

Abstract AB1156 – Table 1

Age/ Sex	Drug	Late-ncy (weeks)	Type of rash	Systemic involv- ment	TLC/AEC /cu.mm	ALT/AST IU/L	Score ¹	Initial Suspected Diagnosis	Followup (months)
14/F	Phenobarbitone	6	F, ED	L, K	23100/693	252/407	5	Lupus	4
33/F	Cefuroxime	2	F, ED	L	27600/ 9936	203/460	6	Acute viral hepatitis, sepsis or Lupus	11
9/M	Carbamazepine or HRZE	2	M, F, ED	L, K	19800/ 2300	2752/ 1385	7	ATT induced hepatitis (for TBM), sepsis	24
53/M	Gabapentin	7	F, ED	L, P	21400/ 3424	63/135	7	Vasculitis, Lupus	Lost FU
50/F	Alternative medicine	3	M	L, P	39630/ 2830	150/202	5	Lupus	24
15/M	SSZ	1	ED	L, G	28300/ 3100	133/92	7	AGE with Sepsis	3
21/M	HRZE	5	F, ED	L, G, K	51900/ 3114	491/478	8	Lupus or Vasculitis	8
20/F	SSZ	3	F, M, ED	L	60820/NA	137/233	6	Lupus Malignancy	3
20/F	Valproate	8	M	L, G,	13500/NA	148/177	5	Lupus	24
53/M	SSZ	6	F, M, ED,	L, K	31300/ 5634	516/436	7	Lupus or Vasculitis	Lost FU
46/M	HRZE	2	M	L, K	24000/NA	135/118	6	Sepsis, ATT induced hepatitis	1
27/F	SSZ	12	M, ED	L, K	38900/850	94/96	6	Sepsis	3
32/F	Leflunomide	4	M	L, P	10500/210	543/297	4	Drug induced transaminitis	4
38/F	SSZ/Leflunomide	20	M,	L, G	36400/ 7200	112/142	8	Sepsis	3



Abstract AB1156 – Figure 1

Conclusions: – Skin rash, arthritis, multi-organ failure of DRESS closely mimic rheumatologic disorders or sepsis (especially with rising TLC)

- As early diagnosis is imperative for successful outcome, low threshold of suspicion is necessary.

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Disclosure of Interest: None declared

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AB1157 CLINICAL IMPLICATIONS OF ULTRASONOGRAPHY (US) IN MONITORING DISEASE ACTIVITY OF RELAPSING POLYCHONDRIITIS (RP) AND COMPARATIVE INVESTIGATION BY US BETWEEN AURICLE OF RP, REPEATED TRAUMA AND HEALTHY SUBJECT

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Background: Relapsing polychondritis (RP) is a rare systemic inflammatory disorder and might often be refractory. Therefore, the discovery of more convenient imaging modality than contrast-CT, MRI and FDG-PET/CT would be required on diagnosis and treatment.

Objectives: To assess the clinical implications of ultrasonography (US) in monitoring disease activity and diagnosis of relapsing polychondritis (RP).

Methods: Firstly, auricular chondritis of patients with RP (n=5) were assessed by US before and after treatments. Second, the relationship between US findings and other serum inflammatory markers were evaluated. Moreover, the comparisons of US findings between the auricle of patients with RP (n=5), repeated trauma (n=5) which is similar to auricle of RP, and healthy subjects (n=5) were also assessed.

Results: US finding before treatment showed low-echoic swollen auricular cartilage with increased power Doppler signals (PDS) in all cases of RP. US findings corresponded to biopsy findings. After treatment with prednisolone (PSL) combined with methotrexate, the swollen ear completely resolved. Then, US findings also showed dramatic reductions in swollen cartilage with the decrease in PDS. When serum inflammatory markers completely improved, but US finding remained in 1 of 5 cases, and this case showed flare due to PSL tapering. Finally, RP could be differentiated from the damage of repeated trauma with producing subperichondrial serous effusion.

Conclusions: US of auricular cartilage in RP possibly facilitates evaluation of auricular lesions and monitoring of disease activity, especially when we consider the treatment response and the timing of drug tapering.

Disclosure of Interest: None declared

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AB1158 MALIGNANCY IN PATIENTS WITH SARCOIDOSIS: A RETROSPECTIVE COHORT STUDY FROM TURKEY

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Background: The relationship between sarcoidosis and malignancy is not clear yet. There is debate with different speculations in the literature in this regard, that this association may be just a coincidence and/or common pathogenetic link.

Objectives: The goal of our study was to evaluate the incidence and characteristics of malignancy in patients with sarcoidosis follow-up in a single centre. Methods: Our study is a retrospective analysis of patients diagnosed with sarcoidosis at the single Rheumatology centre from Turkey. Electronic patient records from the years 2010 to 2016 were screened, and 131 patients with the diagnosis of sarcoidosis were included in the study. Diagnosis of sarcoidosis was either a clinical diagnosis in patients with Löfgren's syndrome or confirmed by tissue biopsy in all other patients. The incidence of malignancies were evaluated in this cohort. Malignant diseases were diagnosed by histopathology. The clinical data of patients with sarcoidosis and malignant diseases were further analysed. Results: A total of 6 patients with malignancy were identified in our cohort of 131 patients with sarcoidosis, representing an incidence of 4.6%.

Among them, Hodgkin lymphoma (HL) were detected in three patients, followed by one patient with breast cancer, one patient with thyroid cancer and one patient with testicular cancer. All patients had chronic sarcoidosis with pulmonary involvement, and only 1 patient (with thyroid cancer) had acute sarcoidosis with Löfgren's syndrome. HL developed concomitantly with sarcoidosis in one patient while other two patients developed disease before and after sarcoidosis diagnosis. Two patients with solid tumours (breast Ca, testicular Ca) developed malignancy years before sarcoidosis diagnosis (1 year and 2 year respectively), while one patient developed thyroid cancer during sarcoidosis follow-up. All 6 sarcoidosis-malignancy patients were survived during six year follow-up.

Conclusions: We found low incidence of malignancy in patients with sarcoidosis in our small cohort. Malignancy may develop in patients with sarcoidosis. Its may occur before, after, or concurrent with the diagnosis of sarcoidosis. The sarcoidosis-malignancy relationship can only be a coincidence and/or can be explained by

a common pathogenesis. New prospective studies involving large patients series are needed in this regard.

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AB1159 VIRUS-NEGATIVE LYMPHOCYTIC MYOCARDITIS: CLINICAL AND DIAGNOSTIC FEATURES FROM A MONOCENTRIC ITALIAN COHORT

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Background: Virus-negative lymphocytic myocarditis (VNLM) is defined by endomyocardial biopsy (EMB) established histological, immunological and immune-histochemical criteria; it may occur as a distinct disease or in the context of systemic autoimmune or inflammatory disorders.

Objectives: To describe the demographic, clinical, histological and immune-histochemical features of VNLM from a monocentric Italian cohort.

Methods: 42 patients (mean age 45.57±14.9 years; male to female ratio 1:1) were diagnosed with EMB-proven VNLM at our Centre from January 2015 to December 2017. In all patients, comprehensive demographic, clinical and histological data were collected.

Results: The most common initial clinical feature was chest pain (40.5%), followed by palpitations (26.2%) and syncope (23.8%). Aborted sudden cardiac death (SCD) was the first manifestation in 3 cases, while arrhythmias were overall present in 47.6% of patients, being life-threatening in 10 of them. Interestingly, 4 patients had only few constitutional symptoms and 2 patients were completely asymptomatic. The distribution of traditional cardiovascular risk factor reflected that of the general population, apart for a more common familiarity for SCD (31.7%) and for autoimmunity (31.7%). Serum levels of troponin T and NT-proBNP were increased in 40.5% and 30.9%, respectively. Both echocardiography and standard ECG were unremarkable in half of the patients, while nearly all patients (92.5%) had at least one Lake-Louise criterion at cardiac magnetic resonance (CMR) evaluation. The most common CMR finding was delayed enhancement in 90% of cases, while T2-oedema was found in 21 patients (50%). Left ventricular ejection fraction was reduced in 50% of patients; a concomitant peri-cardial effusion was detected in 22.5% of cases. Abnormalities on 24h-ECG-Holter tape were overall detectable in 20 patients (47.6%), with ventricular ectopic beats and non-sustained ventricular tachycardia being the most common findings.

Despite positivity for ANA in 42.8% patients, only 4 patients could be diagnosed with a systemic autoimmune disease. Anti-heart antibodies (AHA) and anti-intercalated disks antibodies (AIDA) were positive in 21 patients (50%) and 12 (28.6%) patients, respectively.

On EMB, myocarditis was classified as active in 23 cases (54.8%) and as chronic in 18 (42.3%), while 7 patients (16.7%) had evidence of both features. CD3 +T-lymphocytes>7/mm² were detectable in 27 patients (64.3%), necrosis in 20 patients (47.6%), oedema in 28 patients (66.7%), while only 4 patients showed signs of vasculitis or thrombotic microangiopathy. At time of diagnosis, myocardial fibrosis was evident in 73.8% of EMBs and dilated cardiomyopathy in 6 patients (14.3%). All patients were treated with steroids and azathioprine as first line therapy, and 17 patients (40.5%) were initially referred for device implantation.

Conclusions: VNLM is an overlooked disease characterised by a broad spectrum of clinical features and peculiar immune-mediated hallmarks. The early recognition of myocarditis, allowing a prompt therapeutic intervention, should be a major goal for rheumatologists.

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AB1160 A SYNDROME OF RECURRENT IDIOPATHIC HYDROPS FETALIS, RESPONDING TO ANTI-PLATELETS/ANTI-COAGULANT PROPHYLAXIS. IS IT A NEW ENTITY OR A PART OF MATERNAL HYPERCOAGULATION STATE; THROMBOPHILIA OR ANTI-PHOSPHOLIPID SYNDROME (APS)?

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Background: Anti-phospholipid (aPL) syndrome (APS) is defined in the presence of anticardiolipin (aCL), anti-b2 glycoprotein-I antibodies, or lupus anticoagulant (LAC) with hypercoagulability, including arterial/venous thrombosis episode (s), or pregnancy morbidity; early spontaneous abortions, stillbirth or prematurity with (pre) eclampsia. Beside these classification criteria the increased awareness for seronegative APS and non-criteria clinical manifestations, necessitate additional laboratory diagnostics for aPL's. Use of preventive treatment protocol; low-dose (100 mg/d) aspirin (ASA) and/or daily subcutaneous (SC) heparin, is highly effective and improves fetal vitality and pregnancy outcome. Beside APS, stillbirth and fetal loss may result from thrombophilia and Hydrops fetalis (HF). HF is described as fetal pathological fluid accumulation in serous cavities and soft tissues. It is accompanied with placental thickening and hydramnios. Most of the cases refer to non-immune (NIHF), not caused by red cell alloimmunization. Almost third of NIHF are idiopathic (iNIHF). The pathogenesis of tissue hypoxia with capillary leakage remains unclear. With advanced in-utero therapy mortality is still high (50%–95%).

Objectives: To describe the entity of recurrent idiopathic NIHF, resulting in habitual miscarriages. To document the use of APS prophylactic regimen for fetal loss, and its effect on iNIHF occurrence.

Methods: Data from medical files of women with previous iNIHF, who were treated in the rheumatology clinic, Hadassah Mount Scopus Hospital in Jerusalem, between years 2002–2017 were summarised (table 1).

Results: The present series illustrates the impact of the prophylactic regimen of APS in preventing obstetrical morbidities, including miscarriages and fetal death due to Hydrops fetalis (table 1). Thrombophilia and aPL profiles were normal. Five women who had multiple early abortions and 8 pregnancies with iNIHF, following treatment had a total of 12 successful pregnancies with uneventful delivery to healthy babies.

ANA:anti nuclear antibodies, IUFD:intra uterine fetal death, IVF:in vitro fertilisation, CLX:Clexan=Enoxaparin (SC 40 mg/d), HCQ:Hydroxy-Chloroquine (200 mgx 2/d), PRD:Prednisone (10–20 mg/d)

Abstract AB1160 – table 1. summary of cases

patient	Age (yrs)	Pregnancies before prophylaxis	Outcome (mother age, outcome, gestation age wks)	Treatment (Prophylaxis during pregnancy)	Pregnancies on prophylaxis (maternal age)	Outcome	Positive findings
1	38	10(x7 IVF)	x1(21, preterm, 30), x2(22, 23, iNIHF/IUFD, 31), x7 abortions,<10)	CLX, ASA, HCQ	x3 ^(25, 27, 29) Spontaneous (no IVF), on HCQ, PRD 5 mg/d	Uneventful	ANA+, aCL+, LAC+, Raynaud's, Livedo Reticularis
2	43	2	x2(20, 21 IUFD/iNIHF, 27,23)	ASA, PRD	x3 ^(23, 26, 29)	1-mild HF, live birth 34 wks. 2+3 uneventful	Migraine, low platelets
3	31	2	x1(22, abortion, 8). x1(23 IUFD/iNIHF, 24)	ASA, CLX	x2 ^(25, 27)	Uneventful	None
4	27	3	x1(20, IUFD/iNIHF, 25), x1(21, abortion,<10), x1(22 IUFD/iNIHF, 26)	ASA, CLX, PRD	x2 ^(23, 25)	Uneventful	Raynaud's, Alopecia
5	48	4	x1(25 normal, term), x2(26, 27, abortions,<10), x1(29, IUFD/iNIHF, 30)	CLX 60 mg/d	x2 ^(30, 33)	Uneventful	None