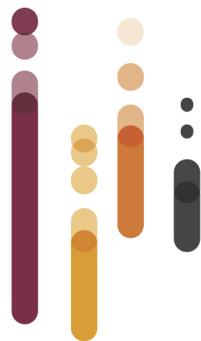


10 al 14 de
noviembre
2024
Ciudad San Diego
(USA)


Sociedad Española de
Reumatología

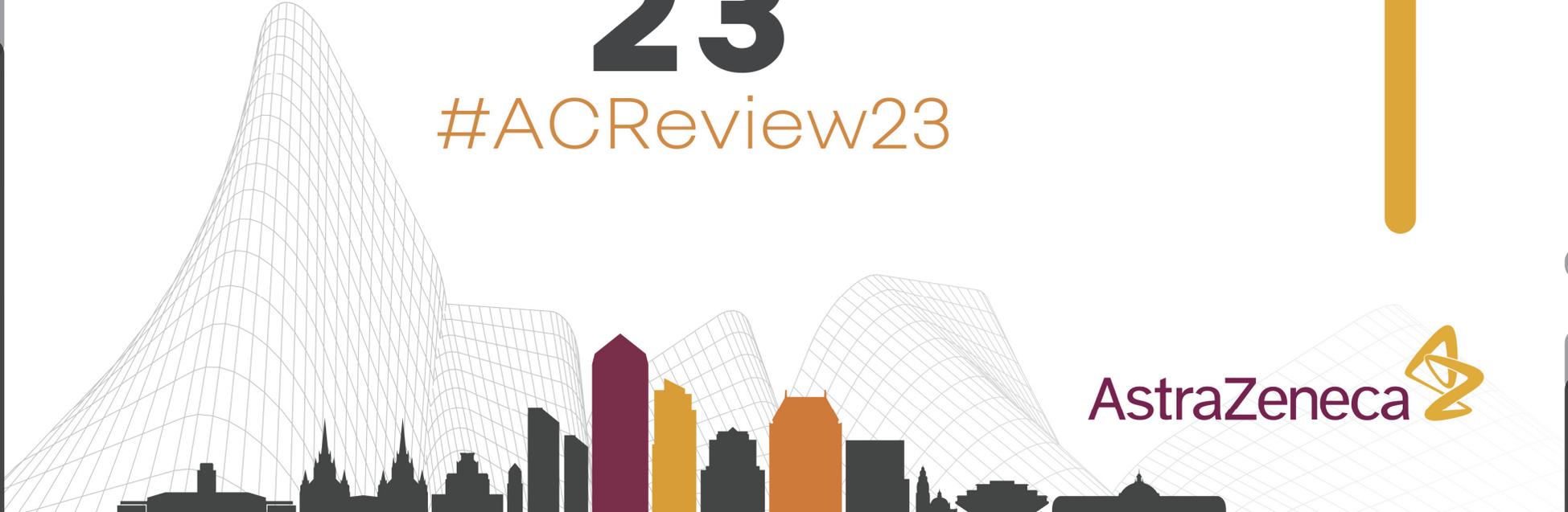


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Enfermedades Autoinmunes Sistémicas: LES-Sjögren y Esclerosis

Dra. Irene Altabás González

Servicio de Reumatología
Complejo Hospitalario Universitario de Vigo. Pontevedra



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ESCLEROSIS SISTÉMICA -Tratamientos-



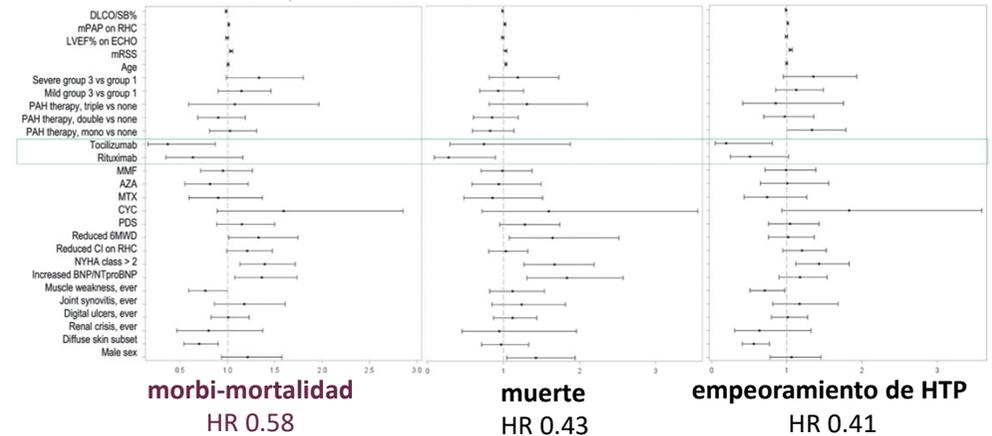
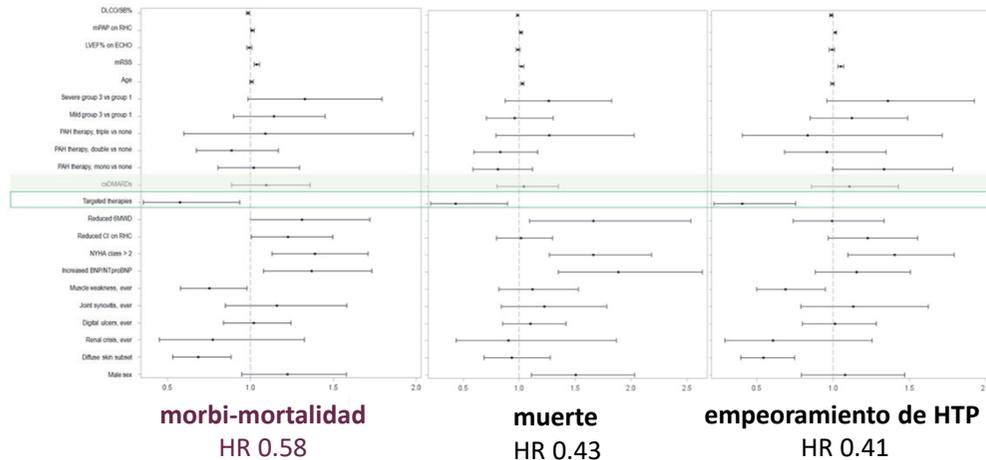
Immunosuppression with Targeted Therapies Reduces Morbidity and Mortality in Pre-Capillary Pulmonary Hypertension Associated with Systemic Sclerosis: A EUSTAR Analysis

Evaluar si IMS afecta la morbi-mortalidad en HTP precapilar asociada a Esclerosis sistémica en la cohorte EUSTAR

755 pacientes con HTP (edad media 63 años, duración media de enfermedad 11 años, 60% EPID en TACAR)

-377 (50%) recibieron IMS: 365 (47%) FAMEc y 68 (9%) terapias biológicas.

* Pacientes con IMS presentaban más EPID, SSd, afectación articular y muscular.



En esta gran cohorte EUSTAR, las terapias biológicas se asocian con un menor riesgo de mortalidad y de empeoramiento de la HTP.

Los ECA deberían explorar más a fondo el impacto de las terapias biológicas para el tratamiento de HTP.

Outcomes in Systemic Sclerosis Patients Treated with Rituximab and Mycophenolate Mofetil Combination Therapy Compared to Autologous Hematological Stem Cell Transplantation

Comparar de forma retrospectiva pacientes con criterios de trasplante autólogo de células hematopoyéticas (AHSCT) que recibieron **combinación de RTX+ MMF (n=21) vs AHSCT(n=16)**.

End-points 1º:

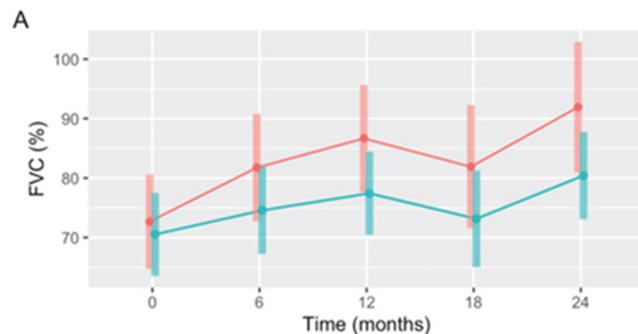
-a 12 meses: descenso de mRSS >25%, o aumento de FVC >10%

13 (81%) **AHSCT** vs 18 (86%) **combinación** $p=0.7$

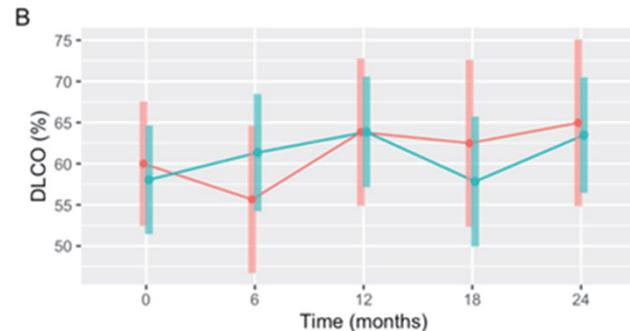
-a 24 meses: supervivencia libre de evento: supervivencia sin fallo de órgano mayor (cardíaco, pulmonar, renal)

-muertes: 3/16 (18.7%) **AHSCT** vs 0 en **combinación**. $HR=0.09$ a favor de combinación

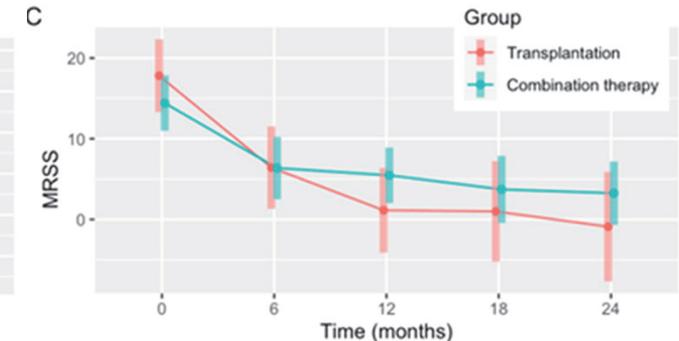
End-points 2º: cambios de mRSS, FVC y DLCO desde basal



Aumento similar en ambos grupos de forma significativa



No se observó incremento de DLCO

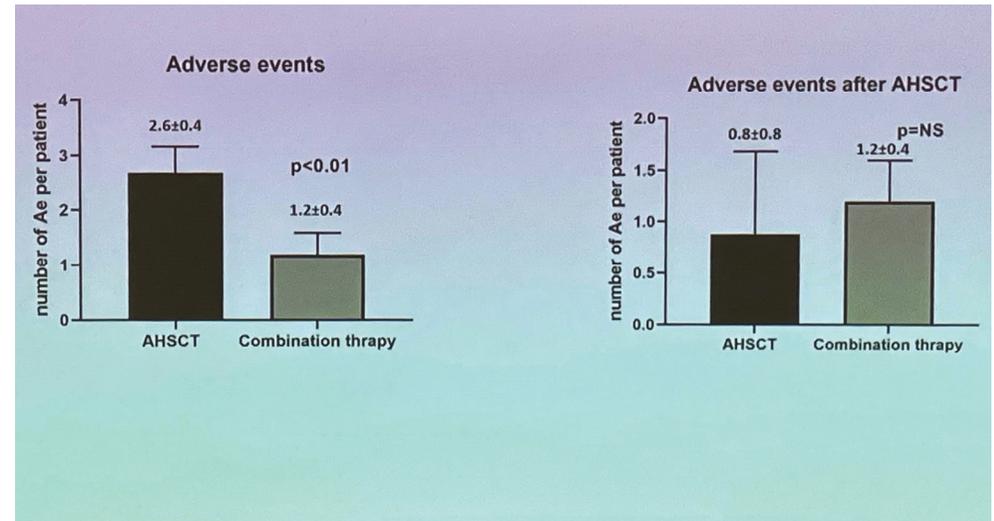


Reducción similar en ambos grupos de forma significativa

Seguridad

- **Más eventos adversos en AHST vs Combinación: 43 vs 25 ($p < 0.001$)**
- Mayor incidencia de **eventos adversos serios en AHST vs combinación: 21 vs 3 ($p < 0.001$)**
- **Tras AHST los eventos adversos fueron similares entre ambos grupos**

Safety- adverse events		AMERICAN COLLEGE of RHEUMATOLOGY <i>Empowering Rheumatology Professionals</i>	
	AHST group (n=16)		Combination group (n=21)
	Adverse events during hospitalization for AHST	Adverse events following AHST	
Serious adverse events	Neutropenic fever (n=8)	Guillain-Barre syndrome	Severe Covid-19 (n=2)
	Pneumonia (n=2)	CMV pericarditis	Severe pneumonia
	Invasive pulmonary aspergillosis	Scleroderma renal crisis	
	Cellulitis	Late cyclophosphamide cardiotoxicity	
Mild adverse events	Perimyocarditis (n=3)	RSV infection	
	Capillary leak syndrome		
	Mucositis (n=6)	Mild Covid-19 (n=7)	Mild Covid-19 (n=10)
	GI bleeding (n=2)	Influenza infection	Pneumonia (n=5)
	Clostridium difficile-associated diarrhea	New onset hypothyroidism	Allergic reaction to rituximab (rash)
	Urinary tract infection	Herpes Zoster reactivation	Allergic reaction to MMF (rash)
	Superficial vein thrombosis		Herpes Zoster reactivation
	Fever		Fungal skin infection
			Scabies rash
			Campylobacter-associated diarrhea
		Nausea	



En esta cohorte, la **terapia combinada de MMF y rituximab** en comparación con AHST en pacientes con ES que cumplen criterios para AHST dio como resultado **una mejoría clínica similar en piel y pulmón con un mejor perfil de seguridad después de 24 meses.**



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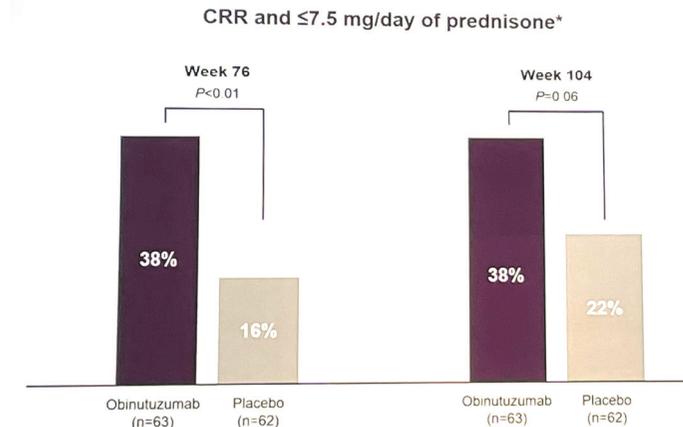
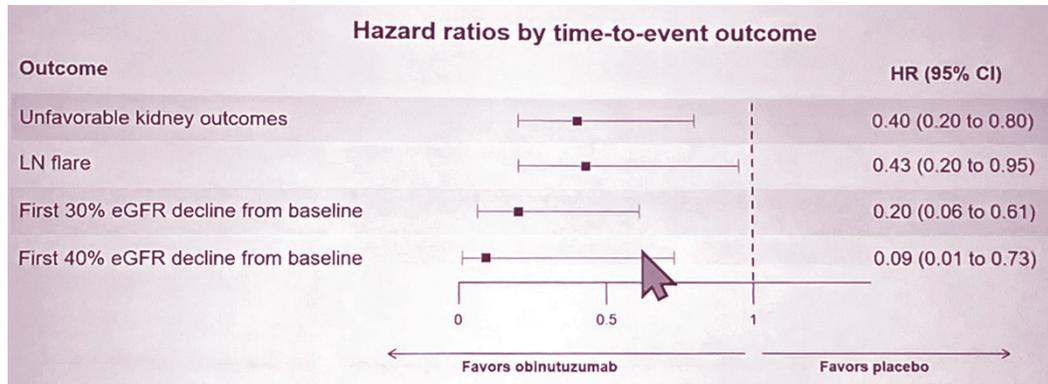
LUPUS ERITEMATOSO SISTÉMICO -Nefritis lúpica-



Kidney-Related Outcomes and Steroid-Sparing Effects in Patients with Active Lupus Nephritis Treated with Obinutuzumab: A Post Hoc Analysis of a Phase 2 Trial

Análisis post-hoc del NOBILITY (Obinutuzumab en NL)

Se evaluó **tiempo hasta primer evento renal** (muerte, duplicación de creatinina o fallo de tto), **brote de renal, caída del 30% y 40% del eGFR, y ahorro de GC.**



Mayores tasas de CRR con ≤ 7.5 mg/día de prednisona
38% vs 16.1 (p=0.001)

- Obinutuzumab reduce de forma significativa el riesgo de eventos renales adversos, deterioro de la función renal y tiempo hasta brote renal, lo que sugiere que este fármaco asociado a SoC puede suponer un impacto en outcomes renales.
- Adicionalmente se observó efecto ahorrador de corticoides a la semana 76.
- Fase 3: REGENCY trial en curso.

Comparison of Dual-immunosuppressive Therapy with a Voclosporin-based, Triple-immunosuppressive Regimen for Lupus Nephritis in the ALMS and AURORA 1 Studies

Comparar y analizar la seguridad y eficacia mediante propensity score de pacientes del ensayo ALMS vs AURORA1.

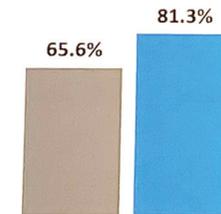
- **ALMS:** MMF 3g/día + GC
- **AURORA 1:** Voclosporina 23.7mg/2 veces día + MMF 2g/día + GC Distinto protocolo de GC.

Mediante propensity matching se identificaron 96 parejas de pacientes con similares características
*más pacientes hispánicos y afroamericanos en AURORA 1 y más asiáticos en ALMS.

	3 Months		6 Months		
	ALMS N=96	AURORA 1 N=96	ALMS N=96	AURORA 1 N=96	
Glucocorticoids	Daily dose n	92	95	86	92
	Mean (SD), mg	21.8 (5.8)	6.13 (3.7)	10.1 (2.8)	5.1 (11.2)
Cumulative exposure	Mean (SD), mg	2849.8 (544.6)	1104.9 (142.8)	3818.5 (777.5)	1502.3 (410.0)
	Daily dose n	89	92	80	90
MMF	Mean (SD), g	2.8 (0.6)	1.9 (0.5)	2.8 (0.4)	1.9 (0.6)
	Cumulative exposure Mean (SD), g	209.8 (45.2)	158.4 (40.5)	414.3 (117.9)	315.3 (86.9)

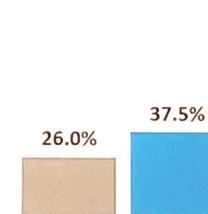
UPCR Reduction >25% at 3 Months

OR 2.50
CI 1.23, 5.08
p = 0.011



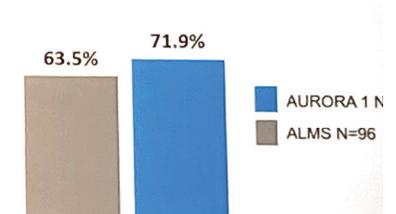
UPCR ≤0.5 g/g at 6 Months

OR 1.85
CI 0.94, 3.65
p = 0.075



UPCR Reduction >50% at 6 Months

OR 1.54
CI 0.8, 2.95
p = 0.196



- La **exposición a MMF y GC** fue **mayor en ALMS**
- A 6 meses, el **porcentaje de pacientes con ≤ 7.5mg prednisona: ALMS 9.4% vs AURORA 85%**

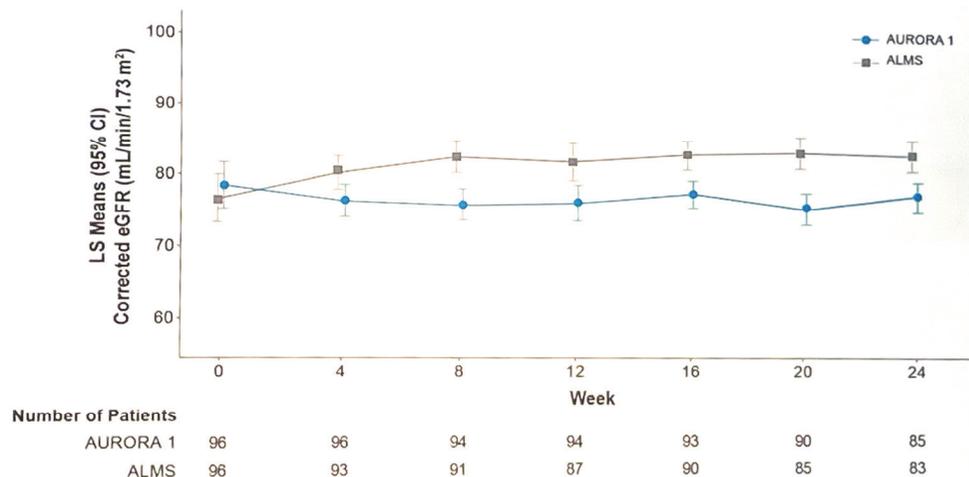
Mayor proporción de pacientes alcanzan **objetivos de proteinuria** a 3 y 6 meses en **AURORA 1**.

Menos efectos adversos en AURORA 1

n (%)	3 Months		6 Months	
	ALMS N=96	AURORA 1 N=96	ALMS N=96	AURORA 1 N=96
Adverse Event (AE)	89 (92.7)	81 (84.4)	92 (95.8)	88 (91.7)
Serious Adverse Event	18 (18.8)	13 (13.5)	22 (22.9)	18 (18.8)
AE leading to study drug discontinuation	7 (7.3)	4 (4.2)	10 (10.4)	7 (7.3)
Death	3 (3.1)	0 (0.0)	3 (3.1)	0 (0.0)

System Organ Class	Preferred Term	3 Months		6 Months	
		ALMS N=96	AURORA 1 N=96	ALMS N=96	AURORA 1 N=96
Infections and infestations		57 (59.4)	45 (46.9)	69 (71.9)	60 (62.5)
Serious Infections and infestations		9 (9.4)	7 (7.3)	12 (12.5)	8 (8.3)
Gastrointestinal disorders		49 (51.0)	30 (31.3)	57 (59.4)	38 (39.6)
Musculoskeletal/connective tissue disorders		38 (39.6)	17 (17.7)	45 (46.9)	22 (22.9)
Skin and subcutaneous disorders		30 (31.3)	17 (17.7)	41 (42.7)	22 (22.9)
Renal/urinary disorders		6 (6.3)	8 (8.3)	11 (11.5)	11 (11.5)
Psychiatric disorders		16 (16.7)	2 (2.1)	16 (16.7)	4 (4.2)
GFR decreased*		0	16 (16.7)	0	20 (20.8)
Hypertension		11 (11.5)	14 (14.6)	14 (14.6)	16 (16.7)
Hyperglycemia		2 (2.1)	0	5 (5.2)	0
Cushingoid/Cushing's syndrome		8 (8.3)	0	8 (8.3)	0

La función renal (eGFR) se mantuvo estable en los 6 meses de evaluación.



La combinación de voclosporina con dosis bajas de GC y MMF en AURORA 1 se asoció a mejor perfil de seguridad a 3 y 6 meses en comparación con el régimen de tratamiento del ALMS. Los pacientes del AURORA 1 experimentaron reducciones de proteinuria más tempranas. Estos resultados podrían justificar la triple terapia de inicio.

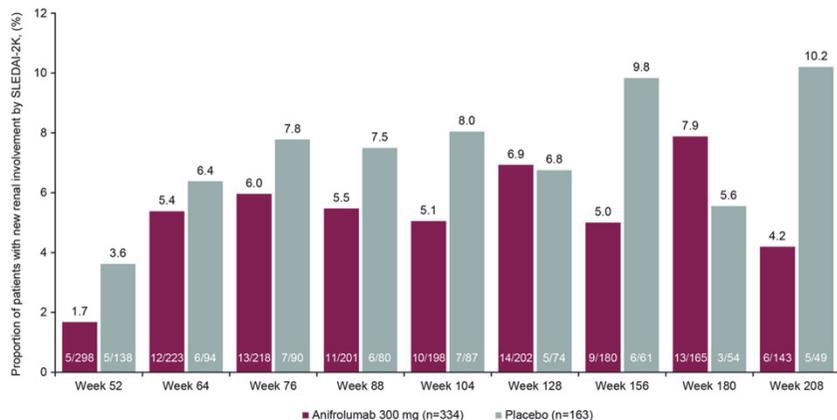
Renal Involvement in Patients with Systemic Lupus Erythematosus Treated with Anifrolumab Compared with Placebo over a 4-Year Period

Análisis post-hoc del dominio renal en la fase de extensión del TULIP (anifrolumab)

Pacientes que completaron TULIP 1 y 2 (52 semanas), que se incluyeron en fase de extensión (3 años).

*TULIP excluidos pacientes con proteinuria UPCR>2mg/g o creatinina >2 mg/dl.

- Compararon 2 grupos: afectación renal por SLEDAI vs no afectación renal desde semana 52.



Subgrupo de pacientes con afectación renal al inicio del estudio: mayor proporción de los tratados con **anifrolumab** lograron una **mejoría renal en SLEDAI-2K** en comparación con el placebo durante el ensayo TULIP-LTE de 4 años.

De los pacientes sin afectación renal al inicio del estudio, **menos pacientes tuvieron nueva actividad renal con anifrolumab** en comparación con placebo.

En curso ensayo clínico de anifrolumab en nefritis lúpica.



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SÍNDROME ANTIFOSFOLÍPIDO



¡ NUEVOS CRITERIOS !

2023 ACR/EULAR antiphospholipid syndrome classification criteria

Criterio de entrada

Al menos **1 criterio clínico** documentado (*de la lista)

+

Ac antifosfolípido positivo

(anticoagulante lúpico o títulos moderados-altos de anticardiolipina o a-B2-GPI (IgG o IgM) **en los últimos 3 años.**

CRITERIOS CLÍNICOS (6 Dominios)

- Macrovascular (tromboembolismo venoso)
- Macrovascular (trombosis arterial)
- Microvascular: sospecha o establecido
- Obstétrico
- Valvular cardiaco
- Hematológico

CRITERIOS LABORATORIO (2 dominios)

- Anticoagulante lúpico: **en 1 sola ocasión ya puntúa**
- Anticardiolipina y anti- β 2-glicoproteína I (anti- β 2GPI)***se da distinto peso a a los títulos de Ac.**

Calculadora online: <https://rheumcalc.com/APS/>

New

Domain 1 — Macrovascular (venous thromboembolism)

Venous thromboembolism (otherwise unexplained* and confirmed by appropriate testing): Includes (but is not limited to) pulmonary embolism, deep vein thrombosis of the legs/arms, splanchnic thrombosis, renal vein thrombosis, cerebral venous thrombosis, and retinal vein thrombosis/occlusion.

Domain 2 — Macrovascular (arterial thrombosis)

Arterial thrombosis (otherwise unexplained* and confirmed by appropriate testing): Includes (but is not limited to) myocardial infarction (coronary artery thrombosis), peripheral/splanchnic/retinal artery thromboses, stroke based on international definitions,^{35 36} and other organ infarcts (eg, kidney, liver, or spleen) in the absence of visualised thrombus.

Domain 3 — Microvascular

Suspected:

Livedo racemosa (by physical examination): Otherwise unexplained* violaceous, "net-like," blotchy mottling of the skin. Note: livedo racemosa with nonuniform, irreversible, broken, and asymmetric persistent discoloration should be scored; *livedo reticularis with uniform, reversible, unbroken, and symmetric discoloration should not be scored*.

Livedoid vasculopathy lesions (by physical examination): Otherwise unexplained* painful papules and erythematous-violaceous purpuric plaques, which may rapidly evolve into haemorrhagic vesicles or bullae. Note: if ruptured, can result in painful small ulcers or reticulate, confluent, geometric, and painful ulcers.

Antiphospholipid antibody (aPL) nephropathy (by physical examination or laboratory tests): Otherwise unexplained persistent: (a) new-onset hypertension or deterioration of previously well-controlled hypertension; (b) proteinuria ≥ 0.5 gm in 24-hour urine specimen or protein:creatinine ratio ≥ 0.5 mg/mg (50 mg/mmoles); (c) acute renal failure (increased serum creatinine levels above normal); or (d) glomerular microscopic hematuria.

Pulmonary hemorrhage (by clinical symptoms and imaging): Respiratory symptoms (eg, dyspnoea, cough, hemoptysis) AND otherwise unexplained* pulmonary infiltrates on imaging suggestive of pulmonary hemorrhage.

Established

Livedoid vasculopathy (by pathology once livedoid vasculopathy lesions described above are present): Otherwise unexplained thrombosis of the small dermal vessels and/or endothelial proliferation.

aPL nephropathy (by pathology once suspected aPL-nephropathy definition above is fulfilled)³⁷: (a) **Acute renal vascular or glomerular thrombotic microangiopathy lesions**, including fibrin thrombi in arterioles or glomeruli without inflammatory cells or immune complexes; and (b) **chronic renal vascular or glomerular lesions**, described as arterial or arteriolar organised microthrombi with or without recanalisation, fibrous and fibrocellular (arterial or arteriolar) occlusions, focal cortical atrophy with or without thyroidization, fibrous intimal hyperplasia, or chronic/organised glomerular thrombi. Note: in patients with systemic lupus erythematosus, aPL nephropathy occurs independent of lesions attributable to lupus nephritis.

Pulmonary hemorrhage (by bronchoalveolar lavage [BAL] or pathology once suspected pulmonary hemorrhage definition above is fulfilled): Otherwise unexplained* progressive haemorrhagic return on BAL with aliquots or hemosiderin-laden macrophages (>20%), OR lung biopsy demonstrating capillaritis or microthrombosis.

Myocardial disease (by imaging or pathology): Otherwise unexplained* non-ST segment elevation myocardial infarction with a normal coronary angiogram (myocardial infarction with nonobstructive coronary arteries, or MINOCA) AND cardiac magnetic resonance imaging (CMRI) abnormalities as per the 2018 Society for CMRI expert consensus³⁸ including: (a) late gadolinium enhancement either transmurally or subendocardially; (b) T2 abnormalities (weighted imaging or mapping); or (c) perfusion MRI abnormalities, OR histologically by thrombosis of the small vessels of the heart.

Adrenal hemorrhage or microthrombosis (by imaging or pathology): Otherwise unexplained* CT or MRI demonstrating hemorrhage, OR histologically by thrombosis of the adrenal (micro)vasculature, for example, adrenal plexus, adrenal vein.

Confirmed
por biopsia

Domain 4 — Obstetric

Prefetal death (preembryonic or embryonic loss): Otherwise unexplained* pregnancy loss before 10 weeks 0 days of gestation. *Ya no es necesario 3 o más

Fetal death: Otherwise unexplained* pregnancy loss between 10 weeks 0 days and 15 weeks 6 days gestation (early fetal death), or between 16 weeks 0 days and 34 weeks 0 days gestation. Note: if a detailed analysis of the fetal morphology or genetic constitution is not performed or unavailable, reasonable clinical judgement should be used based on careful history and review of available medical records.

Preeclampsia with severe features:³⁹ Preeclampsia defined as a systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg on 2 occasions at least 4 hours apart after 20 weeks of gestation in a previously normotensive or hypertensive (chronic†) patient **AND** new onset of one or more of the following: (a) proteinuria ≥ 0.3 mg/mg (30 mg/mmoles) in a random urine specimen or (b) dipstick protein $\geq 2+$ if a quantitative measurement is unavailable **AND** one or more of the following "severe features":

Severe blood pressure elevation: Systolic blood pressure ≥ 160 mm Hg or diastolic blood pressure ≥ 110 mm Hg on 2 occasions at least 4 hours apart while the patient is on bed rest (antihypertensive therapy may be initiated on confirmation of severe hypertension, in which case severe blood pressure elevation criteria can be satisfied without waiting until 4 hours have elapsed).

Central nervous system dysfunction: New-onset headache unresponsive to medication and not accounted for by alternative diagnosis.

Visual disturbances.

Pulmonary oedema.

Impaired liver function: Abnormally elevated blood concentrations of liver enzymes (more than twice the upper limit of normal concentrations), or severe persistent right upper quadrant or epigastric pain unresponsive to medications, not accounted by alternative diagnosis.

Renal dysfunction: Serum creatinine concentration >1.1 mg/dL or a doubling of the serum creatinine concentration in the absence of other renal disease.

Thrombocytopenia: platelet count of $<100 \times 10^9$ /liter.

Placental insufficiency with severe features Intrauterine fetal growth restriction defined as biometry indicating estimated fetal weight of less than the 10th percentile for gestational age or postnatal birth weight less than the 10th percentile for gestational age in the absence of fetal-neonatal syndromes or genetic conditions associated with growth restriction **AND** one or more of the following "severe features":

Abnormal or non-reassuring fetal surveillance test(s) suggestive of fetal hypoxemia, e.g., a nonreactive non-stress test

Abnormal Doppler flow velocimetry waveform analysis suggestive of fetal hypoxemia, e.g., absent end-diastolic flow in the umbilical artery.

Severe intrauterine fetal growth restriction suggested by fetal biometry indicating an estimated fetal or postnatal birth weight of <3 rd percentile for gestational age.

Oligohydramnios, e.g., an amniotic fluid index ≤ 5 cm, or deepest vertical pocket <2 cm.

Maternal vascular malperfusion on placental histology suggested by placental thrombosis/infarction, inadequate remodelling of the uterine spiral arteries (decidual vasculopathy), decreased vasculosyncytial membranes, increased syncytial knots, or decidual inflammation.⁴⁰ Note: Maternal vascular malperfusion on placental histology can be detected in the placentas of aPL-negative patients with intrauterine growth restriction and/or preeclampsia, and even in normal pregnancies; thus, these findings are not specific for APS.

*Ya no es necesario nacimiento prematuro por preeclampsia o insuficiencia placentaria. La patología en sí misma cuenta.

New

Domain 5 – Cardiac valve

Valve thickening (otherwise unexplained*): Based on World Heart Federation echocardiographic criteria,⁴¹ mitral valve thickening is defined as >4 mm between ages 20–39 years and >5 mm for those older than age 40 years, and >3 mm for other valves for any age (valve thickening can be associated with valvular dysfunction (regurgitation or stenosis)).

Valve vegetation (otherwise unexplained*): Based on the American Society of Echocardiography guidelines,⁴² valve vegetation is defined as shaggy, lobulated, or rounded masses typically located on the atrial side of atrioventricular valves (mitral valve and tricuspid valve) or ventricular side of the aortic valve, but can be located on any side of any valve (size is highly variable but usually <1 cm); on echocardiogram, despite the “echo texture” and location of aPL-associated vegetations resembling infective endocarditis, they may appear less amorphous, more rounded, and not associated with valvular destruction, in contrast to a true infective endocarditis; they can be associated with valvular dysfunction (regurgitation or stenosis).

Domain 6 – Haematology

New

Thrombocytopenia: Otherwise unexplained* lowest platelet count ever between 20 and 130×10^9 /liter, confirmed on peripheral blood smear and by repeat testing.

Entry Criteria^(a)
 At least one documented^(b) clinical criterion listed below (domains 1-6)
plus
 A positive antiphospholipid antibody (aPL) test
 (a lupus anticoagulant test, or moderate-to-high titers of anticardiolipin or anti-β₂-glycoprotein-I antibodies [IgG or IgM])
 within three years^(b) of the clinical criterion



If absent, do not attempt to classify as APS - If present, apply additive criteria

Additive clinical and laboratory criteria^(a)		
Do not count a clinical criterion if there is an equally or more likely explanation than APS. Within each domain, only count the highest weighted criterion towards the total score.		
Clinical domains and criteria	Weight	Weight
D1. Macrovascular (Venous Thromboembolism [VTE])		
VTE with a high-risk VTE profile ^(c)	1	
VTE without a high-risk VTE profile ^(c)	3	
D3. Microvascular		
Suspected (one or more of the following)	2	
Livedo racemosa (exam)		
Livedoid vasculopathy lesions (exam)		
Acute/chronic aPL-nephropathy (exam or lab)		
Pulmonary hemorrhage (symptoms and imaging)		
Established (one of more of the following)	5	
Livedoid vasculopathy (pathology ^(d))		
Acute/chronic aPL-nephropathy (pathology ^(d))		
Pulmonary hemorrhage (BAL or pathology ^(d))		
Myocardial disease (imaging or pathology)		
Adrenal hemorrhage (imaging or pathology)		
D5. Cardiac Valve		
Thickening	2	
Vegetation	4	
D2. Macrovascular (Arterial Thrombosis [AT])		
AT with a high-risk CVD profile ^(c)		2
AT without a high-risk CVD profile ^(c)		4
D4. Obstetric		
≥3 Consecutive pre-fetal (<10w) and/or early fetal (10w 0d -15w 6d) deaths		1
Fetal death (16w 0d – 33w 6d) in the absence of pre-eclampsia (PEC) with severe features or placental insufficiency (PI) with severe features		1
PEC with severe features (<34w 0d) <u>or</u> PI with severe features (<34w 0d) with/without fetal death		3
PEC with severe features (<34w 0d) <u>and</u> PI with severe features (<34w 0d) with/without fetal death		4
D6. Hematology		
Thrombocytopenia (lowest 20-130x10 ⁹ /L)		2

Laboratory (aPL) domains and criteria ^(a)	Weight
D7. aPL test by coagulation-based functional assay (lupus anticoagulant test [LAC])	
Positive LAC (single – one time)	1
Positive LAC (persistent)	5
D8. aPL test by solid phase assay (anti-cardiolipin antibody [aCL] ELISA and/or anti-β₂-glycoprotein-I antibody [aβ₂GPI] ELISA [persistent])	
Moderate or high positive (IgM) (aCL and/or aβ ₂ GPI)	1
Moderate positive (IgG) (aCL and/or aβ ₂ GPI)	4
High positive (IgG) (aCL <u>or</u> aβ ₂ GPI)	5
High positive (IgG) (aCL <u>and</u> aβ ₂ GPI)	7

CLASIFICACIÓN DE SAF
 Al menos **3 puntos** de dominios clínicos
Y
 Al menos **3 puntos** de laboratorio



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SÍNDROME DE SJÖGREN

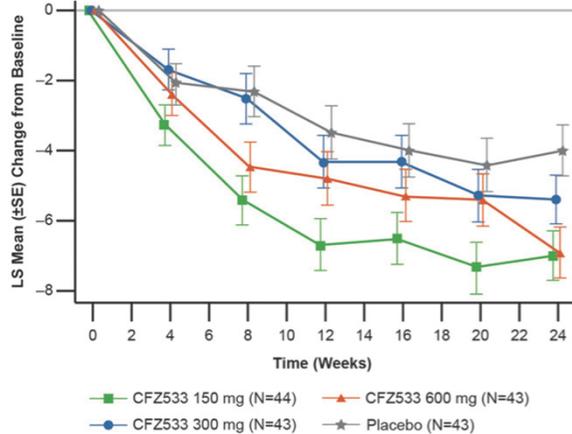


ABS: 1634 Fisher B. et al.

Iscalimab (CFZ533) in Patients with Sjögren's Disease: Week 24 Efficacy and Safety Results of a Randomized, Placebo-controlled, Phase 2b Dose-ranging Study

Cohorte 1: 1º end-point cambio ESSDAI a s24
Mejoría significativa en ESSDAI dosis 150 y 600mg

A. ESSDAI scores over Week 24



Cohorte 2: 1º end-point cambio ESSPRI a s24

- Mejoría ESSPRI y fatiga (no significativa)
- Mejoría en sequedad ($p=0.01$)

Ac monoclonal contra CD40

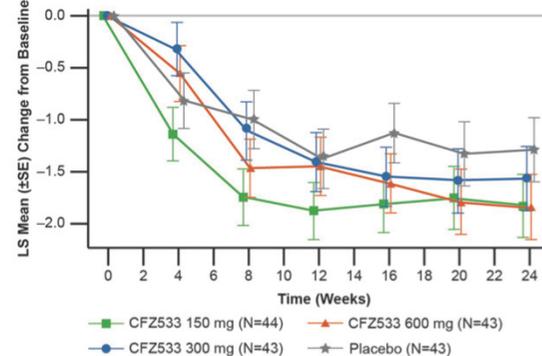
Evaluar seguridad y eficacia de distintas dosis de Iscalimab en

- Cohorte 1: SS con enf. Sistémica moderada-severa (ESSDAI ≥ 5 y ESSPRI ≥ 5).
 - Cohorte 2*: SS con enf. Sistémica leve (ESSDAI < 5 y ESSPRI ≥ 5 en fatiga y sequedad)
- * solo dosis 600mg

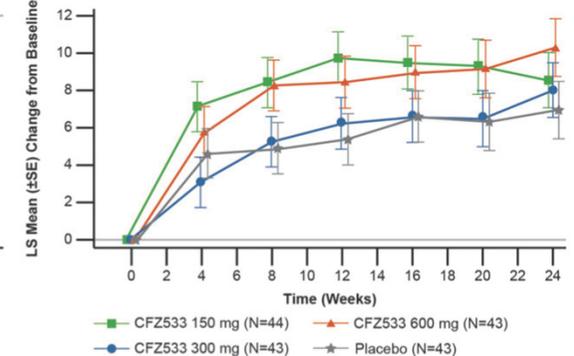
2º End-points

Tendencia a mejoría en ESSPRI, FACIT, flujo lacrimal y salival (n/s)

A. ESSPRI



B. FACIT-F



Iscalimab mostró una mejora clínicamente importante en comparación con placebo en 2 poblaciones distintas de pacientes con Sd Sjögren, bien tolerado y sin problemas de seguridad.

SJOGRENSER Registry: Prospective Evaluation of a Cohort of Patients with Primary Sjögren's Syndrome After 8 Years of Follow-up

Describir la evolución de los pacientes con síndrome de Sjögren (SS) en relación con la **aparición de nuevas manifestaciones** sistémicas y la actividad de la enfermedad, así como los factores de mal pronóstico.

Se comparó fase SS-TRANS y SS-PROS (8 años de diferencia)

180 pacientes (96% mujeres, edad media 68 años, evolución media de la enfermedad 18 años).

Nuevas manifestaciones desarrolladas PROS:

- 47% articular
- 44% citopenias
- 14% afectación pulmonar
- 13% renal
- 13% parotiditis
- 17% digestivo
- 4% SNC
- 3% SNP
- 2.7% cardiaca

ESSDAI medio: SS-TRANS: 3.66 vs SS-PROS 2.78 ↓

ESSPRI medio: SS-TRANS: 4.47 vs SS-PROS 5.2 ↓

- 3/180 (**1.6%**) nuevos **linfomas**
- **27/180 fallecieron** desde SS-TRANS.

Factores de riesgo de mortalidad:

mayor edad, mayor duración de la enfermedad y mayor ESSDAI

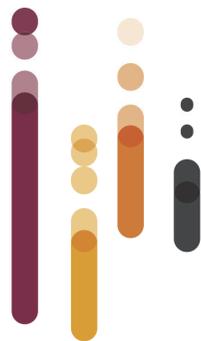
Los pacientes con **SS desarrollan nuevas manifestaciones sistémicas**, a pesar de mantener o mejorar el ESSDAI, lo que sugiere la **necesidad de un seguimiento estrecho**. La ESSPRI varía poco en el tiempo.

La mortalidad en esta cohorte es del 15%.

Mayor edad, tiempo de evolución y ESSDAI basal se asociaron con peor evolución.

10 al 14 de
noviembre
2024
Ciudad San Diego
(USA)


Sociedad Española de
Reumatología



ACRreview 23

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