PERINATAL MORTALITY IN TOXAEMIA OF PREGNANCY

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While all the workers are convinced of the adverse effects of the toxaemic process on the foetus, it is surprising to find that some severely toxaemic women and even eclamptic mothers produce normal healthy babies at full-term and in spite of heavy sedation. On the other hand even mild pre-eclamptic toxaemia causes intra-uterine death. The obvious disparity in foetal results has prompted the present study with the aim of finding out, if possible, which of the numerous variable factors comprising this syndrome of toxaemia of pregnancy are responsible for the adverse effects on the foetus.

The perinatal mortality has been correlated with severity of hypertension (Townsend 1959), duration of hypertension (Carey & Liley 1959), albuminuria (Dieckmann 1941 & Theobold 1955) and maternal age and parity (Peckham 1933 and Hibbard 1962).

One hundred and fifty cases of toxaemia of pregnancy were studied from 1st August 1964 to 30th March 1965.

Hypertension at or above 140/90 mm of Hg. with or without albuminuria and oedema was taken as the criterion and divided into mild, moderate, severe and eclamptic groups.

	Blood pressure	Albuminuria	Oedema
Mild	140/90 mm. of Hg.	0.5 gm. or less	Variable
Moderate	Above 140/90 to 160/110 mm. of Hg.	0.5 to 5 gm./24 Hrs.	—do—
Severe	Above 160/110 mm. of Hg.	Above 5 gm./24 Hrs.	—do—

Eclamptic. All the above signs + convulsions.

The mild group was separated from the moderate group only to see whether toxaemia at this blood pressure level (140/90) affects the foetal prognosis.

Observations

The effect of age: Table I shows that the rise in perinatal mortality is

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proportionate to age i.e. 33% in cases above 35 years.

Effect of parity & Socio-economic status: The incidence of toxaemia was highest in primigravidae while the foetal loss was lowest in this group (21%) and increased to 36% in para 4 and above. The study of socio-economic status revealed that perinatal loss was highest (34%) in the lowest income group i.e. below Rs. 40/- per month per capita.

Effect of haemoglobin level: is shown in Table II. The perinatal death rate was 60% when haemoglobin was below 5 gm. It was very significant. Further it was noted that majority of these were neonatal deaths, indicating that anaemia hampers the chances of survival in neonatal period.

Effect of cardinal signs

The effect of hypertension is shown in Tables III and IV. There is a sudden increase in perinatal loss from 18% to 60-64% above 180 mm of Hg. systolic and 120 mm of diastolic blood pressure.

TABLE I

Age and perinatal mortality

Age Group	No. of cases	Perinatal Death		
		No.	%	
Up to 25 years	67	16	24%	25%
25 to 35 years	61	16	26%	
Above 35 years	22	7	33%	

TABLE II

Haemoglobin level and perinatal mortality

Hb level	No. of cases	Still Birth		Neonatal death		Perinatal mortality	
		No.	%	No.	%	No.	%
Below 4.9 gm.	5	1	20	2	40	3	60
5—6.9 gm.	8	1	13	2	35	3	38
7—8.9 gm.	15	5	53	1	7	6	39
Above 9 gm.	122	14	12	13	10	27	22

TABLE III

Systolic blood pressure and perinatal mortality

Systolic B.P.	No. of cases	Perinatal mortality		
		No.	%	
140 to 159 mm of Hg	74	15	20	18%
160 to 179 mm of Hg	51	8	16	
180 to 199 mm of Hg	18	11	61	64%
200 and above	7	5	71	

TABLE IV

Diastolic blood pressure and perinatal mortality

Diastolic B.P.	No. of cases	Perinatal mortality No.	%	
90—99 mm of Hg.	28	5	17	
100—109 mm of Hg.	59	5	17	18%
110—119 mm of Hg.	34	7	21	
120 and above	29	17	60	

The effect of albuminuria: This is shown in Table V. Out of 150 cases only 88 had albuminuria. The perinatal mortality of 34% in the group with albuminuria was 3 times higher than in the group without (14%) and further it rose steeply from 25% when it was below 5 gms to 65% when it was above 5 gms.

Effect of severity of toxaemia: When the highest blood pressure reading was 140/90 mm of Hg. the perinatal mortality was 9%, which compared favourably with the general perinatal mortality of 11.9% in non-toxaemic cases. The perinatal mortality increased in proportion to the severity of the toxaemic process.

While the presence of albuminuria caused a marked increase in perinatal mortality in the severe group, the same effect was not noted in the moderate group as albuminuria of more than 5 gm per 24 hours appeared only when blood pressure exceeded 160/110 mm of Hg (table VI).

Effect of duration of toxaemia: The perinatal mortality was highest (49%) when the duration was not known, as all these cases were admitted as emergencies with no antenatal care or with complications like accidental haemorrhage.

When the duration was known, a significant association was found bet-

TABLE V

Albuminuria and perinatal mortality

Albuminuria	No. of cases	Perinatal death No.	%		
Without Albumin	62	9	14%		
With Albumin					
.1—1.9 gm/24 Hrs.	24	6	25%		
2—4.9 gm/24 Hrs.	44	11	25%		
5—8.9 gm/24 Hrs.	17	11	64%	65	34%
9 gm and above	3	2	66%		

TABLE VI

Severity of toxaemia and perinatal mortality

Severity of toxaemia	No. of cases	Perinatal mortality No.	%
Mild	11	1	9
Moderate	95	19	20
Severe	36	14	39
Eclamptic	8	5	62

ween the perinatal mortality and term of onset (Table VII) but there was no apparent correlation with the duration of toxaemia (Table VIII).

Effect of Blood Urea Levels is shown in Table IX. Perinatal mortality rose to 71% when blood urea level rose above 40 Mgm. and to 100% when blood urea level rose above 50 Mgm.

Discussion

At the outset it is important to note that the factors that significantly influence foetal survival and maturity at delivery are not independent variables; marked hypertension is more likely to be associated with severe proteinuria and the severe type often appears earlier in pregnancy.

Throughout this study a correlation of foetal outcome has been made with the highest systolic and the highest diastolic recordings during pregnancy. Such single readings can hardly be considered reliable or representative but as a sufficient period assessment was not possible in all cases (49% of which were emergency admissions) it was decided to take the highest reading as a yardstick for comparison of results.

TABLE VII

Term of onset of toxaemia and perinatal mortality

Term of onset of toxaemia	No. of cases	Perinatal mortality	
		No.	%
Not known	54	27	49
Below 5.9 months	4	1	25
6—7.9 months	26	4	16
8—8.9 months	32	3	9
9 months to term	34	4	10

TABLE VIII

Duration of toxaemia and perinatal mortality

Duration	No. of Cases	Perinatal death	
		No.	%
Not known	54	27	49
Upto 2 months	69	10	14
Upto 4 months	17	1	5
Above 4 months	10	1	10

TABLE IX

Effect of blood urea levels and perinatal mortality

Blood urea levels	Total No. of cases	Perinatal deaths	
		No.	%
20—29.9 Mgm.	117	25	22
30—39.9 Mgm.	24	7	28
40—49.9 Mgm.	7	5	71
50 and above	2	2	100

In mild toxaemia the perinatal mortality was 90% which is the same as in the non-toxaemic group. Carlwood and Pinkerton (1961) have made the same observation. Perinatal mortality rises with the severity of the toxaemic process and reaches a maximum in the eclamptic group (62.5%). Browne and Dodds (1942) have noted a perinatal mortality of 48% in the eclamptic group as compared to the over-all perinatal mortality of 13%.

The perinatal mortality associated with hypertension alone was 14% and with hypertension and albuminuria it was 34%. Theobold (1958) states that perinatal mortality associated with hypertension and albuminuria is almost three times higher than with hypertension alone. It was further observed that the significant correlation between the albuminuria and perinatal mortality appeared only when blood pressure exceeded 160/100 mm of Hg.

The appearance of albuminuria between the blood pressure range of 140/90 and 160/100 mm of Hg. is worth nothing specially when we consider the explanation offered by Browne (1950) that when blood pressure rises above 160/100 mm of Hg. the spasm of the glomerular arterioles causes albuminuria. Theobold (1955) has also shown the association of albuminuria with blood pressure level below 160/100 mm of Hg.

A steep rise in perinatal mortality was noted when the highest recorded systolic blood pressure exceeded 180 mm of Hg. and diastolic blood pressure exceeded 120 mm of Hg. and when albuminuria exceeded 5 gms/24 hours, which is to be expected since severe albuminuria is associated with high blood pressure.

Another factor which indicates the severity of the toxaemic process is the blood urea level. The perinatal mortality rose steeply when levels exceeded 40 mgm% and rose almost to 100% over 50 mgm. In all these cases blood pressure and albuminuria levels were high and the placenta was severely infarcted in 5 among 9 cases.

It was observed that the earlier the onset of symptoms the higher was the perinatal loss. At the same time, no significant relationship was noted with duration.

This was due to the fact that when toxaemia appears early in pregnancy, the chances of early intra-uterine death or premature spontaneous labour with neonatal death are higher. Hence it is the stage of pregnancy at which the foetus is exposed to the faulty environment rather than the length of exposure that is important. Carey and Liley (1959) concluded that the perinatal mortality rises with the early onset of symptoms of toxaemia.

Dieckmann (1941), observed that the daily excretion of less than 3 gms of protein in the urine can be continued for a period of weeks with no danger of foetal death, provided systolic blood pressure remains below 150 mm of Hg. The daily excretion of more than 5 gm. per 24 hours for a period of 10 days or more, irrespective of blood pressure level, is quite often accompanied by foetal death in utero. In this series, however, albuminuria of more than 2 weeks' duration, regardless of seve-

rity was associated with poor foetal prognosis.

As compared to a loss of 49% where no treatment was possible the perinatal mortality was definitely lower when the patients were given some treatment to control the toxaemic process before delivery. Gibson (1954) also showed that conservative treatment for 14 days or more is of undoubted benefit to the baby before 35 weeks. In this series, however, as antenatal attendance in booked cases was haphazard and at long intervals, the proplylaxis and control of toxaemia left much to be desired.

The increase in perinatal mortality with the age of toxaemic mothers has also been noted by Peckham, Nelson and Dieckmann. While Nelson found a higher perinatal mortality in primigravidae below 19 years, in this study it was lowest i.e. 8%.

Though the high incidence of toxaemia was in primigravidae, the perinatal mortality was lowest in this group. Perinatal mortality increases with the parity of toxaemic mothers. This finding has been confirmed by Peckham and Hibbard.

The perinatal loss was 60% when haemoglobin level was below 5 gms%. While anaemia did not cause higher antepartum and intrapartum death rate, it hampered the chances of survival of a live birth in the neonatal period, especially the premature live birth.

Conclusions

One can conclude that on statistical basis the correlation between foetal prognosis and the severity of each variable factor of the toxaemic process (except oedema) can be demonstrated in the very severe pre-eclamptic and eclamptic groups only. In moderate toxaemia with blood pressure below 160/110 mm of Hg. and albuminuria below 5 gm/24 hours, which constituted the majority of cases (63%) no particular clinical criteria were a reliable gauge of the individual foetal risk. The foetal loss is not insignificant in cases considered mild by classical criteria. Though statistically the perinatal mortality of 9% in mild cases compares favourably with 11.9% in the non-toxaemic population, it must be remembered that this non-toxaemic group was associated with all the other complications of pregnancy and labour excluding toxaemia.

Therefore, the early intervention in these cases to be effective must be based not on these maternal clinical criteria but on impending death in utero by more sophisticated methods, such as hormone assays. To quote Carey and Liley "The question is not really whether any clinical criterion is a reliable gauge of individual foetal risk, but whether the maximum information being extracted from the factors known to affect the intrauterine death rate. It would seem reasonable that the best estimate of foetal risk is one which makes accurate allowance for the uneven influence and partial interdependence of the relevent variables."

Summary

The effect of hypertension, albuminuria, severity, duration of toxaemia and the effect of age, parity and socio-economic status on foetal outcome was studied in 150 cases of toxaemia of pregnancy. There was a sudden

rise in perinatal mortality, where blood pressure exceeded 180 mm of Hg. systolic and 110 mm diastolic, albuminuria exceeded 5 gm/24 hours, blood urea exceeded 40 mg and in the eclamptic group.

Mild toxaemia was associated with a perinatal mortality of 9% which compared favourably with non-toxaemic group (11.9%). In the moderate group perinatal mortality was 20%, in severe pre-eclampsia it was 39%, and in eclampsia 60%. Excluding emergency admissions which were associated with a perinatal mortality of 49%, the perinatal loss was twice as high when symptoms appeared before 32 weeks than when symptoms appeared after 32 weeks.

It is concluded that in the moderate group which constitutes the majority of toxaemic patients, no clinical citerion was a reliable gauge of individual foetal risk and hence the time for inducing labour to improve foetal survival cannot be based with any degree of precision on clinical criteria.

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