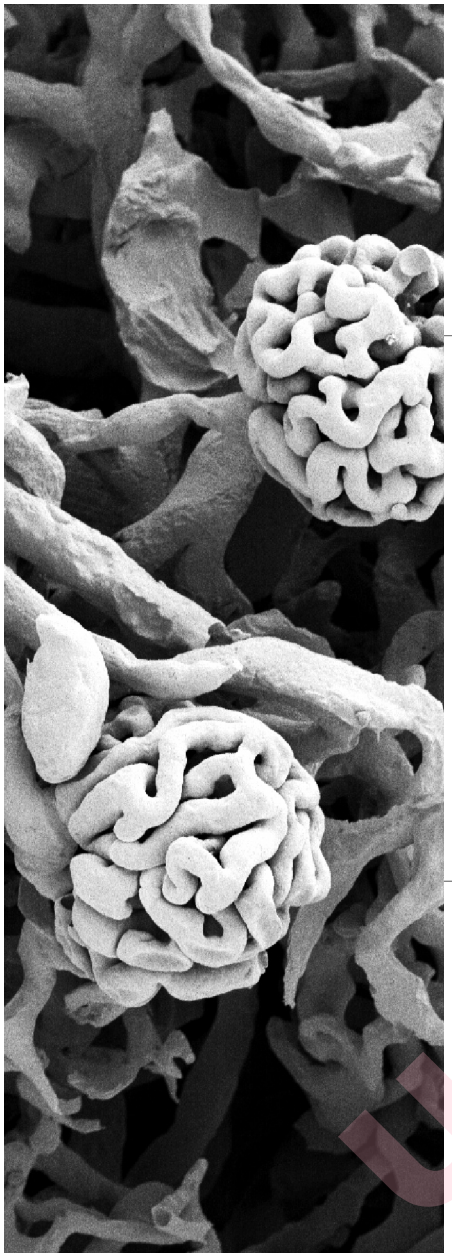


HOSPITAL DE CLINICAS
Dr.Manuel Quintela

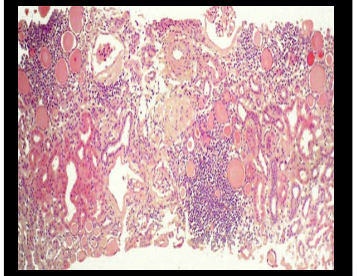
JORNADAS
GLOMERULOPATÍAS

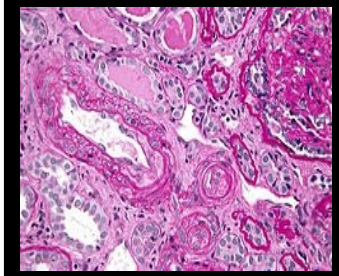
Nefropatía asociada aPL

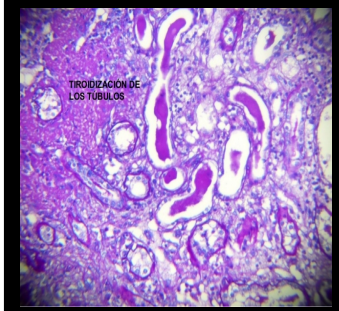
Dr. RUBEN COITIÑO - Asistente Nefrología

14 y 15 de Setiembre 2015

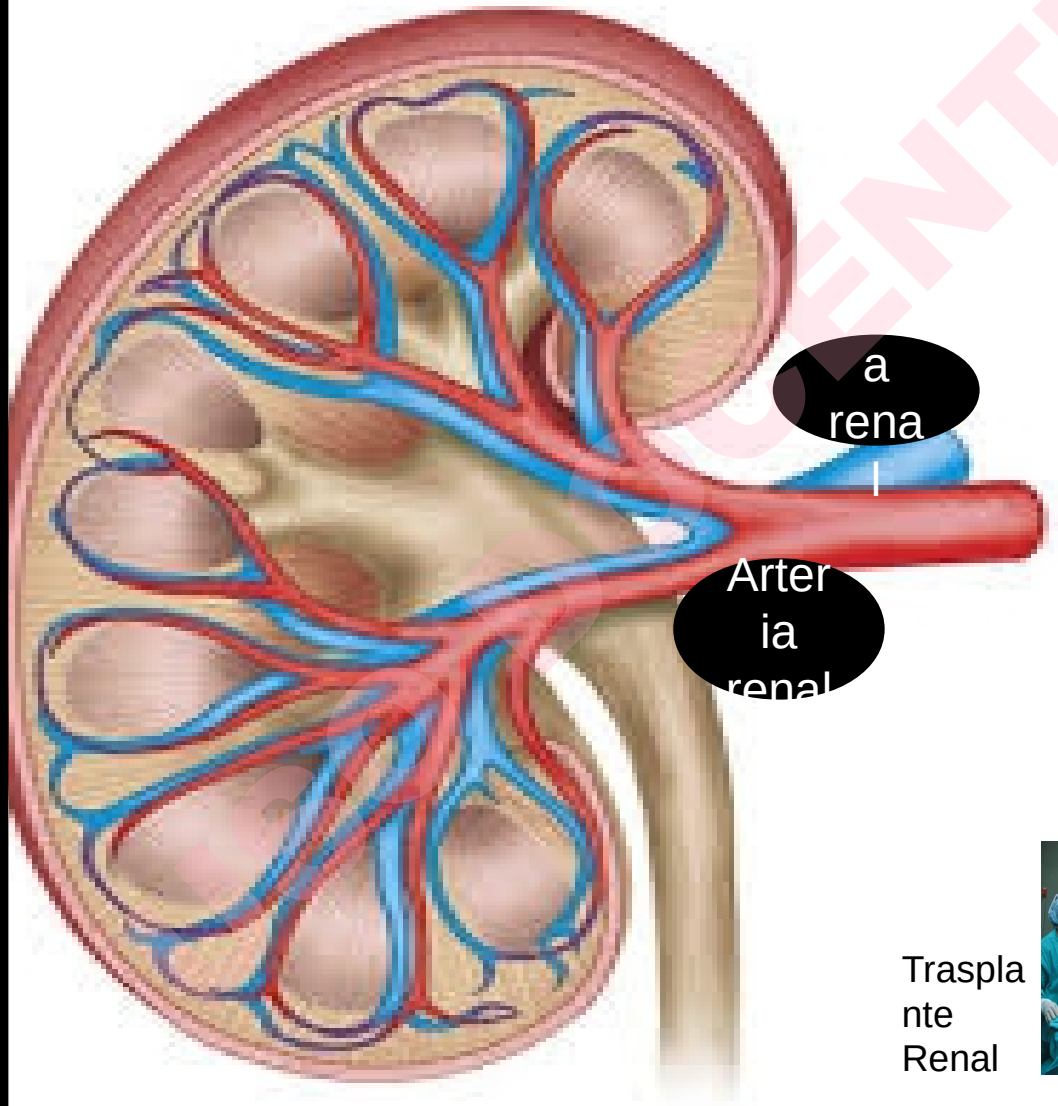
Nefropatía





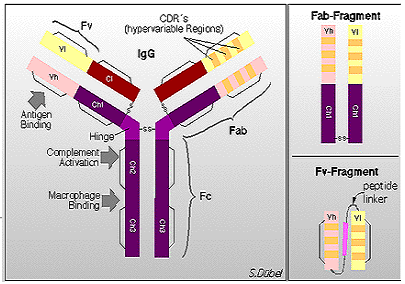


Asociada a aPL



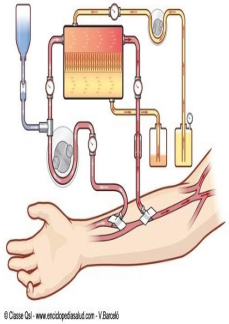
a rena l

Arter ia renal

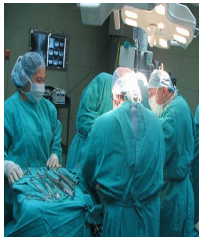


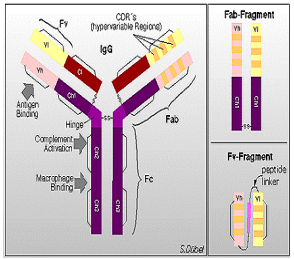
HT A

Diálisis



Traspla nte Renal





International consensus statement on an update of the classification criteria for definite antiphospholipid syndrome (APS)

S. MIYAKIS,* M. D. LOCKSHIN,† T. ATSUMI,‡ D. W. BRANCH,§ R. L. BREY,¶ R. CERVERA,** R. H. W. M. DERKSEN,†† P. G. DE GROOT,†† T. KOIKE,‡ P. L. MERONI,‡‡ G. REBER,§§ Y. SHOENFELD,¶¶ A. TINCANI,*** P. G. VLACHOYIANNOPOULOS††† and S. A. KRILIS*

Antiphospholipid antibody syndrome (APS) is present if at least one of the clinical criteria and one of the laboratory criteria that follow are met*

Clinical criteria

1. Vascular thrombosis†

One or more clinical episodes‡ of arterial, venous, or small vessel thrombosis§, in any tissue or organ. Thrombosis must be confirmed by objective validated criteria (i.e. unequivocal findings of appropriate imaging studies or histopathology). For histopathologic confirmation, thrombosis should be present without significant evidence of inflammation in the vessel wall.

2. Pregnancy morbidity

(a) One or more unexplained deaths of a morphologically normal fetus at or beyond the 10th week of gestation, with normal fetal morphology documented by ultrasound or by direct examination of the fetus, or

(b) One or more premature births of a morphologically normal neonate before the 34th week of gestation because of: (i) eclampsia or severe pre-eclampsia defined according to standard definitions [11], or (ii) recognized features of placental insufficiency¶, or

(c) Three or more unexplained consecutive spontaneous abortions before the 10th week of gestation, with maternal anatomic or hormonal abnormalities and paternal and maternal chromosomal causes excluded.

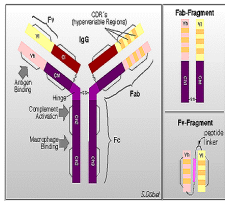
In studies of populations of patients who have more than one type of pregnancy morbidity, investigators are strongly encouraged to stratify groups of subjects according to a, b, or c above.

Laboratory criteria**

1. Lupus anticoagulant (LA) present in plasma, on two or more occasions at least 12 weeks apart, detected according to the guidelines of the International Society on Thrombosis and Haemostasis (Scientific Subcommittee on LAs/phospholipid-dependent antibodies) [82,83].

2. Anticardiolipin (aCL) antibody of IgG and/or IgM isotype in serum or plasma, present in medium or high titer (i.e. > 40 GPL or MPL, or > the 99th percentile), on two or more occasions, at least 12 weeks apart, measured by a standardized ELISA [100,129,130].

3. Anti-β_2 glycoprotein-I antibody of IgG and/or IgM isotype in serum or plasma (in titer > the 99th percentile), present on two or more occasions, at least 12 weeks apart, measured by a standardized ELISA, according to recommended procedures [112].



"Europhospholipid" Project

ARTHRITIS & RHEUMATISM
Vol. 46, No. 4, April 2002, pp 1019–1027
DOI 10.1002/art.10187

Antiphospholipid Syndrome

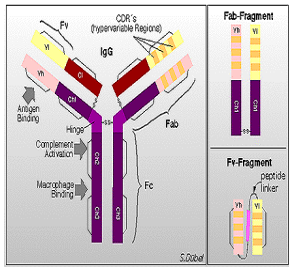
Clinical and Immunologic Manifestations and Patterns of Disease Expression in a Cohort of 1,000 Patients

Ricard Cervera,[1] Jean-Charles Piette,[2] Josep Font,[1] Munther A. Khamashta,[3] Yehuda Shoenfeld,[4] María Teresa Camps,[5] Soren Jacobsen,[6] Gabriella Lakos,[7] Angela Tincani,[8] Irene Kontopoulou-Griva,[9] Mauro Galeazzi,[10] Pier Luigi Meroni,[11] Ronald H. W. M. Derksen,[12] Philip G. de Groot,[12] Erika Gromnica-Ihle,[13] Marta Baleva,[14] Marta Mosca,[15] Stefano Bombardieri,[15] Frédéric Houssiau,[16] Jean-Christophe Gris,[17] Isabelle Quéré,[17] Eric Hachulla,[18] Carlos Vasconcelos,[19] Beate Roch,[20] Antonio Fernández-Nebro,[21] Marie-Claire Boffa,[2] Graham R. V. Hughes,[3] and Miguel Ingelmo,[1] for the Euro-Phospholipid Project Group

Tabla 4
Prevalencia de algunas manifestaciones clínicas en cohorte europea de 1.000 pacientes SAFL

Trombosis venosa profunda (38,9%)
Pérdida fetal temprana (<10 sem) (35,4%)
Pérdida fetal tardía (>10 sem) (16,9%)
Preeclampsia (9,5%)
Trombocitopenia (29,6%)*
Livedo reticularis (24,1%)*
Engrosamiento o disfunción valvular (11,6%)*
Anemia hemolítica (9,7%)
Embolia pulmonar (14,1%)
Hipertensión pulmonar (2,2%)
Vegetaciones valvulares (2,7%)*
Compromiso renal (2,7%) **
Migraña (20,2%)
Stroke (19,8%)
Epilepsia (7%)
Corea (1,2%)
Úlcera de piernas (5,5%)
Necrosis ósea avascular (2,2%)

* Manifestaciones no criterio candidatas a ser incluidas en criterios de clasificación.
** Nefropatía SAFL no fue analizada en este estudio.
Adaptado de: Cervera R, *et al*. Antiphospholipid syndrome: clinical and immunologic manifestations and patterns of disease expression in a cohort of 1,000 patients. Arthritis Rheum 2002; 46:1019-27.



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J Thromb Haemost 2006

Table 5 Definition of aPL-associated nephropathy (APLN)

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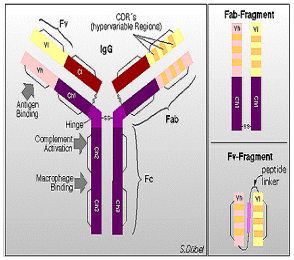
If SLE is also present, the above lesions should be distinguished from those associated with lupus nephropathy.



The Relevance of "non-criteria" Clinical Manifestations of Antiphospholipid Syndrome: 14th International Congress on Antiphospholipid Antibodies Technical Task Force Report on Anti...

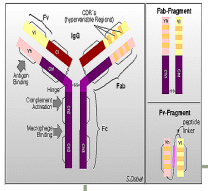
ARTICLE *in* AUTOIMMUNITY REVIEWS · JANUARY 2015

Table 2

Table 2
Result of the complete critical analysis.

Criteria		PIO[a]	Relevance category of PIO	Overall GRADE analysis	Recommendation[b,c]
Superficial venous thrombosis		Recurrent thrombotic events, including life threatening event	Important but not critical for decision-making	Low	Suggestion to be included
Thrombocytopenia		Recurrent thrombotic events, including life threatening event	Critical for decision making	Low	Recommended to be included
APS Nephropathy		Kidney failure, death by kidney failure	Critical for decision making	Moderate	Strongly recommended to be included
Valve heart lesions		Heart failure, valve replacement, death by heart failure	Critical for decision making	Moderate	Strongly recommended to be included
Livedo reticularis		Recurrent thrombotic events, including life threatening event	Important but not critical for decision-making	Moderate	Recommended to be included
Neurological manifestations	Migraine	Recurrent thrombotic events, including life threatening event	Important but not critical for decision-making	Very low	Not recommended to be included
	Chorea	Stroke, neurological arterial and venous thrombosis	Critical for decision making	Low	Recommended to be included
	Seizures	Stroke, neurological arterial and venous thrombosis	Critical for decision making	Very low	Not recommended to be included
	Longitudinal myelitis	Stroke, neurological arterial and venous thrombosis	Critical for decision making	Low	Recommended to be included
Seronegative APS		Recurrent thrombotic events, including life threatening event	Critical for decision making	Low	Recommended to be included

[a] PIO — Patient-important outcome.
[b] Based on patient important outcome + relevance category + overall GRADE analysis.
[c] Recommendation regarding the inclusion of this non-criteria manifestation as part of APS criteria revision.



Estenosis de la Arteria renal : 2 patrones

Rheumatology 2005;44:372–377 doi:10.1093/rheumatolgy/keh490
Advance Access publication 30 November 2004
Concise Report

Renal artery stenosis in hypertensive patients with antiphospholipid (Hughes) syndrome: outcome following anticoagulation

S. R. Sangle, D. P. D'Cruz, I. C. Abbs, M. A. Khamashta and G. R. V. Hughes

Renal artery stenosis: a new facet of the antiphospholipid (Hughes) syndrome

Lupus (2003) 12, 803–804

Renal artery thrombosis and hypertension in a 13 year old girl with antiphospholipid syndrome

Pier Antonio Ostuni, Paolo Lazzarin, Vittorio Pengo, Amelia Ruffatti, Franco Schiavon, Pierfranca Gambari

Annals of the Rheumatic Diseases 1990;

Vasculopathy and arterial stenotic lesions in the antiphospholipid syndrome

C. Christodoulou, S. Sangle and D. P. D'Cruz

Rheumatology 2007;

Nephrol Dial Transplant (2010): Editorial Reviews

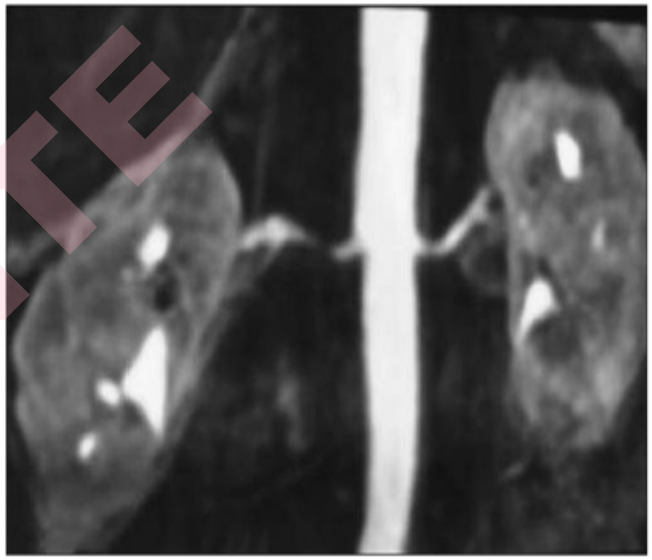
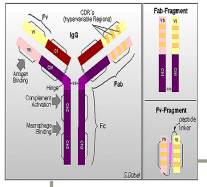


Fig. 1. Right renal artery stenosis associated with hypertension in antiphospholipid syndrome. The aorta is smooth, suggesting that the stenosis is not related to atherosclerosis. Reprinted from reference [9] with permission.

Estenosis distal al ostium de la arteria renal



Atheroma: links with antiphospholipid antibodies, Hughes syndrome and lupus

D. HARATS, J. GEORGE[1], Y. LEVY[1], M.A. KHAMASHTA[2], G.R.V. HUGHES[2] and Y. SHOENFELD[1]

Q J Med 1999; 92:57–59

The anti-phospholipid (Hughes) syndrome: a crossroads of autoimmunity and atherosclerosis

Keywords: atherosclerosis; APS; endothelial cells; autoimmunity

Lupus (1997) 6, 559–560

Premature atherosclerosis in primary antiphospholipid syndrome: preliminary data

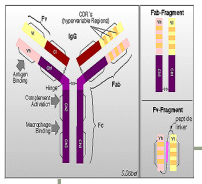
P R J Ames, A Margarita, K B Sokoll, M Weston, V Brancaccio

Ann Rheum Dis 2005;64:315–317. doi: 10.1136/ard.2004.023952

Estenosis de la Arteria renal

Estenosis proximal (igual patrón que aterosclerosis) con compromiso de aorta en general

¿Aterosclerosis acelerada vinculada a aPL?



Renal manifestations of the antiphospholipid syndrome

DP D'Cruz*
The Lupus Research Unit, The Rayne Institute St Thomas' Hospital, London, UK

Lupus (2005) 14, 45–48

Renal vein thrombosis in Chinese patients with systemic lupus erythematosus

Ning-Sheng Lai, Joung-Liang Lan

Annals of the Rheumatic Diseases 1997

Mintz G et al.
Renal vein thrombosis and inferior vena cava thrombosis in systemic lupus erythematosus. Frequency and risk factors. *Arthritis Rheum 1984*

Nephrol Dial Transplant (1989) 4: 854-858

research-article

Recurrent Thrombosis and Renal Vascular Disease in Patients with a Lupus Anticoagulant

D. Kleinknecht[1], G. Bobrie[2], O. Meyer[3], L. H. Noël[2], P. Callard[4] and M. Ramdane[1]

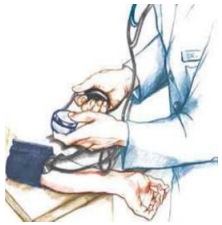
Trombosis de Vena Renal

Grupo de mayor riesgo trombótico:

- Nefropatía Lúpica asociada a AAF
- Pacientes con SAF primario

Frecuencia particularmente incrementada:

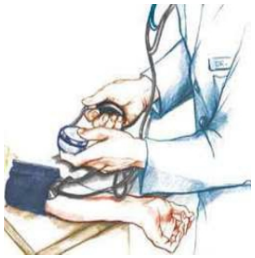
- Portadores de LA circulante



Hipertensión arterial y aPL

- Asherson RA, Clin Exp Rheumatol 1993
- Cacoub P, Clin Exp Rheumatol 1993
- Durand JM, Clin Rheumatol 1994
- Sirvent AE, Nephrol 1996
- Ohtomo Y, Acta Paediatr 1998
- Nochy D, J Am Soc Nephrol 1999
- Aizawa K, Jpn Circ J 2000
- Riccialdelli L, Am J Hypertens 2001
- Nzerue CM, Kidney Int 2002
- Daugas E, J Am Soc Nephrol 2002
- Karim MY, Lupus 2002.
- Sangle S, Lupus 2002
- Rollino C, Lupus 2004
- Chen W, Clin Appl Thromb Hemost 2004

frecuentemente hallada tanto en el SAF primario como secundario



Hipertensión arterial y aPL

J Am Soc Nephrol 10: 507–518, 1999

The Intrarenal Vascular Lesions Associated with Primary Antiphospholipid Syndrome

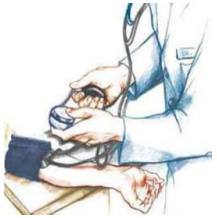
DOMINIQUE NOCHY,* ERIC DAUGAS,* DOMINIQUE DROZ,† HELENE BEAUFILS,‡ JEAN-PIERRE GRÜNFELD,† JEAN-CHARLES PIETTE,‡ JEAN BARIETY,* and GARY HILL*

Presente en 93% de los pacientes

En muchos: único signo sugestivo de nefropatía

Atribuyen HTA a lesiones vasculares.

- Arteriosclerosis (75%),
- Hiperplasia fibrosa intimal arterial o arteriolar (75%)
- Oclusión vascular fibrosa o fibrocelular (68%)
- Microangiopatía trombótica (31%).



Hipertensión arterial y aPL

Antibodies to Endothelial Cells in Borderline Hypertension
Johan Frostegård, Ruihua Wu, Caroline Gillis-Haegerstrand, Carola Lemne and Ulf de Faire
Circulation 1998;98:1092-1098

Niveles elevados de B2GPI en pacientes con Hipertensión Arterial Borderline en 73 pacientes comparados con igual población (sexo y edad) de pacientes normotensos

1: Clin Exp Rheumatol. 1993 Sep-Oct;11(5):479-85.

Malignant hypertension in antiphospholipid syndrome without overt lupus nephritis.

Cacoub P, Wechsler B, Piette JC, Beaufils H, Herreman G, Bletry O, Godeau P.

Department of Internal Medicine, Hôpital de La Pitié-Salpetrière, Paris, France.

Pacientes con SAF primario desarrollan HTA maligna con I Renal
Angiografía de vasos renales NORMAL

Nefropatía por aPL

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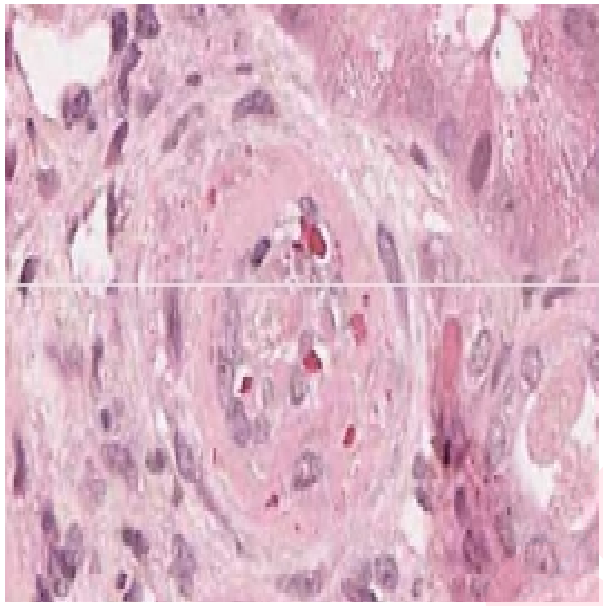
J Thromb Haemost 2006



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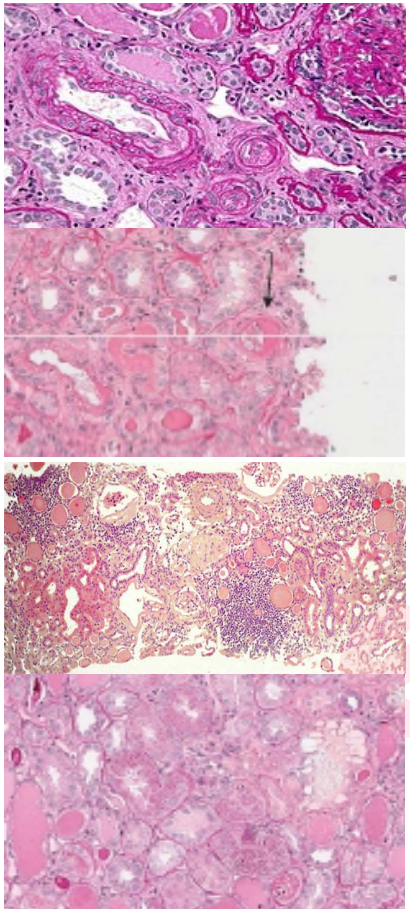
J Thromb Haemost 2006



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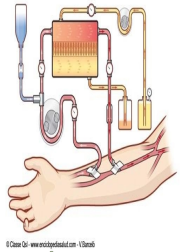


Enfermedad Renal Terminal

Dayal NA et al. **Endstage renal failure in primary antiphospholipid syndrome—case report and review of literature.** *Rheumatology (Oxford) 2003*

Prieto LN et al. **Frequent hemodialysis graft thrombosis: association with antiphospholipid antibodies.** *Am J Kidney Dis 1994*

Brunet P et al. **Antiphospholipids in hemodialysis patients: relationship between lupus anticoagulant and thrombosis.** *Kidney Int 1995*

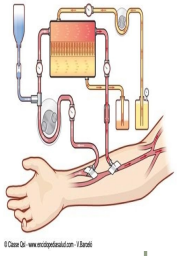
Gronhagen-Riska C et al. **Raised concentrations of antibodies to cardiolipin in patients receiving dialysis.** *BMJ 1990*

Sitter T et al. **Anticardiolipin antibodies and lupus anticoagulant in patients treated with different methods of renal replacement therapy in comparison to patients with systemic lupus erythematosus.** *Ann Hematol 1992*

Garcia-Martin F et al. **Anticardiolipin antibodies and lupus anticoagulant in end-stage renal disease.** *Nephrol Dial Transplant 1991*

Mayor frecuencia que la poblaciòn general de AAF entre quienes ERT, independientemente de:

Edad
Sexo
Tiempo en plan de HD
Tipo de membrana utilizada en HD
Fármacos
Presencia de Hepatitis Crónica C y D



Significado clínico de aPL en

1: Ann Hematol. 1992 Aug;65(2):79-82.

Anticardiolipin antibodies and lupus anticoagulant in patients treated with different methods of renal replacement therapy in comparison to patients with systemic lupus erythematosus.

Sitter T, Spannagl M, Schiffl H.

Medizinische Klinik, University of Munich, Federal Republic of Germany.

1: Nephron. 1993;65(3):350-3.

Prevalence of lupus anticoagulant and anticardiolipin antibodies in haemodialysis patients.

Phillips AO, Jones HW, Hambley H, Hillis AN, Hendry BM.

Renal Unit, King's College Hospital, London, UK.

1: Nephrol Dial Transplant. 1992;7(12):1194-8.

Are antiphospholipid antibodies clinically relevant in dialysis patients?

Chew SL, Lins RL, Daelemans R, Zachee P, De Clerck LS, Vermylen J.

Department of Nephrology-Hypertension, Stuivenberg General Hospital, Antwerp, Belgium.

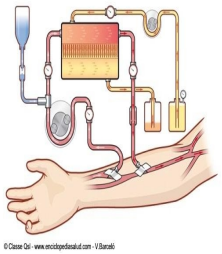
1: J Nephrol. 1999 Mar-Apr;12(2):89-94.

Antiphospholipid (aPL) antibodies in end-stage renal disease.

Fabrizi F, Sangiorgio R, Pontoriero G, Corti M, Tentori F, Troina E, Locatelli F.

Nephrology and Dialysis Division, Lecco Hospital, Italy. nefrolec@tin.it

"Los aPL no son patogénicos"



Significado clínico de aPL en ERCT

1: Am J Kidney Dis. 1994 Apr;23(4):587-90.

Frequent hemodialysis graft thrombosis: association with antiphospholipid antibodies.

Prieto LN, Suki WN.

Department of Medicine, Baylor College of Medicine, Houston, TX.

1: Am J Kidney Dis. 1995 Aug;26(2):347-52.

Anticardiolipin antibody in patients on maintenance hemodialysis and its association with recurrent arteriovenous graft thrombosis.

Prakash R, Miller CC 3rd, Suki WN.

Department of Medicine, Baylor College of Medicine, Houston, TX, USA.

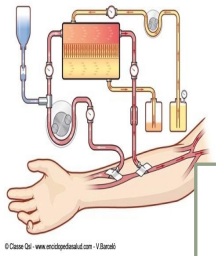
Association of Anticardiolipin Antibodies with Vascular Access Occlusion in Hemodialysis Patients: Cause or Effect?

Y.G. Ilaviv

Nephrology and Hypertension Services, Hadassah-Hebrew University Medical Center, Jerusalem, Israel

Nephron 2000;86:447–454

mayor incidencia de trombosis en accesos vasculares de HD



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Y.S. Haviv

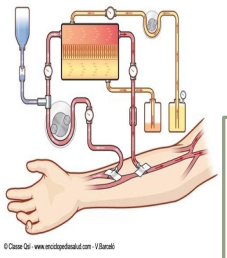
Nephrology and Hypertension Services, Hadassah Hebrew University Medical Center, Jerusalem, Israel

LA y aCL:

igual riesgo de trombosis

LA:

mayor riesgo trombótico



Significado clínico de aPL en ERCT

	Moss *et al.* *n* (%) [5]	Nochy *et al.* *n* (%) [9]	Erkan *et al.* *n* (%) [8]	Vlachoyiannopolous *et al.* *n* (%) [6]	Amigo *et al.* *n* (%) [3]	Griffiths *et al.* *n* (%) [7]
No. of patients	20	16	39	78	20	8
Type of study	Retrospective analyses	5-yr prospective	13-yr prospective	Retrospective analyses	5-yr prospective	Histological analysis
Renal						
Nephrotic	1 (16.7)	1 (6)		4 (25)	2	1
Hypertension	5 (83.3)	15 (93)		44 (56)	5	8 (100)
Reduced GFR	5 (30)	16 (100)		8 (10)	5 (100)	8 (100)
Haematuria			9 (56)		1	
Microangiopathic anaemia			1 (16)			
Non-renal						
Thrombosis (venous) arterial/s			15		7	5
CNS		13 (81)	26 (66)		4	2 (25)
Cutaneous		9 (56)			4	6 (75)
Pregnancy complications		3/6 (50)	23 (77)		4	2 (25)
Pulmonary		4 (25)	1			1
Thrombocytopenia		9 (56)	15 (41)			2 (25)
Adrenal failure						
Outcome						
ESRF	1		1		2	
Deaths	0	3			2	1

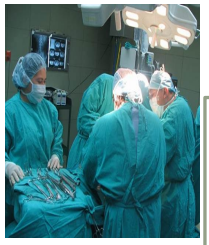
ERT:

Rara complicación del SAF primario

Endstage renal failure in primary antiphospholipid syndrome—case report and review of literature

N. A. Dayal and D. A. Isenberg[1]

Rheumatology 2003; 42: 1128-1129



Trasplante Renal

PREVALENCE AND CLINICAL SIGNIFICANCE OF ANTIPHOSPHOLIPID ANTIBODIES IN RENAL TRANSPLANT RECIPIENTS

Ducloux, Didier[1,2]; Pellet, Emmanuelle[1]; Fournier, Véronique[1]; Rebibou, Jean-Michel[1]; Bresson-Vautrin, Catherine[1]; Racadot, Evelyne[3]; Fellmann, Dominique[4]; Chalopin, Jean-Marc[1]

Transplantation. 1999 Jan 15;67(1):90-3

Incremento en la prevalencia de aPL en trasplante renal, aún sin LES

Stone JHet al. **Antiphospholipid antibody syndrome in renal transplantation: occurrence of clinical events in 96 consecutive patients with systemic lupus erythematosus.**
Am J Kidney Dis 1999

Vaidya S, et al. **Relative risk of posttransplant renal thrombosis in patients with antiphospholipid antibodies.**
Clin Transplant 1998

Wagenknecht DR et al. **Antiphospholipid antibodies are a risk factor for early renal allograft failure.**
Transplantation 1999

Wagenknecht DR et al. **Risk of early renal allograft failure is increased for patients with antiphospholipid antibodies.**
Transpl Int 2000

trombosis
vascular renal
y
falla de transplante

Introducción: En Uruguay existe el PPTG/PSR. Registro de causas de MAT pero no existe una distinción específica de MAT por aPL(sino de todas las causas que presentan dicho patrón AP).

Objetivo General:

Analizar las características clínicas, paraclínicas, la evolución y el pronóstico de una cohorte de pacientes con nefropatia asociada a SAF primario.

Objetivos específicos:

Describir las características clínicas y de laboratorio de pacientes portadores de SAFp al momento del debut de la nefropatía.

Describir la presencia, tipo y títulos de aPL en la población estudiada, su evolución y relación con manifestaciones renales.

Describir el tratamiento instaurado, la evolución posterior a la biopsia renal, recurrencias (morbilidad obstétrica y/o trombosis arterial o venosa) y muerte.

Describir la evolución del compromiso renal

Metodología

Serie de casos retrospectiva de 9 pacientes, cohorte española-uruguaya (Hospital Clinic de Barcelona-Hospital Ramón y Cajal de Madrid, Sanatorio Casa de Galicia Montevideo y Hospital de Clínicas Montevideo).

En base a informes AP de biopsias renales con MAT y aPL detectables en sangre a títulos medio-altos.

Diagnóstico de SAF.

Exclusión de LES y otras causas que podrían explicar MAT (ACN, HTA en acelerada, embarazo, PTT, SUH, Esclerosis sistémica, vasculitis sistémicas, VIH).

Biopsias renales fueron realizadas entre 1993 y 2013. Seguimiento clínicos de pacientes al menos de 1 año.

Registro en cada paciente al diagnóstico y seguimiento: *sexo, edad, trombosis, morbilidad obstétricas cifras de PA, creatinina, FG (MDR/CKD-EPI), Pru, ANA, C3, C4, ANCA, elementos de AH, trombocitopenia, livedo reticularis, manif, neurológicas, hepatocíticas, valvulares. Complicaciones de tto. Perfil de AL, aCL, antib2GPI (presencia y títulos).*

Resultados

Paciente	Edad años /Sexo	Cr(s) (mg/dl) inicio/final	Proteinuria (mg/d)	Hematuria	HTA PreBR/PosBR	Eventos trombóticos ≥2 en evolución	Tiempo meses SAF/BR(meses)	TSR/TR	Seguimiento meses/ Muerte	Tratamiento
1	42/F	2/6,3	3370	+	-/+	+	144	+/-	179/-	AC
2	66/F	1,6/2,3	2464	+	+/+	+	72	-/-	49/+	AC,CC,RPT
3	38/M	1,4/2.1	1360	+	+/+	-	48	-/-	192/-	AC,AAS,CC
4	24/F	0,7/1,1	2282	+	-/+	-	50	-/-	146/-	AC
5	29/F	1/0,85	10912	+	+/-	-	5	-/-	52/-	AC, AAS, CC
6	45/M	1.8/1.5	1000	+	+/+	-	20	-/-	42/-	AC
7	75/F	2.3/2.7	820	+	+/+	-	71	+/-	16/-	AC, CC
8	26/F	1.95/4.2	950	+	-/+	+	62	+/-	62/-	AC,CC
9	19/M	1.67/1.4	551	+	-/-	-	0	-/-	12/-	AC

Tabla 1: Manifestaciones renales clínicas, analíticas y tratamiento.

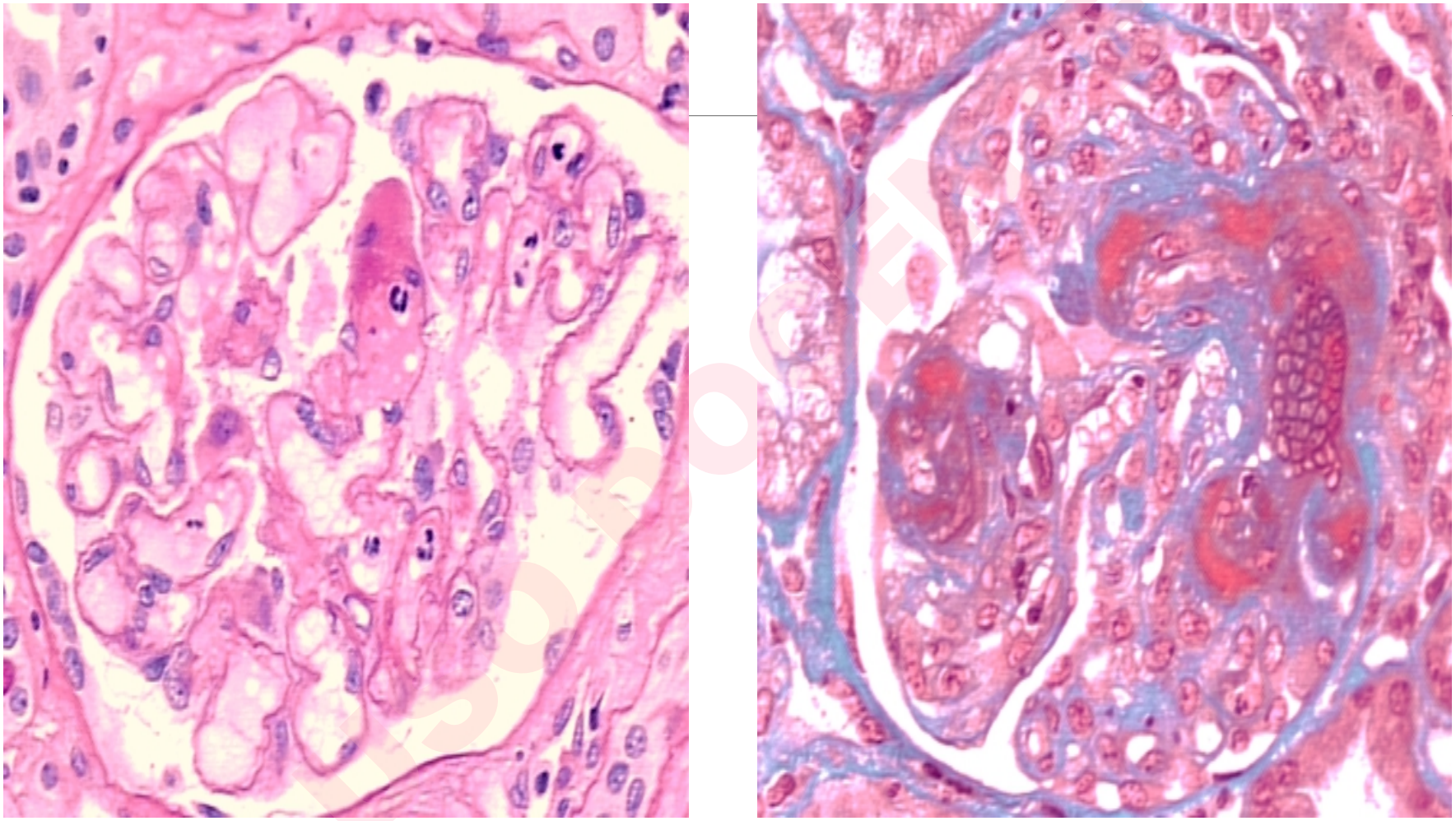
F/M: Femenino/Masculino. Cr(s): Creatinina sérica. BR: Biopsia Renal. HTA: Hipertensión arterial sistémica. SAF: Sindrome antifosfolipídico. TSR: Tratamiento de sustitución renal. TR: Trasplante renal. AC: Anticoagulación. CC: Corticoides. AAS: Ácido acetil salicílico. RPT: Recambio plasmático terapéutico.

Resultados

Paciente	AL	aCL títulos hasta 25 unidades	aCL títulos 26-40 unidades	aCL títulos >40 unidades	aCL IgM/IgG	Anti b2GPI ≤25 unidades	Anti b2GPI ≥26 unidades	ANA/Anti DNA	TV/TA/complic. Obstétricas	AH/Trombocitopenia	SAF C
1	+			+	+/+			-	-/-/-	+/+	-
2	+			+	-/+			+/-	+/+/+	+/+	+
3	+		+		-/+			-	+/-/-	-/-	-
4	+			+	-/+			-	-/+/-	-/-	-
5	+			+	-/+			+/-	+/-/-	+/+	-
6	+			+	+/+	+		-/-	-/-/-	-/+	-
7	-		+		+/+		+	+/-	+/-/-	-/-	-
8	-			+	-/+		+	+/-	+/+/+	-/+	-
9	-	+			-/+		+	-/-	-/-/-	-/-	-

Tabla 2: Tipos de anticuerpos aPL y eventos clínicos AL: Anticuerpo anticoagulante lúpico. aCL: Anticuerpo anticardiolipina. B2GPI: Anticuerpo anti b2 glicoproteína I. ANA: Anticuerpos antinucleares. AH: Anemia hemolítica. SAF C: Sindrome antifosfolipídico catastrófico.

Figura 1: Fotos histología renal caso 7



Trombosis capilar glomerular:

Discusión y Conclusiones

- Retraso considerable entre diagnóstico SAF y biopsia renal (excepción de un caso con SNo). Media de 52,04 meses.
- AUA e HTA con o sin caída del FG son las manifestaciones nefrourológicas más frecuentes.
- Pru y microHu presente en todos los pacientes. HTA al inicio o evolución en 8/9 de pacientes.
- Descenso del FG fue el hallazgo observado en 6/9 pacientes al seguimiento.

- No siempre existe presentación como IRRP. ¿ causa de demora en la solicitud de biopsia renal?.
- 3 pacientes requirieron diálisis en forma definitiva (los 3 tenían Cr mayor a 1,9 mg/dl inicio renal).

- Eventos trombóticos estuvieron presentes en 6/9 pacientes previo a PBR.
- 2/6 pacientes tuvieron complicaciones obstétricas. Un caso de SAFc y muerte.

- La doble positividad para inhibidor lúpico y anticuerpos anticardiolipina IgG fue el perfil de aPL´s más frecuentemente hallado en nuestra serie.

Discusión y Conclusiones

Recomienda:

- aPL a todo paciente que se presente con alteraciones del sedimento urinario con o sin insuficiencia renal.
- La presencia de microhematuria con proteinuria e hipertensión arterial debe hacer sospechar la presencia o concomitancia de esta patología como forma de manifestación renal.
- Abocamos por la realización precoz del estudio histológico renal dada la *variedad de posibilidades histológicas que podemos encontrar y destacando que el hallazgo de microangiopatía trombótica puede presentarse aún sin insuficiencia renal severa*.
- Correlación entre MAT renal asociada a aPL y manifestaciones clínicas como Pru-HTA y descenso de FG.
- Seguimiento importa por causas secundarias.
- Modificar criterios Sídney diagnósticos a la luz de conocimientos actuales, incluir nefropatía asociada a aPL y otros “no criterios” a la definición.

PENSAR en SAF ante AUA + HTA ± Descenso de FG.

Muchas Gracias!