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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Disclosure of Interests: None declared.

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POS0084 SEPTIC ARTHRITIS IN CHILDREN. A LONGITUDINAL POPULATION-BASED STUDY IN WESTERN AUSTRALIA

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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 looking back (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<3 years of age) had at least one admission for SA. AIR was 9.85 (CI 4.79-14.4) overall with higher rates in males (11.9/7 vs 5.0/1) 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 readmission 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 (CCl0.0, 34.6 vs 272%, p=0.02) including diabetes (4.2% v 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 (10.5, 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 (subclinical) comorbidity may thus be a contributing factor to SA development and to the increased mortality risk in female SA patients.

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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

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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 (TNFα) inhibitors; on this therapy patients can achieve clinically inactive disease and long-term remission.

Currently, little is known about when or how to stop TNFα 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 TNFα inhibitors in patients with non-systemic juvenile idiopathic arthritis (JIA).

Methods: A total of 393 patients with JIA who were treated with TNFα 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, 0.8mg per kg of body weight per dose, with adalimumum 24mg/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 TNFα 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 = 1). The clinical subtypes of JIA were RF-negative polyarticular JIA -28 (40.58%) oligoarthritis—38 (55.07%), enthesitis-related arthritis—3 (4.35%). TNFα inhibitors were started after a mean 46.43 (range 1–144) months of disease. The mean duration of therapy with TNFα 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 TNFα inhibitors. 49/69 (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 stopping anti-TNFα therapy, were observed in 4/69 (5.8%) patients.

29 (42.03%) patients restarted TNFα inhibitors after exacerbation, due to lack of improvement after no biological DMARDs. All patients in whom TNFα inhibitors were reinitiated responded satisfactorily.

Conclusion: Among patients with JIA in whom TNFα inhibitors were discontinued after inactive disease was achieved, 57.97% had disease in clinical remission more than 24 months after stopping anti-TNFα therapy. No association was observed between the duration of remission prior to TNFα 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-TNFα therapy after inactive disease was achieved. In our population, TNFα antagonists were withdrawn within a median of 38 (4-107) months after inactive disease was achieved. Data from our experience with anti-TNFα agents in the treatment of JIA suggest that 57.97% of patients can be successfully withdrawn from TNFα antagonists for at least 24 months.

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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.