Obstetric and vascular antiphospholipid syndrome. Two sides of the same coin?

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Antiphospholipid syndrome (APS) is an autoimmune disease characterized by the presence of thrombosis and/or obstetric events, and persistent high titers of circulating antiphospholipid antibodies (aPL). Most patients display both clinical signs, although some patients can have isolated vascular or obstetric variants. aPL are a heterogeneous group of autoantibodies directed against negatively charged phospholipids or phospholipid-binding proteins. It seems that the same aPL are present in the two clinical manifestations of APS, but it is evident that vascular APS and obstetric APS have different pathogenic mechanisms, the first being caused by a state of hypercoagulability and the second by an inflammatory phenomenon. It is not yet clear if there are different specificities of these aPL. Sera or polyclonal IgG from women with several clinical manifestations of APS show differential effects on various cellular models in *in vitro* assays. On trophoblast cells there is a decrease of trophoblast invasion. On endothelial cells these aPL stimulated nitrotyrosine expression, reduction in eNOS phosphorylation at Ser1177, mitochondrial hyperpolarization and activation of autophagic pathways, expression of adhesion molecules and the generation of procoagulant microparticles. In conclusion, whether vascular APS and obstetric APS are two sides of the same coin, or two different coins altogether, has not yet been clarified. It would be very useful both for the diagnosis and for the therapeutic approach of the patients to define whether the determining factors of each variant of the disease are the aPL *per se* or whether there are other factors that influence the onset and pathogenesis of obstetric or vascular APS.