Methods: 94 patients diagnosed with JIA (mean age 8.78±5.21), 95 children with articular syndrome (mean age7.90±4.81) and 164 hospital controls without any signs of autoimmune or inflammatory diseases (mean age13.99±2.68) were included in the study. The JIA patients were divided into subgroups according to IIAR classification criteria; among them 63 patients were diagnosed with oligoarthritis and 16 with RF- polyarthritis. Genomic DNA was extracted from blood samples by means of phenol-chloroform method. SNPs were genotyped using PCR-RFLP or fragmental analysis.
Results: The allele frequencies for all SNPs in the hospital control group were similar to those characteristic of other Europeans. No differences were found between the frequencies of the TNFα risk alleles across all three groups. Hence, both SNPs in the TNFα locus were not associated with JIA and other articular pathologies in our study. The same is true for -318C>T *CTLA4* polymorphism. Unlike these genes, *PTPN22* C1858T polymorphism influenced developing arthritis in children since heterozygous CT genotype was associated with articular pathology (rather than JIA) in the total group (OR=1.87, 95% CI [1.06–3.30], p=0.04), especially in males (OR=3.50, 95% CI [1.61–7.63], p=0.0016). In the latter case, it was effective even being combined with any genotypes in the -308G>A TNFα locus (OR=2.73, 95% CI [1.19–6.24], p=0.018). When analyzing *MIF* polymorphisms (rs755622 and rs5844572), the evident trend to increased carrying genotypes containing the risk allele *MIF*-173C was observed in females with JIA as compared to controls and significantly elevated frequency of this risk allele in females with RF- polyarthritis as compared to males (p =0.037). In females with JIA, increased frequency of heterozygous *MIF*-794 $CATT_{7,8}$ genotype as compared to the controls (OR =7.78, 95% CI [0.95 – 63.8], p=0,056) was also revealed. *STAT4* polymorphism (rs7574865) demonstrated subtype-related association with JIA due to increased frequency of the minor allele in patients with polyarthitic form of JIA as compared to both hospital controls (p=0.01; OR =2.45; 95% CI [1.19–5.04]) and other articular pathology (p=0.001; OR =3.37; 95% CI [1.56–7.28]). The same SNP was also associated with developing arthritis in females. As to role of DNA repair genes, carriers of *XPD* heterozygous Asp/Asn genotype had an enhanced risk of JIA in females (OR=2.14; 95% CI [1.05–4.35]; p=0.05).
Conclusions: Thus, the gender- and subtype-specific associations of some SNPs studied with developing joint pathologies including JIA are found in the Belarusian child population.
Disclosure of Interest: None declared
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Paediatric rheumatology

THU0486 ANGIOPOETIN-2 AS A NEW VALUABLE MARKER OF DISEASE ACTIVITY IN CHILDREN WITH JUVENILE IDIOPATHIC ARTHRITIS

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Background: Angiopoetin-2 (ANG-2) is one of the main angiogenesis modulators. As synovial angiogenesis is considered to be an important early step in the course and pathogenesis of juvenile idiopathic arthritis (JIA), it may be a valuable marker of activity of the disease.
Objectives: To evaluate ANG-2 serum and synovial fluid levels in JIA patients and its possible correlation with disease activity and the degree of ultrasound-detected synovial angiogenesis.
Methods: Serum levels of vascular markers were measured in 63 patients with JIA (aged 1,5–17) and 31 age-matched healthy controls. Synovial fluid was collected from 17 JIA patients. Disease activity (low, medium and high) was assessed by JADAS-27 scale. Ultrasound examination of inflamed joints was performed in each JIA patient and synovial angiogenesis was evaluated by means of Power Doppler ultrasonography (PDUS) and the 4th grade vascularity scale (0–3).
Results: ANG-2 serum levels were higher in children with JIA comparing to the healthy controls (5,5±1,2 ng/ml vs. 1.9±1.4 ng/ml). Serum concentration of ANG-2 in JIA children increased with disease activity (1,4±0,9 ng/ml vs. 1,4±1,0 ng/ml vs. 1,8±2,2 ng/ml), whereas Ang-2 levels in the synovial fluid were the highest in the high disease activity (3,4±2,5 ng vs. 12,5±9,5 ng/ml vs. 26,4±9,0 ng/ml, $p<0,05$). Concentration of ANG-2 in serum correlate with the synovial vascularization obtained by PDUS (0–3, accordingly 1,5±0,8 ng/ml vs. 1,3±0,9 ng/ml vs. 1,5±1,0 ng/ml vs. 13,2±8,7 ng/ml, $p<0,01$) The same pattern was observed for ANG-2 levels in synovial fluid and PDUS grade (0–3, accordingly 3,8±1,0 ng/ml vs. 5,1±4,1 ng/ml vs. 13,5±2,0 ng/ml, $p<0,01$).
Conclusions: ANG-2 might be a valuable marker in JIA children with high disease activity. Together with ultrasound examination, it may add more information about disease severity, what may be helpful in introducing the correct therapy.
Disclosure of Interest: None declared
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THU0487 REAL-LIFE TREATMENT WITH CANAKINUMAB IN SYSTEMIC JUVENILE IDIOPATHIC ARTHRITIS – FIRST EXPERIENCE FROM THE BIKER REGISTRY

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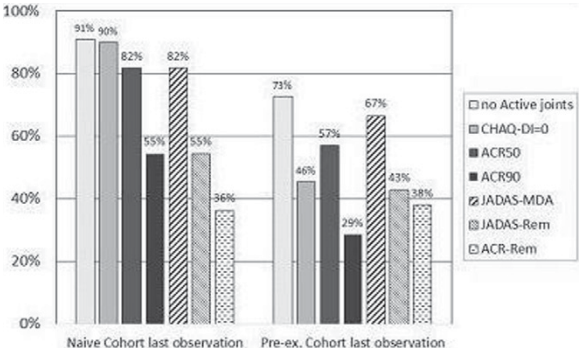
Background: Canakinumab (CAN) had demonstrated its efficacy and safety in SJIA pts (1).
Objectives: To report on the experience with CAN treatment in SJIA in the clinical practice in Germany.
Methods: Data on patients' and disease characteristics, disease activity and safety reports from the German BIKER registry were analysed.
Results: Until Dec. 2016, 37 pts exposed to CAN were identified. In 12 pts used CAN as first biologic, 25 pts were pretreated: Tocilizumab 15, Anakinra 11, Etanercept 9, Adalimumab 9. 3 patients in the pre-exposed cohort had experienced a macrophage activation syndrome. Pts' and disease characteristics are outlined in table 1. Pts pretreated were older, had a longer disease duration and more comorbidities than naïve patients. The proportion of pts with active arthritis, active systemic features and both were comparable. Disease activity at baseline was higher in the naïve cohort suggesting some clinical benefit from pretreatment. Dosing of CAN was comparable (*3.9+/-0.4 vs. 3.5+/-0.7mg/kg)* as well as the median treatment duration (0.8vs.1year). Treatment efficacy at last follow up was better in the naïve cohort with more pts reaching a PedACR 30/50/70/90 response while JADAS- or ACR-remission rates were comparable. Treatment was discontinued by 42% in the naïve and 48% in the exposed cohort. Reasons were inefficacy (n=7;19%), intolerance (n=2;5%) and remission (n=7;19%) of the disease and other (n=2;5%).

Table 1. Baseline characteristics

	Biologics naive	Biologics pre-exposed
N (female gender)	12 (17%)	25 (52%)
Age at JIA onset (years); mean ± SD; Median (IQR)	4.4±4.0; 3.0 (2.0–4.9)	6.0±4.8; 3.7 (2.6–7.1)
Age start of Canakinumab (years); mean ± SD; Median (IQR)	7.1±4.9; 5.6 (3.1–10.2)	9.8±4.8; 10.6 (5.5–14)
Disease duration (years); mean ± SD; Median (IQR)	2.8±4.1; 0.7 (0.3–3.9)	3.8 ± 4.2; 1.9 (0.6–8.7)
Concomitant treatment at baseline: NSAIDS/Steroids/MTX	6 (50%)/6 (50%)/3 (25%)	10 (40%)/11 (44%)/4 (16%)
Patients with active joints	7 (58.3%)	10 (47.6%)
Patients with active systemic features	7 (58.3%)	10 (47.6%)
Patients with active arthritis and systemic features	4 (36.4%)	4 (19.1%)
Active joint count	1.8±1.7; 2.5 (0–3)	2.1±3.3; 0 (0–3)
Physician global VAS (0–10)	5.2±2.8; 6.2 (3.2–7.2)	3.8±3.4 ; 3.7 (0.7–6.3)
Patient Global VAS (0–10)	4.8±2.9; 4.6 (2.7–7)	3.3±2.9; 2.6 (0.6–5.1)
CHAQ-DI (0–3)	0.71±0.65	0.65±0.84
ESR (mm/h)	28.2±22.8	15.4±20.1
CRP (mg/l)	70.5±58	12±23
JADAS10	15.7±8.3	10.5±7.9



Conclusions: First experience with CAN for treatment of sJIA in clinical practice is presented. A high proportion of pts gained significant improvement. JADAS remission was reached by about 50% and ACR remission by 25–57% in both biologics pre-exposed and naïve pts while few pts discontinued treatment in remission so far. Intolerance was rare. The further long term surveillance of sJIA pts exposed CAN is intended by the registry.
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