and safety in children with severe course of FOP. It showed their advantages over the use of steroids and possibility to inhibit the rate of progression.

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POS0084

SEPTIC ARTHRITIS IN CHILDREN. A LONGITUDINAL POPULATION-BASED STUDY IN WESTERN AUSTRALIA

J. Nossent^{1,2}, D. Preen³, H. Keen^{1,4}, W. Raymond¹, C. Inderjeeth^{1,2}, ¹The University of Western Australia, Medical School, Rheumatology Section, Perth, Australia; ²Sir Charles Gairdner Hospital, Rheumatology, Nedlands, Australia; ³The University of Western Australia, School of Population and Global Health, Bentley, Australia; ⁴Fiona Stanley Hospital, Rheumatology, Murdoch, Australia

Background: The incidence of Septic arthritis (SA) in adults is rising, but few data are available for children (1). SA symptomatology in young children is often atypical and delayed diagnosis can cause significant morbidity.

Objectives: To describe the incidence, risk factors and long-term outcomes in children hospitalised with septic arthritis (SA) in Western Australia (WA).

Methods: We extracted population-based longitudinally linked administrative health data for patients under 16 years with a first inpatient primary or secondary code of 711.xx (ICD9-CM) and M00.xx (ICD10-AM) in WA for the study period 1990-2010 (to allow a minimum 5 year followup). We report annual incidence rates per 100,000 (AIR), prior conditions during lookback (median 15 months, IQR 5-45) as well as joint and other comorbidities including Charlson comorbidity index (CCI) and standardised mortality rates (SMR) during a median follow-up of 10 years. Age and gender specific population and mortality rate data were obtained from the Australian Bureau of Statistics.

Results: A total of 891 patients (62% male, median age 6.4 (IQR 1.9-10.6) years with 34% <3 years of age) had a first admission for SA. AIR was 9.85 (CI 4.79-14.41) overall with higher rates in males (11.9 vs 7, p<0.01) and no apparent period (Figure 1) or seasonal variation. Knees (43.9%), hips (34.6%), and ankles (13.3%) were most frequently affected with *Staphylococci* (49%) the predominant organism in patients with positive cultures (41.5%). Prior infections (40.4%) and respiratory disease (7 %) were the main preexisting morbidities. Mean hospital stay was 5.78 (± 6.4) days with ICU admission required in 1.9%, while 30-day readmittance rate was 10.4%. During follow-up 25 patients (3%) had recurrent/persistent osteomyelitis, nine patients were diagnosed with osteoarthritis (1.1%) and five patients (0.6%) underwent joint replacement. More female patients developed new comorbidity (CCI>0, 34.6 vs 27.2%, p=0.02) including diabetes (4.2% vs 0%, p=0.001), cardiovascular events (4.2 vs 1.4%, p=0.002) and chronic arthritis (1% vs 0, p=0.05). While the crude mortality rate was low (0.3%) SMR was significantly increased for female patients (10.52, CI 1.59-41.6).

Conclusion: The statewide incidence of septic arthritis in children in WA is similar to a recent report (1) and did not change over a 20-year period. In this large population based study, subsequent bone/joint disease occurred in 4.6 %, while a third of patients developed other comorbidity before the age of 18. Such (sub-clinical) comorbidity may thus be a contributing factor to SA development and to the increased mortality risk in female SA patients.

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Figure 1. Annual incidence of septic arthritis per 100,000 population <16 years in Western Australia over period 1990-2010 by gender.

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POS0085

EVALUATION OF DURATION OF CLINICAL REMISSION IN CHILDREN WITH NON-SYSTEMIC JUVENILE IDIOPATHIC ARTHRITIS AFTER WITHDRAWAL OF ANTI-TUMOR NECROSIS FACTOR - ALPHA THERAPY

I. Tsulukiya¹, E. Alexeeva^{1,2}, T. Dvoryakovskaya^{1,2}, K. Isaeva¹, R. Denisova¹, O. Lomakina¹, A. Mamutova¹, A. Chomakhidze¹, A. Fetisova¹, M. Gautier¹, I. Kriulin^{1,2}, E. Krekhova^{1,2}, ¹National Medical Research Center of Children's Health, Rheumatology, Moscow, Russian Federation; ²Sechenov First Moscow State Medical University, Pediatric, Moscow, Russian Federation

Background: Juvenile idiopathic arthritis (JIA) is the most common and prevalent rheumatic disease in childhood which is based on a chronic autoimmune inflammation. Inactive disease and remission are now the primary treatment goal in JIA and biologics have been playing an important role to reach this objective. The biologics of the first choice for the treatment of non-systemic JIA are the Tumor Necrosis Factor - alpha (TNF α) inhibitors; on this therapy patients can achieve clinically inactive disease and long-term remission.

Currently, little is known about when or how to stop TNF α inhibitors, when a good clinical response is achieved, and therefore no guidelines are available.

Objectives: To estimate the length of clinical remission after discontinuation of treatment with TNF α inhibitors in patients with non-systemic juvenile idiopathic arthritis.

Methods: A total of 393 patients with JIA who were treated with TNF α inhibitors at the Rheumatology Department of the National Medical Research Center of Children's Health (Moscow, Russia) were screened for inclusion in this retrospective study.

Patients were treated with etanercept 1 times a week, 0.8 mg per kg of body weight per dose, with adalimumab 24 mg/m² body surface area administered every other week until the end of therapy. Treatment was terminated abruptly. Inactive disease was defined according to the preliminary criteria of Wallace et al.[1]

Results: 77 patients (27—male, 50—female) with a mean age at diagnosis of 4 years (range 1–18 years) were included in the analysis. Of those, 69 of them discontinued TNF α inhibitors due to a long-term remission on treatment, 8 patients as a result of side effects, and there were excluded from our study:

allergic reaction (n = 5), development of uveitis (n = 1), alopecia (n = 1), recurrent infection (n=11). The clinical subtypes of JIA were RF-negative polyarticular JIA -28 (40,58%) oligoarthritis—38 (55,07%), enthesitis-related arthritis—3 (4,35%). TNF α inhibitors were started after a mean 46,43 (range 1–144) months of disease. The mean duration of therapy with TNF α inhibitors were 46,63 (range 10-113) months, with a mean duration of remission on medication 40,63 (range 6-107) months before withdrawal of TNF α inhibitors.

40/69 (57,97 %) patients did not develop a disease exacerbation and remained in long-term remission off medication—more than 24 months.

Early flares, that is less than 6 months after termination of TNF α inhibitors, were observed in 4/69 (5,8%) patients.

29 (42,03%) patients restarted TNF α inhibitors after exacerbation, due to lack of improvement after no biological DMARDs. All patients in whom TNF α inhibitors were reintitiated responded satisfactorily.

Conclusion: Among patients with JIA in whom TNF α inhibitors were discontinued after inactive disease was achieved, 57,97 % had disease in clinical remission more than 24 months after stopping anti-TNF α therapy. No association was observed between the duration of inactive disease prior to TNF α inhibitors cessation and the time to disease relapse. In addition, we also observed no correlation between the risk of flare and the length of anti-TNF α therapy after inactive disease was achieved. In our population, TNF α antagonists were withdrawn a median of 38 (4-107) months after inactive disease was achieved. Data from our experience with anti-TNF α agents in the treatment of JIA suggest that 57,97 % of patients can be successfully withdrawn from TNF α antagonists for at least 24 months.

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