

patterns over 3 consecutive years, patterns have been defined as: Persistent Long Quiescent (pLQ), Persistent Relapsing-Remitting (pRR), Persistent Chronic Active (pCA) and Mixed, at least 2 different pattern types. Predictors of pCA (vs. pLQ, pRR and mixed) and pLQ (vs. pCA, pRR and mixed) were identified by univariate and multivariate logistic regression analyses. Several baseline demographics (age, sex, ethnicity, disease duration, tobacco use, years of education and combined annual family income), disease characteristics at baseline (SLEDAI, PGA) and treatment categories (hydroxychloroquine, prednisolone and cytotoxic treatment followed at $\geq 75\%$ of visits vs $< 75\%$ of visits) were used as independent variables. **Results:** 916 patients were identified. The results of the univariate analyses for pCA are shown in table 1. In the multivariate model, African American ethnicity (OR: 2.43, 95% CI: 1.19–4.94, $p: 0.01$) and high baseline SLEDAI (OR: 1.09, 95% CI: 1.03–1.16, $p: 0.004$) remained significant predictors of pCA. Higher education (> 12 years; OR: 2.16, 95% CI: 1.11–4.20, $p: 0.02$) and low baseline SLEDAI (OR: 0.62, 95% CI: 0.52–0.75, $p: < 0.001$) were significant predictors of pLQ in the multivariate analysis while African American ethnicity (OR: 0.36, 95% CI: 0.16–0.78, $p: 0.01$) and female patients (OR: 0.26, 95% CI: 0.12–0.56, $p: 0.001$) were less likely to achieve persistent long quiescence.

Table 1. pCA (vs. pLQ, RR and mixed)

	OR (95% CI)	p-value
Sex (female vs. male)	1.6 (0.4–6.7)	0.54
Age (> 40 vs. ≤ 40 yrs)	1.4 (0.7–2.8)	0.32
Disease Duration	1.0 (0.9–1.1)	0.17
African American (vs. other)	2.6 (1.3–5.3)	0.007
Years of education (> 12 vs. \leq yrs)	0.7 (0.3–1.4)	0.27
Income		
<30000\$	Ref	
30000–65000\$	0.4 (0.2–1.0)	0.06
≥ 65000 \$	0.5 (0.2–1.2)	0.12
Smoking baseline (yes vs. no)	2.1 (1.0–4.6)	0.06
SLEDAI baseline	1.1 (1.0–1.2)	0.002
PGA baseline	1.5 (1.0–2.2)	0.04
Cytotoxic therapy ($\geq 75\%$ vs. $< 75\%$ of visits)	1.1 (0.5–2.3)	0.85
Antimalaria therapy ($\geq 75\%$ vs. $< 75\%$ of visits)	0.5 (0.3–1.0)	0.06
Prednisolone therapy ($\geq 75\%$ vs. $< 75\%$ of visits)	1.1 (0.6–2.2)	0.82

Conclusions: In this large SLE cohort, African American ethnicity and high disease activity at the time of diagnosis are predictors of chronic activity, regardless of treatment, even after adjustment for education years and income, while higher education and low disease activity at baseline predict long-term quiescence.

References:

[1] Györi N, et al. Disease activity patterns over time in patients with systemic lupus erythematosus – Analysis of the Hopkins Lupus Cohort. *Lupus Sci Med.* 2017. In press.

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OP0046 RISK FACTORS FOR ADVERSE PREGNANCY OUTCOME IN ANTIPHOSPHOLIPID ANTIBODIES CARRIERS: RESULTS FROM A MULTICENTER ITALIAN COHORT OVER 20 YEARS OF EXPERIENCE

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Background: Antiphospholipid antibodies (aPL) are risk factors for Adverse Pregnancy Outcome (APO). In particular, for aPL carriers (patients with aPL positivity with no thrombotic or obstetric history) pregnancy outcome and treatment are still undefined.

Objectives: To review pregnancy outcome in a large multicenter cohort of aPL carriers patients, according to different serological profile and treatment protocols.

Methods: We reviewed 69 pregnancies in 61 patients, prospectively followed in 3 Italian centers (1994–2015). Patients with any complication in previous pregnancies or concomitant autoimmune diseases were excluded. aPL profile was defined as the combination of the 3 criteria tests for aPL (Lupus Anticoagulant, anti-cardiolipin, anti-Beta2 Glycoprotein I). APO was defined as: miscarriage (< 10 w), fetal death (≥ 10 w), severe preterm delivery (≤ 34 w) with or without preeclampsia (PE), HELLP syndrome or perinatal death.

Results: Regarding aPL profile: 44 (64%) were single positive, 16 (23%) were double and 9 (13%) were triple. aPL non-criteria manifestations were present in 6 (9%) and lupus-like manifestations in 5 (7%). Eight pregnancies had a combination treatment with prophylactic low molecular weight heparin (LMWH) plus low dose aspirin (LDA), 37 had LDA alone and 24 had no treatment. We observed 2 thrombotic events (3%, 1 deep venous thrombosis and 1 ischemic stroke) and 6 APO (9%): 3 fetal deaths and 3 pre-term birth ≤ 34 w with PE. We compared 8 complicated (6 APO, 2 thrombosis) with 61 non-complicated pregnancies (Table 1). Acquired risk factors ($p: 0.006$), non-criteria aPL ($p: 0.018$) and lupus-like manifestation ($p: 0.010$), triple positive aPL profile ($p: 0.001$) were associated with APO. The combination treatment was also more frequent in APO pregnancies ($p: 0.005$).

	Complicated pregnancies (n=8)	Non-complicated pregnancies (n=61)	P-value *	OR (95% CI)
Mean age at pregnancy (mean (SD))	28.3 (5.8)	32.7(4.6)	0.015 †	
Acquired risk factors ‡	6 (75%)	14 (23%)	0.006	10.7 (1.55-82.4)
Congenital thrombophilia	2 (25%)	4 (7%)	0.140	
Non criteria aPL manifestations †	3 (38%)	3 (5%)	0.018	11.6 (1.37-108)
Lupus-like manifestations	3 (38%)	2 (3%)	0.010	17.7 (1.77-213)
Single positive	3 (38%)	41 (67%)	0.128	
Double positive	0 (0%)	16 (26%)	0.183	
Triple positive	5 (63%)	4 (7%)	0.001	23.7 (3.22-211)
No treatment	1 (13%)	23 (38%)	0.246	
LDA	3 (38%)	34 (56%)	0.457	
LDA+LMWH	4 (50%)	4 (7%)	0.005	14.2 (1.98-116)

Table 1. Complicated pregnancies (APO and thrombosis) compared with non-complicated pregnancies – univariate analysis

* Fisher's exact test, except otherwise indicated; † significant < 0.05

‡ Student's T- test

§ Acquired risk factors: Obesity (BMI > 30), hypertension ($\geq 140/90$), hyper-cholesterolemia, hyper-triglyceridemia, hyper-homocysteinemia, diabetes mellitus

† Non-criteria aPL manifestations were defined as the presence of at least one of the following: [livedo reticularis, thrombocytopenia, headache, hemolytic anemia, cardiac valvulopathy, epilepsy]

Conclusions: Acquired risk factors, aPL profile (triple positivity), lupus-like and non-criteria aPL manifestations could represent risk factors for pregnancy complications and could determine failure to conventional treatment. These patients may deserve additional treatment, for example LMWH at higher/anti-coagulant dose or immunomodulatory treatment (e.g. hydroxychloroquine, also considering its anti-platelet/anti-thrombotic effects).

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Osteoporosis treatment gap, new options and new strategies

OP0047 OSTEOPOROTIC HIP FRACTURE PREVENTION: ARE WE IN A CRISIS?

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Background: Osteoporotic hip fractures are a significant cause of morbidity and mortality in the elderly. With the aging of the US population, it was expected that there would be a nationwide "epidemic" of osteoporotic fractures; however, Medicare reimbursement for osteoporosis screening and the rapid uptake of bisphosphonate therapy in the late 1990s and early 2000s probably forestalled this epidemic. In fact, osteoporotic hip fractures numbers declined during this time, despite the dramatic growth of the elderly population (1). However, bisphosphonate prescriptions have dropped precipitously by over 50% from their peak in 2008 (2), likely because of concerns regarding their adverse events. In this era of "crisis" in osteoporosis prevention, has the pendulum swung back on hip fracture numbers?

Objectives: To study the number and prevalence of osteoporotic hip fractures in patients 50 years and older over the last 22 years (1993–2014) in the US?

Methods: The Nationwide Inpatient Sample (NIS) is a stratified random sample of all US community hospitals. It is the only US national hospital database with information on all patients, regardless of payer, including persons covered by Medicare, Medicaid, private insurance, and the uninsured. We examined all inpatient hospitalizations in NIS from 1993 to 2014 with a primary diagnosis of non-traumatic hip fractures, and calculated age-adjusted prevalence rates. We excluded patients with concomitant diagnosis of major trauma, primary or secondary tumors locally, osteopetrosis and procedures for revision surgery or surgery for complications.

US population estimates and projections for the resident US population were obtained from the US Census Bureau.

Results: There were 407 million all-cause hospitalizations in 1.88 billion person-years of observation among people 50 years of older from 1993 to 2014 (21,626 hospitalizations per 100,000 person-years). During this time-period, there were 6.15 million hospitalizations for osteoporotic hip fractures in this population (327 per 100,000 person-years). After an initial increase in hip fractures from 1993 to 1996, there was a decline of 10.6% in total number of hip fractures from 300,154 in 1996 to 268,356 in 2010 even though population of 50 years and older increased from 69.9 million to 99.6 million in the same time. After bottoming out in 2010, the total number of hip fractures increased by 6.2% to 285,050 in 2014 (Figure, blue broken line). However, the age-standardized prevalence rate of hip fractures declined steadily (from 414/100,000 in 1996 to 269/100,000 in 2014, $p < 0.0001$, Figure solid red line), although the rate of decline has ameliorated somewhat from 2010 onwards (not statistically significant).

Conclusions: The total number of osteoporotic hip fractures in 50 years and