



Impact of Fetal Weight in Assessment of Fetal Cardiac Output in Three Cases of High Output Cardiac Failure

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ABSTRACT

Introduction Many different Doppler-derived techniques have been proposed for overall assessment of cardiovascular well-being. In this report, 3 cases of high output cardiac failure were presented with the aim of evaluating the effect of fetal weight on fetal combined cardiac output (CCO).

Patients Information The first and second cases were referred to Yas Hospital for the management of fetal anemia at 31 and 29 weeks of gestation, respectively, and the third case had a large solid and vascular sacrococcygeal teratoma at 17 weeks. The fetal weight in the first case was less than 10 percentile, in the second case was in normal limit, and in the third one was above 90 percentile. Left ventricle, right ventricle, CCO, and weight-indexed CCO were assessed for both fetuses and all were above normal limit, indicating high output cardiac failure. Right to left cardiac output ratio also increased obviously.

Conclusion The fetal growth restriction overestimates the weight-indexed CCO and the weight above 90 percentile underestimates it. In these conditions, CCO seems more precise in demonstrating the true burden on fetal heart in comparison with weight-indexed CCO.

Keywords Cardiac Output; Teratoma; Fetal Anemia; High Output; Fetal Weight

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[1] Fetal cardiac function: Technical considerations and potential research and clinical applications [2] Techniques for assessing cardiac output and fetal cardiac function [3] Fetal cardiac output and its distribution to the placenta at 11-20 weeks of gestation [4] Diagnosis and management of heart failure in the fetus [5] High-output cardiac failure in fetuses with large sacrococcygeal teratoma: Diagnosis by echocardiography and Doppler ultrasound [6] Prenatal diagnosis of concurrent facial and cerebral vascular malformation which caused congestive heart failure [7] Fetal cardiac output, distribution to the placenta and impact of placental compromise [8] Cardiac output and central distribution of blood flow in the human fetus [9] Fetal cardiac output estimated by Doppler echocardiography during mid- and late gestation

Introduction

Heart assessment was initially used to detect structural abnormalities in fetus; but, it has recently been employed for the evaluation of cardiac function and its physiology [1, 2]. Many different Doppler-derived techniques have been proposed for overall assessment of cardiovascular well-being. One of them is the calculation of fetal combined cardiac output (CCO) [3].

So, a high output cardiac failure antenatally can be a result of many different situations such as Sacrococcygeal teratomas, pump twin in the twin-reversed arterial perfusion, fetal anemia, and arteriovenous malformation [4-6].

The CCO can be measured as ml/min or weight-indexed CCO as ml/min/kg. The first one gradually increased, but the second one was nearly stable during the entire second half of the normal pregnancy (400ml/min/kg) [7].

The aim of the present case report was to evaluate the effect of fetal weight effect on fetal combined cardiac output.

Patients Information

In this report, 3 cases of high output cardiac failure were represented. The informed consent was given to 3 cases and the study protocol was approved by our institute's ethics committee on human research.

Case 1: A 39-year-old gravida 4, para 2, female was referred to Yas Hospital for the management of fetal anemia by intrauterine transfusion (IUT) at 31 weeks of gestation according to the first-trimester ultrasound exam. Maternal red blood cell alloimmunization resulted in fetal anemia. The ultrasound (US) examination, using transabdominal convex array transducer with 2-6MHz frequency was performed in our center by Philips ultrasound machine (KPI, USA). The estimated fetal weight (EFW) was about 1330g, which was in favor of fetal growth restriction (FGR) and less than 10 percentile for gestational age. Cardiomegaly, mild tricuspid regurgitation and mild pericardial effusion were noted without any evidences of hydrops fetalis. The heart was structurally normal. Doppler studies showed a high peak systolic velocity (PSV) in the middle cerebral artery (MCA) of 76cm/s more than 1.5 MOM, suggestive of anemia.

Doppler velocity waveforms were obtained from the aorta and pulmonary artery just after the valve level (Figure 1). The left ventricle cardiac output (LVCO) and right ventricle cardiac output (RVCO) were calculated separately. The LVCO was 396ml/min and 297ml/kg/min and RVCO was 1309ml/min and 984ml/kg/min (the right to left CO ratio was 3.3). The CCO was 1700ml/min and 1280ml/kg/min. It has obviously increased in favor of high output cardiac failure.

IUT was performed during the following days when the two subsequent IUTs were administered at 32

and 33 weeks of gestation and the hemoglobin reached 13g/dl. Then, the mother was discharged from the hospital. A male newborn was delivered at 35 weeks due to premature rupture of membrane and received no other blood transfusion after birth. In follow-up echocardiography on the 7th day, only mild cardiomegaly and mild tricuspid regurgitation were identified with normal cardiac function.

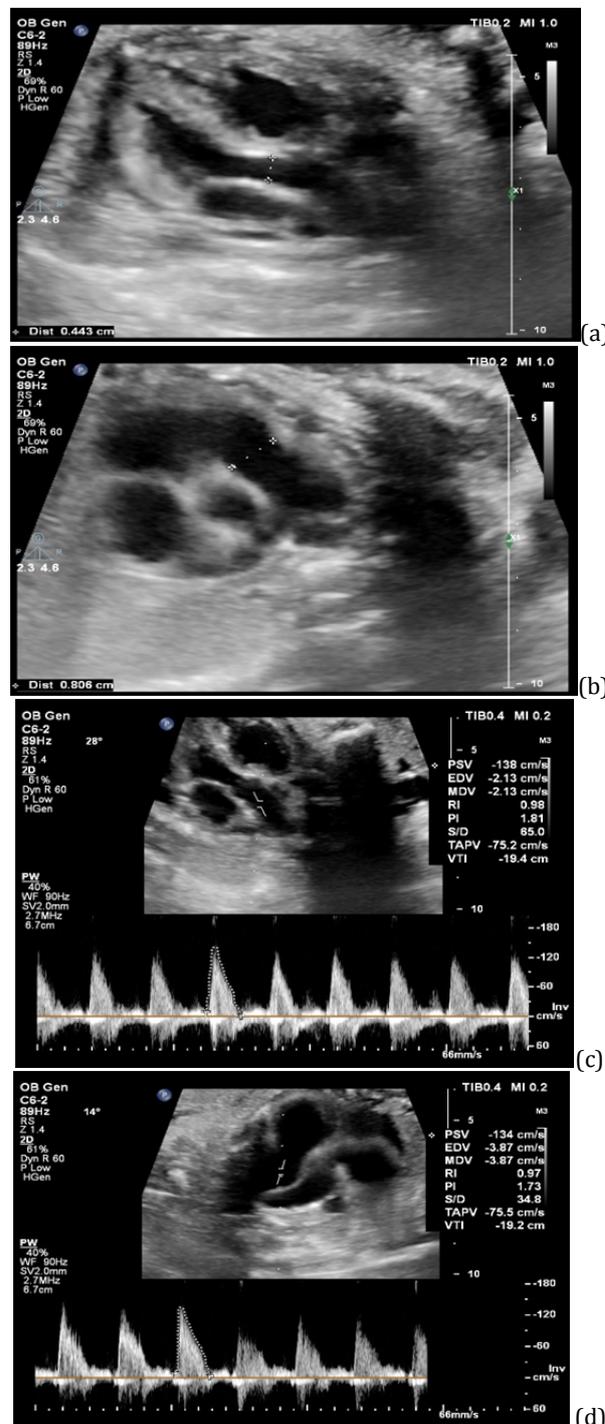


Figure 1) A 39-year-old female was referred at 31 weeks of gestation for management of fetal anemia by intrauterine transfusion. Diameter measurement (a, b) and Doppler study (c, d) at the level of the aortic Valve (a, c) and at the pulmonary Valve (b, d)

Case 2: A 30-year-old gravida 2, para 1, female was referred to radiology department for the management of fetal anemia (with unknown cause) by IUT at 29 weeks of gestation. The US examination in our center revealed severe cardiomegaly, mild pericardial effusion, and mild tricuspid regurgitation. The heart was structurally normal. The fetal EFW was in a normal limit for gestational age. Doppler study of MCA was suggestive of severe anemia.

Doppler study was performed with the same protocol of the first case. The LVCO and RVCO were calculated separately as 207ml/min (177ml/kg/min) and 486ml/min (415ml/kg/min) with the right to left CO ratio of 2.3. The CCO was about 693ml/min and 592ml/kg/min and mildly increased in favor of high output cardiac failure.

IUT was performed in the following day. Unfortunately, the fetus died after a few days.

Case 3: A 26-year-old gravida 1, para 0, woman was referred to Yas Hospital at 17 weeks of gestation with the diagnosis of a large solid mass in the sacral region typical for sacrococcygeal teratoma. The personal and familial history of the parents were unremarkable. A large 97×70mm solid vascular mass in sacral spine with an intra-pelvic extension in a female fetus was revealed in our center (Type 2 according to the Altman classification). Evidence of obvious hepatomegaly and mild ascites was noted, which resulted in vivid increase in abdominal circumference (around 20 weeks+ 3 days) and also fetal weight (234g, more than 95 percentile). Bilateral plural effusion and club feet were other findings.

In echocardiographic evaluation, severe cardiomegaly and moderate tricuspid regurgitation were detected in a structurally normal heart. The LVCO and RVCO were calculated separately as 51ml/min (220ml/kg/min) and as 115ml/min (494ml/kg/min) with the right to left CO ratio of 2.8. The CCO was about 166ml/min and 714ml/kg/min, which had increased.

After detailed discussion with parents, they chose the termination of pregnancy due to large tumor size, heart failure, and hydrops in such early weeks.

Discussion

A variety of volume loading conditions have been used to determine CCO. In fetal anemia, cardiac output will be increased in order to maintain adequate tissue oxygenation. Since adaptation mechanisms can no longer be compensated, they will inevitably be ended in tissue ischemia and diminished function and, then, a dilated cardiomyopathy. The highly vascularized sacrococcygeal teratomas can lead to an increased circulating blood volume and preload returning to the heart will be resulted in high output cardiac failure in fetus [4, 5].

The LVCO, RVCO, and CCO all increased with advancing gestational age. A right ventricular dominance has consistently been shown in the second half of fatal period. About 60-65% of CCO was ejected by the right ventricle with a ratio of RVCO to LVCO of 1.5 to 1.85 [8, 3]. In our cases, this ratio has vividly increased as 3.3 in the first case, 2.3 in the second case, and 2.8 in the third one.

It has been shown that fetuses with combined cardiac outputs more than 800ml/kg/min are at the greatest risk of developing hydrops fetalis [4]. In our second case, weight-indexed CCO was 529ml/min/kg and the presence of severe hydrops and fetus outcome may indicate that the fetus had higher cardiac output before, but now reach a decompensated state.

Some studies assessed and reported the CCO during pregnancy. Vimpeli *et al.* have evaluated CCO at 11–20 weeks of gestation and found a mean of 278ml/min/kg in their study for an estimated fetal weight of 51–296g. In their study, both CCO and weight-indexed CCO gradually increased over time, but the first one had steeper curve [3].

Kiserud *et al.* found an average weight-indexed CCO about 400ml/min/kg during the entire second half of the normal pregnancy. But, the mean fetal CCO increased over time and it was 80ml/min at 18 weeks and 1370ml/min at 40 weeks [7]. Similar results were achieved by Mielke *et al.* and De Smedt *et al.*, showing nearly constant weight-indexed CCO during about 15-40 weeks of gestational age (425ml/min/kg and 553ml/min/kg, respectively) [8, 9].

Our first case had CCO of 1700ml/min and 1280ml/kg/min, which obviously increased in favor of high output state. The fetal weight was less than 10 percentile. We compared the CCO of this fetus with Kiserud *et al.* nomograms and the CCO was 2.8 times above the value of 50 percentile for gestational age and weight-indexed CCO was 3.2 times higher than that. On the other hand, our third case weight was above 90 percentile and had a CCO of 166ml/min and 714ml/kg/min over the normal range (high output state). In comparison with the Vimpeli *et al.* charts, the CCO was 2.7 times higher than the value of 50 percentile for gestational age, and weight-indexed CCO was 2.3 times higher. Also, if we include the weight of sacrococcygeal teratoma, the weight-indexed CCO is even lower. On the other hand, in normal weight second fetus, both CCO and weight-indexed CCO were about 1.4 times higher than the normal value. These findings may show when the fetal weight is less than 10 percentile or above 90 percentile, weight-indexed CCO overestimates or underestimates, respectively, the true burden on fetal heart.

In a study conducted by Schmidt *et al.*, they calculated only weight-indexed CCO in 3 cases of sacrococcygeal teratoma with high output cardiac

failure. The weight of their fetuses was in normal range for the gestational age. In another study carried out by Kiserud *et al.*, fetuses with FGR had lower CCO compared to the normal group, but the weight-indexed CCO was not different [5, 7].

In conclusion, FGR state or weight above 90 percentile is not uncommon among these fetuses. Although using weight-indexed CCO is much easier in routine practice, when the fetal weight is not between 10 and 90 percentile, the CCO will be more accurate compared to the weight-indexed CCO in showing the true burden on fetal heart. However, this idea needs more attention and investigations to be confirmed thoroughly.

The main limitation of this study is the limited number of cases and research on multiple cases with similar situation as fetal weight of less than 10 percentile or above 90 percentile is needed.

Conclusion

The fetal growth restriction overestimates the weight-indexed CCO and the weight above 90 percentile underestimates it. In these conditions, CCO seems more precise in demonstrating the true burden on fetal heart in comparison with weight-indexed CCO.

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