

STUDY OF AMNIOTIC FLUID GLUCOSE AND CREATININE IN NORMAL AND HIGH RISK PREGNANCY

G.K. BEDI ● A.S. SAINI ● H.JASWAL

SUMMARY

Amniotic fluid glucose (AFG) and crteatinine (AFC) levels were determined in 125 cases of normal and high risk pregnancy and correlated with foetal weight and Apgar score. Concluded that there was significant downward linear trend of AFG levels with advancing gestation, which served as a good parameter for foetal well-being and maturity and in placental insufficiency there was significant fall in the AFG levels whereas levels of AFC showed insignificant rise with advancing gestation and remained within normal limits in high risk pregnancy.

INTRODUCTION

A majority of our hospital patients do not furnish reliable data of their last menstrual period, and in order that foetus may not be delivered prematurely and succumb later to respiratory complications, the obstetrician has to assess foetal well-being and foetal maturity especially where induction of labour may become necessary to save the foetus from an increasingly unfavourable intrauterine environment.

The amniotic fluid by virtue of its ready accessibility and certain changes in its biochemical constituents at various periods of gestation, has been used to assess foetal age and well-being

Dept. of Obst. and Gyn, Govt. Medical College, Amritsar

Accepted for Publication on 1/12/90

(Pitkin and Zwirek, 1967; Brosgen and Gordon, 1966, Chandiok et al, 1971). The present study was undertaken to evaluate the usefulness of amniotic fluid glucose and creatinine levels as a parameter of foetal well-being in normal pregnancy as well as pregnancy with complications.

MATERIAL AND METHODS

The present study was done on 125 pregnant females of different age groups and parity admitted to the antenatal ward/labour room of Govt. Hospital for women, Medical College, Amritsar. Out of these, 75 cases were of normal pregnancy and 50 cases were of complicated pregnancy (Post-maturity, P.E.T. severe anaemia, IUGR). A detailed history and examination was carried out. Diabetics were not included in the study.

Amniotic fluid samples were taken by amniotomy (85), from amniotic sac at the time of Caesarean section (29) and amniocentesis (11). Blood stained and meconium stained samples were discarded. These samples were collected in Sodium fluoride vials (to prevent glycolysis), kept at 4°C and estimation carried out within 1-2 hours of collection. Glucose was estimated by method of Winckeris and Jacob (1971) and creatinine estimation was done by Jaffe-Hoppe-Seyler's Picric acid technique (1986).

Outcome of labour, weight of the baby and Apgar score at one minute and 5 minutes were recorded. Clinical findings were correlated with the biochemical parameters.

OBSERVATIONS AND DISCUSSION

Distribution of cases in normal and high risk pregnancy groups and mean values of AFG, AFC, mean birth weight as well as mean Apgar score at 1 minute and 5 minutes are shown in Table I. Our findings (20.0 mg% \pm 4.36) were comparable to Dhar and Eduljee (1980) who reported mean AFG levels as 18.22mg% \pm 2.79

and 17.38 mg% \pm 3.34 respectively. Mukherjee et al (1985) however, have reported much lower values (12.02 mg% \pm 6.12) at term pregnancy.

Downward linear trend of AFG concentration as gestation advances (Table I) may be explained on the basis of progressive maturity of foetal kidney i.e. increasing tubular fluid, as well as maturity of the liver to store glycogen with advancing gestation.

In cases of placental insufficiency glucose transfer capacity across the placenta is reduced thus resulting in fall in the AFG levels which were highly significant ($p < 0.01$) in severe PET group in the present study i.e. 2.15 mg% \pm 1.76. The carbohydrate reserves of foetal liver may also be lower in hypoxic states. In the present study (Table II), with negative AFG levels (4 cases) the Apgar score was below 7, with AFG levels between 1-8 mg%, 23 cases had Apgar score below 7 while 20 cases had Apgar score between 8-10, with AFG levels above 8 mg% all the 78 (100%) cases had Apgar score between 8-10.)

TABLE I
SHOWING DISTRIBUTION OF CASES AND MEAN VALUES

Group	No. of Cases	Amniotic fluid glucose mg% mean \pm SD	Amniotic fluid creatinine mg% mean \pm SD	Mean birth weight (g)	Mean Apgar score at 1 minute	Mean Apgar score at 5 minutes
Normal Pregnancy						
37 — 38 Wks	28	20.00 \pm 4.36	1.98 \pm 0.16	2860	8.12	9.22
39 — 40 Wks	47	18.00 \pm 4.53	2.00 \pm 0.17	2955	8.84	9.64
Post maturity	21	5.90 \pm 1.82	2.10 \pm 0.12	3050	7.82	8.80
Mild PET	11	4.70 \pm 1.11	2.10 \pm 0.14	2618	7.00	9.18
Severe PET	6	2.15 \pm 1.76*	2.28 \pm 0.15	2433	6.33	7.92
Severe anaemia	5	4.17 \pm 1.85	1.98 \pm 0.15	2716	7.14	8.66
IUGR	7	4.80 \pm 1.30	1.90 \pm 0.11	2414	6.28	7.84

* Highly significant ($p < 0.01$)

TABLE II

SHOWING CORRELATION OF AMNIOTIC FLUID GLUCOSE AND CREATININE VALUES WITH THE APGAR SCORE OF THE NEWBORN

	Values (mg%)	No. of cases	Apgar score below 7	Apgar score 8-10
Mean amniotic fluid glucose	Negative	4	4	Nil
	1-8	43	23	20
	More than	78	Nil	78
Mean amniotic fluid creatinine	1.5-2.0	56	11	45
	2.1-3.0	69	12	57

Amniotic fluid creatinine levels at term were 1.98 mg%+0.16 which showed a marginal rise with the advancing gestation (Table I). The values are comparable to Gupta et al (1978) and Padubidri et al (1983) who reported AFC values at term to be 2.20 mg% and 2.50 mg% respectively. The rise in creatinine levels, as gestation advances may be caused by more urinary excretion of protein end products or diminishing volume of amniotic fluid.

In Toxaemias of pregnancy, the AFC levels showed an insignificant rise ($p < 0.5$) whereas in dysmaturity the levels were low (1.90 mg%) therefore indicating that AFC levels are reliable index of foetal maturity only where foetal growth in normal. Apgar score was not affected by

AFC levels.

REFERENCES

1. Brosen, I and Gordon, H: *J. Obstet. Gynec. Brit. C'wealth* 73:88, 1966.
2. Chandio, S; Gupta, A.N. and Devi, P.K.: *J. Obstet. Gynec. India*, 30:794, 1980.
3. Gupta, S; Diskshit, J; Tuli and Bodhe, R.R.: *J. Obstet. Gynec. India*, 28:250, 1978.
4. Jaffe, Hoppe-Seyler, Z: *Physiol. Chem.*, 10:391, 1986.
5. Mukherjee, G; Mall, A; Mukherjee, K and Sinha, S.N.: *J. Obstet. Gynec. India*, 35:1070, 1985.
6. Padubidri, V; Singh, N and Dutta, S: *J. Obstet. Gynec. India*, 33:622, 1983.
7. Pitkin, R.M. and Zwirek, S.J: *Am. J. Obstet. Gynec.*, 98:1135, 1967.
8. Winckeris, P.L.M. and Jacob, P.H: *Clin. Chem. Acta*, 34:401, 1971