TOTAL PLATELET COUNT, ADHESIVE PLATELET COUNT AND PLATELET ADHESIVENESS IN INCOMPLETE ABORTION, MISSED ABORTION AND SEPTIC ABORTION†

by

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Changes in platelet count causing severe haemorrhage after abortion was reported more in cases of septic and missed abortions. Prolonged retention of dead foetus and septic abortion cause haemostatic defects, presumably, due to a deficiency of fibrinogen, factor VIII and platelets which are consumed when intravascular coagulation occurs. Autolysis of placenta and decidua allows entry into the maternal circulation of thromboplastic substances which convert prothrombin into thrombin, which in turn converts fibrinogen into fibrin with associated depletion of platelets and other clotting factors.

In 1936 Gemell demonstrated that the lack of circulating fibrinogen is not an isolated defect, but low platelet count together with low levels of factors V, VIII and IX are usually present at the same time.

Material and Methods

The present study was carried out in the Departments of Obstetric & Gynaecology and Physiology of Medical College, Jabalpur from October 1973 to August 1974.

Twenty-seven cases of incomplete abortion acted as control, while 10 cases of missed abortion and 22 of septic abortion formed the study group.

Platelet count was estimated on admission, 24 hours after evacuation and 7 days after evacuation. The platelet estimations were estimated by Rees and Ekers method.

Observations

Patients of similar age and parity were selected in each group. Age, parity and duration of pregnancy had no influence on platelet values. The Table give the platelet values in cases of incomplete abortion, missed abortion and septic abortion cases.

Comments

The total platelet count was lower in cases of septic and missed abortions as compared to incomplete abortion. The values in missed abortion were lowest, which is due to reversal of pregnancy changes to non-pregnant levels.

The platelet adhesiveness was highest in septic abortion group.

24 hours after evacuation, the platelet count showed a little rise in incomplete and missed abortion but slight fall in

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TABLE I

Platelet Count in Incomplete Septic and Missed Abortions (mean values)

	On admission	24-hours after evacuation	7-days after evacuation
Incomplete abortion			
Total platelet count	220074	232962	221100
Adhesive platelet count	22777	25850	24200
Platelet adhesiveness	12.12%	11.4%	13.0%
Septic abortion			
Total platelet count	217545	209909	223333
Adhesive platelet count	31277	24600	26166
Platelet adhesiveness	14.0%	9.2%	11.6%
Missed abortion			
Total platelet count	199800	198600	191600
Adhesive platelet count	23400	27400	25520
Platelet adhesiveness	13.3%	13.8%	12.2%

septic abortion group. The platelet adhesiveness also showed a fall. This is compatible with the findings in labour and immediate postpartum. The effect of operation and anaesthesia is also evident.

Mor *et al* (1960), Slunsky (1964) and Shukla *et al* (1974), also noted a fall in platelet values after interruption of pregnancy. This was attributed to the destruction of tissues and their absorption and utilization of thrombocytes for haemostasis. Genell (1936) Dawbarn *et al* (1928) and Windfield and Genell (1936) noted a rise in platelet values.

The anaesthetic agents inhibit platelet aggregation. The exact mechanism is not known, but the anaesthetics stabilise the membranes in various ways. They also alter the surface characteristics of blood platelets (Udea, 1971).

Therefore, the factors affecting the platelet values are complex—they are, labour, puerperium, anaesthetics and operative trauma.

The platelet stickiness fell 24 hours after evacuation, and then rose again. The fall may be due to effects of anaesthetic agents, otherwise a rise of platelet stickiness in postoperative period is expected. The rise after seven days corresponds to the period when thromboembolic phenomenon are observed.

Septic Abortion

Endotoxin, even in small concentration, which doesnot produce shock, has a direct action on blood platelets. It is assumed that a subnormal platelet count with an otherwise normal coagulation profile in septic patients is suggestive of endotoxaemia and gram negative organism insection. There is disseminated platelet accumulation in capillaries and arterioles of heart, kidneys, liver and lungs, causing thrombocytopenia (Oscar, 1955, Jack and Pritchard 1959, Louse Lang 1967, Mckay 1967, Fritzk 1973). Thus, we find lower platelet count and raised platelet stickiness in septic abortion cases.

Missed Abortion

Platelet count was lower in missed abortion cases but platelet stickiness was raised. The exact mechanism for this variation is not known, but is probably

due to autolysis of placenta and decidua, which allows entry into the maternal circulation, of thromboplastic substances, thereby reducing platelet count and promoting intravascular clotting and depleting other clotting factors, especially factors V and VIII and increasing the risk of bleeding. (Jack and Pritchard 1955, Jackson and Hartman 1955, Oscar *et al* 1955, Hodgkinson *et al* 1954, Mor *et al* 1960, Talbert *et al* 1960).

Thus, the variations in platelet count and stickiness in septic and missed abortions explain the phenomenon of hypofibrinogenemia, so commonly observed in these cases.

Summary

Total platelet count, adhesive platelet count and platelet adhesiveness was estimated in 27 cases of incomplete abortion, 22 cases of septic abortion and 10 cases of missed abortion.

Total platelet counts were lower in septic and missed abortion cases.

There was a fall in the levels of platelets soon after evacuation. This could be attributed to absorption of necrotic material from endometrium, and effect of anaesthesia. This fall was followed by a rise which corresponded to the period when the thrombo-embolic complications are expected.

The fall in the total platelet count and rise in platelet stickiness in cases of septic and missed abortion cases may be explained by intravascular clotting in capillaries and arterioles, thereby depleting the circulating platelets and increasing the risk of haemorrhage due to hypofibrinogenemia.

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