Background: The assessment of disease activity plays a pivotal role in the management of children with juvenile idiopathic arthritis (JIA). Most recent recommendations require that parents’ and children’s perception is incorporated in the evaluation of the disease course and of effectiveness of therapeutic interventions. A new disease activity tool, named parent Juvenile Arthritis Disease Activity Score (parJADAS) and based only on parent-centered outcome measures, is currently under development (1).

Objectives: To demonstrate, in a large multinational dataset of JIA patients, the discriminant ability of the parJADAS.

Methods: The parJADAS includes 4 measures: 1) parent assessment of disease activity; 2) assessment of pain intensity; 3) proxy assessment of joint disease; 4) assessment of morning stiffness (MS). Disease activity and pain are assessed on a 21-numbered circle VAS (0 = best and 10 = worst). The active joint count is based on the count of any swollen or painful joint, irrespective of its type, up to a maximum of 10 joints. MS duration is assessed on a Likert scale ranging from no MS (0 points) to > 2 hours of MS (10 points). Validation was conducted on a dataset of 8,656 children with JIA from 49 countries, enrolled in the study of Epidemiology, treatment and Outcome of Childhood Arthritis (2), a dataset of 8,656 children with JIA from 49 countries, enrolled in the study of Epidemiology, treatment and Outcome of Childhood Arthritis (2), who had all the variables included in the parJADAS available. Discriminant ability was evaluated by comparing parJADAS levels (median, [IQR]) among patients with inactive disease (ID), low disease activity (LDA), moderate disease activity (MDA), and high disease activity (HDA) according to the C-JADAS10; patients in remission, continued activity, or flare according to the attending physician; patients whose parents were satisfied or not with current disease state. To assess the possible influence of the articular and extra-articular damage on the parJADAS, the levels of the score in patients with or without damage (Juvenile Arthritis Damage Index > 0) were compared. For this analysis, only subjects in inactive disease and with at least 2 years of disease course were considered (n = 2,423).

Results: The levels of parJADAS in patients in ID, LDA, MDA, and HDA were 0.0 [0.0, 1.0], 3.0 [1.0, 6.0], 6.0 [2.0, 11.5], an 14.5 [8.5, 21.0], respectively (Kruskal-Wallis test, p < 0.001). The levels of parJADAS in patients in remission, continued activity, or flare according to the attending physician were 0.5 [0.0, 3.5], 9.0 [3.5, 17.0], 12.0 [5.5, 20.0], respectively (Kruskal-Wallis test, p < 0.001). Median parJADAS in patients whose parents were satisfied or not satisfied with disease course is 1.5 [0.0, 7.0] and 13.0 [6.6, 20.5], respectively (Mann-Whitney test p < 0.001). ParJADAS was not different in JIA patients in remission with or without damage measured with the JADI (Mann-Whitney test p = 0.08).

Conclusion: The parJADAS showed excellent discriminate ability in a large multinational cohort. The score did not show to be relevantly influenced by disease damage in JIA patients in remission.

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REFERENCES

**Background:** The patient global assessment (PtGA) is a core set variable to assess disease activity in rheumatoid arthritis (RA). It is strongly linked to patient-reported pain and has shown to be a limiting factor for reaching remission in patients with remittent joint inflammation and normal acute phase response, particularly when the ACR/EULAR Boolean criteria are used. In these, the PtGA may not be greater than 10 on a 0-100 scale to reach remission.

**Objectives:** To analyse the impact of higher cut-offs for or removal of the PtGA applied in the ACR/EULAR Boolean remission on the consistency with the SDAI remission definition, and with respect to long-term structural and functional outcomes.

**Methods:** We retrieved data from six clinical trials testing the efficacy of tumor-necrosis factor inhibitors vs MTX or placebo/MTX. Three trials depicted early RA: ASPIRE (infliximab), Go Before (golimumab), PREMIER (adalimumab); and three late RA: ATTRACT (infliximab), DE019 (adalimumab) and Go Forward (golimumab). We increased the cut-off of the PtGA gradually by 5mm (mBoolean15-REM) up to 30mm, and also omitted the criterion completely (Boolean-NO REM, i.e. requiring only CRP, SJC, TJC<1). We assessed frequencies of remission by these definitions at 6 and 12 months and evaluated agreement with the Index based (SDAI) definition of remission (which does not include an inherent cut-off for PtGA). Further the impact on functional and structural outcomes after 1 year were explored based on achievement of each of these remission definitions at 6 months.

**Results:** We included 3293 patients in our study, 2121 in early and 1172 in late RA (mean disease duration: 1.5±3.0 and 9.8±8.6 years, respectively). The rates of patients achieving Boolean remission increased with higher allowance for PtGA from 11.9% to 18.8% in early RA, and from 5.9% to 12.4% in late RA at 6 month, and from 19.7% to 29.9% and from 10.7% to 20.9%, respectively, at 1 year. Best agreement with SDAI occurred at a PtGA cut-off of 15mm and 20mm, while with higher allowances for PtGA the disconnect between Boolean and Index based remission increased again (Figure). Radiographic progression was very similar in the different Boolean groups (ranging from a mean of 0.27±4.7 to 0.41±5.1). As expected, removing the PtGA increased functional impairment in remission (% of patients scoring HAQ=0 in Boolean 10, 15, 20, 25, 30, and in the “no PtGA” definition were 68.2%, 63.7%, 60.4%, 56.0%, 54.6% and 47.9%, respectively. Functional scores in late RA were generally worse than in early RA. CRP levels across all definitions, including “no PtGA”, were similar (data not shown).

**Conclusion:** Increasing the PtGA cut-off to 15mm would provide highest consistency between Boolean with the Index based remission, while the integer cut-off of 20mm (or 2/10) would also allow the use on numerical rating scales. This new cut-off would discount the overly stringency of the PtGA in the remission context, while keeping the patient perspective in the core of RA disease activity evaluation.

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The diagnostic value of serum KL-6 in connective tissue disease associated interstitial lung disease

Wu Xue, Lijun Wu, Cai Nan Luo, Ya Mei Shi.
People’s Hospital of Xinjiang Uygur Autonomous Region, Department of Rheumatology and Immunology, Urumqi, China

Background: The connective tissue diseases (CTDs) are a group of inflammatory, immune-mediated disorders. Involvement of the respiratory system, particularly interstitial lung disease (ILD), is common and is an important contributor to morbidity and mortality. Currently, high-resolution computed tomography (HRCT), bronchoscopic examination and surgical lung biopsy (SLB) are the basic methods for the diagnosis of ILD. But these tests require specific inspection machines, are less repeatable and cause considerable discomfort to the subject.

Objectives: To evaluate the diagnosis of the serum Krebs von den Lungen-6 (KL-6) for the interstitial lung disease (ILD) associated with connective tissue diseases (CTD).

Methods: Patients with CTDs who visited our Hospital between January, 2016 and December, 2017, and whose serum KL-6 level was measured were included. We analyzed 175 patients with CTDs, 84 CTDs associated ILD, 91 CTDs patients without ILD. Record age, gender, diagnosis, serum KL-6 levels, pulmonary function tests and performed in parallel were reviewed. Statistical analysis was performed using SPSS (version 20.0) statistical package.

Results: The significantly higher levels of KL-6 were determined in the CTD-ILD group than in the CTDs without pulmonary involvement group (P<0.05 (Figure 1). By the ROC curves of serum KL-6 levels in 175 patients. The optimal cutoff value of serum KL-6 for a diagnosis of CTD-ILD was 409U/ml, and the sensitivity and specificity were 82.1% and 86.8%, respectively. The AUC was 0.905 (Figure 2). Serum KL-6 correlated negatively with vital capacity (VC) (%predicted), forced vital capacity (FVC) (%predicted), forced expiratory volume in one second (FEV1) (%predicted) and diffusing capacity of the lung for carbon monoxide (DLcoSB) (% predicted) (Table 1).

Table 1 Correlation between KL-6 level and pulmonary function

<table>
<thead>
<tr>
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<th>KL-6</th>
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<tbody>
<tr>
<td>VC%</td>
<td>-0.196 0.009</td>
</tr>
<tr>
<td>FVC%</td>
<td>-0.158 0.037</td>
</tr>
<tr>
<td>FEV1%</td>
<td>-0.173 0.022</td>
</tr>
<tr>
<td>FEF25%</td>
<td>-0.090 0.237</td>
</tr>
<tr>
<td>FEF50%</td>
<td>-0.058 0.447</td>
</tr>
<tr>
<td>FEF75%</td>
<td>-0.044 0.560</td>
</tr>
<tr>
<td>MMEF%</td>
<td>-0.075 0.323</td>
</tr>
<tr>
<td>DLcoSB</td>
<td>-0.470 0.000</td>
</tr>
</tbody>
</table>

Conclusion: The serum KL-6 is a valuable biomarker for CTD-ILD diagnosis, and it is an important serum marker for detection of CTD-ILD activity.

References:

Disclosure of Interests: None declared


Anti-C1q antibodies have higher correlation with lupus nephritis disease activity

Wu Xue, Lijun Wu, Cai Nan Luo. People’s Hospital of Xinjiang Uygur Autonomous Region, Department of Rheumatology and Immunology, Urumqi, China

Background: Systemic lupus erythematosus (SLE) is the prototype of autoimmune disease and is characterized by the production of a variety of autoantibodies, and lupus nephritis continues to be a principal cause of morbidity and mortality. The focus is to finding biomarkers that can monitor renal activity and predict prognosis for early diagnosis and treatment. Complement C1q is the starter molecule of the classical pathway.

Fig.1: Comparison of serum KL-6 concentrations in CTD-ILD group and CTD-ILD group.

Fig.2 Receiver-operating characteristic curve (ROC) of KL-6 for the diagnosis of CTD-ILD.
of complement activation and plays an important role in the clearance of immune complexes and apoptotic cell debris. Hereditary homozygous deficiency of C1q has been described to be the strongest risk factor for developing SLE. There are some cross-sectional studies on anti-C1q in which the antibody was found to have a significant association with renal involvement but the association of anti-C1q antibodies (antiC1q) with lupus nephritis (LN) still a matter of debate.

Objectives: We assessed the association between lupus nephritis disease activity and anti-C1q antibodies

Methods: We retrospectively analyzed the medical records of 88 patients with lupus nephritis, aged 35.7±10.8 years on the average, with SLE of average duration 12 (3, 57) years. In all examines the levels of anti-dsDNA and anti-C1q antibodies were measured using the ELISA, C3, C4, 24-hour urinary protein performed in parallel were reviewed. The clinical manifestations of SLE was also collected. Lupus nephritis disease activity was measured by The SLICC Renal Activity Score of 2004. All biopsied tissues were scored based on the to ISN/RPS2003 lupus nephritis pathological typing standards. Acute Index, Chronic Index Score were used to evaluated the activities of lupus. All the analyses were performed by SPSS 20.0 software.

Results: Patients with active lupus nephritis had a higher levels of anti-C1q antibodies than inactive lupus nephritis (68.9(34.1, 140.1) vs. 11.6(5.5, 44.1); P<0.001) (Figure 1). Anti-C1q antibody levels were positively correlated with levels of 24-hour urinary protein (r=0.605; P=0.000), AI score (r=0.337; P=0.001), and negatively correlated with serum C3 (r=-0.573; P=0.000) and C4 (r=-0.509; P=0.000) (Figure 2).

Conclusion: Anti-C1q antibodies are more closely correlated with renal disease activity.

REFERENCES

Disclosure of Interests: None declared

SAT0674 ADIPOKINES AND ENDOTHELIAL DYSFUNCTION IN SYSTEMIC LUPUS ERYTHEMATOSUS

ahmed yamany1, Menvat Behiry1, Ahmed Fayed2, Ahmed Soliman2, 1Kasr El Aini Teaching Hospital, internal medicine, cairo, Egypt; 2Kasr El Aini Teaching Hospital, internal medicine, cairo, Egypt

Background: Premature atherosclerosis is clearly described in systemic lupus erythematosus as a main cause of poor outcomes and mortality(1). Pleiotropic adipokines including adiponectin and visfatin are implicated in the inflammatory process of lupus disease that promote cytokines signaling leading accelerated endothelial disruption(2)

Objectives: To evaluate the endothelial dysfunction by measuring serum visfatin, adiponectin, leptin and HOMA-insulin Index as reliable biomarkers of atherosclerosis & estimating the FMD of brachial artery among lupus patients and correlating these parameters with clinical characteristics

Methods: A case-control study in which 150 systemic lupus patients who were fulfilling American College of Rheumatology revised classification were recruited consecutively from Internal Medicine department at Cairo University. They were compared to 90 age & sex matched healthy controls. Patients who were pregnant, smoker, diabetic, those with hepatic and