PATOGENIA SINDROMULUI ANTIFOSFOLIPIDIC

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Thrombosis is the defining feature of Hughes syndrome. Simple thrombosis does not explain many of the syndrome`s aspects, however, and it is possible that anticoagulation will prove to be an incomplete, insufficient treatment for many of its symptoms. We do not yet understand the pathogenesis of, and cannot assume the effectiveness of anticoagulation for livedo, valvulopathy, cognitive dysfunction, or renal thrombotic microangiopathy. We know that aPL arises years if not decades before clinical illness, a fact that encourages us to maintain clear distinctions between etiological agents and triggers of clinical events and between aPL and Hughes syndrome itself. The risk in a given circumstance for a new thrombosis, the effects of contributing factors, such as smoching,oral contraceptive treatment , or surgery on thrombosis risk remain to be defined.We now know about the binding properties of aPL;the relationships between aPL and β2-glycoprotein I and antibodies to other phospholipid binding proteins, such as prothrombin;and the roles in the syndrome of plasmin, tissue factor pathway inhibitor, complement activation, endothelial cell and platelet activation.