Introduction

Preeclampsia is a common complication of pregnancy, particularly in primiparous woman, and can result in serious consequences for the mother and the infant ***(Janes and Goodal, 1994)***.

Although the exact cause of preeclampsia remains to be elucidated, evidence shows that platelets play a substantial role in the pathogenesis of this disease. Local or systemic immunologic processes, endothelial cell damage, and placental activation are supposed to play an important role in this disease with involvement of multiple organs ***(Konijenberg et al., 1997)***.

Activation causes a conformational change in the platelet membrane GPIIb-IIIa exposing the receptor site for fibrinogen, and leading to binding plasma fibrinogen. The exposed fibrinogen binding site can be recognized with IgM monoclonal antibody (MAB), PAC-1, whilst plasma fibrinogen bound to platelet GPIIb- IIIa can be detected using either monoclonal or polyclonal anitifibrinogen antibodies ***(Janes and Goodall, 1994)***.

PECAM-1 (platelet endothelial cell adhesion molecule-1) or CD31 is a 130-KD integral membrane GP expressed on the surface of platelets, endothelial cells, monocytes and neutrophils.

Recently, they found that PECAM-1 becomes phosphororylated and associates with the platelet cytoskelton on platelet activation suggests that PECAM-I may be a major mediator of post-activation events on platelet.***(Cramer et al.,1994).*** P-selectin (CD62P) is an adhesion protein which was initially characterized in platelets where it was termed PADGEM (platelet activation dependent granule external membrane protein) ***(Carlos and Harlan, 1994).***

Janes and Goodall(1994) found that significant increased platelet surface expression of CD63, (a marker for secretion of platelet lysosomal granules) in preeclampsia compared with normotensive pregnancy. In a prospective study ***Janes et al.,*** also found an increased expression of CD63 at 28 weeks, gestation in pregnant women before the development of preeclampsia ***(Janes and Goodall, 1994).***

Aim of Work

The aim of the work is to investigate by means of flow cytometry using a panel of activation markers, the extent of platelet activation in normal pregnancy and whether this activation is more extensive during preeclampsia or not.