REMISSION OF DIABETES MELLITUS IN PREGNANCY

(A Case Report)

by

SHAIL KUMARI SINHA

SUMMARY

The concept that insulin requirement in pregnant diabetics increases, and if it falls it reflects fetal compromise, has achieved studied acceptance. A case is reported here in which a patient with a fasting blood sugar (FBS) of 220 mg%, requiring 40 units of lente insulin at 20 weeks gestation, started showing a steady decrease in insulin needs from 27 weeks onwards, till at 34 weeks FBS was 80 mg% and insulin was discontinued. There was no evidence of fetal compromise and pregnancy was allowed to continue. Eventually induction of labour was required for worsening pre-eclampsia at 36 weeks. The baby had a good Apgar score and continued to do well. The patient reverted to her diabetic state 4 weeks after delivery. Cases like this just go to prove that even established concepts have their exceptions, and each patient accordingly requires individualised attention.

Introduction

The insulin requirements in diabetic women usually increase during pregnancy, especially during the second and third trimesters, is an unchallenged statement. Placental production of human placental lactogen, progesterone, estrogen, cortisol and insulinase is thought to cause these changes. An obvious corollary of this is that a significant fall in insulin requirements means a decrease in placental function with resulting fetal compromise often intrauterine death. This has been stresed by several authors (Barnes, 1974; White, 1974). A small decrease in insulin needs shortly before term is, however, not unusual.

It is the purpose of this case report to document the possibility of a marked fall in insulin requirements in pregnancy without evidence of fetal compromise; and to emphasize the importance of recognising this in the evaluation and treatment of the pregnant diabetic women. Premature induction can be avoided in such cases, and knowing the propensity for infants of diabetic mothers delivered before the 37th week to develop respiratory distress syndrome even when LS ratios are 2:1 more (Mueller-Heubach *et al* 1978), this could be useful.

From: Medical Officer, Sadar Block, Darbhanga, Station Road, Laheriasarai-846 001.

Accepted for publication on 22-6-85.

Case Report

The patient was a 30 year old third gravida. She had eclampsia in her first pregnancy at 8½ months gestation, and delivered a stillborn male child. There was no antenatal check up. During the 2nd pregnancy she had regular antenatal care and a hospital delivery of a 4 kg. infant. Fasting and post-prandial blood sugars done at her first visit during the 4th month were normal. Glucose tolerance test (GTT) was not done, nor were blood sugars repeated later in pregnancy.

In the 3rd pregnancy which is reported here, the patient sought medical advice for severe itching vulva and burning micturition at 20 weeks gestation. On examination, her weight was 52 kg. B.P. was 120/70 mm Hg and the uterus was 20 weeks in size. There was severe monilial vulvovaginitis. Urine examination showed sugar 2+, but no acetone or albumin. Bacterial culture of urine was sterile. Fasting blood sugar (FBS) was 220 mg%. She was started on a 2000—calorie diet and 20 units of Lente insulin before breakfast. The dose of insulin was gradually increased to 40 units till good control was established with a FBS level of 126 mg%. Urine was free of sugar. She was followed up with weekly FBS estimations. From 27 weeks onwards, the FBS started reducing and insulin was correspondingly reduced.

At 32 weeks, she developed oedema of the feet and her B.P. was 130/90 mm Hg. Urine was free of albumin and sugar. FBS was 108 mg%. Insulin was reduced to 10 units and phenobarbitone and frusemide were started. At 34 weeks the FBS had further reduced to 80 mg% and insulin was discontinued. The B.P. had risen to 140/100 mm Hg and methyl dopa was added. The size of the uterus corresponded to the period of gestation and acceleration of the fetal heart rate was consistently observed with fetal movements. Pregnancy was allowed to continue.

At 36 weeks, however, the patient showed puffiness of the face and oedema of the hands. B.P. was 158/110 mm Hg and albumin was 1+ in urine. Blood sugar remained normal. Retinoscopy showed changes consisent with severe pre-eclampsia. With these indicators of deteriorating pre-eclampsia, labour was induced. Artificial rupture of the membranes was followed by syntocinon drip. Liberal sedation was given. Fetal heart rate remained regularly between 136-152/min. She delivered vaginally after 8 hours, a 2.7 kg. infant with a 1 min. Apgar score of 8 and a 5 min. Apgar of 10. The plancenta appeared normal.

Two days after delivery the B.P. was 150/100 mm Hg and FBS was 98 mg%. Two weeks later, B.P. was normal and antihypertensives were stopped. At 4 weeks postpartum, her blood sugar levels were grossly raised, indicating a reversion to her diabetic state. FBS was 250 mg% and, 2 hours after a glucose load of 75 gm., it was 458 mg%. The baby had no problems.

Discussion

This cases represents a deviation from the usual effect of pregnancy on diabetes and also shows that significantly decreased insulin requirements during pregnancy does not necessarily indicate impending fetal demise. Two similar cases have been reported by Sheldon and Coleman (1974) in which diabetes remitted during pregnancy. They attributed it to increased insulin sensitivity due to some unknown factor specifically related to pregnancy as there was rapid deterioration after delivery. One case has been reported by Gabbe *et al* (1974). This patient also required induction for worsening pre-eclampsia. An interesting speculation was whether a portion of placental function is reduced while other functions remain normal, specifically whether endocrine function is reduced while respiratory function remains normal. This is significant not only as a clue to the mysterious working of the placental laboratory, but also in clinical practice where delaying induction is a consideration so as to improve chances of fetal lung maturity and hence neonatal survival. It takes on an added significance in our country where sophisticated fetal monitors and laboratory parameters of fetal

well being are available only in select centres. Without these facilities one runs the risk of being left with no elasticity in patient management. In this particular case, if pre-eclampsia had not become dangerous to the mother and baby, and if she did not have an ominous history of eclampsia, she might have continued uneventfully to term, inspite of a marked fall in blood sugar levels.

Despite extensive literature, we still do not understand fully the degree to which pregnancy alters glucose tolerance. Till the precise behaviour of blood glucose levels during pregnancy is clearly established, diabetes mellitus will remain, at best, an enigmatic area in which to be dogmatic. That every pregnant diabetic woman requires constant, close and competent supervision cannot be overstressed. Cases like this one increase one's awareness of when not to intervene, as opposed to our concern with when to intervene in high risk pregnancies.

References

1. Barnes, C. S.: Medical Disorders in Obstetric Practice. ed. 4, Oxford, 1974, Blackwell Scientific Publications: p. 290.
2. Gabbe, S. G., Dizerega, G. S. and Mestman, J. H.: Am. J. Obstet. Gynec. 125: 264, 1974.
3. Mueller-Heubach, E., Caritis, S. N., Edelstone, D. I. and Turner, J. H.: Am. J. Obstet. Gynec. 130: 28, 1978.
4. Sheldon, J. and Coleman, T.: Brit. Med. J. 1: 55, 1974.
5. White, P.: Clin. Perinatol. 1: 331, 1974.