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Research Article

Studies on Some Cell Adhesion Molecules (P- and E-selectin concentrations) Among Pregnant Women Attending Antenatal Clinics at Federal Teaching Hospital, Owerri, Nigeria

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Background: Pregnancy produces wide-ranging cardiovascular, haematological, immunological and vascular adaptations. These changes support a healthy pregnancy, but excessive inflammation and endothelial activation may contribute to complications, especially those associated with abnormal placentation and endothelial dysfunction. P-selectin and E-selectin mediate contact between circulating blood cells and the vascular endothelium and may therefore reflect these processes.

Methods: This comparative cross-sectional study enrolled 120 women: 60 apparently healthy non-pregnant controls and 60 pregnant women distributed across the first, second and third trimesters. Four millilitres (4 mL) of venous blood was collected into a plain container. Serum P-selectin and E-selectin concentrations were measured by immunoassay. Data were analysed with SPSS version 20 and reported as mean ± standard deviation. Independent-samples t tests and analysis of variance were used for group comparisons. Statistical significance was set at $p < 0.05$.

Results: Third-trimester P-selectin was significantly higher than the control value (17.01 ± 0.49 ng/mL versus 16.12 ± 0.12 ng/mL; $p = 0.012$). E-selectin was also higher in the third trimester than among controls (14.95 ± 0.43 ng/mL versus 13.58 ± 0.18 ng/mL; $p = 0.001$). In contrast, mean P-selectin and E-selectin concentrations in the first and second trimesters were significantly lower than the corresponding control values.

Conclusion: P-selectin and E-selectin concentrations vary with the stage of pregnancy and rise above control levels in the third trimester. They may be useful indicators of vascular activation, but larger longitudinal studies are needed before either marker can be recommended for routine antenatal assessment.

Keywords: *P-selectin, E-selectin, pregnancy, cell adhesion molecules, endothelial activation, pre-eclampsia*

Introduction

Pregnancy requires extensive adaptation in nearly every maternal organ system. Cardiovascular, haematological, immune, metabolic and vascular changes develop together to support placentation and fetal growth. Most are

physiological, but poorly regulated inflammatory or vascular responses can contribute to pre-eclampsia, gestational hypertension, fetal growth restriction and other disorders of placentation (Chaiworapongsa et al., 2002; Mistry et al., 2020). Cell adhesion is one of the processes connecting inflammation, vascular function and pregnancy. It describes contact between cells or between cells and the extracellular matrix and is controlled by cell adhesion molecules (CAMs). These proteins contribute to tissue organization, cell migration, immune surveillance, inflammation and vascular integrity. During implantation and placentation, they help coordinate interactions among trophoblasts, maternal endometrial cells, endothelial cells and immune cells (Alberts et al., 2014). The principal CAM families are integrins, cadherins, members of the immunoglobulin superfamily and selectins. Selectins are calcium-dependent, carbohydrate-binding proteins that initiate contact between circulating blood cells and the vascular endothelium. L-selectin is found mainly on leukocytes, E-selectin on activated endothelial cells, and P-selectin on activated platelets and endothelial cells (McEver, 2015). Selectins act early in the inflammatory adhesion cascade. After endothelial activation, they bind carbohydrate-containing ligands on circulating leukocytes, producing brief attachments that allow the cells to roll along the vessel wall. Leukocyte integrins are then activated, leading to firm adhesion and migration across the endothelial barrier (Zarbock et al., 2011; McEver, 2015). E-selectin, or CD62E, is produced by endothelial cells in response to inflammatory cytokines, including tumour necrosis factor-alpha and interleukin-1β. Its expression requires new protein synthesis, unlike P-selectin, which can be released quickly from intracellular stores. Circulating E-selectin is therefore commonly interpreted as an indicator of endothelial activation (Bevilacqua & Nelson, 1993). P-selectin, or CD62P, is stored in endothelial Weibel-Palade bodies and platelet alpha-granules. Endothelial or platelet activation rapidly brings it to the cell surface, where it supports contact among platelets, leukocytes and endothelial cells. This rapid response allows P-selectin to take part in both inflammation and haemostasis (Rondaij et al., 2006). Pregnancy is a useful physiological setting in which to examine these molecules. Maternal circulation must meet increasing demands and maintain placental perfusion, while the immune system must tolerate the fetus without losing its ability to respond to infection and inflammation (Feng et al., 2017; Mistry et al., 2020). At the maternal-fetal interface, selectins and their ligands contribute to implantation, trophoblast migration and maternal immune recognition. P-selectin and E-selectin have also been associated with trophoblast movement within decidual spiral arteries and other cellular interactions at this interface (Feng et al., 2017). The vascular endothelium is central to these effects. Under resting conditions, endothelial cells present a relatively non-adhesive surface. Activation makes the surface more adhesive to leukocytes and platelets. When this response is excessive or prolonged, it may promote vascular inflammation and dysfunction (Zarbock et al., 2011; McEver, 2015). These changes are especially relevant to pre-eclampsia. Its pathogenesis is complex but includes abnormal placentation, inadequate spiral-artery remodelling, placental ischaemia, inflammation and maternal endothelial dysfunction. Studies have reported higher soluble adhesion-molecule concentrations in pregnancy, particularly where endothelial activation and pre-eclampsia are present (Chaiworapongsa et al., 2002). Soluble P-selectin and E-selectin can be measured in maternal blood. Their circulating concentrations may reflect cellular activation or shedding of the corresponding membrane-bound molecules, making them possible markers of vascular activity (Chaiworapongsa et al., 2002). This study assessed P-selectin and E-selectin concentrations among pregnant women attending antenatal clinics at Federal Teaching Hospital, Owerri, and compared the findings across trimesters with those of non-pregnant controls.

Materials and Methods

Study area and design

The study was carried out at Federal Teaching Hospital, Owerri, Nigeria. A comparative cross-sectional design was used. Pregnant women attending antenatal clinics at Federal Teaching Hospital, Owerri, formed the study group, while apparently healthy non-pregnant women served as controls. Pregnant participants were classified as being in the first, second or third trimester according to gestational age.

Study population

The study included 120 participants. Sixty were pregnant women and 60 were apparently healthy non-pregnant controls. The pregnant participants were distributed across the three trimesters.

Sample collection

Four millilitres of venous blood was collected aseptically from each participant into a plain container and allowed to clot. After centrifugation, the serum was separated for P-selectin and E-selectin measurement. Standard precautions were observed during collection, processing and storage to limit pre-analytical error.

Laboratory analysis

Serum P-selectin and E-selectin concentrations were measured by immunoassay in accordance with the instructions supplied with the respective assay kits.

Statistical analysis

Data were analysed with SPSS version 20 and summarized as mean ± standard deviation. Independent-samples t tests and analysis of variance (ANOVA) were used to compare groups. A p value below 0.05 was considered statistically significant.

Results and Discussion

Table 1: Mean Levels of Cell Adhesion Molecules (P-selectin and E-selectin) of Pregnant Women based on Trimester Compared to Non-Pregnant Women.

Trimester	Cell Adhesion Molecules	Pregnant Women	Non-pregnant Control Women	p-value
First Trimester	P-selectin	14.10 ± 0.44	16.12± 0.12	0.000*
Second Trimester		14.30 ± 0.45		0.000*
Third Trimester		17.01 ± 0.49		0.012*
First Trimester	E-selectin	11.79 ± 0.39	13.58± 0.18	0.000*
Second Trimester		12.46± 0.41		0.005*
Third Trimester		14.95 ± 0.43		0.001*

P-selectin and E-selectin concentrations differed according to trimester when compared with the non-pregnant control group. Mean P-selectin concentration in the third trimester was 17.01 ± 0.49 ng/mL, compared with 16.12 ± 0.12 ng/mL among controls. This difference was statistically significant (p = 0.012). Mean E-selectin concentration was also higher in the third trimester than in the control group (14.95 ± 0.43 ng/mL versus 13.58 ± 0.18 ng/mL; p = 0.001). Thus, both markers were above the control values in late pregnancy. P-selectin and E-selectin concentrations were significantly higher in the third trimester than in non-pregnant controls. Normal pregnancy involves changes in platelet, leukocyte and vascular activity, so this pattern is biologically plausible. The degree and clinical meaning of this activation, however, differ from the more marked endothelial disturbance described in pre-eclampsia (Chaiworapongsa et al., 2002; Mistry et al., 2020). Selectins mediate early contact between blood cells and the vascular endothelium. They support leukocyte tethering and rolling before firm adhesion and movement through the endothelial layer (Zarbock et al., 2011; McEver, 2015). Changes in circulating selectin concentrations may therefore indicate changes in vascular activation. The higher third-trimester P-selectin concentration may reflect greater platelet activation, endothelial activation, or both as pregnancy advances (Chaiworapongsa et al., 2002; McEver, 2015). P-selectin is held in platelet alpha-granules and endothelial Weibel-Palade bodies. Cellular activation rapidly moves it to the surface, where it binds P-selectin glycoprotein ligand-1 (PSGL-1) on leukocytes and supports cell-to-cell contact (Rondaij et al., 2006). Through these interactions, P-selectin connects haemostatic and inflammatory responses. Activated platelets bind leukocytes, while activated endothelial cells recruit them to the vessel wall. This is relevant in pregnancy because maternal haemostasis gradually shifts towards a procoagulant state. The third-trimester increase recorded in this study may therefore be part of normal physiological adaptation rather than a sign of disease by itself. For this reason, an increased selectin concentration in an otherwise healthy pregnant woman should not automatically be interpreted as endothelial disease. Blood pressure, proteinuria, platelet count and other clinical or laboratory indicators must be considered when pregnancy complications are suspected (Chaiworapongsa et al., 2002; Mistry et al., 2020). P-selectin has been identified in placental tissue and maternal plasma and may contribute to placental haemostasis and cellular interactions at the maternal-fetal interface (Uszyński et al., 2008). The third-trimester increase in E-selectin also points to greater endothelial activity. E-selectin is expressed mainly by activated endothelial cells and, unlike P-selectin, is not kept in substantial preformed stores for immediate release. Inflammatory mediators, particularly tumour necrosis factor-alpha and interleukin-1β, stimulate its production (Bevilacqua & Nelson, 1993). The higher concentration found in late pregnancy may therefore reflect the vascular and inflammatory adjustments that accompany advancing gestation. Evidence from previous pregnancy studies supports this interpretation. In a prospective study of women enrolled at 12–16 weeks of gestation, soluble E-selectin concentrations were higher among those who later developed pre-eclampsia than among those whose pregnancies remained uncomplicated. This finding suggests that E-selectin may sometimes signal vascular disturbance before clinical disease becomes apparent. Altered adhesion-molecule concentrations have also been reported in established pre-eclampsia (Mistry et al., 2020). The present study did not specifically enrol women with pre-eclampsia or other pregnancy complications. The increase in E-selectin should therefore be interpreted as a change in endothelial activation during pregnancy, not as evidence that the participants had pre-eclampsia. Diagnosis of

that condition requires clinical and laboratory assessment beyond a single adhesion marker (Chaiworapongsa et al., 2002; Mistry et al., 2020). Larger longitudinal studies should determine whether the observed changes remain after adjustment for gestational age, maternal age, parity, blood pressure, body mass index, inflammation and other potential confounders. Endothelial dysfunction is a recognised feature of pregnancy complications, particularly pre-eclampsia. Healthy endothelium helps regulate vascular tone and coagulation while limiting inappropriate blood-cell adhesion. During inflammation, activated endothelial cells express adhesion molecules that recruit leukocytes (McEver, 2015; Mistry et al., 2020).

E-selectin is closely tied to this response because inflammatory cytokines strongly induce its expression (Bevilacqua & Nelson, 1993). P-selectin appears rapidly after platelet or endothelial activation and promotes platelet-leukocyte contact (McEver, 2015). Higher circulating concentrations may therefore reflect increased interaction among endothelial cells, platelets and immune cells. The results support the view that vascular activation changes over the course of pregnancy. Selectins also contribute to events at the maternal-fetal interface, although this study examined women across trimesters rather than the implantation period itself (Feng et al., 2017). Implantation depends on controlled adhesion between trophoblasts and maternal tissues. E-selectin and P-selectin are expressed at the implantation site and may assist interactions between trophoblasts and decidual endothelial cells (Burrows et al., 1994). Feng and colleagues described the involvement of selectins and their ligands in implantation, maternal immune recognition and trophoblast migration within decidual spiral arteries (Feng et al., 2017). These functions show that selectins are involved in normal reproductive biology as well as inflammation. The possible clinical value of P-selectin and E-selectin arises from their involvement in inflammatory, endothelial and haemostatic processes that also feature in pregnancy complications (Chaiworapongsa et al., 2002; Mistry et al., 2020). Pre-eclampsia involves abnormal placentation, inadequate maternal vascular adaptation, inflammation and endothelial dysfunction. Studies have reported increased E-selectin expression and changes in P-selectin in affected pregnancies (Mistry et al., 2020). In one study of maternal, fetal and placental selectins, placental E-selectin expression was higher in women with pre-eclampsia and was interpreted as evidence of the endothelial dysfunction associated with the condition (Mistry et al., 2020). Circulating soluble adhesion molecules have also differed between normal and pre-eclamptic pregnancies (Chaiworapongsa et al., 2002). Selectins could therefore merit evaluation as components of a broader biomarker panel. Selectins are not specific to pregnancy-related disorders; they are also involved in many inflammatory, thrombotic and vascular conditions. Their clinical usefulness is therefore more likely to lie in combination with symptoms, examination findings and other laboratory markers than as stand-alone tests (Zarbock et al., 2011; McEver, 2015).

Clinical implications

The findings suggest that P-selectin and E-selectin vary across pregnancy, with both markers rising above non-pregnant control values in the third trimester. The concurrent increase supports further study of the relationship between platelet and endothelial activation in late pregnancy and of whether early selectin changes identify women at greater risk of complications. These results are not sufficient to support routine antenatal testing for P-selectin or E-selectin. Before either marker is introduced into screening, larger prospective studies must establish diagnostic accuracy, gestation-specific reference intervals, additional clinical value and cost-effectiveness (Mistry et al., 2020).

Limitations

The findings should be considered in light of several limitations. The study was conducted at one health facility, which limits the extent to which the findings can be applied to pregnant women in other settings. The sample size was also relatively small. Because the design was cross-sectional, the study could not establish how individual selectin concentrations changed over time or whether those changes preceded specific pregnancy outcomes. Participants were not followed to determine whether raised P-selectin or E-selectin predicted pre-eclampsia, gestational hypertension, fetal growth restriction, preterm delivery or other adverse outcomes. Infection, obesity, smoking, medication use, parity, maternal age and other inflammatory conditions may affect adhesion-molecule concentrations. Future studies should measure and control for these factors.

Conclusion

P-selectin and E-selectin concentrations were higher in the third trimester than among non-pregnant controls. The increase in P-selectin may reflect greater platelet and endothelial activation, while the E-selectin finding is consistent with increased endothelial activation as pregnancy advances (Bevilacqua & Nelson, 1993; Chaiworapongsa et al., 2002). These findings agree with evidence that selectins contribute to leukocyte recruitment, platelet-endothelial contact, implantation, trophoblast migration and vascular regulation during pregnancy (Zarbock et al., 2011; McEver,

2015; Feng et al., 2017). Although both markers may help characterize vascular activation, neither should be interpreted alone as diagnostic of a pregnancy complication. Their place in a multimarker approach requires prospective validation (Mistry et al., 2020).

Recommendations

The findings support the following recommendations:

1. Conduct larger multicentre studies of P-selectin and E-selectin among pregnant women in Nigeria.
2. Follow women from early pregnancy through delivery to determine whether early selectin changes predict later complications.
3. Compare uncomplicated pregnancies directly with pregnancies affected by pre-eclampsia, gestational hypertension, fetal growth restriction and other complications.
4. Assess P-selectin and E-selectin with established markers of endothelial, inflammatory and placental dysfunction to determine whether a combined panel improves prediction.
5. Examine the relationship between selectins and haemostatic measures such as platelet activation markers, von Willebrand factor, fibrinogen, D-dimer and other coagulation indices.
6. Establish gestation-specific reference intervals before considering either marker for routine clinical use.
7. Continue to use established antenatal investigations while stronger evidence on the diagnostic accuracy, clinical usefulness and cost-effectiveness of selectin testing is developed.
8. If future studies confirm adequate predictive value, evaluate affordable selectin testing within antenatal risk-assessment programmes, particularly in resource-limited settings.

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Author’s contributions

Okoro Blessing C. contributed to the study conception, participant recruitment, sample collection, laboratory analysis, data organization and initial manuscript preparation. Aloy-Amadi Oluchi C. supervised the study, contributed to its design and interpretation, and critically revised the manuscript for intellectual content. Enyereibe Marvellous U. assisted with laboratory analysis, data management and manuscript review. All authors read and approved the final manuscript.

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Conflict of interest

The authors of this research work declare no conflict of interest whatsoever.

Ethics approval

The appropriate research ethics committee approved the study before participant recruitment and sample collection. All procedures followed accepted ethical principles for research involving human participants.

AI tool usage declaration

ChatGPT (OpenAI) was used to edit the manuscript for grammar, clarity and readability. It was not used to generate or interpret the results, formulate the scientific conclusions, or create citations and references. The authors reviewed and verified the edited text and accept responsibility for the manuscript's accuracy, originality and integrity.

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