Platelets in pregnancy

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Summary

As stated in this review, platelets functions and their important role in coagulabity in pregnancy must be well understood, not only in thrombosis related complications in pregnancy (i.e., hypertension, diabetes, thrombophilia).
Clinical findings suggest that a periodical monitoring of haematological markers such as MPV and coagulation markers may be associated to Doppler velocimetry, keeping in mind also that the incidence of complications is increased in women who have heritable platelet function disorders.

Key Words: *platelets, coagulation, high risk pregnancies.*

Introduction

The platelet is one of the key elements of human blood. Platelets play an essential role in the process of thrombogenesis, as well as an important role in atherogenesis and the progression of atherosclerotic lesions (1). The interaction of the platelets with the vessel wall and its subsequent contribution to atheroma formation and thrombosis is of pivotal importance in the aetiology and pathogenesis of peripheral, coronary, cerebrovascular and other vascular diseases (2). Acknowledgment of the fact that platelets have a central role to play in these disease states has led to a considerable amount of research into its pathophysiology and understanding (3). Inappropriate platelet activation is common in atherosclerosis and many of its risk factors, such as smoking and diabetes, play a prime role in the increasing heart disease burden of society. There is still no generally accepted ideal measure of platelet activation that would indicate a state of 'high risk'. There is also a need for the objective assessment of the relative effectiveness and safety of antiplatelet agents (4,5). A normal platelet count in a healthy individual is between 150,000 and 450,000 per µl (microlitre) of blood (150-450 x 10^9/L). Ninety-five percent of healthy people will have platelet counts within this range. Some will have statistically abnormal platelet counts while having no demonstrable abnormality. However, if it is either very low or very high, the likelihood of an abnormality being present is higher. Early diagnosis of progressive activation of coagulation may help manage these diseases successfully (6,7). Platelet activation markers could be a useful guide in order to distinguish different subgroups of patients, following the observation that stroke patients with carotid artery disease exhibit significantly more platelet activation than those strokes with a cardioembolic aetiology (8). There is also the potential of confounding effects from concomitant drug therapy, such as the non-steroidal antinflammatory agents or steroids.

Material and Methods

With introduction of Coulter counter new hemathologic parameters have became available to the clinician, one of those is mean platelet volume (MPV). MPV is a measurement of the average size of platelets found in blood and is typically included in blood tests. Since the average platelet size is larger when the body is producing increased numbers of platelets, MPV test results can be used to make inferences about platelet production in bone marrow (9).

Results

Hereafter, some researches regarding platelets involvement in several pregnancy conditions.
Pregnancy and platelets: after a brief presentation regarding platelets function in normal population, the following paragraphs represent the objective of this review, the platelets function in pregnancy and related complications.
Normal platelets changes in pregnancy: pregnancy is characterized by a physiological rise in the strain exerted upon the endothelium. Maternal constitutional factors giving rise to endothelial stress may represent a predictive value on pregnancy outcome regarding the development of hypertensive disorders in high-risk pregnancies. Normal pregnancy is characterized by an increase in platelet aggregation and a decrease in the number of circulating platelets with gestation (10).

Platelet lifespan declines and the MPV increases minimally during pregnancy (11)**.** Increased consumption of platelets in the uteroplacental circulation has been suggested to be the explanation of the reduction in the number of circulating platelets.

Steroids in pregnancy: by decreasing inflammation, corticosteroids that are potent anti-inflammatory drugs, may be an effective treatment for thrombocytopenia in pregnant women. A recent study performed by Giannubilo et al (12) observed the effect of betamethasone for fetal lung maturity promotion on platelets count in both thrombocytopenic and normal pregnant women. Low doses of betamethasone (12 mg I.M. in two consecutive days) showed no effect on platelets parameters in pregnant women affected by threatened preterm delivery and with normal platelet count (13).

Hypertension in pregnancy and platelets: platelet count falls early in hypertension and precede renal changes, proposing an active role of platelet consumption in the pathophysiology of this disorder. A reduction in platelet count and an elevated platelet size are common features of hypertension in pregnancy (14-18). In the early stages of hypertension in pregnancy, platelet aggregation is increased, in established severe disease it is decreased (19). In the clinical phase of hypertension in pregnancy, the typical case picture is one of a vasoconstrictive state with low plasma volume and cardiac output, high blood pressure and systemic vascular resistance in combination with signs of organ damage [proteinuria, hemolysis elevated liver enzymes low platelets (HELLP) syndrome]. Hemodynamic management is necessary in severe disease to prevent maternal complications (20-22). The significant higher volume of platelet and lower volume of RBC in hypertension in pregnancy, may indicate the severity of disease.

Insufficiency of the uteroplacental circulation due to failure of trophoblastic invasion of the spiral arteries is supposed to be a common etiological factor in both hypertension and IUGR (20). Inadequate cytotrophoblast invasion may constitute the impetus to endothelial cell dysfunction and increased activation of platelets. It is well known that there is platelets consumption because of uncontrolled intravascular platelets activation and fibrin deposition in hypertension in pregnancy (23,24). Increased platelets turnover and consequently more immature platelets in the maternal circulation may explain why MPV is increased. Several studies have proved that the changes in PTL aggregation and MPV occur in association with hypertensive states (9,25-28). Although an accepted model is abnormal placentation leading to widespread maternal endothelial dysfunction, interest has also been demonstrated in the role of platelets in the pathophysiology of hypertension in pregnancy.

The contact of platelets with the injured endothelium may represent the initial step of a coagulatory cascade which leads to increased consumption of platelets in the uteroplacental circulation with resultant reduction in the number of circulating platelets in the first phase of the process. Subsequently, there may be a compensatory increase in bone marrow production. In fact, there is evidence that in hypertension in pregnancy, the platelets production time is significantly reduced in comparison with normal pregnancies (29,30). Young platelets thrown in circulation are bigger and present a higher tendency to aggregation. Other studies showed previously that pregnancies with abnormal Doppler and linked hypertension show an enhancement of MPV and platelets aggregation (31).

Although the essential pathogenetic mechanism of preeclampsia is only partly understood, there is general according about a central role for dysfunctional endotheliumin triggering the typical clinical symptoms. Conditions such as hypertension, diabetes mellitus and thrombophilia are associated with extra strain upon the endothelial lining of the vascular bed (32). The deleterious consequence of increasing pressure, presumably to maintain blood flow, is partially compensated for by concomitant decrease in red cell volume, thus attempting to counteract the viscous effects of a larger relative red blood cell mass with smaller cell size characteristics.

Platelets and Diabetes in pregnancy: no significant changes in platelet count but higher MPV were described in diabetic pregnancies respect to controls (33,34). This may explain in part pathogenesis and development of diabetic complications. Higher MPV values denote higher platelets activity that may lead to hypercoagulability in the placental bed with consequent vascular events, which may responsible for fatal pregnancy complications.

Inherited coagulability defects and platelets: the incidence of pregnancy complications is increased in women who have genetic platelet function disorders (35,36). Defects affecting platelets during pregnancy can lead to heterogeneous complications, such as thrombosis, first trimester miscarriage and postpartum haemorrhage.

Conclusions

As stated in this review, platelets functions and their important role in coagulabity in pregnancy must be well understood, not only in thrombosis related complications in pregnancy (i.e., hypertension, diabetes, thrombophilia). Clinical findings suggest that a periodical monitoring of haematological markers such as MPV and coagulation markers may be associated to Doppler velocimetry, keeping in mind also that the incidence of complications is increased in women who have heritable platelet function disorders.

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