Correlation between Fetal Renal Volume and Renal Artery Doppler in Normal and Growth Restricted Fetuses

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By

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List of Abbreviations

Abb.	Mean
2D US	2 dimensional ultrasound
3D US	3 dimensional ultrasound
AC	Abdominal circumference
AFI	Amniotic fluid index
AGA	Appropriate for gestational age
BMI	Body mass index
BMUS	British Medical Ultrasound Society
BPD	Biparietal diameter
DV	Ductus venosus
EDV	End-diastolic volume
EFW	Estimated fetal weight
FGR	Fetal growth restriction
FL	Femur length
GA	Gestational age
GI	Gastrointestinal
НС	Head circumference
IUGR	Intrauterine growth restriction
LMP	Last menstrual period
LRV	Left renal volume
MCA	Middle cerebral artery
MVP	Maximum vertical pocket
PI	Pulsatility index
PO_2	Partial pressure of oxygen
PSV	Peak systolic volume
Pw Doppler	Pulsed wave Doppler

Abb.	Mean
RI	Resistive index
RRV	Right renal volume
S/D	Systolic / diastolic
SD	Standard deviation
SGA	Small for gestational age
TRV	Total renal volume
UA	Umbilical artery
UV	Umbilical vein
VOCAL	Virtual organ computer aided analysis

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Abstract

Background: Fetal kidney weight cannot be measured in utero; renal volume measured by U/S is a valid substitute. With the latest developments in the field of three dimensional U/S, accurate assessment of the fetal organ volume has become feasible and this technique has gained widespread application in different medical fields.

We also aimed to examine the association of renal artery, umbilical artery with fetal kidney volume.

<u>Objective</u>: Intrauterine growth restriction may lead to renal disease and hypertension in adult life. This study was performed to determine whether renal volume differs between fetuses that are intrauterine growth restricted and fetuses that are not. We also aimed to examine the association of fetal blood flow parameters with the fetal renal volume.

Design: Observational case-control study.

Setting: Special Care Centre for the fetus in Ain Shams University Maternity Hospital.

<u>Patients and methods:</u> Examinaton of 40 pregnant women between 36-40 weeks of gestation. Women was divided into 2 groups; mothers with IUGR (group A, n=20) and mothers within normal range fetal biometry i.e. control group (group B, n=20). In all participants, total renal volume was measured using 3D U/S and umbilical as well as fetal renal artery.

Doppler which was calculated using color Doppler technique. All measured parameters were compared to fetal growth stage and correlated to renal volume.

Results: The mean total fetal renal volume was 9.97 ± 0.3 cm³ in group A and 15.15 ± 0.24 cm³ in group B (p value <0.001). Estimated fetal weight were positively correlated with the total renal volume "TRV" in both groups with a statistically significant P value for both gorups. Renal artery pulsitilaty index (PI)and resistance index (RI) showed significant correlation with the TRV in both groups.

<u>Conclusion:</u> The conclusion of our study is that intrauterine growth restriction appears to be associated with a decrease in the estimated fetal weight with a significant difference in the fetal renal volume between the two groups this study supports the hypothesis that intrauterine growth restriction is linked to renal disease and hypertension in late life.

<u>Keywords</u>: (Intrauterine growth restriction-fetal renal volume-fetal renal artery, 3D U/S).

Introduction

Intrauterine growth restriction (IUGR) is defined as estimated fetal weight less than 10th centile (Unterscheider et al., 2014). It complicates up to 10% of all pregnancies (Creasy, 1999). The cause of IUGR is multifactorial and complex, including intrinsic fetal conditions as well as maternal and environmental factors (Brodsky and Christou, 2004).

Placental insufficiency is the most common cause and it is associated with raised placental blood flow resistance (Baschat and Hecher, 2004). Researchers have focused on the long term morbidity that is associated with this condition. Epidemiological studies have demonstrated low birth weight and fetal growth restriction to be risk factors contributing to renal disease and hypertension in adult life (Lackland et al., 2003). It has been hypothesized that an adverse fetal environment leads to fetal growth restriction and impaired renal growth and development, with a subsequent smaller kidneys due to reduced number of nephrons and glomerular filtration surface area. Because nephrogensis continues until 36 weeks of gestation and the induction of nephron number ceases thereafter, suboptimal

kidney growth and development in fetal life may have life long consequences (Silver et al., 2003).

In vivo studies of kidney size in human fetuses of known gestational age have shown that intrauterine growth restriction is accompanied by decreased kidney volume compared to fetuses with appropriate weight for gestational age (Verburg et al., 2007).

Under physiological conditions the fetal renal blood flow represents 2-3 % of the cardiac output because of the very high resistance in the human fetal renal artery. During the third trimester of pregnancy, fetal renal arterial resistance decreases moderately, with increase in the End-Diastolic Velocity (EDV) and mean velocity and only minor changes seen in the Peak Systolic Velocity (PSV) possibly related to the increased blood flow of the renal circulation. During hypoxemia, the renal blood flow decrease by 25–50% as compared to the baseline values, but the exact mechanism of this reduction has not been elucidated (**Arduinid and Rizo, 1990**).

A direct relationship has been reported between hypoxia and the renal artery pulsatility index (Vajda and Pal, 1994).

This condition occurs because blood flow from peripheral organs (kidneys) is diverted to the brain (Veille and Kanaan et al., 1989).

As fetal kidney weight cannot be measured in-utero, renal volume measured by ultrasound is a valid substitute (**Konje et al., 1997**). Accurate assessment of the fetal organ volume has become feasible by using three-dimensional ultrasonography. Numerous investigators have demonstrated that three-dimensional ultrasonography is superior to two-dimensional ultrasonography in fetal organ measurement (**Strommen et al., 2004**).

The main component of amniotic fluid is fetal urinary production, which may therefore be related to kidney volume and reflect kidney function (**Aditya et al.**, **2016**). An amniotic fluid index of less than 5cm increases the risk of IUGR (**Phelan et al.**, **1987**).

Provocative epidemiologic studies have suggested that IUGR is a risk factor for the development of essential hypertension and hyperlipidemia in later life (Godfery and Barker, 2000).

The actual pathways by which IUGR could lead to hypertension in adult life are unknown, but several plausible theories have been put forward (**Brenner and Chertow**, 1993).

Because no therapy at present has been shown to significantly improve placental function, the goal of prenatal testing in such cases is to optimize the timing of delivery, late enough to avoid the sequel of iatrogenic severe prematurity, yet early enough to avoid fetal death (Cohn et al., 1974).