

third trimester of normal pregnancy as compared with PE (3.4 µg/ml vs. 3.1 µg/ml) or before as compared with after leukocyte therapy (4.5 µg/ml vs. 4.3 µg/ml). In conclusion, the data of this study indicated that the concentration of sCD30 in serum during pregnancy period is not correlated with RSA or PE. Furthermore, leukocyte therapy does not alter the level of sCD30 in serum. Finally, it seems that the measurement of sCD30 in serum may not be a good index for Th2 immune responses in pregnancy.

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## P28

### Anti-fibrillin-1 autotibodies in normal pregnancy and recurrent pregnancy loss

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Fibrillin-1 is an extracellular matrix glycoprotein, a main component of microfibrils as free bundles or in association with elastin-containing elastic fibrils. It is present in the structures undergoing intensive remodeling during menstrual cycle and pregnancy, hence endometrium and decidua. The aim of this study was to investigate anti-fibrillin-1 autoantibodies in patients with a history of recurrent pregnancy loss (RPL) and during normal pregnancy. Anti-fibrillin-1 IgG and IgM autoantibodies were measured by a home-made ELISA in serum samples of 48 medically and obstetrically normal pregnant women, classified to three trimester groups, 15 female patients with RPL, and 25 healthy non-pregnant women with a history of successful pregnancies. One-way analyses of variance and Least Significant Difference method were used for a statistical analysis. The levels of IgM anti-fibrillin-1 autoantibodies were significantly decreased in all pregnancy groups compared with the controls. Comparing RPL patients with the healthy non-pregnant controls showed significantly increased IgM anti-fibrillin-1 antibody levels. There were no significant differences in the levels of IgG anti-fibrillin-1 autoantibodies between the investigated groups. Variations in the serum levels of anti-fibrillin-1 IgM autoantibodies were established in normal pregnancy as well as in the

RPL patients compared with the healthy non-pregnant women. Increased anti-fibrillin-1 autoantibodies may contribute to the pathogenesis of immune-mediated pregnancy losses.

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## P29

### PGE1 versus PGE2: protective rather than inducing agent for endometriosis

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The ectopical implantation of endometrium leading to endometriosis has been correlated to various immune as well as non-immune parameters. Thus, inflammatory cytokines including IL-2, IFN-γ, TNF-α, IL-4 have been found to be increased in serum and peritoneal fluid of endometriotic patients at the same time that the elevated levels of prostaglandins PGE1 and PGE2 have been accused to facilitate the inflammation process. Using the experimental model of L-carnitine (L-Cn)-induced endometriosis, the aim of the present study was to explore the role of PGE1 and PGE2 in endometriosis development. During the treatment for endometriosis development with L-Cn, experimental mice were injected with specific PGE1 and PGE2 receptor inhibitors and their effect on “endometriosis specific” markers was examined. Thus, the levels of cytokines in the serum and peritoneal fluid were evaluated by ELISA experiments, whereas the immune cell content of the peritoneal cavity and uterus was tested by immunofluorescence and immunohistological techniques, respectively. The results showed that PGE1 but not PGE2 inhibition further increased the levels of IL-2, IFN-γ, TNF-α, GM-CSF, IL-4 and IGF-1 in the serum and to a lesser degree in the peritoneal fluid, while the number of immune cells was also found to be affected as compared to the L-Cn-treated endometriotic controls. Inhibition of PGE2, however, was shown to reverse the L-Cn-induced profile towards control values. The results indicated that at least PGE1 was not part of the endometriosis-inducing mechanism, but rather part of an anti-endometriotic resistance of the organism, trying to maintain a state of down-regulation of immune cells and inflammatory cytokine production.

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