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Abstracts

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nancy loss was 60% and 59% for SLE patients with Lupus Anti-Coagulants or Anti-Cardiolipin respectively, and 13% and 5% for SLE patients without Lupus Anti-Coagulants or Anti-Cardiolipin respectively. Pregnancy loss can occur in the first, second or third trimester of pregnancy. Even in the case of a successful pregnancy, Phospholipid antibody have been associated with other unfavorable outcomes such as preeclampsia, HELLP syndrome, chorea gravidarum, intrauterine growth retardation, stillbirth, preterm births, and thrombosis. In spite of considerable interest in these autoantibodies, the mechanism(s) implicated in the association between Phospholipid antibody and fetal wastage remains unclear. There are still doubts whether Phospholipid antibody has a pathogenic potential on their own, or whether they are epiphenomena. The cause of fetal wastage is thought to be thrombosis (or eventually vasculopathy) in placental and decidual vessels. A large number of potential pathological mechanisms inducing thrombosis have been proposed for Phospholipid antibody such as increased tissue factor generation, inhibition of the thrombomodulin-protein C pathway (the placenta is rich in thrombomodulin), interference with the prostacyclin-thromboxane balance, inhibition of the fibrinolytic system and so on, as well as combinations of them. Based on the clinical and laboratory heterogeneity of Phospholipid antibody, it is logical to assume a similar diversity for the pathophysiology of pregnancy loss. In this article a 31 year old woman will be presented without any clinical criteria of SLE. She experienced five consecutive fetal losses since 22-years of age. Laboratory evaluation revealed high titer ANA and Anti ds-DNA, associated with low complement levels. There was a history of ITP in her past history when she was eighteen. Neither dermatologic nor renal and CNS manifestations of SLE was detected.

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Lack of association between the TGF-β1 gene polymorphisms and recurrent spontaneous abortion

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Transforming growth factor-β1 (TGF-β1) is produced by T regulatory lymphocytes (Treg), which play an important role in the physiology of pregnancy. Several polymorphisms of the TGF-β1 gene (*TGFB1*) have been reported, some with an important correlation with TGF-β1 production and disease severity. We performed an association study between *TGFB1* polymorphisms and recurrent spontaneous abortion (RSA). We first used a PCR-RFLP method to detect three known *TGFB1* cSNPs (coding single nucleotide polymorphisms) among 111 RSA and 110 normal control women from southern Iran, such as 29 T–>C (Leu 10 Pro), 74G–>C (Arg 25 Pro) and 788C–>T (Thr 263Ile), and compared their frequencies between the two groups of subjects. To confirm results of the RFLP study and to identify new SNPs in the RSA women, we then sequenced their DNA samples for seven exons and adjacent intronic regions of *TGFB1*. Consequently, ten SNPs were detected, one (-14G–>A) was located in the upstream region of exon 1, three in exons (two in exon 1 and one in exon 5) and six in intronic regions. Two (IVS 5+18G–>C and IVS 6+910G–>A) of the ten SNPs were novel. Statistical analysis on the frequency of six most frequent SNPs including the three cSNPs, as well as on the frequencies of genotypes and 13 haplotypes regarding the 6 SNPs, revealed no significant difference between RSA and control women. Therefore, it is less likely that exonic and adjacent intronic polymorphisms of *TGFB1* are associated with RSA.