization of the right ventricular cavity were demonstrated on the tenth week after birth (Figure 2).

**DISCUSSION**

Some congenital heart anomalies, such as pulmonary stenosis, tetralogy of Fallot or truncus arteriosus, may cause RVH. Echocardiographic examination was normal for these anomalies in our infant. Other causes of RVH include metabolic and heredofamilial disease, maternal diabetes and premature closure of DA (1,2,4-7). Also, we could not explain RVH with metabolic or heredofamilial disease because of the transient nature of RVH and the lack of familial history about metabolic and heredofamilial diseases. It is well known that maternal diabetes mellitus may cause transient RVH, but blood sugar analyses were within normal ranges during her pregnancy. Transient RVH in neonates may result from remodeling of pulmonary vasculature secondary to acute perinatal distress (8). Our case was also born with cesarean section because of perinatal distress. The reason of idiopathic or spontaneous intrauterine closure of the DA is not known exactly (9,10). Intrauterine closure of the human DA has been reported following maternal administration of the prostaglandin synthetase inhibitors such as aspirin, glucocorticoids and NSAIDs (5,11-14). There was no history of such medications during pregnancy in our case.

On the third day echocardiographic examination, DA was diagnosed as closed in our patient. We really do not know whether the closure of DA was in utero or after birth. Recently, clinical and experimental evidence showed that maternal consumption of polyphenol-rich substances (PRFs), such as

![Figure 1](image1.png)  
**Figure 1:** A subcostal four-chamber view on the third day of life revealed severe isolated RVH with reduced right ventricular cavity while the left ventricle was normal. **RV:** right ventricle, **LV:** left ventricle.

![Figure 2](image2.png)  
**Figure 2:** The comparable subcostal four-chamber view at ten weeks of life showed complete regression of the RVH. **RV:** right ventricle, **LV:** left ventricle.
herbal teas, orange and grape juice, chocolate, and others, may interfere with fetal DA dynamics and may cause fetal ductal constriction (15,16). Also, we found that a large amount of herbal tea rich in PFRs was consumed during the last 3 months of her pregnancy. The need to limit ingestion of foods with high concentrations of PRFs to avoid fetal ductal constriction in the third trimester of pregnancy is well known (15).

It is well known that maternal consumption of PRFs during pregnancy may cause fetal ductal constriction (15,16). Although we were not sure whether the ductal closure had occurred in utero or after birth, probably ductal narrowing had taken place during the third trimester in our case owing to consumption of a large amount of herbal tea as a kind of PFR. We wanted to emphasize the importance of nutrition during pregnancy with our case with transient RVH.

REFERENCES