

compared to a healthy control group (HC, n=11). Samples were analysed with Electrochemiluminescence, Mesoscale®.

**Results:** The level of INF  $\gamma$  was significantly higher in NS group compared to non-NS group in CSF (median 11.3 pg/mL vs. 3.3 pg/mL) and in plasma (median 21.6 pg/mL vs. 4.5 pg/mL).

In CSF and not in plasma, the level differs significantly in NS group compared to non-NS group, IL12/IL23p40 (median 54.05 pg/mL vs. 3.61 pg/mL), IL16 (median 8.7 pg/mL vs. 4.3 pg/mL), IL17A (median 1.37 pg/mL vs. 0.19 pg/mL), TNF  $\beta$  (median 0.37 pg/mL vs. 0.02 pg/mL), IL8 (median 55.4 pg/mL vs. 37.8 pg/mL), CCL11 (median 36.1 pg/mL vs. 16.4 pg/mL), CCL26 (median 6.7 pg/mL vs. 3.7 pg/mL), CXCL10 (median 4981 pg/mL vs. 771 pg/mL), CCL13 (median 24.5 pg/mL vs. 10.0 pg/mL), CCL22 (median 129.8 pg/mL vs. 22.6 pg/mL), CCL3 (median 54.1 pg/mL vs. 20.7 pg/mL), CCL17 (median 54.0 pg/mL vs. 6.9 pg/mL), ICAM1 (median 13901 pg/mL vs. 7327 pg/mL), and VCAM1 (median 18594 pg/mL vs. 12132 pg/mL).

A cut-off level for each cytokine was set at 20% above the maximum values of both non-NS group and HC. Using this, the ratio of patients in NS group over were: -INF  $\gamma$ , 57% had level over 6.2 pg/mL in CSF, and 50% had level over 21.6 pg/mL in plasma.

-IL12/IL23p40, 71% had level over 16.7 pg/mL in CFS.

-CXCL10, 79% had level over 1614 pg/mL in CFS.

-CCL22, 79% had level over 49.8 pg/mL in CFS.

**Conclusion:** In NS patients, INF  $\gamma$  was elevated in both CSF and plasma, and multiple cytokines, chemokines and vascular biomarkers were elevated in CSF.

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SAT0512

#### OCULAR INVOLVEMENT AND TREATMENT IN SARCOIDOSIS. STUDY OF 41 PATIENTS OF A SERIES OF 383 PATIENTS FROM A SINGLE UNIVERSITY HOSPITAL

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**Background:** The eye is a common and potential severe complication of sarcoidosis. Topical and systemic corticosteroids are the first-line treatment. Conventional and biological immunosuppressants (IS) are frequently needed (1-5).

**Objectives:** To assess the frequency, clinical and treatment of ocular involvement of sarcoidosis.

**Methods:** Study of a large cohort (n=383) of systemic sarcoidosis from a single university hospital. All consecutive patients diagnosed with sarcoidosis from January 1, 1999 to January 1, 2019 according the ATS/ERS/WASOG criteria (*Eur Respir J* 1999;14:735-737) were included.

**Results:** 41 (22 women/19 men) of 383 (10.7%) patients had ocular involvement, mean age 44.8±16 years. Uveitis (n=34; 82.9%) was the most common ocular manifestation, especially anterior uveitis (n=18; 52.9%). Ocular surface and eye orbit may also be affected (Table). In addition to topical and systemic

corticosteroids, conventional (n=23; 56.1%) and biologic (n=14; 34.1%) IS drugs were required. Adalimumab and Infliximab were the most used biologic treatments (Table). Cystoid macular edema (CME) and Retinal Vasculitis was observed in both cases in 3 (7.3%) patients, 2 of them (66.7%) required biological treatment. Papillitis appeared in 7 (17.1%) cases, biological treatment was needed in 3 (42.9%) patients. The most frequent sequels were cataract (n=9, 21.9%), intraocular hypertension (n=5; 12.2%) and pupil alterations (n=4; 9.7%). The average of the best corrected visual acuity was 0.6±0.3 at diagnosis and 0.7±0.3 after one year follow up.

**Conclusion:** Ocular involvement of sarcoidosis is a relative frequent and potential severe complication, especially if panuveitis is presented.

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SAT0513

#### ASSOCIATION OF SERUM OMENTIN LEVELS WITH COLCHICINE RESISTANCE IN FAMILIAL MEDITERRANEAN FEVER

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**Background:** Omentin is an anti-inflammatory adipokine, which plays important roles in the adjustments of glucose metabolism, cardiovascular homeostasis, atherosclerosis (1).

**Objectives:** To investigate the omentin levels in Familial Mediterranean fever (FMF) patients and to assess the association with markers of subclinical inflammation in FMF patients such as serum amyloid A (SAA), erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP).

**Methods:** This cross-sectional study included 54 consecutive adult FMF patients (27 male, 27 female) and 28 healthy individuals (16 male, 12 female). The demographic and clinical features and MEFV gene mutations were recorded. The FMF patients were separated into 3 groups: 1) attack-free group, 2) active attack group and 3) colchicine-resistant group. Serum omentin levels were compared between the FMF patients and the healthy control group.

**Results:** Serum omentin and SAA levels were higher in the study group than in the control group (108.05(19.97-343.22) vs. 199.5(42.98-339.41) p<0.05, 3.69(1.18-22.75) vs. 1.31(0.95-3.16) p<0.001) (Table 1). When the FMF patients were examined as separate groups, serum omentin values were lower in the colchicine resistant group than in the groups without resistance (Table 2). The correlation analysis showed a negative correlation between omentin and SAA levels (r = -0.240, p = 0.030).

Table. Ocular manifestations of sarcoidosis and treatment with corticosteroids, conventional and biological IS.

OCULAR INVOLVEMENT	CONVENTIONAL IS								BIOLOGICAL IS							
	Cases	TCS	OCS	MD of OCS	IVMP	CIS	MTX	AZA	CFM	MMF	BT	ADA	IFX	TCZ	GLM	ETN
SURFACE n(%)	3(7.3)	2(66.7)	2(66.7)		2(66.7)	2(66.7)	2(66.7)	2(66.7)	1(33.3)	0(0)	2(66.7)	2(66.7)	2(66.7)	0(0)	0(0)	0(0)
-CG/N, n(%)	1(33.3)	1(100)	0(0)		0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
-PUK, n(%)	2(66.7)	1(50)	2(100)	60	2(100)	2(100)	2(100)	2(100)	1(50)	0(0)	2(100)	2(100)	2(100)	0(0)	0(0)	0(0)
UVEITIS n(%)	34(82.9)	25(73.5)	28(82.3)		10(29.4)	19(55.6)	18(52.9)	7(20.6)	1(2.9)	1(2.9)	12(35.3)	11(32.3)	4(11.8)	3(8.8)	2(5.9)	1(2.9)
-Anterior uveitis, n(%)	18(52.9)	11(61.1)	13(72.2)	30	1(5.5)	5(27.8)	5(27.8)	1(5.5)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
-Posterior uveitis, n(%)	4(11.7)	2(50)	3(75)	60	1(25)	3(75)	2(50)	2(50)	0(0)	0(0)	2(50)	1(25)	0(0)	0(0)	1(25)	0(0)
-Panuveitis, n(%)	12(35.3)	12(100)	12(100)	60	8(66.7)	11(91.7)	11(91.7)	4(33.3)	1(8.3)	1(8.3)	10(83.3)	10(83.3)	4(33.3)	3(25)	1(8.3)	1(8.3)
EYE ORBIT n(%)	4(9.7)	2(50)	3(75)		2(50)	2(50)	2(50)	2(50)	0(0)	0(0)	2(50)	1(25)	1(25)	1(25)	0(0)	0(0)
-Proptosis, n(%)	2(50)	1(50)	1(50)	30	1(50)	1(50)	1(50)	1(50)	0(0)	0(0)	1(50)	1(50)	0(0)	1(50)	0(0)	0(0)
-Strabismus, n(%)	2(50)	1(50)	2(100)	60	1(50)	1(50)	1(50)	1(50)	0(0)	0(0)	1(33.3)	0(0)	1(33.3)	0(0)	0(0)	0(0)
TOTAL, n(%)	41(100)	29(70.7)	33(80.5)	50±15.5	14(34.1)	23(56.1)	22(53.7)	11(26.9)	2(4.9)	1(2.4)	14(34.1)	14(34.1)	7(17.5)	3(7.3)	2(4.9)	1(2.4)

TCS: topical corticosteroids; OCS: oral corticosteroids; MD: maximum dose; IVMP: intravenous methylprednisolone; CIS: conventional immunosuppressors; BT: biologic therapy; CG/N: conjunctival granuloma/nodule; PUK: peripheral ulcerative keratitis

**Table 1. Laboratory results of the FMF and the control group**

Variables	FMF patients (n=54)	Control (n=28)	P value
Omentin, pg/mL	108.05(19.97-343.22)	199.5(42.98-339.41)	0.03
SAA, ng/mL	3.7 (1.18-22.75)	1.31(0.95-3.16)	<0.001
ESR, mm/h	15(2-68)	12(7-17)	<0.001
CRP, mg/L	12(1-194)	2.5(1-8)	<0.001

Variables were given as median (IQR).

Calculated using Mann-Whitney U test for non-normal distribution.

FMF=Familial Mediterranean fever, SAA=serum amyloid A, ESR=erythrocyte sedimentation rate, CRP=C-reactive protein, IQR=interquartile range.

**Table 2. Laboratory results of FMF patients with and without colchicine resistance**

Variables	With resistance (n=16)	Without resistance (n=38)	P value
Omentin, pg/mL	76.64(19.77-224.33)	186.47(28.41-343.21)	0.006
SAA, ng/mL	3.69(1.18-22.75)	3.77(1.18-21.49)	0.784
ESR, mm/h	25.5(2-68)	15(2-60)	0.835
CRP, mg/L	11(1-67)	19(1-194)	0.111

Variables were given as median (IQR).

Calculated using Mann-Whitney U test for non-normal distribution.

FMF=Familial Mediterranean fever, SAA=serum amyloid A, ESR=erythrocyte sedimentation rate, CRP=C-reactive protein, IQR=interquartile range.FOM

**Conclusion:** FMF patients with colchicine resistance are associated with decreased omentin concentrations, probably mediated by inflammation-driven mechanisms.

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#### SAT0514 FATIGUE IN FAMILIAL MEDITERRANEAN FEVER

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**Background:** Fatigue is an important and common symptom in rheumatologic diseases. It causes disability and worsens patients quality of life. Familial Mediterranean Fever (FMF) is a hereditary autoinflammatory disease characterized by recurrent episodes of fever and serositis. Mutations in the MEFV gene that encodes pyrin protein are responsible for the disease. Most frequent mutation is M694V and FMF patients with M694V/M694V genotype have more severe disease.

**Objectives:** The aim of this study is to investigate fatigue and its impact on quality of life of FMF patients who are attack-free for more than one year.

**Methods:** Seventy-seven FMF patients and 70 age and sex matched healthy controls were enrolled in the study. Fatigue severity scale (FSS) was used to evaluate fatigue level. Disease severity was evaluated via FMF disease severity score. Short form-36 (SF-36) was used to evaluate the quality of life of the participants and Beck depression inventory (BDI) was used to evaluate depression.

**Results:** No statistically significant difference was found when FMF group and healthy controls were compared for demographic variables and laboratory markers (complete blood count, erythrocyte sedimentation rate, c-reactive protein levels, liver and kidney function tests). All of the subscale scores of SF-36 were lower in FMF group when compared with the control group. BDI and FSS scores were higher in the FMF group.(Table 1)

**Table 2. Correlation of fatigue, depression, disease severity and duration with quality of life in FMF patients**

	Physical functioning	Social functioning	Role Physical	Role emotional	Mental health	Vitality	Pain	General health
FMF disease severity scale	r= -0,223	r= -0,319**	r= -0,313**	r= -0,184	r= -0,124	r= -0,188	r= -0,191	r= -0,351**
Beck Depression Inventory	r= -0,418**	r= -0,554**	r= -0,461**	r= -0,470**	r= -0,660**	r= -0,594**	r= -0,308**	r= -0,576**
Fatigue severity scale	r= -0,391**	r= -0,426**	r= -0,475**	r= -0,324**	r= -0,650**	r= -0,739**	r= -0,410**	r= -0,521**
Disease duration	r= -0,40**	r= -0,218	r= -0,232*	r= -0,287*	r= -0,026	r= -0,211	r= -0,189	r= -0,109

\*\* p<0.01

\*P<0.05

**Table 1. Comparison of FMF and control groups for fatigue, depression and quality of life**

	FMF group (n: 77) median(min-max)	Control group (n: 70) Median (min-max)	p
Fatigue severity scale (FSS)	31(27-47)	28 (24-37)	p<0.05
Beck depression inventory (BDI)	15 (0-35)	12 (0-19)	p<0.05
SF-36 Physical functioning	80.00(25.00-100.00)	95.00 (50.00-100.00)	p<0.01
SF-36 Social functioning	55.50 (40.00-88.80)	77.70 (22.20-88.80)	p<0.01
SF-36 Role physical	25.00 (0.00-100.00)	100.00 (0.00-100.00)	p<0.01
SF-36 Role emotional	33.30 (0.00-100.00)	100.00 (0.00-100.00)	p<0.01
SF-36 Mental health	56.00 (24.00-88.00)	72.00 (24.00-100.00)	p<0.01
SF-36 Pain	55.50 (0.00-100.00)	77.70 (33.30-100.00)	p<0.01
SF-36 Vitality	45.00 (15.00-95.00)	65.00 (5.00-100.00)	p<0.01
Sf-36 General health	40.00 (5.00-92.00)	67.00 (20.00-100.00)	p<0.01

Twenty-three of FMF patients had M694V/M694V genotype and those patients had higher disease severity scores (p<0.01), higher FSS (p<0.01) and higher BDI scores (p<0.05) when compared with other FMF patients. Regarding the quality of life, patients with M694V/M694V genotype had lower scores in social functioning, role physical, role emotional and pain subscales of SF-36. In the correlation analysis depression and fatigue were found to be the major determinants of quality of life in FMF. Disease severity or duration were not strongly correlated with the SF-36 scores (Table 2).

**Conclusion:** Despite being attack-free for more than one year, FMF patients had poor quality of life and fatigue when compared with the healthy controls. The quality of life in FMF patients, whose attacks are well controlled, is mainly determined by fatigue and depression rather than disease severity.

#### References:

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#### SAT0515 COMPLEX HYPERMOBILITY EHLERS-DANLOS SYNDROME (HEDS): MAPPING THE PATIENT'S JOURNEY OVER 40 MONTHS IN A TERTIARY REFERRAL CENTRE

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**Background:** Ehlers-Danlos Syndromes are heritable connective tissue disorders. They are multisystemic and patients can present with several symptoms such as joint pain and instability, visceral and autonomic dysfunction, as well as significant psychosocial sequelae. Managing this cohort of young patients is usually challenging as many patients present late due to delayed diagnosis, often with several complications, problems with mobility and opioid use. Furthermore, there is often a prolonged lack of coordinated healthcare and access to social care services. A recent parliamentary debate in the U.K. highlighted that hEDS services are excluded from specialist Rheumatology commissioning services. In order to ascertain the relevance and utility of specialist services in this population, we conducted this study.

**Objectives:** The objective of this study was to map the patient experience following a referral to the specialist clinic in order to assess the need for an integrated, multidisciplinary approach to treating patients with hypermobility EDS.

**Methods:** We retrospectively reviewed the records of 50 patients with the diagnosis of hypermobility EDS who were seen in a specialist hypermobility clinic at University College Hospital UCLH between January 2016 and March 2016. Relevant data was collected regarding their medical care in our hospital up to October 2019.

**Results:** The median age was 37 (range 21-59). We had 10 males and 40 females. The diagnosis of hypermobility EDS was based on the 1997 criteria as these patients were seen prior to the 2017 classification. Overall, the study yielded 6 key themes: 1. All patients experienced chronic pain, with 36% reporting use of opioids for pain management. 2. Patients were referred to multiple