were no unexpected new AEs observed in patients who continued with voclosporin treatment compared to control-treated patients for more than one year.

Table 1. UPCR

	Control (n=100)		Voclosporin (n=116)		Treatment Comparison of Voclosporin to Control	
	n	UPCR (mg/mg)	n	UPCR (mg/mg)	UPCR (mg/mg)	p-value
Pre-treatment baseline, mean	100	3.87	116	3.94	NC	NC
Change from pre-treatment baseline, LS mean						
Year 1	100	-2.4	116	-3.0	-0.6	0.0080
Year 2	51	-2.1	73	-3.1	-1.0	0.0004

LS, least squares; NC, not calculated; UPCR, urine protein creatinine ratio. Mixed effects model for repeated measures (MMRM) analysis of LS mean change from pre-treatment baseline for UPCR included terms for baseline covariate, treatment, visit and treatment by visit interaction. Integrated results include data from pre-treatment baseline of AURORA 1, the one-year treatment period in AURORA 1 and up to a one-year treatment period in AURORA 2.

Conclusion: Patients in the voclosporin treatment arm maintained meaningful reductions in proteinuria with no change in mean eGFR at two years of treatment. Additional AURORA 2 efficacy and safety data will be provided at the conclusion of the study.

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POS0682

RETROSPECTIVE ANALYSIS OF PREGNANCY OUTCOMES IN PATIENTS WITH OBSTETRIC ANTIPHOSPHOLIPID SYNDROME (OAPS), NON-CRITERIA OAPS (NC-OAPS) AND ANTIPHOSPHOLIPID CARRIERS

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Background: Antiphospholipid (aPL) antibodies are considered in obstetric morbidity even when Sydney criteria for OAPS are not met. Classification and treatment of NC-OAPS patients and aPL carriers during pregnancy are still debated.

Objectives: To increase knowledge, we evaluated and compared aPL serum profiles, exposure to antithrombotic therapies and pregnancy outcomes in OAPS, NC-OAPS and aPL carrier patients, accessing to our centre.

Methods: A retrospective observational study was conducted on pregnant outpatients from January 2003 to April 2020. According to Sydney revised classification criteria, we considered lupus anticoagulant (LA), IgM and IgG anti-cardiolipin antibodies (aCL), IgM and IgG anti-beta2 glycoprotein I antibodies (aβ2-GPI), to stratify aPL risk profiles [Ref]. In each pregnancy, after case stratification into high (≥ 2 aPL or LA serum positivity) versus low (single aPL positivity) risk profile, we evaluated antithrombotic treatment strategy and subsequent pregnancy outcomes as live-births, spontaneous abortions (SA) or foetal losses.

Results: A total of 78 pregnancies were followed: 17 in OAPS, 9 in NC-OAPS and 52 in aPL carrier patients. Rheumatic diseases (RD) coexisted predominantly in carriers (73.1%), mainly systemic lupus erythematosus (57.9%). As presented in Table 1, in OAPS and aPL plus RD carrier groups the association of acetyl-salicylic acid (ASA - mean dose 100 mg q.d.) and low-molecular weight heparin (LMWH - mean dose 4000 UI q.d.) showed a better rate of positive outcomes (97.8% of pregnancies) in high aPL risk profile, compared to monotherapy, especially with LA or triple aPL positivity. Conversely, negative outcomes occurred mostly with triple aPL positivity in the first group and double aPL in the second, despite therapy approaches. No significant data were obtained in NC-OAPS group, due to its paucity, though adverse outcomes were observed with monotherapy both in high and low risk profiles. Except aPL carriers with RD, in all other low risk subgroups, a prevalence of negative outcomes occurred using ASA alone, without statistical significance (OR 0; p= 0.45). Similarly, considering the whole population, the use of a mono or a combination therapy in high risk subgroups had not a significant correlation with pregnancy outcomes (OR 1.79; 95%CI 0.31-10.15; p= 0.50).

Conclusion: In our study, negative pregnancy outcomes were sporadic, occurring mostly with ≥ 2 aPL positivity. Combination treatment showed better results overall in high aPL risk profile patients, both in OAPS and NC-OAPS or aPL carriers. Though no significant correlation between outcomes and treatments were

found, we hinted how aPL-based risk stratification may be useful in adopting personalised therapies to prevent obstetric failures.

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Table 1. Population groups, aPL risk profiles, antithrombotic therapies and pregnancy outcomes.

Population group	aPL risk profile	Type/n° of aPL	Therapy (n° of pregnancies)	Pregnancy outcomes	
OAPS	High	LA	ASA (1)	1 live-birth	
		2 aPL 3 aPL	ASA + LMWH (1) ASA + LMWH (3) ASA + LMWH (9)	1 live-birth 3 live-births 7 live-births	
			Low	1 aPL	ASA (2)
	ASA + LMWH (1)	1 foetal loss			
	NC-OAPS	High		LA	-
			2 aPL	ASA (1)	- 1 live-birth
Low			3 aPL 1 aPL	LMWH (1) -	1 SA -
	ASA (6)	5 live-births			
	aPL carriers without RD	High	LA	ASA (1)	1 SA
ASA + LMWH (1)				1 live-birth	
ASA (1)				1 live-birth	
Low		1 aPL	LMWH (1) ASA (1)	1 live-birth 1 live-birth	
			LMWH (2) ASA (1)	2 live-births 1 live-birth	
			ASA + LMWH (3) ASA (5)	3 live-births 4 live-births	
aPL carriers with RD	High	LA	ASA (4)	1 foetal loss 3 live-birth	
			ASA + LMWH (7)	1 foetal loss 6 live-births	
			2 aPL	ASA (6)	1 SA 6 live-births
	Low	3 aPL	ASA + LMWH (4)	2 live-births	
			ASA + LMWH (5) ASA (8)	1 SA 1 foetal loss 2 live-births	
			ASA + LMWH (3)	5 live-births 8 live-births 3 live-births	

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NOVEL STRINGENT OUTCOME MEASURES APPLIED TO THE PHASE 2 AND 3 ANIFROLUMAB TRIALS

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