A STUDY OF TOTAL PLATELET COUNT, ADHESIVE PLATELET COUNT AND PLATELET ADHESIVENESS IN TOXAEMIA OF PREGNANCY

(With Special Reference to Foetal Weight)

by

S. C. SAXENA,* M.S. N. KHIRWARKAR,** M.D. H. SUBHEDAR,*** M.S.

> and R. APTE,† M.S.

In pregnancy toxaemia there is disseminated intravascular coagulation leading to fibrin deposition which causes an alteration in clotting factors, fibrinogen, and platelets. Fibrin deposition in maternal blood spaces of placenta causes placental insufficiency resulting in foetal growth retardation. Low platelet count is probably the single most important finding in diagnosis of consumptive coagulopathy. Because of the key role of platelets in coagulation mechanism, it seems likely that chronic platelet damage is one of the central mechanism in perpetuating this chronic low grade intravascular coagulation.

The present study deals with the changes in platelet count, adhesive platelet count and platelet adhesiveness in toxaemia of pregnancy, with special reference to foetal weight and well-being.

*Reader in Obstetrics and Gynaecology.

**Professor of Physiology.

Accepted for publication on 8-7-80.

Material and Methods

The present study was carried out in the Dept. of Obst. & Gynaecology and Physiology, Medical College, Jabalpur from January 1977 to June 1978. The material consisted of 25 normal pregnant patients in second and third trimesters of pregnancy, and 50 cases of toxaemia of pregnancy of varying severity. There were 12 cases of eclampsia (7 antepartum and 5 postpartum). The severity of toxaemia was based on the following criteria:-Mild-B.P. 130/80 mm.Hg. to 140/90 mm.Hg., and oedema, no albuminuria, Moderate-B.P. 140/90 to 160/100 mm. Hg., with oedema, with or without albuminuria. Severe-B.P. above 160/100 mm.Hg., with oedema and albuminuria.

The platelet count was estimated according to Rees and Ecker's method.

Observations

The ages of these patients varied from 15 to 40 years, maximum cases were in the age group of 21-25 years.

Nine cases in the control group and 30 cases in the study group were primigra-

Table I shows the mean platelet values

^{***}Lecturer in Obstetrics and Gynaecology.
†Asstt. Surgeon, Medical College, Jabalpur.

^{****}Paper presented at 22nd All India Congress in Obstetrics and Gynaecology, Bangalore, December 1979.

TABLE I

Mean Platelet Values in Normal and Toxamic Pregnancy

Type of cases	Total platelet count	Adhesive platelet count	Platelet adhesiveness
II trimester			
control	268000	249000	9.90%
Toxaemia	183300	150300	18.70%
III trimester			
control	283000	238900	14.58%
Toxaemia	150800	139800	23.18%

in control and study group in second and third trimesters of pregnancy. Platelet counts were lower and platelet adhesiveness were higher in toxaemia cases as compared to control cases.

Table II shows the relation of platelet values to baby weight in control group. Tables III & IV show the platelet values in toxaemia cases according to the seve-

rity of toxaemia, and its relation to baby weight and condition. Platelet count went on falling and platelet adhesiveness went on rising with severity of toxaemia. The baby weight went on falling with severity of toxaemia. Platelet counts were higher in mothers who gave birth to a living child as compared to those who delivered a still born child. With increase in baby weight the platelet count also increased.

TABLE II
Relation of Platelet Values to Baby Weight in Control Cases

Baby weight	Total platelet count	Adhesive platelet count	Platelet adhesiveness
Below 2000 gms.	225000	205000	8.10%
2001-2500 gms.	275000	240000	9.5%
2501-3000 gms.	303630	256810	14.59%
3001-3500 gms.	282500	212750	23.58%
Above 3500 gms	286600	260000	9.87%

TABLE III
Relation of Platelet Values to Severity of Toxdemia of Pregnancy and Average Baby Weight

Severity	Total platelet	Adhesive platelet count	Platelet adhesiveness	Average
	count			baby weight
Mild	153250	119910	19.58%	2179 gms.
Moderate	149000	111200	30.00%	2160 gms.
Severe	144930	102370	31.40%	1877 gms.
Antepartum				The same of
eclampsia	122850	94710	32.70%	2150 gms.
Postpartum				
eclampsia	114000	80000	27.29%	2537 gms.

TABLE IV

Relation of Platelet Values to Baby Weight and Condition in Toxaemia Cases

Baby weight	Condition of birth	Total platelet count	Adhesive platelet count	Platelet adhesiveness
Below 1500 gms.	S.B.	103300	76220	24.75%
	alive	50000	40000	33.40%
1501-2000 gms.	S.B.	85330	55220	34.45%
	alive	144410	106410	28.30%
2001-2500 gms.	S.B.	75000	68000	10.0%
	alive	198000	150000	22.0%
Above 2500 gms.	S.B.	178330	141660	19.16%
	alive	225620	168500	31.80%

Comments

From the study it is evident that platelet counts are lower in toxaemia of pregnancy as compared to normal pregnancy. Similar results were obtained by Howie et al (1976). With severity of toxaemia the platelet count goes on falling (Imrie and Raper, 1972; Davidson and Lang, 1972; Howie et al, 1976; Kennan and Bell, 1957). In absence of other disorders, the development of thrombocytopenia reffected the participation of platelets in the process of disseminated intravascular coagulation (Davidson and Lang, 1972). Platelet values are lower in eclampsia than in severe pre-eclampsia (Pritchard et al, 1954; Kennan and Bell (1957), Beecham (1974) states that the severity of clotting mechanism did not always correlate with the severity of toxaemia. Convulsive thresholds were not particularly related to severity of coagulopathy. Baby weight and platelet counts go on falling with severity of toxaemia (Howie and McNicol, 1971). In the present study, the babies of eclamptic mothers weighed more than those of severely toxaemic though the platelet count in the former were lower than in the latter group. This can be explained by the fact that oedema is associated with higher concentration of oestrogen and the latter results from an

increased mass of well functioning placenta which in turn is associated with larger babies (Page, 1972). The weights of babies go on increasing with duration of pregnancy in normal as well as toxaemia cases (Das and Rosario, 1968; Chakrayarty, 1967; Handricks and Brenner, 1971; Peters and Arora, 1973; Kitzmiller et al, 1974; DeSouza et al, 1976). This is due to the fact that intravascular coagulation is not often disseminated in systemic circulation in pre-eclampsia, but is more likely a subtle localised chronic process organising from or occurring in placental bed. Platelet counts were higher in mothers who delivered a live born baby than those who delivered a still born child. Stillbirth rate goes on decreasing as the birth weight goes on increasing (Das and Rosario, 1968; Neutre, 1975). Platelet adhesiveness was higher in the study group as compared to control, and went on increasing with severity of toxaemia. Howie and McNicol (1971) did not find any corelation between platelet adhesiveness and severity of toxaemia. McKay (1964) found an increase in platelet adhesiveness with severity of toxaemia, but did not observe any change in platelet adhesiveness in essential hypertension cases. The increased platelet adhesiveness is due to platelet damage, which may be caused by damaged placental trophoblast and is related to a slowly progressive low grade process of disseminated intravascular coagulation in pre-eclampsia. A rise in platelet adhesiveness in mothers with still born babies as compared to live born babies also favour this explanation.

Summary

Total platelet count, adhesive platelet counts and platelet adhesiveness were studied in 25 normal pregnant cases and 50 patients of toxaemia of pregnancy. These values were correlated to the severity of toxaemia, baby weight and condition at birth. An explanation for these variations is sought.

Acknowledgement

We are thankful to Dr. (Mrs.) S. Agrawal, Prof. of Obst. & Gynaecology, and Dr. S. Agrawal, Dean, Medical College, Jabalpur. for their kind permission to publish this series.

References

 Beecham, J. B.: Obstet. Gynec. 43: 576, 1974.

- 2. Chakravarty, A. P.: J. Obstet. Gynec. Brit. C'wlth. 74: 247, 1967.
- 3. Das, S. K. and Pinto Rosario, Y.: J. Obstet. Gynec. India. 18: 53, 1968.
- Davidson, E. Jr. and Lang, L.: Obstet. Gynec. 29: 433, 1967.
- DeSouza, S. W., Joh, R. Q. and Richards, B.: Brit. J. Obstet. Gynec. 83: 292, 1976.
- Howie, P. W. and McNicol, C. P.: J. Obstet. Gynec. Brit. C'wlth. 78: 992, 1971.
- Howie, P. W., Purdie, D. W., Begg,
 C. B. and Practice, C. R. M.: The Lancet. 2: 33, 325, 1976.
- Lancet. 2: 33, 325, 1976.

 8. Handricks, C. H. and Brenner, W. E.:
 Am. J. Obstet. Gynec. 109: 225, 1971.
- Imrie, A. H. and Raper, G. C.: Brit. J. Obstet. Gynec. 84: 71, 1977.
- Kennan, A. L. and Bell, W. N.: Am.
 J. Obstet. Gynec. 73: 57, 1957.
- 11. Kitzmiller, J. L., Lang, J. E. and Partico, F.: Am. J. Obstet. Gynec. 118: 362,
- 12. McKay, D. G.: Circulation (Suppl. 11) 29: 66, 1964.
- Neutra, R.: Brit. J. Obstet. Gynec. 82: 382, 1975.
- Page, E. W.: J. Obstet. Gynec. Brit. C'Wlth. 79: 883, 1972.
- 15. Peters, E. and Arora, S.: J. Obstet. Gynec. India. 23: 293, 1973.
- 16. Pritchard, J. A., Weisman, R. Jr., Ratnoff, O. D. and Vosburgh, G. L.: New Eng. J. Med. 25: 89, 1954.