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The effects of established and gestational diabetes on pregnancy outcome at Harare Maternity Hospital

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SUMMARY

A study was carried out to determine the effect of established and gestational diabetes on pregnancy outcome over a period of two years at Harare Maternity Hospital, Harare, Zimbabwe. During the period, 51 patients with established *diabetes mellitus* and 70 patients with gestational diabetes were treated. The perinatal mortality was higher among this group (124 per 1 000) compared with the rest of the total hospital population (44 per 1 000) who delivered during the same period.

INTRODUCTION

Established diabetes is well recognised as a cause of increased perinatal mortality (Connell *et al* 1985) whilst the clinical significance of abnormal glucose tolerance identified in pregnancy remains controversial (Beard and Hoet 1982, Jarret, R J, 1981, Al-Sawaf *et al* 1988).

In Harare Maternity Hospital, the patients with established *diabetes mellitus* are treated with insulin and those with glucose intolerance first diagnosed in pregnancy (gestational diabetes) are treated with diet alone or with diet and insulin.

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The purpose of this study was to determine the foetal outcome on women with established diabetes and gestational diabetes as measured by perinatal mortality and foetal birth-weight.

MATERIALS AND METHODS

A retrospective analysis of the records of all patients who had established diabetes and those with gestational diabetes and delivered at Harare Maternity Hospital between January 1, 1990 to December 31, 1991 was done.

Diabetes was classified as "established" if the disease was known to be present and was being treated before pregnancy. These were mainly patients referred from city clinics, Harare Hospital Medical OPD, District and Provincial hospitals and general practitioners.

A diagnosis of gestational diabetes was made in a pregnant women whose carbohydrate intolerance was first recognised during that current pregnancy using WHO (1980) criteria.

At booking of a current pregnancy, selective screening with a glucose load test was done in any woman with any of the following risk factors: namely previous history of unexplained still-births, previous delivery of macrosomic baby (birth-weight >4kg), first degree relative with insulin dependent *diabetes mellitus*, polyhydromnious in current pregnancy, glycosuria on more than two occasions and those with bad obstetric history.

To perform the glucose load test, 50gm of oral glucose is given to the pregnant woman without regard to time of day or last meal. If the venous plasma glucose level measures one hour later is ≥ 7.8 mmol/l a complete glucose tolerance test is performed (Beard *et al* 1980, O'Sullivan *et al* 1973b).

At Harare Maternity Hospital, an oral glucose tolerance test (OGTT) is performed with a 75gm glucose load in a patient who has starved from the previous night and glucose intolerance is shown by fasting blood glucose levels at ≥ 6.0 mmol/l and 2-hour levels of ≥ 7 mmol/l.

Once a diagnosis of gestational diabetes is established from OGTT, treatment starts with a diabetic diet and if subsequent glucose profiles do not show acceptable glucose haemostasis of between 4–6mmol/l, insulin treatment is commenced (Fraser R B, 1991, Burdnett, M and Doddridge, M C 1989).

RESULTS

A total of 121 patients were treated from January 1990 to December 1991. This represents about 0,04 pc of all deliveries during that period. Of the 121 cases, 51 (42 pc) were established diabetes and 70 (58 pc) were gestational diabetes. Among the gestational diabetics only 19 (27 pc) were treated with diet compared with 51 (73 pc) who were treated with diet and insulin.

Table I shows that perinatal mortality rate is higher for the diabetic patients (124 per 1000), compared to the total hospital population (44 per 1000). Established diabetics had a higher perinatal mortality (196 per 1000) compared to those with gestational diabetics (71 per 1000).

Table I: Perinatal Mortality

Group	All deliveries	All babies born	Still-births (per 1 000 births)
Total hospital population	34,362	35,454	1 555 (44)
Established diabetics	51	51	10 (196)
Gestational diabetics	70	70	5 (71)

Table II shows details of perinatal deaths. The excess loss of macerated still-births, with no explanation for the deaths, tended to occur in late pregnancy and in macrosomic babies whose mothers were on insulin.

DISCUSSION

It is important to point out the disadvantages of this study: firstly that data collection was retrospective, resulting in possible inaccuracy on total number of patients with diabetes in pregnancy. This may explain why there is such a small number (less than 1 pc) of diabetics in the pregnant population at Harare Maternity Hospital. However, a community-based study to estimate the incidence of gestational diabetes would be more accurate.

In most regions of the world, the incidence of gestational diabetes varies from 1,6 pc to 2,5 pc (Beard R W, 1982, O'Sullivan J V, 1973). A strict criteria of diagnosis should be followed as recommended by W.H.O. (1980).

Table II: Details of Perinatal Deaths

	Age (yrs)	Parity	Type of diabetes	Outcome
1.	38	8	G.D.	Ruptured uterus, fresh SB at term, 4357 gm
2.	32	5	IDDM	Breech, IUD at term 6100 gm
3.	39	3	IDDM	Emergency LSCS for arm prolapse, 4100 gm, died 1/7
4.	17	0	IDDM	IUD at 30 wks, 2630 gm
5.	39	5	IDDM	Macerated SB at term 6400 gm
6.	34	7	IDDM	Macerated SB at 34 wks 2500 gm
7.	19	1	IDDM	SB at 39 wks 3800 gm
8.	38	9	GD + PIH	SB at 35 wks 2100 gm
9.	24	1	GD	Preterm labour at 30 wks, 1900 gm, Neonatal death at 2/7
10.	31	7	IDDM + PIH	Macerated SB at 28 wks 800 gm
11.	39	5	IDDM	Hydrocephalus + spina bifida diagnosed by USS at 35 wks, decompression of head done
12.	26	3	IDDM + PIH	Ketoacidosis at 30 wks macerated SB 1110 gm
13.	32	5	IDDM	Referred at 36 wks on oral hypoglycaemics, macerated SB 6100 gm
14.	35	5	GD + PIH	IUD at 37 wks 4400 gm
15.	40	4	GD + PIH	Macerated SB at 34 wks 2500 gm.

GD = Gestational Diabetes
IDDM = Insulin Dependent Diabetes Mellitus
SB = Still-born
IUD = Intra Uterine Death
PIH = Pregnancy Induced Hypertension
LSCS = Lower Segment Caesarean Section
USS = Ultra Sound Scan

Although the numbers in the study are small, there is, however, a clearly defined poor outcome among the 121 diabetic pregnancies treated at Harare Hospital in 1990/91.

The majority had detailed ultra-sound scan in late second trimester and only one was found to have CNS malformation. Post mortem examinations were not performed on the still-born babies but gross macroscopic inspection did not show malformations.

Intra-uterine death occurred more commonly among women with established diabetes than those with gestational diabetes probably because of poor control in the earlier group. The outcome was even poorer when a women had established diabetes with pregnancy induced hypertension.

Late referral in established diabetes results in poor control and subsequent higher foetal loss as shown by Case 12 who was sent from a district hospital at 30 weeks in ketoacidosis. Case 13 who was referred at 36 weeks on chlopropramide illustrates why we do not use oral hypoglycaemic drugs in pregnancy because of poor control and risk of congenital malformation (Brudness M *et al*, 1989).

The long-term implications for this study are firstly to establish the normal glucose haemostasis among pregnant and non-pregnant Zimbabwean women of reproductive age. Secondly, we would like a prospective study on established and gestational diabetics to see if present management can be improved for a better outcome on this population.

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