THE EUROPEAN REGISTRY ON OBSTETRIC ANTIPHOSPHOLIPID SYNDROME (EUROAPS): A SURVEY OF 1100 CONSECUTIVE CASES

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Background: The obstetric antiphospholipid syndrome (OAPS) is an autoimmune disease defined by the presence of obstetric complications related to antiphospholipid antibodies. EUROAPS is the biggest published European registry on obstetric antiphospholipid syndrome and it is ongoing.

Objectives: To analyse the clinical features, laboratory data and foetal-maternal outcomes, and follow them up on a cohort of 1100 women with obstetric antiphospholipid syndrome (OAPS).

Methods: Thirty hospitals throughout Europe have collaborated to carry out this registry. Cases with obstetric complaints related to antiphospholipid antibodies (aPL) who tested positive for aPL at least twice were included prospectively and retrospectively. The eight-year survey results are reported.

Results: 1100 women with 3653 episodes were included of which 2553 were historical and 1100 were latest episodes. All cases fulfilled the Sydney classification criteria. According to the laboratory categories: 29.2% were in category I, 35.7% in IIa, 22.4% in IIb and 12.7% in IIc. Miscarriages appeared in 38.6%, 30.6%, 24.8% and 21.9% in categories I, IIa, IIb and IIc, respectively. Patients with recommended treatment had a good live-birth rate (85%), while patients with no treatment showed a poor birth rate (49.6%).

Conclusion: In this series, recurrent miscarriage is the most frequent poor outcome. To avoid false-negative diagnoses, all laboratory category subsets were needed. OAPS cases have very good foetal-maternal outcomes when treated. Results suggest that we were able to improve our clinical practice to offer better treatment and outcomes to OAPS patients.

REFERENCES

ENHANCING THE QUALITY OF CLINICAL DATA THROUGH DATA CURATION IN PRIMARY SJÖGREN’S SYNDROME

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Background: Primary Sjögren’s Syndrome (pSS) is a chronic systemic autoimmune disease that is affecting primarily women near the menopausal age, causing exocrine gland dysfunction, with clinical manifestations varying from dry eye and mouth to multi-systemic disorders [1]. The lack of automated means for data quality improvement in pSS cohorts and the huge time effort needed for manual curation, yield data that are irrelevant and incomplete, introducing undesirable implications in their analysis.

Objectives: To enhance the quality of the clinical data in pSS using automated data curation.

Methods: Anonymized clinical data were recruited from 380 patients with pSS from the University of Athens (UoA) cohort (300 patients, mean age 68.79±14.84) and the Harokopio University of Athens (HUA) cohort (80 patients, mean age 59.23±13.92). The features consist of SS-related measures (see [2] for details). The curation tool produces 3 files: (i) a quality report, including the metadata, the presence of outliers (using the z-score [3]), unknown data types, and missing values, on a feature-basis (data imputation [4] is used to fix features with < 50% missing values), (ii) the curated dataset, where the inconsistencies are marked using color notations, and (iii) a standardization report, where the features that share common terminology with those from a reference model [5] (i.e., a set of parameters that describe the pSS minimal requirements) are identified using lexical matching [6].

Results: For the UoA cohort, out of 167 features, 80 were classified as “bad”, 30 with unknown data type, and 12 were marked for outliers (Fig. 1). An example of an outlier was found for the IgM (1370 mg/dL). For the HUA cohort, out of 204 features, 69 were classified as “bad”, 5 with unknown data type, and 13 were marked for outliers. The standardization process successfully matched 82 out of 88 (93.18%) pSS-related terms for the UoA cohort and 61 out of 69 (88.4%) terms for the HUA cohort.

Conclusion: Our strategy enhances the quality of the pSS clinical data through data curation and reduces the time effort needed for manual curation by the clinicians. The tool produces re-usable reports that can be used to fix inconsistencies, outliers, missing values, and harmonize pSS clinical data [8].

Acknowledgement: Elmina Lefkou, Gerard Espinosa, Sara Tabacco, Luca Marozio, Pier Luigi Meroni, Maria Gerosa, Elisa Picardo, Elisa Llurba, Arsene Mekinian, Bettina Toth, Katharina Brenner, Athanasios Tziofas.

Disclosure of Interests: Enrique Esteve-Valverde: None declared, Jaume Alijotas-Reig: None declared, Raquel Fernandez-Oliveras: None declared, Luis Saez-Comet: None declared, Cristina Belizna: None declared, Amelia Rufatti: None declared, Angela Tincani Consultant for: UCB, Pfizer, Abbvie, BMS, Sanofi, Roche, GSK, AlphaSigma, Lilly, Jannsen, Celgene, Novartis, Sara DeCarolis: None declared, Omar Lozano: None declared, Ricard Cervera: None declared.


REFERENCES
Objectives: To evaluate the development and evolution of SLE after 1,25(OH)2D2 supplementation in the PIL model.

Methods: Female BALB/c mice divided into 3 groups: CO, PIL, and PIL+VD. Lupus was induced in PIL and VD groups using pristane. VD group received a subcutaneous injection of calcitriol [2ug/kg] in PBS-Tween 20 buffer every second day during 180 days. Both groups were examined by direct immunofluorescence. IL-2, IL-4, IL-6, IFN-α, and TNF-α levels were measured by Luminex technology in serum. Data was analyzed with ANOVA Two-Way followed by Bonferroni and independent sample t-test. p<0.05 was considered significant. All data are represented as means±SD.

Results: PIL group showed arthritis and kidney injury, characterized by increased proteinuria, glomerular mesangial expansion and inflammation. Moreover, PIL model showed increased levels of IL-6, TNF-α and IFN-α in serum. VD treatment reduced arthritis incidence compared PIL (42vs85%;p<0.01) at the end of the experimental period. The arthritis clinical score (1.00±1.15vs2.85±1.34;p<0.01) and the hind paws edema (0.20±0.03vs0.24±0.05 mL;p<0.05) in the VD group were also attenuated in relation to the PIL at day 180 after induction. VD was able to reduce synovial hyperplasia (0.0±1vs2.23;p<0.05), erosion in bone (0.0±1 vs2.22;p<0.05) and cartilage (11.2±2vs3.23;p<0.05) when compared to the PIL group. Treatment with VD did not affect proteinuria levels (44.28±54.17 mg/dL), decrease mesangial hypercellularity (31.48±2.2;31.3±2.2;31.4±2.2) and IgM (12.62±6.9vs15.57±3.7) deposition in the kidneys. VD supplementation did not alter IL-6, TNF-α, IL-2 and IL-17 cytokine levels, but reduce IFN-γ levels (p<0.01).

Conclusion: VD improves arthritis but does not influence renal injury despite reducing IFN-γ levels. These results support that the role of VD may be different depending on acting site, what could explain different responses according clinical phenotype. Therefore, further investigations of VD are needed to explore the supplement dosage, timing, and the molecular basis in SLE.

REFERENCES

Acknowledgement: FIFE/HCRA, CAPES, CNPq Universal Disclosure of Interests: None declared