

Summary

The period of perinatal growth is vitally important to the child's future well-being. So, study of the nature of human life in utero, especially the growth process, has been and will continue to be among the most fascinating and rewarding in all of biological researches.

Fetal growth is a multifactorial phenomenon. It is basically the result of the relationship between the genetic drive to growth, environmental factors and the supply of substrata to the fetus. The genetic control is the dominant factor in early gestation.

General factors as age of the mother, parity, the maternal size, endurance exercise during pregnancy, smoking and social class are important factors influencing fetal growth. The endocrinological control of fetal growth is very important late in gestation. Insulin and insulin-like growth factors are the most important hormones in fetal growth. Insulin has been implicated as the primary growth hormone for intra-uterine growth and development. For example, when fetal insulin is absent, marked IUGR occurs. On the other hand, when fetal hyperinsulinism is present as a result of maternal and fetal hyperglycaemia, marked anabolic actions of insulin become manifest. There ensues an accelerated protein synthesis together with a deposition of

excessive glycogen and fat stores and the typical macrosomic infant results. Similarly, insulin-like growth factors are believed also to be growth hormone dependent and have insulin-like metabolic effects. They can promote fetal growth and cellular differentiation. Insulin-like growth factors (somatomedins) have been implicated as regulators of fetal growth and their circulating levels increase with gestational age and in normal intra-uterine growth and decrease in retarded fetal growth. Other hormones are pituitary growth hormones, human placental lactogen and fetal renal factor, which mediates normal fetal growth.

There are abnormal forms of fetal growth as intra-uterine growth retardation and macrosomia. The IUGR simply is defined as a birth weight below the 10th percentile for gestational age and sex. Most fetal growth retardation occur after the 28th week of gestation with increasing frequency toward term leading to asymmetrical or head sparing growth retardation, but if it occurs early before the 28th week of gestation, symmetrical growth retardation with both somatic and brain lag results.

Macrosomia is another form of abnormal fetal growth. Fetal macrosomia is defined as a birth weight greater than the 90th percentile for gestational age. Hyperinsulinaemia is the main cause of fetal macrosomia.

The present work aimed to determine the correlation between birth weight and the growth factors believed to be involved in fetal growth namely insulin (which is reflected by C-peptide level) and IGF-1 .

Thirty selected pregnant women and their neonates who were admitted in labour to the Galaa Maternity Hospital and the obstetrics department of Benha University Hospitals were the subjects of this study. They were divided into 3 equal groups:

The first group included 10 normally pregnant women who delivered AGA neonates (between the 10th and 90th percentile).

The second group included 10 normally pregnant women who delivered SGA neonates (below the 10th percentile) .

The third group included 10 normally pregnant women who delivered LGA neonates (above the 90th percentile) according to Lubchenco et al., (1972) .

From each subject maternal blood from cubital vein and fetal blood from umbilical cord were collected and the resulting sera were separated after centrifugation and were stored at -20°C till they were assayed for IGF-1 and C-peptide by radioimmunoassay .

Our results have yielded the following findings:

- 1- There was an increase in both maternal and fetal IGF-1 and C-peptide levels among the (LGA) group in comparison

with the (AGA) group. Meanwhile, there was a decrease in both mean maternal and fetal IGF-1 and C-peptide levels among the (SGA) group in comparison with the (AGA) group .

- 2- The differences in the maternal serum mean values of IGF-1 showed a statistically significant increase between the (LGA) and (SGA) groups ($p < 0.05$) .
- 3- The differences in the fetal serum mean values of IGF-1, showed a statistically significant increase between the (LGA) and (AGA) groups ($p < 0.05$) and between (LGA) and (SGA) groups ($p < 0.01$) respectively .
- 4- The differences in the maternal serum C-peptide mean values showed a statistically significant increase between the (LGA) and (AGA) groups ($p < 0.01$), between the (LGA) and (SGA) groups ($p < 0.001$), and between the (AGA) and (SGA) groups ($p < 0.05$) respectively .
- 5- The differences in the fetal serum C-peptide mean values showed a statistically significant increase only between (LGA) and (SGA) groups ($p < 0.05$) .
- 6- There was no significant correlation between serum maternal IGF-1 and serum fetal IGF-1 levels among the 3 studied groups. Also, there was no significant correlation, between the serum maternal IGF-1 levels and birth weight in the 3 studied groups. However, there was a highly significant

correlation between serum fetal IGF-1 levels and birth weight in the (LGA) and (AGA) groups but not in the (SGA) group.

- 7- As regards C-peptide, there was a significant correlation between serum maternal and fetal C-peptide levels in the (LGA) group ($p<0.05$), and in the SGA ($p<0.01$) .

There was no significant correlation between serum maternal C-peptide levels and birth weight in the 3 studied groups. The serum fetal C-peptide levels correlated significantly ($p<0.01$) with birth weight only in the (LGA) group .

From results of this study we can conclude that :

- 1- Insulin (as reflected by C-peptide level) and IGF-1 play an important role in the regulation of fetal weight gain .
- 2- Fetal serum IGF-1 and C-peptide concentrations are positively correlated with birth weight, among LGA newborns indicating that IGF-1 and insulin have a definite role in the pathogenesis of macrosomic fetuses .
- 3- Maternal serum levels of IGF-1 and C-peptide have no correlation with birth weight in normal pregnancy .