

# Review of corticosteroid induction protocols used for children with a new diagnosis of polyarticular course juvenile idiopathic arthritis (pJIA) in an East of England Tertiary Rheumatology Service

## Background

Children with pJIA present with five or more joints affected by pain, swelling and stiffness. Untreated, joint inflammation can lead to irreversible joint damage and disability. Corticosteroids have been used for treatment of JIA since the 1950s. Current clinical practice for treatment of pJIA involves high-dose corticosteroids for a limited period in order to decrease inflammation. The aim is to induce remission whilst systemic treatment, commenced alongside corticosteroids begins to work. No standardised, evidence-based approach currently exists to guide corticosteroid induction regimens in pJIA.

## Methodology

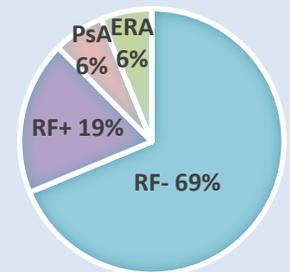
Retrospective chart review of children newly diagnosed with pJIA, January 2019 to December 2020, inclusive. Demographic data collected and steroid regimens documented. Disease activity recorded pre-instigation of corticosteroids, and then at a follow-up appointment on average 5.5 weeks into treatment (range 3-12 weeks). A modified JADAS-27 (27-joint juvenile arthritis disease activity score) was created using active joint count (AJC), CRP and ESR, as physician and patient/parent global assessment scores were missing from the majority of charts. Total score achievable using modified JADAS-27 (mJADAS-27) was 47.

## Objectives

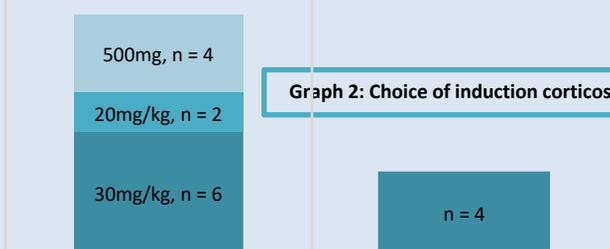
- To describe corticosteroid regimens in children newly diagnosed with pJIA.
- To compare disease activity at diagnosis, with follow-up review after treatment with corticosteroids.

## Results

Sixteen-children were diagnosed with a polyarticular course JIA (pJIA) between January 2019 and December 2020, (male = 5, 15%). Eleven-children (69%) had polyarticular RF-negative JIA (RF-), three (19%) polyarticular RF-positive JIA (RF+), one (6%) Psoriatic-JIA (PsA) and one (6%) HLA-B27 positive enthesitis-related arthritis (ERA) (**graph 1**). All children were commenced on non-steroidal anti-inflammatory drugs (Naproxen, n=11; Ibuprofen, n=5) and subcutaneous Methotrexate (15mg/m<sup>2</sup>) alongside corticosteroids.



Graph 1: ILAR classification of cohort



Graph 2: Choice of induction corticosteroids

### IV METHYLPREDNISOLONE PO PREDNISOLONE

Finally, median mJADAS-27 pre-corticosteroids was 19.4 (5-43.5). Follow-up mJADAS-27 was calculated about 5.5 weeks (3-12) into corticosteroid treatment. Median follow-up mJADAS-27 was 3.5 (0-8). On average, mJADAS-27 improved by 81% (0-100%) following corticosteroids. Of the four children that did not receive ivMP, one child with RF- had 100% improvement in mJADAS-27, the other experienced no improvement. The child with PsA experienced 86% improvement; the child with ERA, only a 20% improvement.

A three-day course of intravenous methylprednisolone (ivMP) was the initial corticosteroid of choice in 12/16 (75%) children. Median AJC of these children was 18 (range 8-27). Six children were given a dose of 30mg/kg (maximum 1 gram), two children 20mg/kg, and four 500mg (weight 30.9-44.2 kg; two of these children were on oral prednisolone (POPred) prior to admission for ivMP; one child had T1DM) (**graph 2**). Following 3-days of ivMP, all 12 children were commenced on POPred. Six children (50%) at a dose of 0.5-1mg/kg with a 6-week weaning plan. The remaining six children were started on a relatively low dose ( $\leq 0.2$ mg/kg) and advised to continue until review.

The children that did not receive ivMP were commenced on POPred at a dose of 0.5-1mg/kg, with an initial weaning plan of 5mg/week. Two of these children had RF-, one had ERA and the other PsA. Median AJC was 9 (range 5-12).

## Conclusion

Corticosteroids lead to improved disease activity in children with pJIA. However, treatment regimens employed vary. Development of a standard operating procedure for corticosteroid induction in pJIA is required. Longitudinal studies would enable evidence-based development of such protocols, and should consider optimal corticosteroid route of administration and dose to achieve maximal benefit, whilst minimising corticosteroid toxicity.