

RELATIONSHIP BETWEEN PLATELETS AND INTRAUTERINE GROWTH RETARDATION IN CASES OF PRE-ECLAMPSIA

by

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Pre-eclampsia and eclampsia are important causes of perinatal mortality. The low birth weight is a major adverse factor associated with proteinuric pre-eclampsia. It has been well known that disseminated intravascular coagulation is a feature of pre-eclampsia leading to impairment of placental blood flow, placental insufficiency and growth retardation (Trudinger, 1976). The deposition of fibrin and platelets in the placenta contributes to the placental insufficiency and lead to depletion of circulating platelets in maternal blood stream.

To detect intravascular coagulation, the single most important sensitive finding is a low platelet count, as fibrinogen level may be normal or depressed (Bonnar, 1969). Therefore, the present study has been undertaken to know the association between intrauterine growth retardation and maternal platelet count in cases of pre-eclampsia and its comparison with

platelet level in third trimester of pregnancy.

Material and Methods

The present study was conducted on 75 patients who were attending the Umaid Hospital attached to Dr. S. N. Medical College, Jodhpur. Out of these, 25 patients served as healthy control group which were in the third trimester of pregnancy (34-40 weeks of gestation). Fifty pregnant women between 34-42 weeks of gestation with all degrees of toxæmia were studied. Out of these 50 patients, 27 had mild pre-eclampsia, 10 had severe pre-eclampsia, 12 were of essential hypertension and remaining 1 had essential hypertension superimposed with toxæmia. Diagnosis of pre-eclampsia was made by presence of at least two out of three signs of toxæmia, blood pressure, proteinuria and/or oedema over the dependent parts of the body. Complete history was taken in each case especially for swelling and its duration, headache, vertigo, blurring of vision, decreasing height of uterus, varinal bleeding and loss of foetal movements. Previous obstetric history was taken in respect to parity, abortion, still birth and early neonatal death. Past history of renal disease,

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hypertension and diabetes was noted in each case. *Discussion*

Routine haematological investigations and complete urine examination was done. Platelet count was done by Rees and Ecker method (1923). The counting was done in haemocytometer. Normal value with this method is 1,40,000 to 3,40,000/cu. mm.

Observations

The mean foetal weight and mean platelet count is unaffected in cases of essential hypertension as compared to normal. As the degree of toxæmia progresses the mean foetal weight and mean platelet count progressively decreased (Table I).

Pre-eclampsia is one of the major cause of perinatal mortality. The incidence of pre-eclampsia in our study was 4.28%, the incidence of premature deliveries in our hospital was 13.32%, out of which 3.29% deliveries were due to pre-eclampsia.

In this study, platelet count and foetal weight were taken to establish relation between platelet count and intra-uterine growth retardation.

In normal pregnancy the mean platelet count was 2,04,000/cu. mm. (S.D. 19600), while in mild pre-eclampsia it was 1,73,000/cu. mm. (S.D. 17400), in severe pre-eclampsia the platelet count further

TABLE I
Mean and Range of Foetal Weight and Platelet Count in Various Types of Toxaemia and in Normal Pregnancy

Types of cases	No. of Cases	Mean & range of Foetal weight (in grams)	Mean & range of Platelet count (/cu. mm.)
Normal Pregnancy	25	3060 (2620 to 3370)	204,000 (198000 to 220000)
Mild pre-eclampsia	27	2948 (1784 to 3253)	1,73,000 (135000 to 220000)
Severe pre-eclampsia	10	2274	1,70,000 (140000 to 200000)
Essential hypertension	12	3038 (2453 to 3791)	1,90,000 (140000 to 200000)
Essential hypertension & toxæmia	1	1333	1,20,000

The mean foetal weight increases in normal pregnancy, mild pre-eclampsia and in essential hypertension upto 40 weeks of gestation. However, in severe pre-eclampsia the mean foetal weight was reduced at 40 weeks of gestation, this may be probably due to marked proteinuria in these cases. The platelet count was progressively reduced as the period of gestation progressed upto 40 weeks (Table II).

reduced to 1,70,000/cu. mm. (S.D. 19000) and in essential hypertension the mean platelet count was 1,90,000/cu. mm. (S.D. 17800). All these findings show that as toxæmia progresses the platelet count reduces, however, the count is near normal in essential hypertension. Howie (1971) also observed normal platelet count in uncomplicated hypertension. Our findings are similar to that of Keenan

TABLE II
 Mean Platelet Count and Mean Foetal Weight at Different Periods of Gestation in Various Types of Toxaemia and in Normal Cases

Period of gestation in week ¹	Normal Pregnancy		Mild Pre-eclampsia		Severe Pre-eclampsia		Essential Hypertension	
	Mean Platelet count /cu. mm.	Mean Foetal weight gms.	Mean Platelet count/cu. mm.	Mean Foetal weight in Grams	Mean Platelet count/cu. mm.	Mean foetal weight in Grams	Mean Platelet count/cu. mm.	Mean foetal weight in Grams
34	220000	2626	—	—	—	—	—	—
36	200000	3050	180000	2007	170000	2118	200000	3010
38	198000	3142	186000	2706	172500	2230	170000	2620
40	198000	3370	160000	2750	165000	2007	186000	3900
42	—	—	160000	3010	160000	2118	200000	2676

and Bell (1957) and Bonnar (1971). Kitzmiller (1974) also reported a platelet count of $164 \pm 62 \times 10^3$ in toxaemia group while the platelet count was $212 \pm 66 \times 10^3$ in normal pregnancy which is much higher than toxaemia group.

In our study, the mean foetal weight in normal cases was 3060 gms. with the S.D. 538.4, in mild preeclampsia 2948 gms. with S.D. 492.6, in severe pre-eclampsia 2274 gms. with S.D. 432.2 and in cases of essential hypertension the mean foetal weight was 3038 gms. with S.D. 522.5. This shows that as toxaemia progresses the foetal weight proportionately reduces, whereas foetal weight remains normal in essential hypertension. Similar findings were also observed by Sinha and Mukherjee (1973) and Rohatgi *et al* (1975).

Trudinger (1976) pointed that there is a definite correlation between reduced level of circulating platelets in pre-eclampsia (not in essential hypertension) and intra-uterine growth retardation. In our study, 1 patient of essential hypertension superimposed with toxaemia of pregnancy had lowest platelet count (1,20,000/cu. mm.) and delivered a still born child of lowest birth weight at 42 weeks of gestation (1333 gms.).

Heavy proteinuria led to fall in mean foetal weight in cases of mild and severe pre-eclampsia. This finding coincides with Morris *et al* (1955), who stated that heavy proteinuria led to retardation of foetal growth.

Summary

Seventy-five patients were subjected to platelet count and foetal weight to assess intrauterine growth retardation. Fifty patients of toxaemia of pregnancy revealed low foetal weight and low platelet count and this reduction in weight and

count was proportionate to the degree of toxæmia as compared to normal. However, in essential hypertension the platelet count and foetal weight were almost similar to normal pregnancy (25 cases).

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