

THE STUDY OF PLACENTAL FUNCTION BY HUMAN PLACENTAL LACTOGEN IN NORMAL AND POST-TERM PREGNANCIES

by

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The recent advances in the management of various obstetric complications have greatly reduced the maternal mortality. Unfortunately, the perinatal mortality has failed to show a similar parallel decline. There is therefore room for research to salvage heavy foetal loss, the most important cause being prematurity. Time and again, the problem crops up regarding foetal maturity and its status in utero prior to induction of labour or elective caesarean section.

The estimation of serum Human Placental Lactogen (HPL) has recently caught the attention of obstetricians in assessing foetal maturity as well as in predicting placental function.

Human Placental Lactogen (HPL) which is a protein hormone secreted by the syncytiotrophoblast, was first isolated from the placental tissue by Josimovich and MacLaren in 1962. This hormone is secreted into the maternal circulation, is probably metabolised by the liver and excreted by the kidneys. The maternal urine contains only a trace of this hormone. (Grumbach *et al* 1968). Its concentration in the cord blood is 1% due to an effective placental barrier. The

amniotic fluid concentration is about 10% of the maternal serum level (Singer *et al* 1972).

This hormone is detected in the maternal serum as early as the fifth week of gestation. The level gradually rises upto 36-38th week, but remains constant thereafter upto term. A drop in the level after 40 weeks is reported by some authors (Verma *et al*). A low HPL is noted in placental insufficiency, postmaturity and when the placenta is damaged as in PET and accidental haemorrhage.

The reliability of HPL as an indicator of placental activity is enhanced by its short life (20 minutes), and by the fact that HPL production is autonomous and not altered by change of posture, stress, maternal metabolic or endocrine factors.

Material and Method

The estimation of serum HPL was done using a radioimmuno assay kit (RIAK-2) obtained from Bhabha Atomic Research Centre. The serum was separated from 5 ml. of venous blood obtained from each pregnant woman by refrigerated centrifuge soon after collecting the sample. This serum was kept at 4°C for the assay on the same day or stored frozen if the assay had to be postponed to a later date.

Group I—65 normal pregnant women

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Accepted for publication on 1-8-1981.

were chosen at different weeks of gestation. (7 cases were between 6-12 weeks, 35 between 16-36 weeks, 23 cases were between 38-40 weeks).

Group II—24 post-dated pregnancies extending upto 42 weeks were included in the H.P.L. study.

Observation

Normal pregnancy—HPL level gradually rose from 0.02 ug/ml at 6th week, and 1 ug/ml at 18 weeks to 8.6 ug/ml at 40 weeks. All these women delivered vaginally at term. The average placental weight was 399.8 gms and the mean birth weight was 2.84 kg.

Graph I shows the distribution of HPL levels at different weeks of gestation in normal pregnancies.

Post-dated Pregnancies—24 cases

The HPL level was found to be within the normal range in all but 1 case. This patient with a low HPL level of 3.8 ug/ml at 41 + 6 days developed foetal distress during induction. The baby delivered by caesarean section showed signs of dysmaturity. Two women delivered spontaneously and 22 were induced with syntocinon. There was no positive correlation between HPL level and foetal, as well as placental weights.

Graph II shows the distribution of HPL values in post-dated pregnancies.

Discussion

In the present series, the HPL level of 0.02 ug/ml was detected at the 6th week, and 1 ug/ml at 18-20 weeks of normal gestation. After 36th week, the level was over 6 ug/ml. However, there was no consistent rise in HPL level after 38th week of gestation. A similar pattern of HPL rise was reported by Kaplan and Grumbach in 1966, Saxena *et al* in 1969 and Genazzani in 1971. HPL values had no bearing on placental and birth weight.

The present study indicates that serum HPL of 6 ug/ml and over would

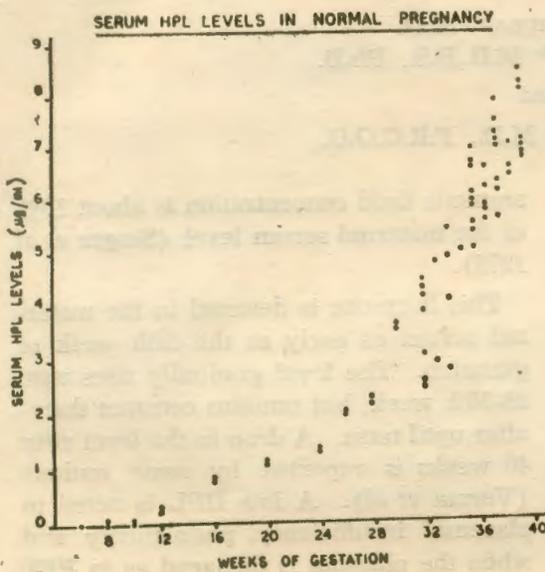


Fig. 1

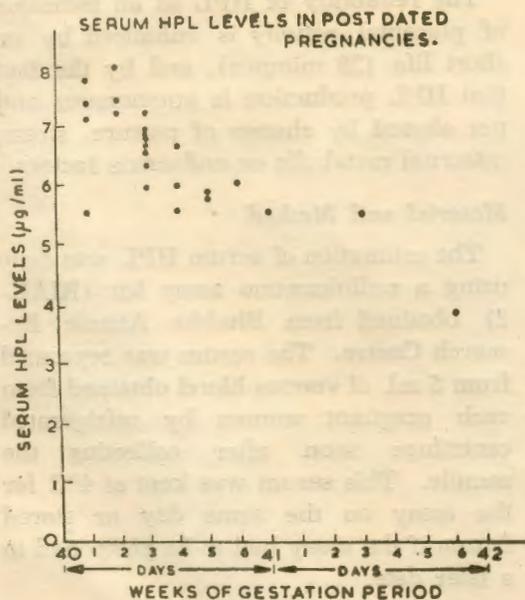


Fig. 2

guarantee foetal maturity of at least 36 weeks in normal pregnancies. Low HPL value for the corresponding period of gestation suggests placental insufficiency and intrauterine foetal stress.

It was not possible to diagnose post-dated pregnancy, as the HPL level failed to demonstrate a linear rise with each week of advancing pregnancy. The HPL values were within the normal range found near term. Spencer and Genazzani in 1971 and later Hobbins and Goldstein (1974) observed that HPL level above 6 ug/ml in post-dated pregnancies ensured a live baby with normal Apgar score at birth. A lower level indicated foetal anoxia and warranted induction or amniocentesis to verify foetal maturity and to detect meconium, a sign of chronic foetal stress.

There was one case showing HPL value of 3.8 ug/ml at 41.6 days, and foetal distress occurred during induction.

Saxena *et al* (1969) and Verma *et al* (1971) observed a gradual fall in the serum HPL with each week of post-maturity, but the fall was less than 50% of the normal value at term. The level dropped by more than 50%, only when the postmaturity was associated with foetal anoxia caused by placental insufficiency. Gudson (1969) and Genazzani (1971) noted a low Apgar score associated with low HPL level. These authors conceded that placental insufficiency in postmaturity could be fairly accurately predicted whenever a low reading was obtained.

Menachem *et al* (1978) showed that

70% of post-term babies with serum HPL below 6 ug/ml were born with low apgar score.

Summary

It is possible to assess foetal maturity of over 36 weeks with serum HPL of over 6 ug/ml.

The diagnosis of postmaturity is not possible. However, the placental insufficiency and foetal anoxia associated with post dated pregnancy can be strongly suspected when HPL level falls by 50%.

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