NA02. PREGNANCY OUTCOME IN LUPUS PATIENTS WITH PREVIOUS ADVERSE OUTCOMES: EXPERIENCE WITH PROTOCOL-BASED, MULTIDISCIPLINARY CARE FROM KERALA, SOUTH INDIA

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Background: In patients with SLE with previous adverse obstetric outcomes the risk of an adverse outcome in subsequent pregnancy increases. In this prospective cohort of such patients the primary objective was to assess the impact of a protocol based care.

Methods: Between March and September 2010 all patients with SLE fulfilling the ACR classification criteria with previous at least one adverse obstetric outcome (maternal; preterm labour, pre eclampsia or previous medical termination of pregnancy (MTP) in view of SLE flare, foetal; miscarriage, IUGR, preterm birth, low birth weight (LBW), intrauterine death or still birth) desiring of having more children were prospectively enrolled. Briefly the protocol comprised of pre natal counselling, pre-natal drug and disease status review, risk stratification, periodic ante-natal visits for the monitoring of pregnancy (including with obstetric ultrasounds) and disease, 2D echocardiogram at weeks 18 and 32 if Ro/La were positive and post natal disease and drug review and contraception advice. Therapeutic changes were made as necessary at each stage.

Results: Fourteen patients (age mean ±SD years; 29 ±3) were enrolled. Previous poor obstetric outcomes were: miscarriage(s) in 6, MTP in 3, preterm labour with IUGR in 2, intrauterine death in 1, four patients had secondary APS and it had both or either Ro/La positive. Three had lupus nephritis (LN); >6 months ago in 2. There were 10 (71%) live births (2 LBW, instrumentation or caesarean section in 3). Three patients had miscarriages (one had ongoing LN). One decided against becoming pregnant after initial counselling. Five patients (35%) had lupus flare (2 mild, 2 moderate and 1 severe based on SLEDAI).

Conclusions: Majority of patients in our cohort had acceptable pregnancy outcome. This highlights that for high risk lupus pregnancy a multidisciplinary input with protocol based care offers a good chance of improved pregnancy outcome.

Disclosure statement: The authors have declared no conflicts of interest.

ES01–ES25

ABSTRACTS FROM EIR SCHOOL

ES01. THE EFFECTIVENESS OF MONOCONAL ANTIBODIES TO TUMOR NECROSIS FACTOR ALPHA: INFILXIMAB IN JUVENILE ANKYLOSING SPONDYLITIS

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Background: Juvenile ankylosing spondylitis (JAS) is a type of arthritis that affects the spine and the sites where the muscles, tendons, and ligaments are attached to bone, with frequent onset before 15 years of age preferably in boys. Currently, besides standard schemes of immunosuppressive therapy, administration of biological agents, influencing on various components of pathogenesis is most perspective. One of them consists in administration of monoclonal anti-TNF-α antibodies, consisting in variable area of murine highly affine, neutralizing monoclonal antibodies to TNF-α (2), combined to fragment of human IgG – infliximab.

Methods: 58 patients with juvenile ankylosing spondylitis were enrolled in the study, from them there were 17 girls and 41 boys. Median age of the patients was 12 ±3,1 years, median duration of disease – 4 ±3,6 years.

Average dose of infliximab comprised to 7,5 mg/kg/administration. Administration of Infliximab was conducted in standard scheme 0, 2, 6 weeks, thereafter every 8 weeks.

Results: Before treatment of infliximab overall assessment score in doctor’s opinion (VAS) comprised to 60(53,72), overall health assessment in patient’s opinion or his parents (VAS)-72(58,82), number of “active” joints-3,93±6, number of joints with function limitation-4(3,6), assessment of functional ability by using questionnaire Childhood Health Assessment Questionnaire (CHAQ)-2(1,62,4), ESR-40(25,58) mm/h, C-PR-3 (1,26)mg%, ACR28 (54) weeks of the drug 89% patients achieved 90%, 100% patients – 70% improvement in criteria ACR-pedi.

Conclusion: infliximab is a highly effective drug in patients with juvenile ankylosing spondylitis.

ES02. RELATIONSHIP BETWEEN FATIGUE AND IL 6, DISEASE ACTIVITY, DEPRESSION IN PATIENTS TREATED WITH TOCELLIZUMAB

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Background: Fatigue is a symptom described as chronic prostration not relieved by appropriate sleep that causes incapacity to participate in social and professional activities as well as in rehabilitation programs. Fatigue is an important symptom in rheumatoid arthritis patients and contributes to loss of quality of life.

There are several theories to explain the appearance of fatigue in RA patients. One of them is that the fatigue is due to high levels of pro-inflammatory cytokines such as IL-6.

Methods: We studied 35 RA patients receiving intravenous infusions of TCZ (8 mg/kg each) at a 4-week intervals in combination steady-state therapy with disease-modifying anti-rheumatic drugs and glucocorticoids. We assessed C-reactive protein, erythrocyte sedimentation rate, serum interleukin (IL) 6 concentration, fatigue (assessed by VAS), depression level by EQ SD scale, VAS for general health, tender joint count (TJC), swollen joint count (SJC), hemoglobin level.

Results: Basic level of fatigue was 5.3 (4.0; 7.0), IL 171.3 (83,98;462,29), DAS-28 6,5(5,97;17,6), pain 66,0 (51,0;74,0), TJC 13(8,7), SJC 12 (8,18). Significant improve in IL6 level, fatigue and DAS28 was on the 4 week of therapy. The improvement continued throughout the 6 months.

In 6 month improve in fatigue was 52%, in DAS28 68%, in IL6 level 80,4%. Fatigue value did not correlated with IL6 level (r = 0,039, p = 0,82), hemoglobin level (r = –0,23, p = 0,05), pain (r = 0,30, p = 0,053), SJC/r = 0,19, p < 0,05). But it was associated with DAS28 (r = 0,36, p = 0,047), depression (r = 0,45, p = 0,02) and VAS for general health (r = 0,52, p < 0,05).

Conclusion: High fatigue levels characterize activity of RA. The decrease in fatigue and depression in patients treated with TCZ is a result of reduction of disease activity, not pro-inflammatory cytokines.

Disclosure statement: The presenting author has declared no conflicts of interest.

ES03. NK CELL DEPLETION AND ITS RELATION TO ELEVATED REDOX POTENTIAL INRHEUMATOID ARTHRITIS

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Background: Reactive oxygen intermediates (ROIs) attack on the mitochondrial Electron transport chain and oxidative phosphorylation and depletes the ATP levels. The cells life cycle is dependent on the level of ATPs produced during mitochondrial processes. The number of natural killer cells in the autoimmune disorders is one of the major prognostic factors. This work is comprised of the correlation between elevated ROIs with the NK cell numbers in Rheumatoid arthritis (RA) patients.

Methods: Peripheral blood of 15 patients of RA and 15 age and sex matched healthy volunteer with ethically approved inclusion and exclusion criteria were collected. The enzymatic activities evaluated were: Superoxide ROIs with the NK cell numbers in Rheumatoid arthritis (RA) patients.

Results: Basic level of fatigue was 5.3 (4.0; 7.0), IL 171.3 (83,98;462,29), DAS-28 6,5(5,97;17,6), pain 66,0 (51,0;74,0), TJC 13(8,7), SJC 12 (8,18). Significant improve in IL6 level, fatigue and DAS28 was on the 4 week of therapy. The improvement continued throughout the 6 months.

In 6 month improve in fatigue was 52%, in DAS28 68%, in IL6 level 80,4%. Fatigue value did not correlated with IL6 level (r = 0,039, p = 0,82), hemoglobin level (r = –0,23, p = 0,05), pain (r = 0,30, p = 0,053), SJC/r = 0,19, p < 0,05). But it was associated with DAS28 (r = 0,36, p = 0,047), depression (r = 0,45, p = 0,02) and VAS for general health (r = 0,52, p < 0,05).

Conclusion: High fatigue levels characterize activity of RA. The decrease in fatigue and depression in patients treated with TCZ is a result of reduction of disease activity, not pro-inflammatory cytokines.

Disclosure statement: The presenting author has declared no conflicts of interest.

ES03. NK CELL DEPLETION AND ITS RELATION TO ELEVATED REDOX POTENTIAL INRHEUMATOID ARTHRITIS
Conclusions: The results explain the effect of ROS on the prognosis of RA. The levels of increased ROS cause lipid peroxidation and decreased GSH levels. The reduced levels of anti-oxidative enzymes are possibly able to keep a check on the concentration of ROS levels. This increased oxidative stress condition is suggestive of diminished NK cell populations which may be due to uncoupled mitochondrial electron transport complexes affecting the oxidative phosphorylation and hence ATP production. The molecular mechanisms underlying this phenomenon need further elucidation.

Sponsorship: The present study was supported by the CSIR, India.

Disclosure Statement: The authors have declared no conflict of interest.

ES04. AVASCULAR NECROSIS IN HIV INFECTED PATIENT

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Introduction: Avascular necrosis is a growing problem among HIV infected patients. The first report was in 1990 in a HIV infected homosexual male. We present a HIV infected patient with bilateral hip AVN and review the literature.

Presentation: 27 years old nalliparous African female presented in May 2009 with bilateral hip and knee pains for eight months. She had to use crutches 4 months later due to hip pain. She was a teetotaller with no history of prior trauma or any past illnesses. Analgesia was not helpful. Family history was non contributory. Physical examination revealed oral thrush, bilateral reduction in internal and external rotation at the hips. Pelvic Xrays showed features of avascular necrosis of the hips, which were confirmed on MRI. There were no abnormalities elsewhere on bone scan. Fasting lipogram was within normal limits. Retroviral screen was reactive. She had a negative antiphospholipid antibody, weakly positive lupus anticoagulant (1.41) and a negative ANF. Protein S was low at 34.2. She was referred to orthopaedic surgeons for hip replacement surgery.

Discussion: The incidence of AVN in HIV has been found to be as high as 4.4% in prospective MRI studies. There is a high incidence among men of 8:1, with a mean age at presentation around 34 years. Possible risk factors include antiphospholipid antibodies, procoagulant states, increased risk of vasaclitis, use of protease inhibitors, direct effects of HIV, low CD4 count. Postulated mechanisms include altered intravascular blood flow, direct cellular toxicity and impaired mesenchymal cellular differentiation. Small studies and case reports suggest increased risk of rapid progression and collapse.

Conclusion: AVN is becoming another important emerging adverse musculoskeletal manifestation in HIV. Further studies are needed to better elucidate risk factors, pathogenetic mechanisms and treatment options available.

Disclosure statement: The author has no conflict of interest.

ES05. EARLY PREDICTORS OF INCREASED BONE RESORPTION IN JUVENILE IDIOPATHIC ARTHRITIS

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Background: In all subgroups of juvenile idiopathic arthritis (JIA), a decrease in bone mass has been described in a high percentage with osteoporosis. The decrease in bone mass is multi-factorial in origin and influenced by inflammation, medication, nutrition, and physical inactivity. Exploring early changes in the predictors of bone turnover and its relation to the parameters of joint inflammation and joint destruction in all subtypes of JIA hold the key to identification of patients at risk and to management of osteoporosis.

Methods: Seventy children with JIA and thirty age, sex, ethnicity, and geography matched healthy controls were included in this study. Both groups had the average dietary intake of calcium products and sun exposure. Disease activity was determined by clinical and laboratory evaluation, Juvenile arthritis disease activity score in 27 joints (JADAS27). All involved joints were assessed by plain radiography. Serum levels of calcium (Ca), phosphorus (Ph), alkaline phosphatase (ALP), osteocalcin (OC), RANKL and (OPG) were measured. Urinary concentration of deoxy-pyridinoline (DPD) was also done.

Results: Significant low serum concentrations of ALP and OPG was observed in all JIA subgroup, while there was a significant increase in serum level of RANKL and urine level of DPD. OPG/RANKL ratio was significantly lower in JIA patients than in controls. OPG/RANKL ratio is correlated with most clinical characteristics, disease activity variables, JIA outcome measures and radiographic findings. DPD, RANKL and OPG/RANKL ratio respectively are considered as independent predictors of joint-articular osteoporosis. OPG/RANKL ratio was the only predictor of bone erosions.

Conclusions: The OPG/RANKL ratio could be an early predictor of increased bone resorption and a valuable biomarker for joint inflammation and bone injury in JIA patients.

Disclosure statement: The authors have declared no conflicts of interest.

ES06. EVALUATION AND MANAGEMENT OF A PATIENT PRESENTING WITH POLYARTHRITIS IN A RESOURCE LIMITED SETTING

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Background: Musculoskeletal complaints are common in paediatrics practice. Causes of these include traumatic, inflammatory and idopathic conditions. It is essential that when children present with such complaints, they are evaluated appropriately and only relevant investigations are requested. It is equally important that basic investigations be available when needed to aid in the prompt evaluation and management of such patients.

Methods: This is a case report on the evaluation of a patient who presented with recurrent joint pains.

Results: An eleven year old boy presented with one week history of pain in the wrists and toes as well as swelling of the toes. There was no associated chronic cough, fever or easy fatigability and no family history of sickle cell disease or joint disease. On examination he was wasted, afibrile, moderately pale and had no lymphadenopathy. The liver and spleen were not palpable. There was positive metacarpo-phalangeal and metatarsophalangeal squeeze tests. The proximal interphalangeal joints of the toes and the first metatarsophalangeal joints were tender and swollen. Tenderness was also elicited in the wrists, and elbows. The laboratory investigation revealed negative Rheumatoid Factor, HIV and Sicking tests. The ESR was 140 mm per hour; and Total blood count results are being waited while the Hb was 5.7 g/dl. The Chest radiograph was normal and wrist radiographs showed some erosion especially 2nd MCP joints. Foot radiographs were not taken. A diagnosis of Juvenile Idiopathic Arthritis (JIA) was made and the patient started on NSAIDS (ibuprofen) while awaiting methotrexate to be imported since the drug is not currently available in South Sudan.

Conclusion: Use of simple algorithms such as the ACR/EULAR classification criteria for RA can greatly aid in the evaluation and diagnosis of rheumatologic disorders in resource constrained settings without resorting to irrelevant expensive investigations that often delay clinical management decisions.

Disclosure statement: The authors have declared no conflicts of interest.

ES07. THE TOLL-LIKE RECEPTORS PATHWAY IN RHEUMATOID ARTHRITIS: GENETIC PLOYMORPHISM STUDY INVOLVING 20 GENES

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Background: The toll-like pathway (TLRs) plays an important role in the pathogenesis of RA. However, the genetic architecture has not been fully examined in Chinese RA patients.

Methods: We carried a case-control study enrolled 300 RA patients and 360 healthy controls from north China. 108 SNPs mapped to 22 genes in TLR pathway were genotyped.

Results: Three SNPs, rs142144857 mapped to TLR4, rs5030445 and rs5030416 mapped to TRAF6 were associated with RA after Bonferroni correction.

Conclusions: The TLR4-TRAF6 interaction has its potential in RA, which need further evidence to replicate.

Disclosure of interests conflict: The authors have declared no conflicts of interest.
ES08. THE RELATIONSHIP BETWEEN C-REACTIVE PROTEIN IN SERUM AND RHEUMATOID FACTOR IN SYNOVIAL FLUID IN RHEUMATOID ARTHRITIS

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Background: Determining the relationship between C-reactive protein (CRP) in serum and rheumatoid factor (RF) in synovial fluid in rheumatoid arthritis (RA).

Material and methods: We have conducted a six-year study (6.3 ± 2.9 years) and monitoring of 80 (average age 51.2 ± 14.7 years) patients with early RA, diagnosed according to criteria ACR (American College of Rheumatology, 1987) Among them 71 women and 9 men. They measured the concentration of C-reactive protein (CRP) and titers of rheumatoid factor (RF) in serum and synovial fluid of different (I, II, III) the degree of disease activity (DAS 28).

Results: The close relationship between the level of CRP in the serum and the titer of RF in synovial fluid has been determined. According to studies, the specificity of RF in the synovial fluid in the diagnosis of RA in the early stages is 81%, the combination of RF in synovial fluid + Erythrocyte Sedimentation Rate (ESR) - 88%, CRP titers in the serum of RF + synovial fluid - 93%, RF + synovial fluid titers CRP in the blood serum + ESR - 96%, RF in synovial fluid + polyarticular pain - 92%, RF in synovial fluid + morning stiffness - 94%. At the same time of the survey, only 37% of patients corresponded to the diagnostic criteria ACR. With a high degree of activity of the process increases the level of CRP in the blood (1:192 mg/L and above) and increases the titer of RF (1:96 and above) in the synovial fluid.

Conclusion: The level of CRP in the blood serum, the rate of change of rheumatoid factor in synovial fluid of joints in patients with articular syndrome, can be used for early diagnosis of rheumatoid arthritis and to assess the severity of the disease. In this context, the definition of RF in synovial fluid can be very useful in early diagnosis of RA, when manifest only a few clinical symptoms.

ES09. DNASEx IGG ACTIVITY IN PATIENTS WITH EARLY ARTHRITIS

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Background: Patients with arthritis need to be diagnosed very early in the disease process, possibly before diagnostic criteria are fulfilled. In order to make the best diagnostic of early arthritis (EA), new biomarkers have to be identified. Abzymes (catalytic antibodies) may serve a new tool in differential diagnostic of EA.

Methods: The patients with EA (48 with early rheumatoid arthritis (ERA), 40 with acute reactive arthritis (ARA) associated with Ch. trachomatis urogenital infection) and 39 healthy persons were examined. The average symptoms duration was 4.29 ± 2.39 months (95% CL: 3.59-4.99) in patients with ERA and 3.8 ± 2.44 months (95% CL: 2.73-4.22) in patients with ARA.

The polyclonal IgG samples were purified from the sera of patients and healthy persons by combined method of affinity chromatography on protein A column. The experiments, confirming that abzyme activity is the essential quality of polyclonal IgG, were performed. The methods for DNsAse activity assessment relied upon rivanol capacity to form a clot with DNA reversely proportional to substrate depolymerisation on the action of DNsAse.

Results: The levels of DNsAse abzyme activity in patients with ERA (4.00 units (95% CI: 3.50-4.50), ARA (2.50 units (95% CI: 2.00-3.00) were (p < 0.001) higher than in controls (0.00 units (95% CI: 0.00-0.00). The levels of DNsAse activity in patients with ERA were significantly higher (p < 0.001) than in patients with ARA. We revealed that determination of elevated levels (3 and more units) of DNsAse IgG activity might be implicated for discrimination of ERA from ARA (sensitivity 86,05 (95% CI: 72,10-94,70), specificity 83,33 (95% CI: 69,80-93,89), PLR 5,16 (95% CI: 4,39-6,29), NLR 0,17 (95% CI: 0,06-0,50).

Conclusions: For the first time we confirmed the presence of elevated levels DNsAse polyclonal IgG activity in patients with EA in comparison with healthy persons and prevalence of this activity in ERA. We designed new test for differentiation of ERA and ARA on the basis of assessment of DNsAse abzyme activity.

Disclosure statement: The presenting author has declared no conflicts of interest.

ES10. A FUNCTIONAL RANKL POLYMORPHISM ASSOCIATED WITH YOUNGER AGE AT ONSET OF RHEUMATOID ARTHRITIS

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Background: We previously reported association of co-occurrence of HLA-DRB1 shared epitope (SE) and RANKL SNPs with younger age of RA onset in 182 rheumatoid factor positive (RF) European American (EA) early RA patients. Here, we fine-mapped the 48 kb RANKL region in the extended 210 EA RF-positive early RA cohort, sought replication of RANKL-associated SNPs in additional 501 EA and 298 African-Americans (AA) RA cohorts, and explored functional consequences of RA-associated SNPs.

Methods: SNP genotyping was conducted using pyrosequencing or TaqMan PCR assays.

Results: Association of rs7984870 with RANKL expression in plasma, PBMC and isolated T cells were quantified using ELISA and RT-PCR. Site-directed mutagenesis of rs7984870 within the 2 kb.

RANKL promoter was performed to drive the luciferase reporter gene in osteoblast and stromal cell lines. Interaction of DNA and protein was determined by electrophoretic mobility shift assay.

Conclusions: A single promoter SNP rs7984870 was consistent significantly associated with earlier age of RA onset in 3 independent seropositive (RF or anti-cyclic citrullinated peptide antibody positive) RA cohorts but not in seronegative RA patients. The risk allele of rs7984870 conferred a 2-fold higher plasma RANKL levels in RF-positive RA patients, significantly elevated RANKL mRNA expression in activated normal T cells, and increased promoter activity after stimulation in vitro via differential binding to a transcription factor SOX5.

Disclosure of interests: All authors declared no conflicts of interest.

ES11. ANTI-INFLAMMATORY AND ANTIARTHRITIC OF THYMOQUINONE AGAINST COLLAGEN INDUCED ARTHRITIS IN WISTAR RATS

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Background: Thymoquinone (TQ) is the major active compound derived from the medicinal plant Nigella sativa. The present study was done in order to assess its anti-inflammatory and antiarthritic effects of TQ in collagen-induced arthritis (CIA) in Wistar rats.

Methods: Wistar rats were immunized with bovine Collagen Type II emulsified with Freund’s adjuvant complete (CFA) intradermally at about 1.5 cm distal from the base of the tail. TQ was administered at a dose of 5 mg kg-1 body weight once daily and methotrexate (MTX) (1 mg kg-1 week−1) for 21 days orally. The severity of clinical arthritis
in each affected paw was graded on a subjective scale of 0–4. The effects of treatment in the rats were assessed by studying levels of cytokines i.e. TNF-α, IL-1β, IFN-γ, IL-6 and IL-10 in the joints and serum. We also determined the effect of TQ on collagen-induced prostaglandin E2.

**Results:** Oral TQ administration to collagen-immunized rats reduced the progression of arthritis by inhibiting the increase in arthritis score and paw swelling as compared to RA rats. TQ significantly inhibited the production of PGE2 and erosion of cartilage dramatically in rat knees. Oral TQ treatment suppressed the overproduction of PGE2 and erosion of cartilage dramatically in rat knees. Conclusion: protective effects of TQ against RA were evident from the decrease in arthritis scoring and it abolishes a number of factors known to be involved in RA pathogenesis renders it a clinically potential value in the treatment of RA. This study indicates that TQ can be used similar to MTX as a safe and effective therapy for CIA and may be useful in the treatment of rheumatoid arthritis.

**Disclosure statement:** The authors have declared no conflicts of interest.

**ES12. EVALUATION OF LIVER FIBROSIS WITH TRANSIENT ELASTOGRAPHY (FIBROSCAN) AND APRI SCORE IN SYSTEMIC SCLEROSIS PATIENTS**

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**Background:** Systemic sclerosis (SSc) is a complex and rare autoimmune disease characterized by vascular damage as well as skin and internal organs sclerosis. Transient elastography (Fibroscan) has been validated for the diagnosis of hepatic fibrosis. Our aim was to study the frequency of liver stiffness detected with Fibroscan in patients with SSc and the association of liver stiffness with liver disease severity measured by the AST platelet ratio index (APRI).

**Methods:** The APRI was calculated in all patients: AST/upper limit of normal x 100/platelet count (109 /l). An APRI < 0.5 has acceptable accuracy for excluding significant fibrosis. Liver stiffness scores were expressed as kilopascals (Kpa). For the diagnosis of fibrosis, a cut-off value of TE > 7.5 kpa has been proposed. Correlation was tested with Spearman’s rho.

**Results:** The study included 40 patients (M/F 6/34) with a median age of 59 years (range: 34-79), HBV, HVC, and anti-mitochondrial antibodies were all negatives, Alcohol consumption was less than 20g/day in all cases. Median liver stiffness score was 5.32 Kpa (range: 3.1-9.4). One patient had a TE > 7.5 Kpa (prevalence 5%, 95%CI 0.1-24.8). This patient had a value of 9.4 Kpa with an ultrasound pattern compatible with fatty liver and an APRI value of 0.18. The median APRI value was 0.29 (range: 0.16-0.58). The patient with the highest APRI value had a liver stiffness score of 0.21 Kpa. There was no correlation between the APRI score and liver stiffness by Fibroscan (rho = 0.11).

**Conclusions:** The prevalence of significant liver fibrosis is moderate to low in SSc patients with no clinical evidence of hepatic disease, as liver disease less prone to SSC-specific pathophysiological events. Fibroscan, a non-invasive method, detected a prevalence of liver stiffness similar to expected. Liver enzymes levels do not correlate well with liver stiffness.

**Disclosure of interests/conflict:** The authors have declared no conflicts of interest.

**ES13. CARDIAC INVOLVEMENTS IN ADULT POLYMYOSITIS OR DERMATOMYOSITIS: A SYSTEMATIC REVIEW**

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**Objective:** To investigate the clinical features of cardiac involvement in polymyositis or dermatomyositis (PM/DM).

**Methods:** All articles published in English were retrieved by searching MEDLINE via PubMed (1970 to July 2011). After selecting eligible articles according to the predefined inclusion and exclusion criteria, a systematic review was carried out.

**Results:** A total of 23 articles enrolled in this study, which included 1530 patients. The incidence of cardiac involvement was 9–72%. Heart failure was the most frequent (50–77%), followed by arrhythmia (44–72%) and pericardial effusion (24–35%). Among the abnormal ECG and UCG, the incidence of conduction abnormalities, left ventricular diastolic dysfunction and hyperkinetic left ventricular contraction were 25–38.5%, 42% and 6–12%, respectively. The pathologic findings in inflammation, degenerative changes and necrosis similar to that in skeletal muscles. Cardiac manifestations of some patients improved after glucocorticoid and immunosuppressant treatment. Thirty-eight patients (25.5%) died as a direct result of heart diseases.

**Conclusions:** Heart abnormalities are frequent in patients with PM/DM, most of which were subclinical. The efficacy of glucocorticoids and immunosuppressants is uncertain. Cardiac involvement is a common cause of death.

**Disclosure statement:** The authors have declared no conflicts of interest.

**ES14. ROLE OF REACTIVE OXYGEN SPECIES AND CASPASE-3 IN CYTOTOXIC T LYMPHOCYTE MEDIATED CELL DEATH IN SYSTEMIC LUPUS ERYTHEMATOSUS**

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**Background:** Cytotoxic T lymphocyte (CTL)-mediated cell death through granzyme B has recently been proposed to be a preferential and selective source of auto-antigens in systemic lupus erythematous (SLE). The aim of this study was to study role of ROS and caspase-3 in CTL mediated cell death in 40 SLE patients and controls.

**Method:** The ROS levels were measured using the dye 2’,7’-dichlorofluorescein diacetate by flow cytometry. Cytotoxic T lymphocyte activity of CD8+ T cells was measured by the intracellular expression of perforin/granzyme B in CD8+ T cells by flow cytometry. The disease activity was determined by using SLE Disease Activity Index (SLEDAI) score.

**Results:** The levels of ROS and intracellular expression of caspase-3 were significantly elevated in SLE patients and positively associated with disease activity in SLE patients. The expression of perforin/granzyme B independently and perforin/granzyme B together in CD8+ T cells were significantly increased in SLE patients and correlated with the disease in SLE patients. Furthermore, the levels of granzyme B and perforin/granzyme B on CD8+ T lymphocytes were strongly associated with increased level of caspase-3 and ROS in SLE patients.

**Conclusion:** The increased levels of caspase-3 and ROS production demonstrate that CTL mediated cell death mediated by caspase-3 dependent pathway and this amplification get enhanced by production of ROS in SLE patients.

**Conflict of Interest:** The authors declare that they have no conflict of interest.

**ES15. THE RELATIONSHIP BETWEEN OPG/RANK/RANKL SYSTEM AND INFLAMMATION IN EARLY SYSTEMIC SCLEROSIS**

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**Introduction:** The pathogenesis of Systemic Sclerosis is complex and incompletely understood. A holistic view of pathogenesis must integrate the three cardinal features of SSc: vascular injury and damage, inflammation, autoimmunity, and generalized interstitial and vascular fibrosis. Although each of these processes occurs in each patient, their relative contribution to the disease varies from one patient to another osteoprotegerin(OPG), also known as an osteoclast inhibitor factor, is a cytokine member of the TNF receptor superfamily, a key factor in bone remodeling, and a decoy receptor for the receptor activator of nuclear factor-ligand (RANKL). Recently it has been demonstrated that OPG is produced not only in bone but also in other tissues, including the cardiovascular system, lungs, kidneys, immune tissues, and blood vessel walls. OPG was implicated in inflammation First, by interfering with RANKL and possibly other members of the TNF family signaling pathway; it blocks some aspects of inflammation. In addition, OPG may block another role of RANKL—activation of lymphocyte differentiation and function. Under normal conditions, RANK-L is most highly expressed in lymph node tissue and it enhances activated T-cell survival. Mice deficient in RANK-L have no lymph nodes and abnormal B- and T-cell differentiation. In peripheral blood monocytes, RANK-L induces expression of a myriad of inflammatory cytokines, including TNF-alpha, interleukin (IL)-6, IL-12, IL-1-beta, and macrophage inflammatory protein (MIP)-1alpha. In conclusion, protection from OPG may block in the clinically significant bone loss seen in association with rheumatoid arthritis. In the vascular system, several lines of evidence suggest a function of OPG in vascular disease. Increased OPG levels may be related to endothelial lesion formation, endothelial dysfunction, smooth muscle hyperplasia, or advanced plaque calcifications.
In animal models of arterial calcifications induced by warfarin or vitamin D intoxication, subcutaneous administration of OPG prevented the appearance of vascular lesions OPG is expressed in the smooth muscle layer of coronary vessels and increases the survival of these endothelial cells by interfering in their apoptosis. In humans submitted to coronary angiography, OPG levels were significantly higher in those with more advanced atherosclerotic disease. Recent data demonstrated that OPG is increased in PAH and that it can regulate PA-SMC proliferation and migration. OPG may provide a common link between the different pathways associated with the disease, potentially playing an important role in the pathogenesis of PAH. Which is a leading cause of mortality in SSC. The outcome in SSc-associated PAH is considerably worse than that of idiopathic PAH

As Micro-vascular damage & inflammation are the main pathogenic events in early systemic sclerosis (SSc). The receptor activator of nuclear factor-κ ligand RANKL/RANK/OPG system is involved in vascular biology & inflammation.

Aim: 1) To assess OPG and soluble RANKL (sRANKL) serum levels in patients with SSc and compare with healthy controls from german population.

2) To prospectively evaluate the relationship between soluble RANKL (sRANKL) serum levels, plasma OPG levels & the skin scoring, arthritis, nail fold capillaroscopy, BMD in early SSc

2) To determine the relationship between OPG and biomarkers of inflammatory arthritis in early SSc patients with SSc and compare with healthy controls from german population.

Exclusion criteria:

- Persons on prolonged steroid therapy.
- Iloprost.
- Oral anticoagulant therapy will not be administrated to patients under study.
- History of any cardiovascular disease, venous thromboembolism or other chronic diseases.
- Persons on prolonged steroid therapy, lloprost, oral anticoagulant, heparin, anti-aggregant therapy will not be administrated to patients investigated in this study. In the observation period, none will receive, methotrexate, cyclophosphamide, azathioprine and D-penicillamine, topical glicerol nitrate, ACE-inhibitors. 15 days before the study.
- Patients on drugs known to interfere with bone metabolism.

Inclusion Criteria

Patients with early Systemic sclerosis ~ 3 years’ duration and 50 healthy controls matched for age, sex, BMD and menopausal status.

Methods:

1. Skin score according to Rodent Reference
2. Nail fold capillaroscopy
3. Blood sample is collected for C-reactive protein, TNF-alpha, interleukin-6, interleukin-10, interleukin-12, interleukin-17 and TGF beta.
4. Soluble vascular cell adhesion molecule (sVCAM; marker of endothelialactivation/injury)
5. Plasma OPG levels measured by an ELISA.

Statistical analysis

1. Anova
2. Mann-Whitney U test linear regression analysis
3. Odds ratio will be calculated

ES16. POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN SYSTEMIC LUPUS ERYTHEMATOUS: A REPORT FROM THE DEVELOPING WORLD

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Background: Since the original description in 1996, 66 cases of PRES have been reported in SLE. PRES frequently occurs in the setting of high disease activity.

Methods: We reviewed the records of patients with SLE and PRES, seen in our department over eight years.

Results: Case 1: A 13 year old Indian female, with SLE based on oral ulcers, arthritis, serositis, haematological involvement, suspected nephritis, positive ANF and anti-dsDNA, was admitted to ICU with a non-resolving bronchopneumonia. Empirical anti-tuberculous therapy was commenced. Her admission was complicated by herpes zoster, bronchopneumonia and active alveolitis. Pharmacotherapy included IVI corticosteroids, oral prednisone, mycophenolate mofetil and IVI cyclophosphamide. She developed headaches, blurred vision, focal neurological deficits, hypertension and rapidly deteriorating renal function. MRI demonstrated T2 and flair hyper-intensities in the occipital, parietal and left frontal lobes. Treatment included antiepileptics and plasmapheresis. Complete clinical and radiological recovery occurred, with no recurrence. She demised 18 months later from pneumonia.

Case 2: A 31 year old Coloured female known with SLE for 6 months, based on mucocutaneous disease, haematological involvement, suspected nephritis and positive ANF, was admitted to ICU, with cognitive disturbance, seizures, hypertension, progressive renal impairment and bronchopneumonia. Neuro-imaging revealed white matter hypodensities in the high parietal, occipital and L fronto-parietal regions. Management included mechanical ventilation, BP control, haemodialysis and IVI antibiotics. ICU recovery was unremarkable. Subsequent complications included recurrent infections, psychosis, alveolar haemorrhage and cutaneous flares, necessitating repeat pulses of IVI corticosteroids and cyclophosphamide. She is currently on ICU with recurrent seizures, visual disturbance and increasing BP. A repeat MRI is pending.

Conclusions: We report two additional cases of PRES, occurring in association with hypertension, renal disease, immunosuppressive therapy and infections. The characteristic features and reversibility of PRES emphasises the importance of distinguishing it from the wide spectrum of neurological manifestations in SLE.

Disclosure statement: The authors have declared no conflicts of interest.
controls. The haplotype MLBL -221/9G/27/54G is more frequent among the controls and is protective for the development of SLE (p = 0.048).

Conclusion: Our results indicate that both TNF-α and MLBL polymorphisms are associated with the development of systemic lupus in Bulgarian patients. The research analysis was fulfilled under projects - MU-Sofia 26/2008 and MU-Sofia 50/2010. The authors have declared no conflicts of interest.

ES18. LIFE-THREATENING CRYOglobulinemic Vasculitis ASSOCIATED WITH HCV Infection. Clinical Description and Outcomes of 279 CASES.

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Objectives: To analyze the clinical characteristics and outcomes of HCV patients presenting with life-threatening cryoglobulinemic vasculitis.

Methods: We evaluated 181 admissions from 89 HCV patients diagnosed with cryoglobulinemic vasculitis consecutively admitted to our Department between 1995 and 2010. In addition, we performed a systematic analysis of cases reported to date through a MEDLINE search. The following organ involvements were considered as potentially life-threatening in HCV patients with cryoglobulinemic vasculitis: cryoglobulinemic, biopsy-proven glomerulonephritis presenting with renal failure, gastrointestinal vasculitis, pulmonary hemorrhage, CNS involvement and myocardial involvement.

Results: A total of 279 patients (29 from our Department and 250 from the literature search) fulfilled the inclusion criteria: 205 presented with renal failure, 45 with gastrointestinal vasculitis, 38 with CNS involvement, 18 with pulmonary hemorrhage and 3 with myocardial involvement; 30 patients presented with more than one life-threatening cryoglobulinemic manifestation. There were 156 (56%) women and 123 (44%) men, with a mean age at diagnosis of cryoglobulinemia of 54 years (range, 25 to 87) and a mean age at life-threatening involvement of 55 years (range, 25 to 87). In 266 (86%) patients, life-threatening involvement was the first clinical manifestation of cryoglobulinemia. Severe involvement appeared a mean of 1.2 years (range 1–11) after the diagnosis of cryoglobulinemic vasculitis. Patients were followed for a mean of 14 months (range, 3 to 120) after the diagnosis of life-threatening cryoglobulinemia. One hundred and thirty-eight patients (44%) died. The highest rate of mortality was found in patients presenting with pulmonary hemorrhage (78%), followed by those with CNS involvement (34%), gastrointestinal vasculitis (33%) and renal failure (30%).

Conclusions: HCV-related cryoglobulinemia may result in progressive (renal involvement) or acute (pulmonary hemorrhage, gastrointestinal ischemia, CNS involvement) life-threatening organ damage. The rate of these manifestations ranges between 30% and 80%. Unfortunately, this may be the first cryoglobulinemic involvement in almost two-thirds of cases, highlighting the complex management and very elevated mortality of these cases.

The presenting authors have declared no conflicts of interest.

ES20. EXPRESSION OF THE TYPE I INTERFERON SYSTEM IN MUSCLE AND LUNG OF EXPERIMENTAL AUTOIMMUNE MYOSITIS

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Background: To investigate the expression levels of the type I interferon system in muscle and lung of experimental autoimmune myositis (EAM) model and to determine any correlation the EAM mouse model associates with the pathogenesis of the EAM model in rats.

Methods: The EAM model was established to determine creatine kinase (CK) in blood serum. The pathology of muscle and lung tissue was examined by hematoxylin-eosin staining. The concentration of type I interferon system mRNA in muscle and lung tissue was detected by real-time PCR.

Results: The concentration of creatine kinase (CK) [209.17 ± 9.69(U/L) in model group was significantly higher than that of control groups [76.16 ± 13.21(U/L), 80.00 ± 22.37(U/L)] (P < 0.05). The scores of muscle and lung in EAM model were significantly higher than that of control groups (all P < 0.05). The expression levels of the type I IFN system in muscle of EAM model were significantly higher than that of control groups (all P < 0.05). The expression levels of the type I IFN system in muscle with EAM model were positively correlated with CK and the scores of muscle (all P < 0.05). The expression levels of IFNα, IFNβ, IFNα1, Signal transducer and activator of transcription 1 (STAT1), Myxovirus resistance protein 1 (MX1) in lung of EAM model were significantly higher than those of control groups (all P < 0.05), but without interferon-induced protein with tetratricopeptide repeats 1 (IFIT1) and IFN-stimulated gene 15 (ISG15). The expression levels of IFNα, IFNβ, MX1 in lung with EAM model were positively correlated with the scores of lung (all P < 0.05).

Conclusions: The type I interferon system probably played a crucial role in the pathogenesis and the pathology of muscular and lung of EAM model.

Keywords: Myositis, Interferon Type I, Muscle, skeletal, Lung.

ES19. LATE-ONSET SYSTEMIC LUPUS ERYTHEMATOSUS IN CHINESE PATIENTS: NOT A BENIGN DISEASE

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Background: Systemic lupus erythematosus (SLE) is a multisystemic disorder which predominantly affects women of child-bearing age. Onset of the disease beyond the age of 50 is rare and considered to be benign. This aim of this study was to assess clinical manifestations, laboratory finding and outcomes of a cohort of Chinese SLE patients with late disease onset.

Methods: All hospitalized SLE patients from 2001 to 2010 in our hospital were retrospectively analyzed. Patients who developed disease at or after the age of 50 were considered as late-onset SLE. 1:1 matched new onset control patients, admitted in the same period with initial symptoms before the age of 50, paired by race and disease duration, were randomly selected. Clinical manifestations, laboratory findings and outcomes as well as outcome of the two groups of patients were compared.

Results: 35 patients (4.3%) were identified as late onset SLE. Lower female predominance was found in late-onset patients (P < 0.05). Compared with early onset controls, the prevalence of malar rash (P < 0.01), oral ulcers (P < 0.01), Raynaud phenomenon (P < 0.05) was lower in late-onset patients, whereas renal involvement (P < 0.01) occurred more frequently. In laboratory findings, the prevalence of anti-dsDNA (P < 0.01), anti-Sm (P < 0.01), anti-SSA (P < 0.05), anti-SSB (P < 0.05) were lower in late-onset SLE patients. Additionally, late-onset SLE patients have lower scores of disease activity index (SLEDAI) at diagnosis (P < 0.05). Immunosuppressive drugs were less frequently used in late-onset SLE patients (P < 0.05). The mortality was increased in late-onset disease group.

Conclusions: Late-onset SLE patients exhibited different clinical and laboratory features. Although disease activity tends to be lower, they tend to experience more severe renal damage and higher mortality.

Disclosure Statement: The authors have declared no conflicts of interest.

ES21. THE PREDICTIVE VALUE OF SMOKING AS RISK FACTOR AT ONSET OF LUPUS

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Background: Patients with systemic lupus erythematosus (SLE) are at increased risk of developing cardiovascular disease and this is associated with significant early morbidity and mortality from coronary heart disease.

Methods: A representative of 102 SLE patients, who fulfilled the 1997 ACR criteria for SLE. They also underwent a carotid examination using B model Doppler depending on presence or absence of plaque or thickening of intima media. The smoking was assessed by “The North Karelia Project” a) actual smoker; b) ex-smoker; c) non-smoker. The assessment included historical taking, information given regarding exercise and family history of cardiovascular disease in first degree relative; level of blood pressure, weight, height and investigations of serum cholesterol, glucose and antiphospholipid syndrome.

Results: In the study were included 90 patients who met the criteria for lupus. It was found a quarter 24 (26.6%) of patients that were current smokers. Based on the results obtained the average age was
ES24. ENGINEERED SMOOTH MUSCLE FROM ADIPOSE TISSUE DERIVED STEM CELLS FOR TREATMENT OF MUSCLE DYSFUNCTION IN RHEUMATIC DISEASES

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Background: Many rheumatic diseases involve smooth muscle cells (SMC) pathology. Adipose derived stem cells (ADSCS) represent ideal source for cellular treatment. The aim of our study was to investigate whether ADSCs can be differentiated into SMCs and whether these differentiated cells can be delivered into various forms including dissociated cells, cell sheets and cell seeded scaffolds.

Material and methods: Rat and Human ADSCs were isolated and expanded. They were induced to SMCs using DMEM with 10% fetal bovine serum and recombinant human TGF-β1. The phenotype of induced cells was checked for SMC markers; actin, calponin and myosin through immunocytochemistry and western blot. Then, the cells were used to construct SMC sheets by plating into thermo-sensitive dishes and SMC grafts by seeding cells onto SIS. The cell sheets were stained with H&E and Masson Trichrome and checked for SM actin, calponin and myosin, phallolidin and collagen IV. The EdU labeled seeded grafts were implanted subcutaneously in rats, and evaluated after 1 week for cell survival, proliferation and phenotype.

Results: The induced cells showed positive staining for SMC markers indicating their phenotype after 4 weeks in rat cultures and 3 weeks in human cells. The western blot confirmed their phenotype conversion. The cell sheets were formed of the 2-5 layers of cells that showed positive staining for SMC markers. They also showed positive staining for collagen IV indicating the formation of extracellular matrix. The seeded grafts showed cells viability, attachment and proliferation and maintained their phenotype. After in vivo implantation, the seeded cells formed SMC tissue and incorporated into new blood vessels.

Conclusion: These results showed the ability of ADSCs to form SMC and form renewable source for cellular therapy. The SMC sheets form new technology for SMC repair in rheumatic patients. Cell-seeded grafts are needed for whole organ replacement in various disease conditions.

Disclosure statement: The authors have declared no conflicts of interest.

ES25. SERUM ALANINE AMINO TRANSFERASE–A DISEASE ACTIVITY MARKER IN INFLAMMATORY MYOSITIS?

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Introduction. Inflammatory myositis is often complicated by raised liver enzymes i.e. aspartate transaminase (AST) and alanine aminotransferase (ALT). AST, being a ubiquitous enzyme is expected to rise in myositis but little is known about alanine amino transferase. The aim is to evaluate the serum level of Alanine aminotransferase as a disease activity marker in inflammatory myositis.

Methods: A total of 70 patients suffering from inflammatory myositis was assessed for disease activity and serum ALT level. The patients were divided into two groups, group A, where inflammatory myositis was active and group B, where inflammatory myositis was inactive.
of this study was to determine whether serum ALT is related to disease activity in myositis.

Methods. Hospital Records of patients with idiopathic inflammatory myositis (IIM), attending rheumatology clinic from January 2008 to August 2011, were studied retrospectively. All patients satisfied Bohan and Peter criteria for definite/probable IIM, while patients with myositis overlap syndrome were excluded. Besides demographic data, diagnosis, laboratory values and treatment details, paired values of serum creatinine phosphokinase (CPK) and liver enzymes (AST, ALT, ALP) were noted at baseline, 1st review between 2 and 4 months and the last visit. Bivariate correlation between log converted values of CPK and AST, ALT and ALP was analysed.

Results. Forty one patients with IIM (Dermatomyositis-29, Polymyositis-9 and unclassified inflammatory myositis-3) were identified. Demography revealed Male:Female of 14:27, Median Age 32 (9-68) years and median disease duration 4.5 (1-96) months. Majority (38/41) of patients had active disease at baseline, as evidenced by muscle weakness and/or active rash and/or raised CPK (31 patients). Baseline serum ALT and CPK levels were 113 (18 to 441 U/L) and 2196 (32 to 74900 U/L) respectively. Serial values of these enzymes were analysed on 99 occasions. All patients were given steroids and 26/41 were initiated on methotrexate or azathioprine in addition.

There was a good correlation between serum ALT and CPK at baseline ($R = 0.43, p = 0.000$) and serial follow up ($R = 0.55, p = 0.000$). The correlation between ALT and CPK was similar in serial values in patients with ($R = 0.59, p = 0.000$) or without ($R = 0.50, p = 0.000$) methotrexate or azathioprine therapy. In 2 out of 31 patients with regular follow up, ALT remained persistently elevated (> 100 IU/L); both had persistently active disease.

Conclusion. In patients with inflammatory myositis, there is a linear correlation of serum ALT with serum CPK, which was unrelated to drug effect of methotrexate or azathioprine.