MEGA JOURNAL OF CASE REPORTS

OPEN ACCESS

Editorial

Compiled Date: June 06, 2025

Ultrasound and Doppler Studies on Impaired Intrauterine Conditions and the Development of Future Disease

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Condensation

This editorial focuses on ultrasound and Doppler studies on intrauterine under and over nutrition effect on several fetal organs, the placenta, and in the fetal circulation.

Editorial

More than 20 years ago, based on human epidemiologic data and animal studies, Barker hypothesis [1] was introduced to the scientific and popular literature. Barker hypothesized that low birth weight subjective to poor nutrition in the intrauterine environment withparadoxically improved standards of living and nutrition after World War IIin western countries, result in later life vascular disease, such as coronary heart disease. This hypothesis led to the second part of the Barker hypothesis, the thrifty phenotype in which adaptation to under nutrition in fetal life that result in Fetal Growth Restriction (FGR), leads to permanent "programmed" metabolic and endocrine changes. Such changes may predispose the individual later in life to obesity and impaired glucose tolerance if nutrition improves, especially in over nutrition, thereby, altering the distribution of cell types, gene expression, or both. The fetus, in response to poor placental blood supply, and in order to maintain sufficient vascular supply to the brain, decreases resistance to blood flow in the middle cerebral artery. Simultaneously, the fetus develops the arterial redistribution process that is accompanied by an increased resistance to flow to

other fetal vital organs, such as heart, kidneys, liver, and pancreas. Therefore, individuals exposed to ischemic changes in utero to these vital organs develop dyslipidemia, lower nephron number, and impaired glucose tolerance, all contributing to metabolic syndrome and end organ damage later in life. However, the support for the hypothesis is weak and comes mainly from studies in rodents [2] and retrospective epidemiological studies [3]. In the last 10 years, ultrasound and Doppler studies on human FGR and its several fetal organs; the placenta, the fetal circulation, the brain, heart, kidneys, adrenal glands, liver, and pancreas, may serve as a significant support to thethrifty phenotype hypothesis. Our group presented a classic case of severe FGR and oligohydramnios, secondary to placental compromise, and arterial redistribution with decreased resistance to flow in the fetal brain (brain sparing), and as a result to probable ischemic changes to other fetal organs, the kidneys and pancreas size were less than 5% percentile for gestational age, with impaired heart function and liver shunting [4].

Our group has recently published a case series of severe FGR and oligohydramnios, in which arterial redistribution by liver shunting served as a rescue mechanism to improve fetal growth and amniotic fluid amount, these shunting resolved up to 30 months follow-up after birth [5]. Other investigators suggest a different pathway that links between intrauterine life and future disease - over nutrition in utero, especially in poor controlled women with gestational and pre-gestational diabetes [6], and too less extend, excessive weight gain during their pregnancy, may expose the fetus to over growth, Large for Gestational Age (LGA) or macrosomia. Indeed, our group found an association between increased fetal pancreatic sizes as a predictor to the later development of gestational diabetes [7]. These intrauterine conditions may lead to childhood obesity, impaired glucose tolerance and later metabolic syndrome, probably, related to the effect of enlarged pancreas that occurred in intrauterine life.

In summary, by using ultrasound and Doppler assessment, further studies are required to complete the association between the effect of the impaired intrauterine conditions, under and over nutrition and the development of pediatric, adolescence and adult disease.

Keywords: Ultrasound; Doppler; Fetal growth restriction; Macrosomia

References

1. Barker DJP. Mothers, babies and health in later life. 1998.
2. Langley-Evans SC, Gardner DS, Welham SJ. Intrauterine programming of cardiovascular disease by maternal nutritional status. Nutrition. 1998;14(1):39-47.
3. Gliksman MD. et al. Childhood socioeconomic status and risk of cardiovascular disease in middle aged US women: a prospective study. J Epidemiol Community Health. 1995;49(1):10-5.
4. Bar J, Weiner E, Levy M, Gilboa Y. The thrifty phenotype hypothesis: The association between ultrasound and Doppler studies in fetal growth restriction and the development of adult disease. Am J Obstet Gynecol MFM. 2021;3(6):100473.
5. Gilboa Y, Drukker L, Bar J, Berbing-Goldstein D, Geron Y, Mozer Glassberg Y, et al. Intrahepatic portosystemic shunt: salvage mechanism for oligohydramnios complicating fetal growth restriction. Ultrasound Obstet Gynecol. 2025;65(3):311-316.

6. Xu Y. et al. Metabolic syndrome risk after gestational diabetes: a systematic review and meta-analysis. PLoS One. 2014;9(1):e87863.
7. Gilboa Y, Geron Y, Perlman S, Drukker L, Ofir K, Ellert A, et al. Predictive capacity of fetal pancreatic circumference for gestational diabetes mellitus. Ultrasound Obstet Gynecol. 2024;64(3):348-353.

Citation of this Article

Geron Y, Gilboa Y and Bar J. Ultrasound and Doppler Studies on Impaired Intrauterine Conditions and the Development of Future Disease. Mega J Case Rep. 2025;8(6):2001-2003.

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