



Assessment of Platelet Profile of Healthy Volunteers in the Trimesters of Pregnancy in Benin City, Nigeria

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ABSTRACT: These studies aimed at assessing platelet profile in the three trimesters of pregnancy. Subjects were apparently healthy volunteers who were staff, students and antenatal patients of a Hospital in Benin. Platelet profile was assessed using Swelab Alfa Basic model Haematological Analyser (Boule Medical AB, Stockholm, Sweden). The results were presented as mean \pm SEM in tables and graphs and analysed using One Way Analysis of Variance and Duncan post Hoc test and $p < 0.05$ was considered significant. The study showed that platelet count $\times 10^3/\mu\text{l}$ (277.83 ± 36.33 ; first trimester $> 193.60 \pm 13.15$; second trimester $> 185.50 \pm 11.58$; third trimester) and plateletcrit in percentage (0.22 ± 0.03 ; first trimester $> 0.17 \pm 0.01$; second trimester $> 0.16 \pm 0.01$; third trimester) reduced progressively as pregnancy advanced and this was statistically significant ($p < 0.05$) when compared to control of $276.17 \pm 34.32 \times 10^3/\mu\text{l}$ and 0.20 ± 0.20 respectively. A similar pattern was observed for Mean Platelet Volume (MPV). However, Platelet Width Density (PDW) in percentage increased (10.52 ± 0.22 ; first trimester $< 13.44 \pm 0.30$; second trimester $< 13.65 \pm 0.41$; third trimester) as pregnancy advanced. Thus PDW compensates for these reductions and a lack of this compensatory increase may aggravate the effect of thrombocytopenia in pregnancy. ©JASEM

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Keywords: Platelet Count, Plateletcrit, Mean Platelet Volume, Platelet Distribution Width, Trimesters, Pregnancy

Platelet studies have evolved from its customary function in hemostasis to other functions such as inflammation, innate immunity, wound healing and angiogenesis (Kakali, 2013). In pregnancy several hematological, biochemical and hemodynamic modifications occur as part of the physiological adaptation of the body to this condition (Rodríguez-Dennen, 2011). These adaptations are meant to maintain the maternal, fetal and uteroplacental homeostasis. Thrombocytes or platelets, red cells and white cells are essential for hemostasis, transport of oxygen and immunity respectively and their physiologic modification are particularly important during pregnancy. However, there have been controversies about the pattern of platelet profile in normal pregnancy, for instance Mohapatra *et al* (2007) reported that platelet count increased as pregnancy advanced. This is in contrast to other studies that reported a fall in platelet count as pregnancy advanced (Bonar *et al.*, 1969; Sejeny *et al.*, 1975; Karim *et al.*, 1992). Also, some researchers reported that there was no change in the platelets count as pregnancy advanced (Todd *et al.*, 1965; Ratnoff., 1954; Tygart *et al.*, 1986) while others reported a fall of platelet count from the first trimester and a rise in the third trimester (Amah-Tariah *et al.*, 2011). Amongst Chinese women, a gradual decrease in platelet count with no change in mean platelet volume has been reported (Liu *et al.*,

2011). Most of the studies done on platelet in pregnancy did not involve other platelet parameters such as Mean Platelet Volume (MPV), Plateletcrit (PCT) and Platelet Density Width (PDW). This study is therefore aimed at assessing plate count and these additional parameters in the three trimesters of pregnancy.

MATERIALS AND METHODS

Study population: This cross sectional study was conducted on students, staff and antenatal patients of a hospital in Benin. A total of 400 apparently healthy subjects aged between 23-27 years and parity between 0-4 volunteered for the study. 100 subjects were in the non-pregnant control group while each of the three trimesters of pregnancy had 100 subjects each. Ethical clearance for the study was obtained from the Ethics and Collaboration Committee of the hospital. Informed consent was also obtained from each subject who volunteered for the study. Volunteers with history of cardiovascular disease in pregnancy, bleeding disorders, diabetes and drugs capable of affecting platelet count were excluded from the study.

Measurement of Blood Pressure: Blood pressure was measured and recorded in mmHg as follows: after a rest period of about 30 minutes, the systolic and diastolic pressures were measured in each subject in

the sitting position on the brachial artery using auscultatory method. Diastolic pressure was determined as the disappearance of the Korotkoff's sound.

Blood Sample collection: 5ml of blood was drawn with minimum stasis from ante-cubital vein of subjects between 9am and 11 am each day in comfortable position and stored in an EDTA anticoagulated tube for platelet analysis. The blood samples were properly labeled and stored at room temperature until ready for analysis. All blood samples were collected between 9am and 11 am each day and analyzed within thirty minutes of collection.

Sample analysis: Analysis of blood was done within thirty minutes of collection and the sample was thoroughly and gently mixed before analysis. Platelet count (PLT), Platelet Width Density (PDW), Plateletcrit (PCT), Mean Platelet Volume (MPV) were determined by flow cytometry using the Swelab Alfa Basic model hematological autoanalyzer (Boule Medical AB, Stockholm, Sweden) at room temperature ($27.5 \pm 0.5^\circ\text{C}$).

Statistical Analysis: Data were presented as mean value \pm standard error of mean. The means were tested for homogeneity using one way analysis of variance of Micro soft excel 2010 and the significance between means was tested using the Duncan post hoc test of SPSS version 20. A difference between two means was considered to be statistically significant with a p value < 0.05 .

RESULTS AND DISCUSSION

Of the four hundred subjects that volunteered for the study, twenty six were disqualified because of the exclusion criteria enumerated in the methodology. Eleven of the disqualified subjects were from the first trimester of pregnancy, while fifteen were from the third trimester of pregnancy. No subject was disqualified from the second trimester of pregnancy and the non-pregnant control. Thus a total of 374 subjects i.e. 93.50% of the volunteers were involved in the study. Table 1 shows the subjects demographics.

Table 1: Subjects

Parameters	CONTROL (n=100)	1 st Trimester (n=89)	2 nd Trimester (n=100)	3 rd Trimester (n=85)
Age(yrs)	24.11 \pm 0.11	23.30 \pm 0.33	25.00 \pm 2.50	25.59 \pm 1.50
Parity (range)	1-3	0-1	1-3	1-3
Blood pressure(mmHg)				
Systolic:	114.17 \pm 2.00	115.44 \pm 2.12	112.23 \pm 2.01	114.05 \pm 2.89
Diastolic:	72.64 \pm 1.81	73.11 \pm 1.34	70.23 \pm 1.09	71.45 \pm 0.78

Table 2: Platelet indices in the non-pregnant control and in three trimesters of pregnancy

Parameters	Control (n=100)	1 st Trimester (n=89)	2 nd Trimester (n=100)	3 rd Trimester (n=85)
PLT ($\times 10^3/\mu\text{l}$)	276.17 \pm 34.32	277.83 \pm 36.33 [#]	193.60 \pm 13.15*	185.50 \pm 11.58*
MPV (fL)	9.14 \pm 0.41	8.22 \pm 0.31 [#]	8.40 \pm 0.23*	8.00 \pm 0.29*
PDW (%)	10.21 \pm 0.18	10.52 \pm 0.22 [#]	13.44 \pm 0.30*	13.65 \pm 0.41*
PCT (%)	0.20 \pm 0.02	0.22 \pm 0.03	0.17 \pm 0.01*	0.16 \pm 0.01*

*P < 0.05 indicates a significant difference when compared to control while [#] P > 0.05 indicates no significant difference when compared to control.

This study revealed a fall in platelet count in the second and third trimesters of pregnancy (Table 2). This pattern of platelet count fall in the three trimesters of pregnancy was also observed in other studies (Sejny *et al.*, 1975; Karim *et al.*, 1992). The progressive fall in platelet count from mid-term to labor indicates a direct relationship between the plasma volume changes and the corresponding platelets counts (Shaper *et al.*, 1968). Nevertheless, the progressive fall in platelet count as pregnancy advanced did not cause thrombocytopenia in these trimesters of pregnancy as the values were still within normal range of 150,000 and 450,000 per microliter

of blood ($150\text{--}450 \times 10^9/\text{L}$). The decrease in the number of circulating platelets as pregnancy advances are due to normal physiologic changes characterized by increased blood volume, increase in platelet activation, increase platelet aggregation and increased platelet clearance (Matthews, 1990; Fay *et al.*, 1983). Furthermore, placenta size enlarges with progression of gestation and consequently more capturing and destruction of platelet ensue (Shehata *et al.*, 1999). Thrombocytopenia is fairly common in pregnancy and it is caused by accelerated platelet consumption and decreased production (Shamoon *et al.*, 2009). The etiology is unknown but is considered

to be due to the relative hemodilution in pregnancy (Coplestone, 1992; Dutta, 1998), amplified by the capture and destruction of platelets in the placenta (Shehata *et al.*, 1999). A decreased platelet count as a function of placenta size is emphasized by a study which reported that 70% of the thrombocytopenic pregnant women were in the third trimester of pregnancy and none in the first trimester (Shamoon *et al.*, 2009; Boehlen *et al.*, 2000). Multiparity with larger placenta size (Monga, 2006) have been shown to have a reducing effect on platelet count possibly due to increased coagulation system in the placenta. Although, Mean platelet volume (MPV) and platelet distribution width (PDW) are known to increase during platelet activation (Vagdatli *et al.*, 2010) only the latter increased in this study (Table 2). The observed increase in PDW is probably a physiologic compensation for the decreasing platelet count and volume as pregnancy advanced. Platelets having denser granules are bigger in size and metabolically more active (Dogan *et al.*, 2012; Valkila *et al.*, 1994). PDW is more specific than MPV for identification of platelet activity and it is a simple, practical and specific marker for enhanced coagulation (Vagdatli *et al.*, 2010). The plateletcrit in this study also decreased as pregnancy advanced possibly because of the hemodilution (Coplestone, 1992) of mid pregnancy till labour.

The findings from this indicates that platelet count, Mean Platelet Volume (MPV) and plateletcrit reduced as pregnancy traverses its trimesters and this reduction is compensated for, by a physiologic increase in Platelet Width Density (PWD). However, pregnant women who have a setting for thrombocytopenia may be at a higher risk of the effect of this disorder in the third trimester of pregnancy. Thus, PDW should be part of the routine investigations for ante natal women especially in the third trimester as a defect in this compensatory increase may be the reason for the observed effects of thrombocytopenia.

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