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

REFERENCES:

- [1] Chen ES, Moller DR. Etiology of sarcoidosis. *Clin Chest Med*. 2008; 29:365–377.
- [2] Iannuzzi MC, Fontana JR. Sarcoidosis: clinical presentation, immunopathogenesis, and therapeutics. *J Am Med Assoc*. 2011;305:391–9.
- [3] Baughman RP, Teirstein AS, Judson MA, Rossman MD, Yeager H Jr, Bresnitz EA, et al. Clinical characteristics of patients in a case control study of sarcoidosis. *Am J Respir Crit Care Med*. 2001;164:1885–9.
- [4] Reich JM, Mullooly JP, Johnson RE. Linkage analysis of malignancy-associated sarcoidosis. *Chest* 1995;107(3):605–13.
- [5] Mantovani A, Allavena P, Sica A, Balkwill F. Cancer-related inflammation. *Nature* 2008; 454:436–444.

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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 pericardial 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-β2 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