

Thrombophilie : prévalence et risque relatif

Facteur de risque	Année	Popul générale	Popul avec TV	Risque relat TV
AT 3	1965	0.18	1-2	5-10
Prot.C	1981	0.2	2-4	6,5-8
Prot.S	1984	1.3	2-4	2,4-8
V Leiden	1993	6	20-30	5-6,5
F VIII	1995	11	25	4,8
F II G20210	1996	2.3	6.2	2,8

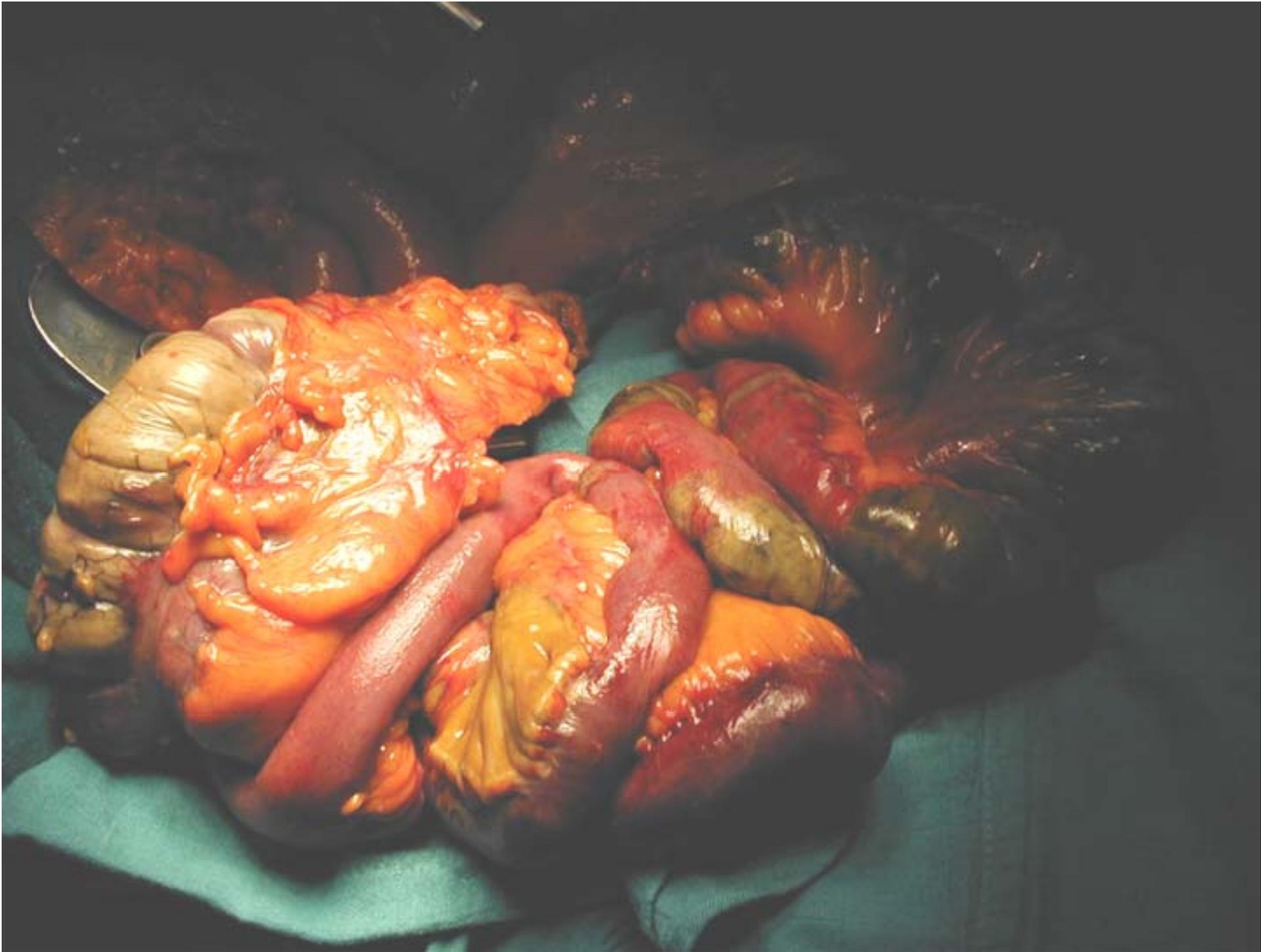
SYNDROME DES ANTIPHOSPHOLIPIDES

➤ BIOLOGIE :

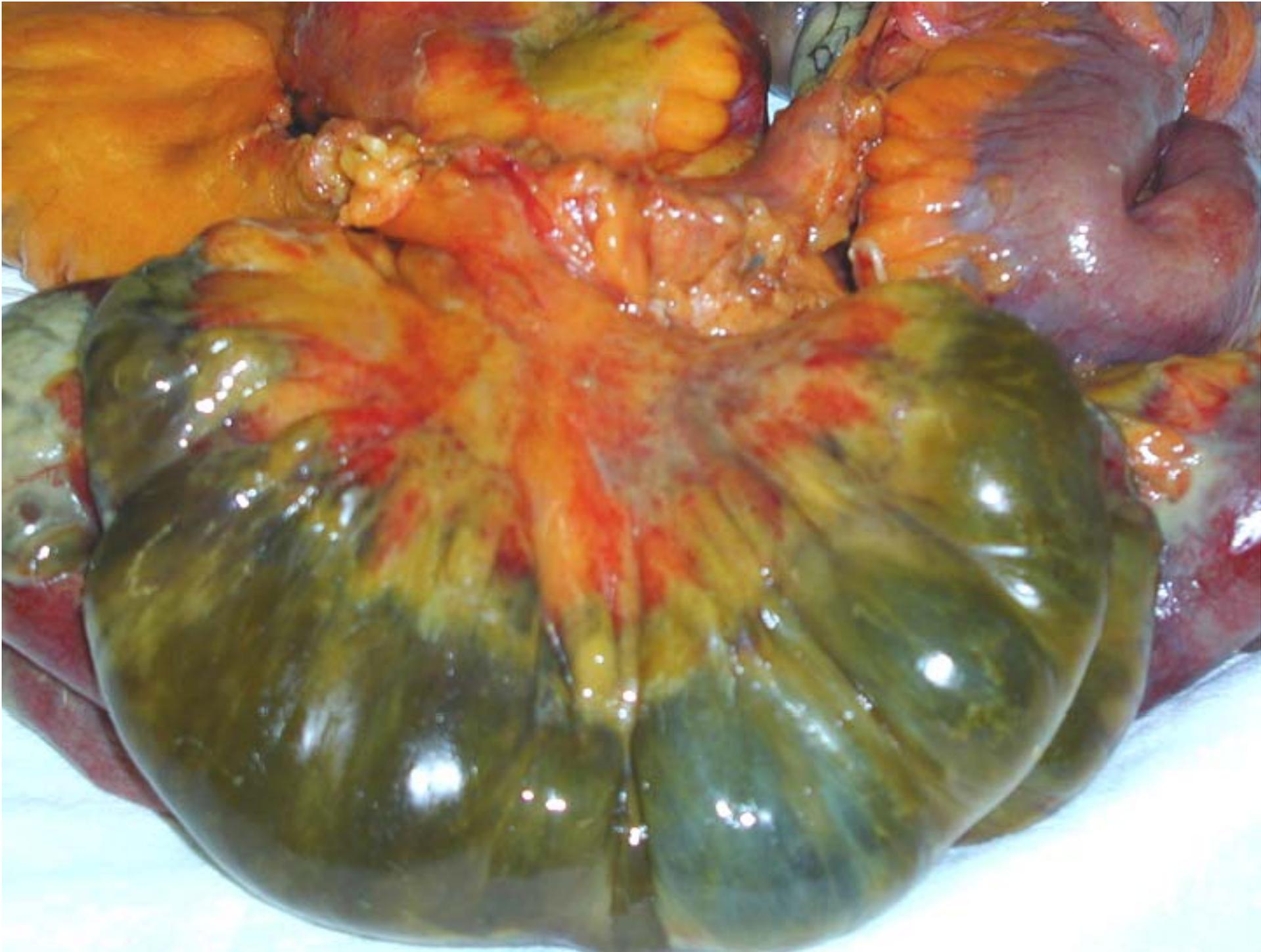
- anticoagulant circulant de type lupique
- anticorps anticardiolipidique
- anticorps antibeta-2 GP1

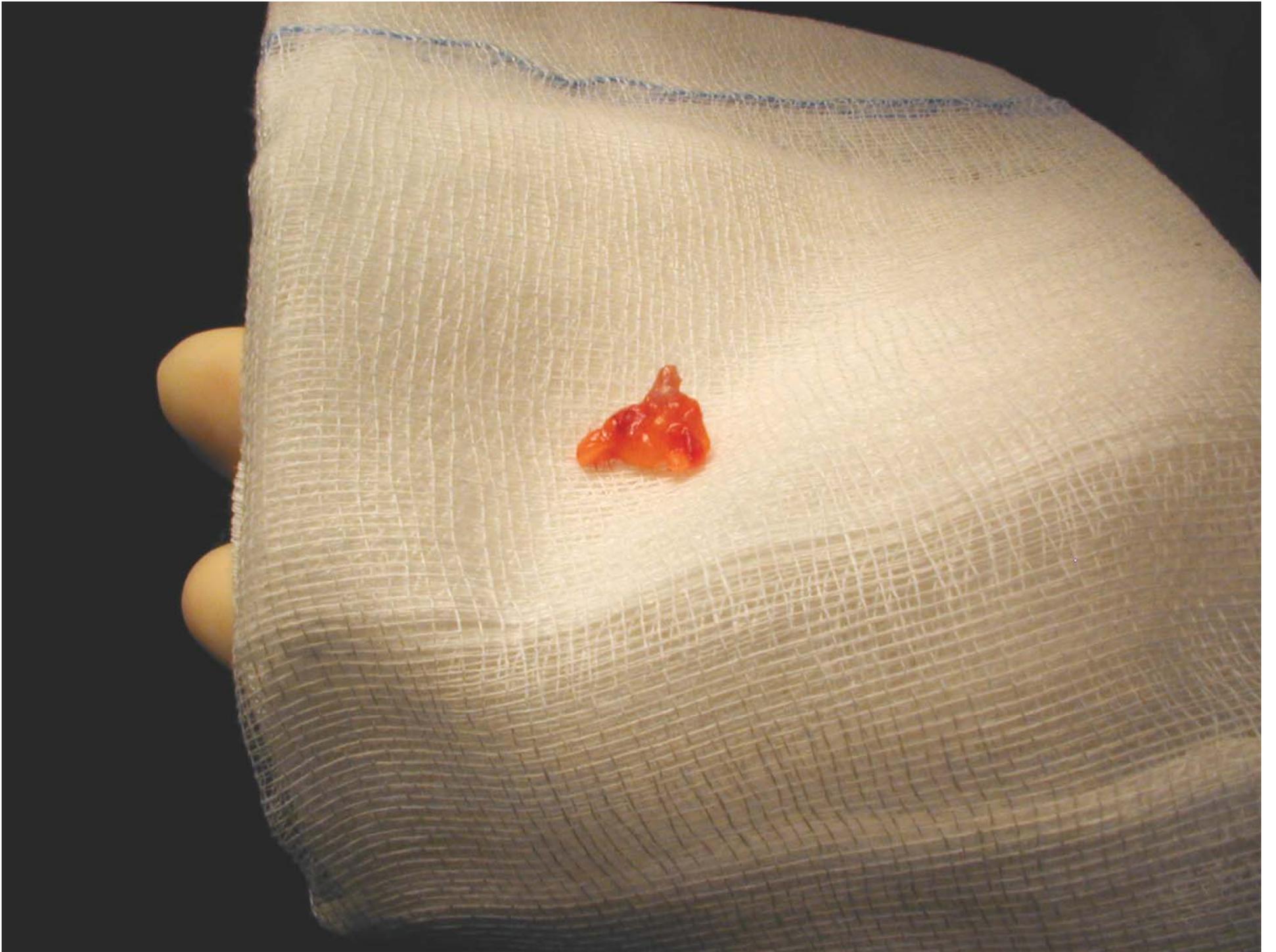
➤ CLINIQUE :

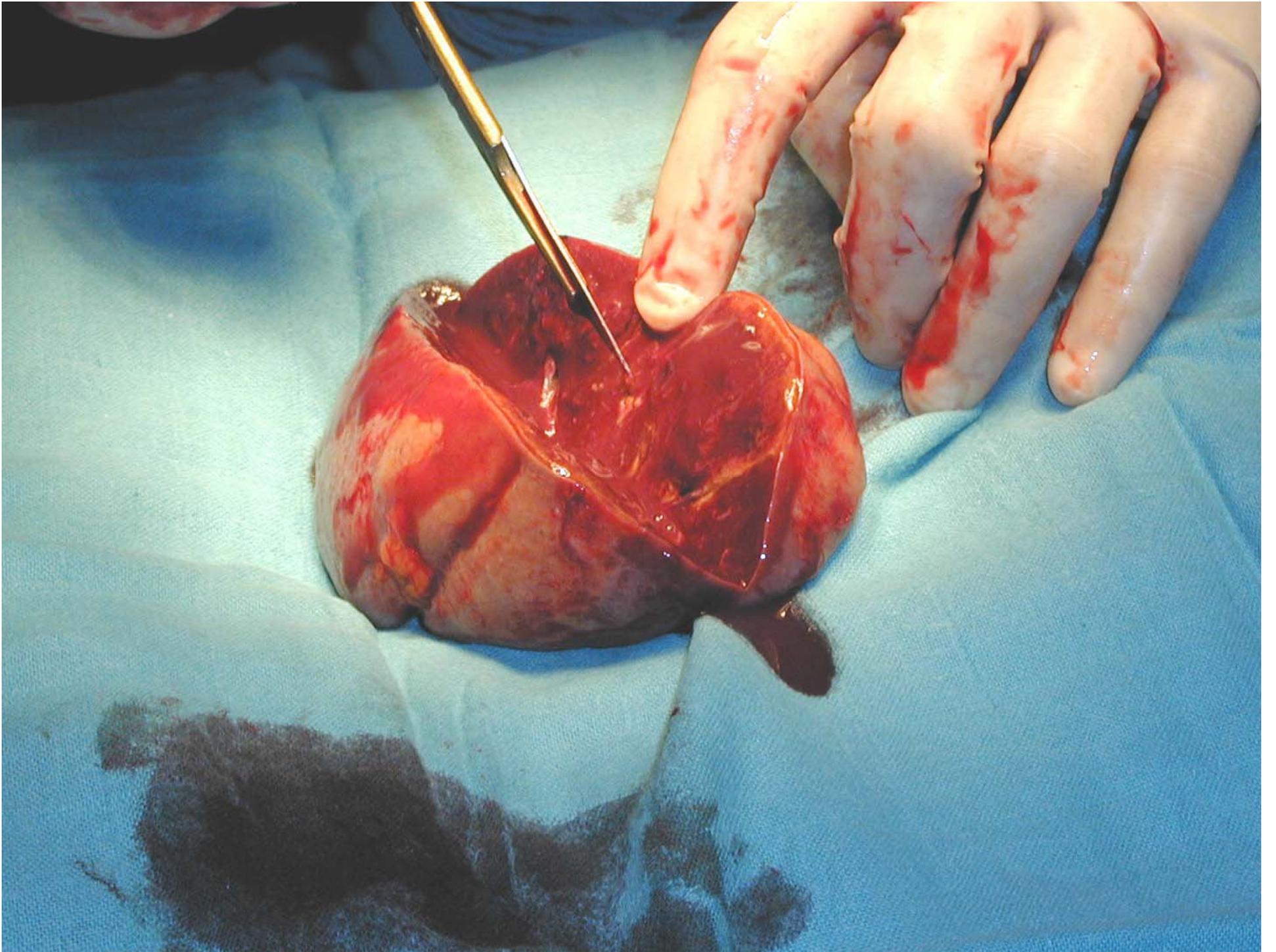
- accident thrombotique artériel ou veineux
- fausses couches spontanées répétées

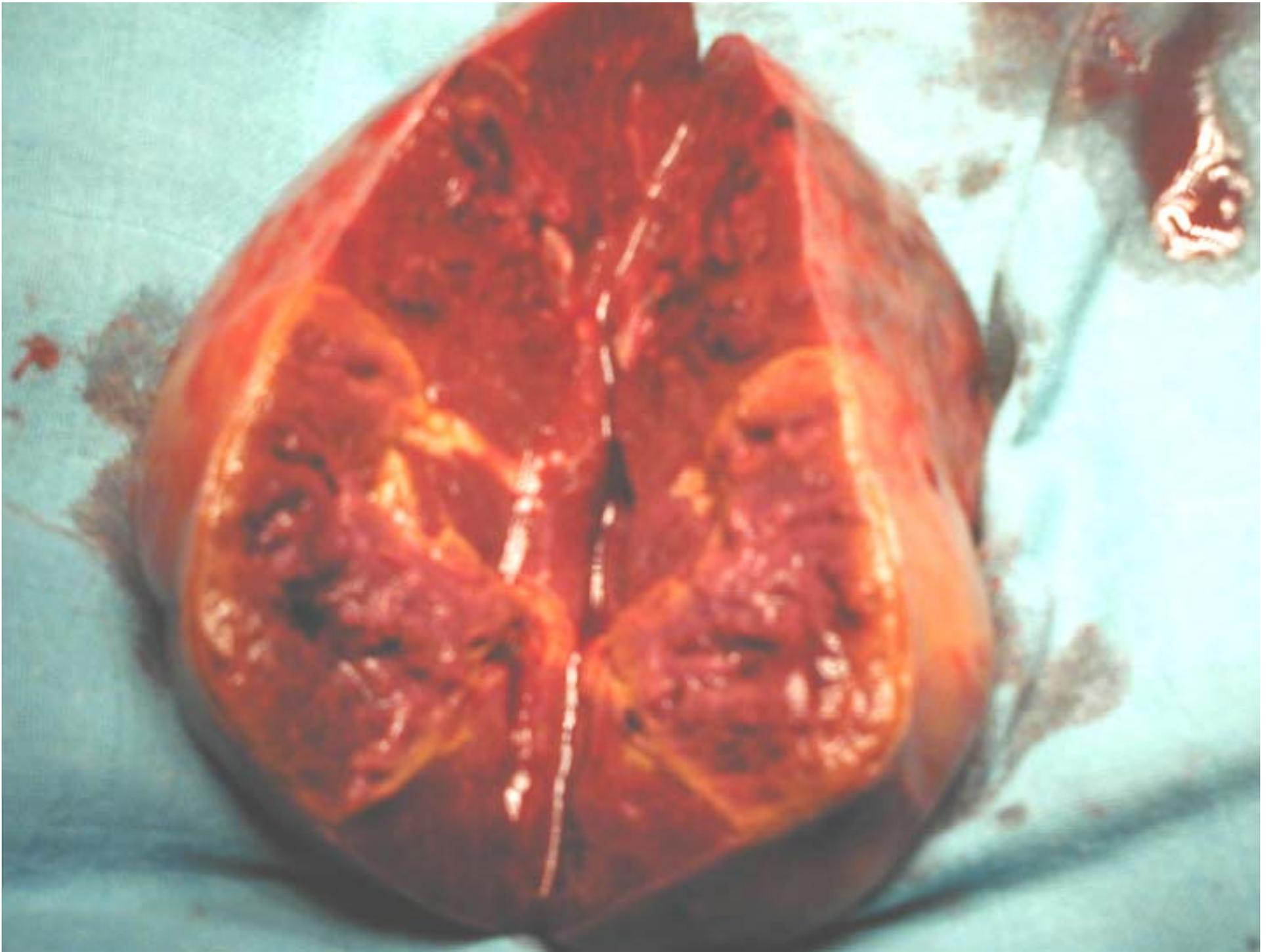










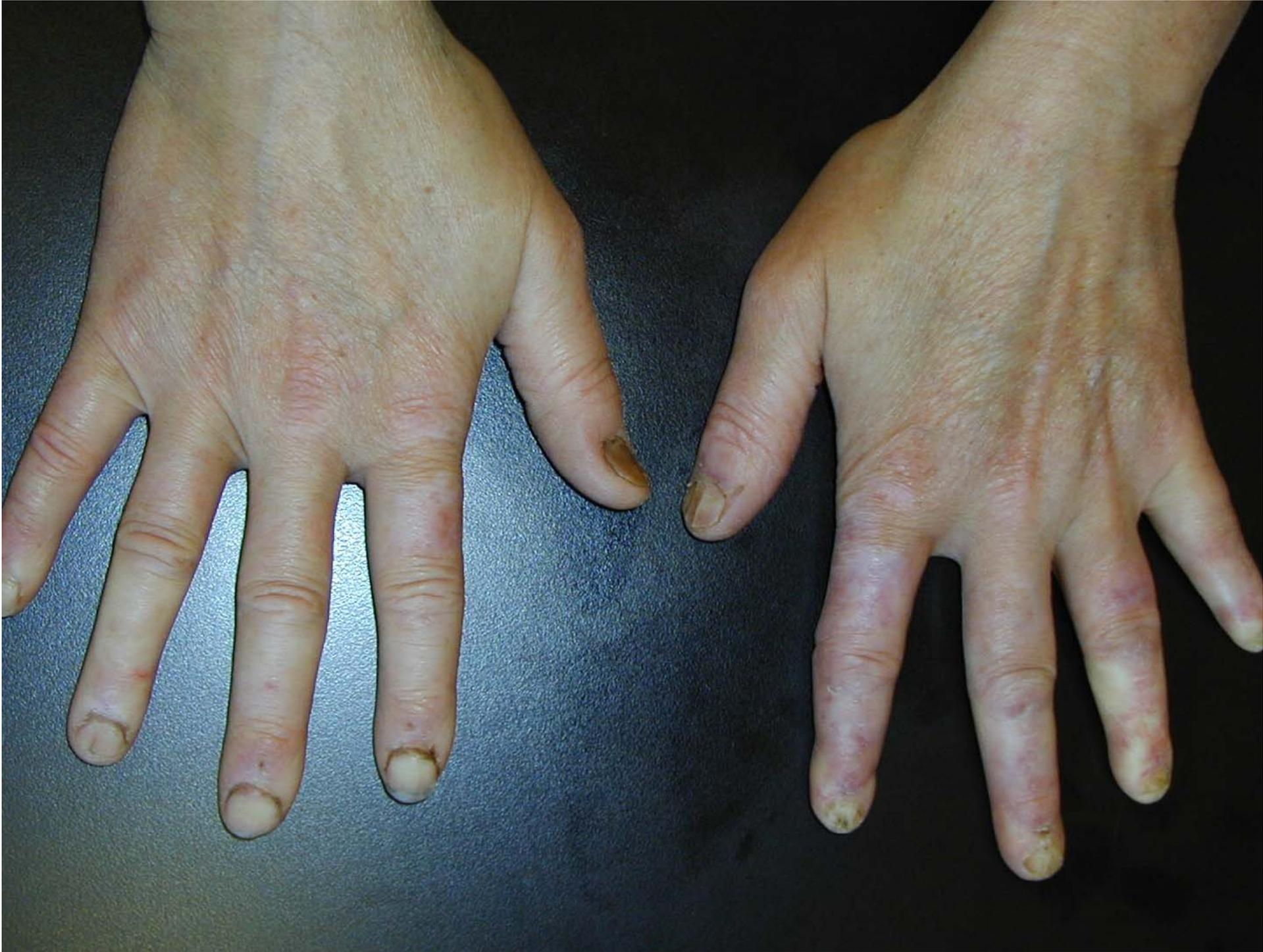


Asherson et al.
Catastrophic antiphospholipid syndrome. Medicine
1998

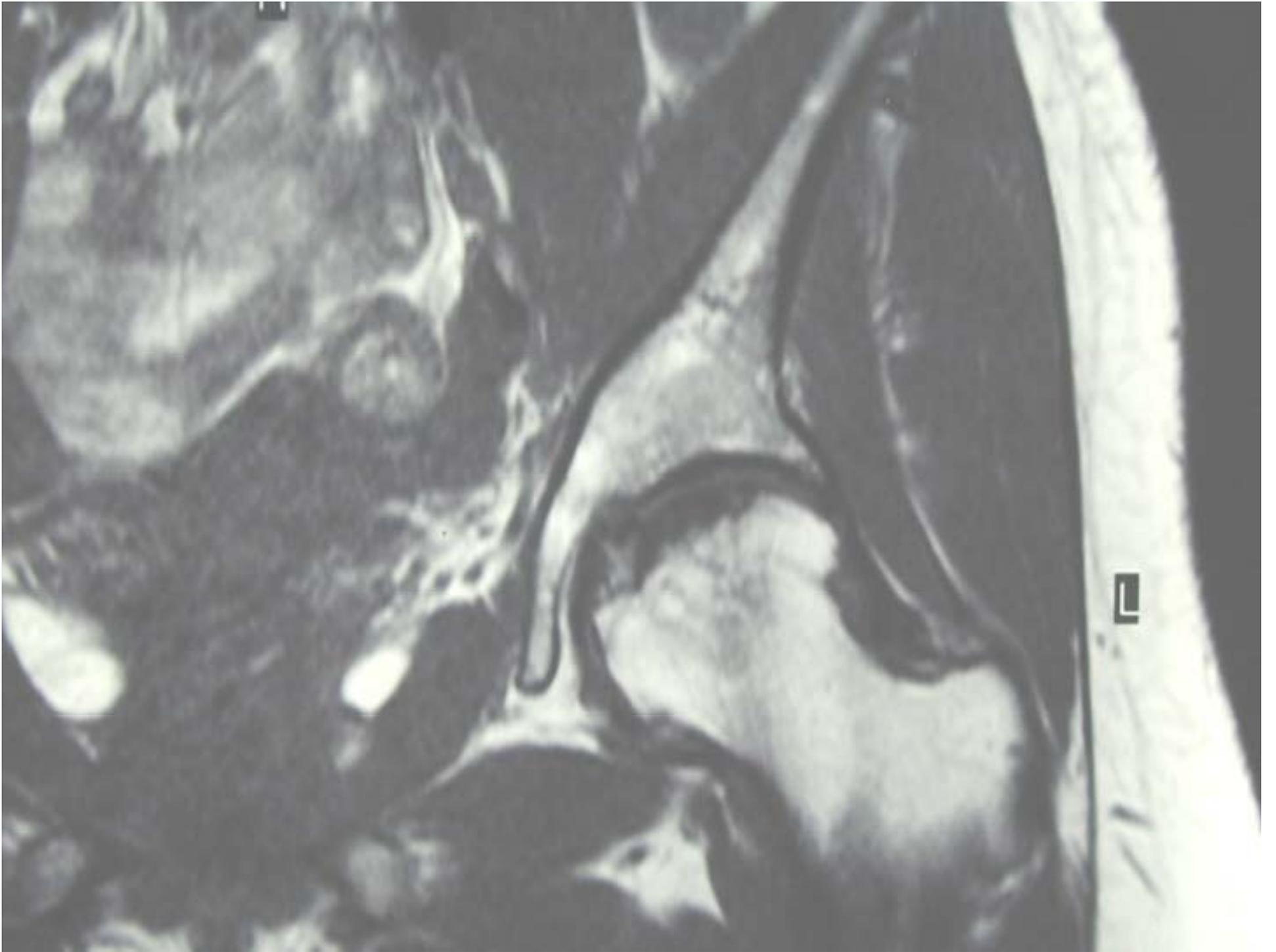
- 50 malades , 1992-1996
- atteinte vasculaire conjointe de plusieurs organes
- issue mortelle fréquente : 50 % des malades
- traitement non codifié











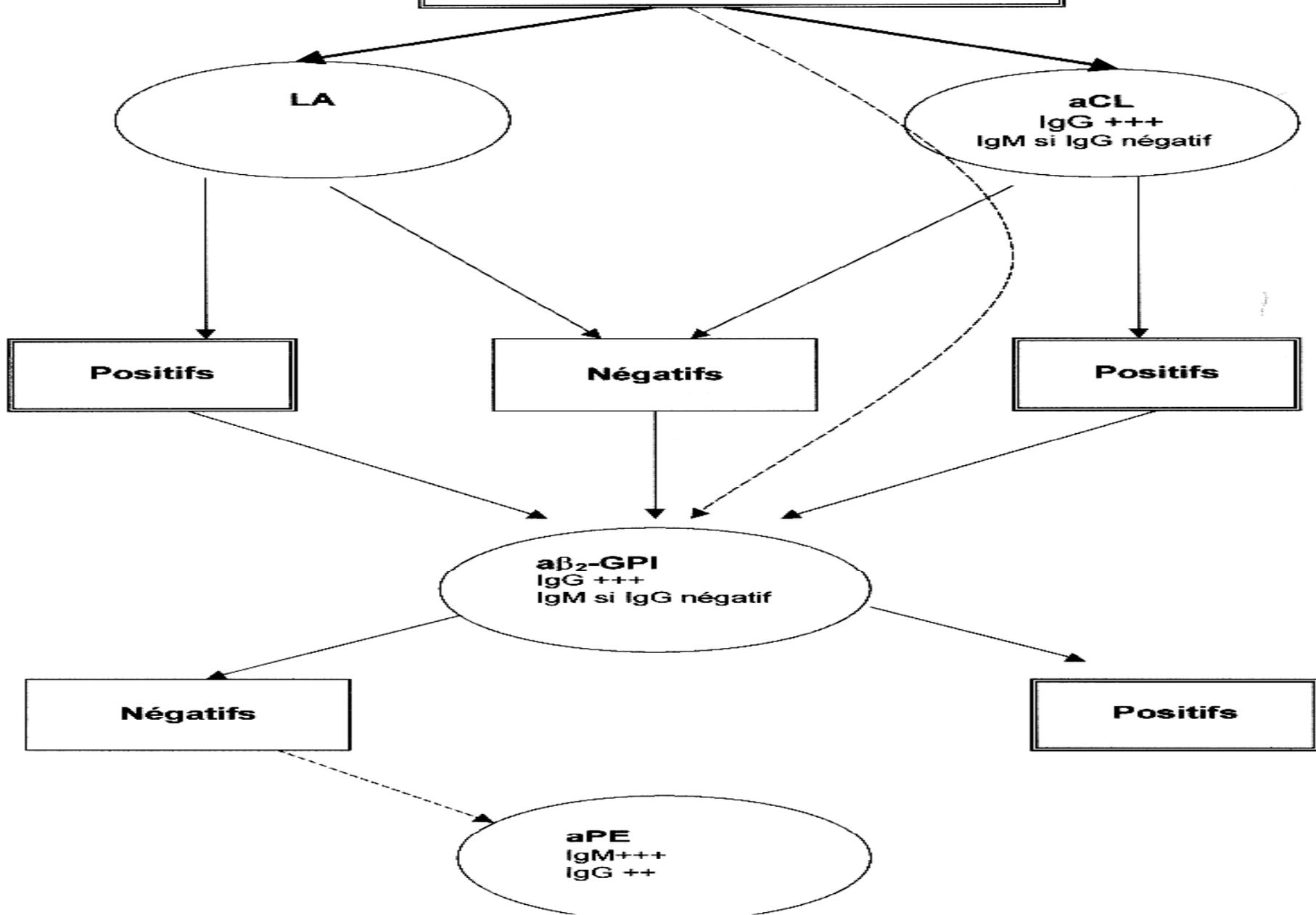


CAROTIDE G



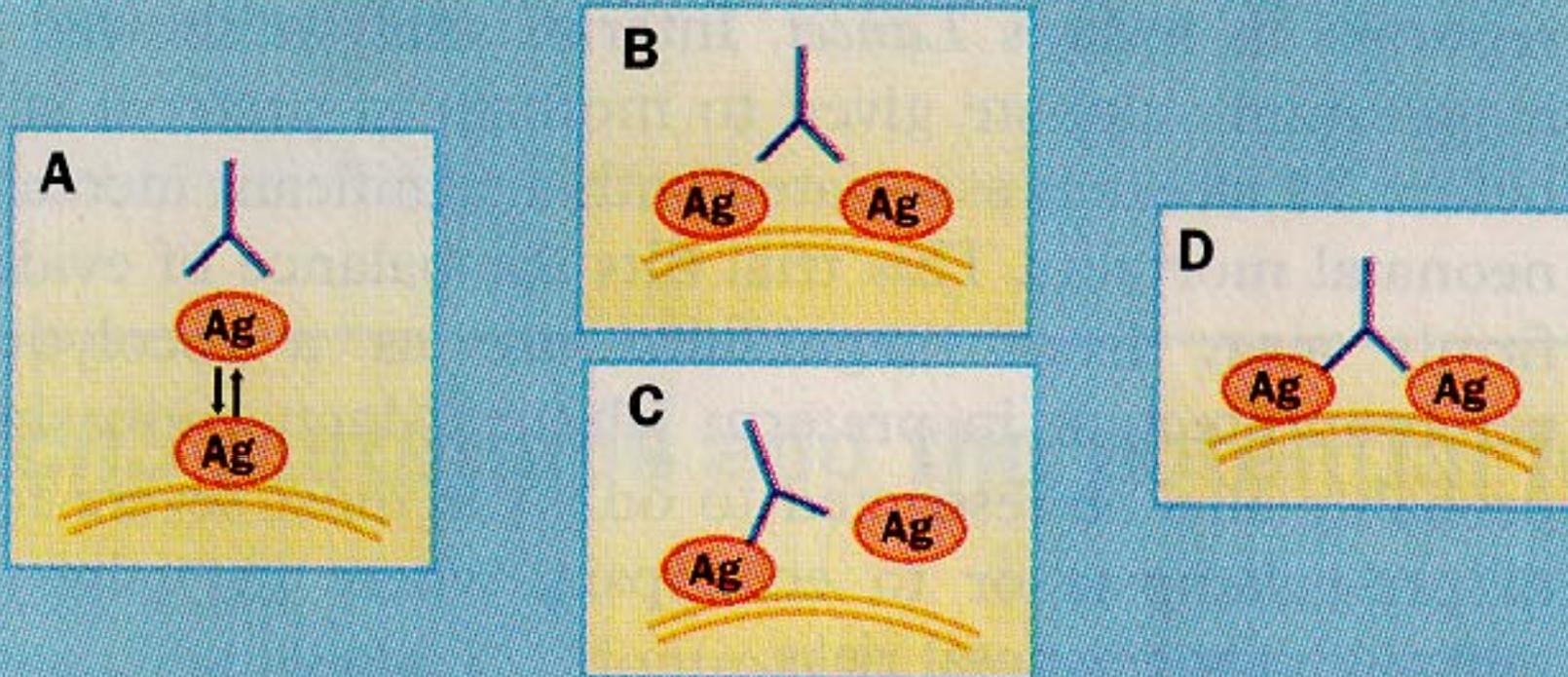
CAROTIDE INTERNE G

Anomalies cliniques d'un SAPL



Possible mechanisms of autoantibody-mediated thrombosis

By cross-linking membrane-bound antigens, autoantibodies may enhance the avidity of the antigen-phospholipid interaction



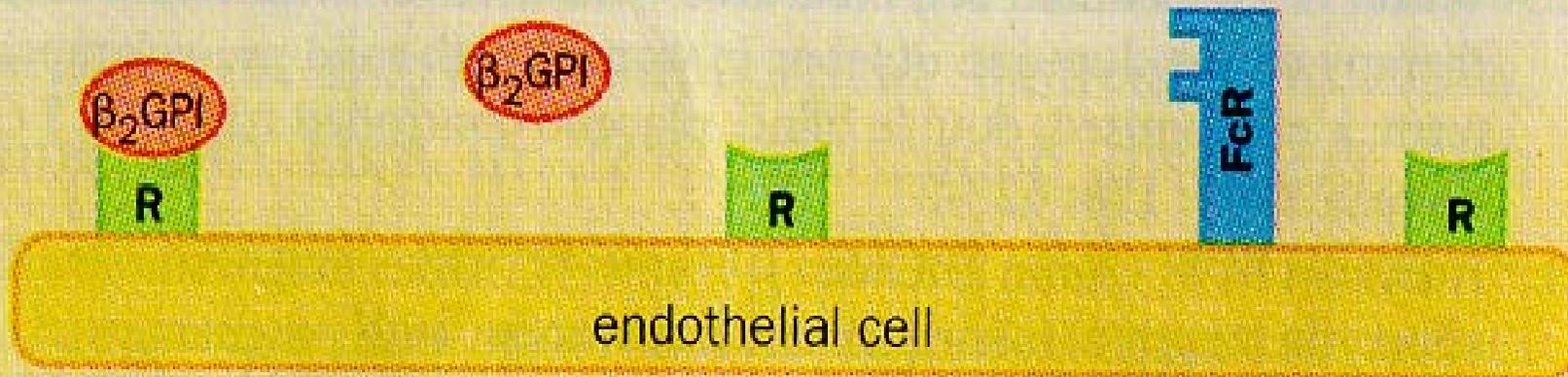
A: fluid-phase plasma protein antigen (Ag) in equilibrium with membrane-bound antigen; low-affinity autoantibody, () circulates free from antigen.

B+C: alternative intermediate stages of bivalent antibody binding.

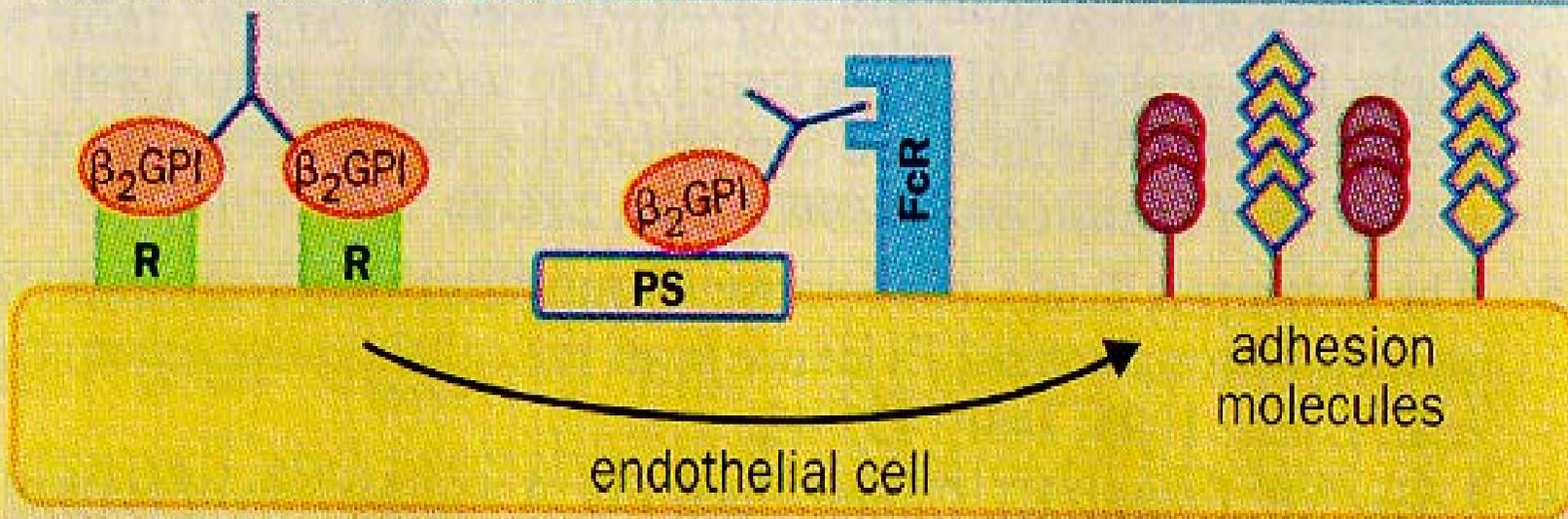
D: bivalent binding of antibody to membrane-bound antigen. Antibodies may alter kinetics of phospholipid-dependent haemostatic reactions in which the antigen is an enzyme, cofactor or substrate. Phospholipid-bound antibody-antigen complexes may also inhibit phospholipid-dependent reactions by competitively blocking the binding of other proteins to anionic phospholipids.

By autoantibody engagement of antigens on cell surfaces, leading to transduction of a signal and altered cell activity

A



B



A: endothelial cell expressing Fc receptors (FcR) and putative β_2 GPI receptors (R).
B: anti- β_2 GPI autoantibodies bind to the endothelial cell surface via β_2 GPI on β_2 GPI receptors and/or β_2 GPI bound to anionic phospholipids such as phosphatidylserine (PS). These antibodies may stimulate the cell via cross-linking of β_2 GPI receptors and/or engagement of Fc receptors, leading to expression of adhesion molecules.

APL dans différentes populations

	Lupus Ac	Anti CL	Beta2 GP1
Pop Nle	0-4%	0-10%	0-2%
Lupus	15-50%	17-86%	10-17%
TV inexpl.	8-15%	-	
Infection	5-70%	5-100%	0-30%
SAPL	-	-	32-88%