Objectives To ascertain whether clinicians have adhered to the new guidance on offering thyroid function testing as per the Down Syndrome Medical Interest Group guidance.

Methods A retrospective notes review was undertaken of all infants with Down syndrome referred to the three child development centres in Leeds during the period February 2020 – February 2021.

Results Electronic case notes of 13 babies with Down syndrome were reviewed. Six infants were offered thyroid function testing between 4–6 months of life. Although two babies had insufficient samples and so did not get a result. At the time of auditing these babies were 26 weeks and 49 weeks old and yet to have repeat blood sampling.

One infant was tested at 7 months and 1 infant was tested at 8 months.

Three infants are yet to have thyroid function tests and are more than six months of life. One infant is only three weeks old and too young to be offered testing but this has been arranged to be taken at 4 months of life.

One infant (male) had thyroid function testing at 21 days of life due to abnormal TSH on newborn screening and has subsequently been diagnosed with congenital hypothyroidism.

One infant had inappropriate thyroid function tests sent at 10 days of life whilst on the neonatal unit.

One baby had a TSH of 6.4 with a normal T4 and the decision was made to repeat the samples in 3 months’ time and not within 1–5 days as per recommended guidance.

Four babies had blood spot TSH (including the two babies with insufficient samples) and 4 babies had venous samples sent.

All babies were offered newborn blood spot screening.

The parents of ten babies were informed on thyroid dysfunction in Down syndrome and the importance of monitoring thyroid function in the initial clinic by the clinician. For the remaining 3 babies there is no documented evidence that this information was conveyed.

Conclusions We can do better in terms of timely offering the initial surveillance testing at 4–6 months of life and following up promptly on arranging repeat testing if there are sampling issues.

We must continue to educate parents on thyroid dysfunction in Down syndrome ensuring they are aware of key signs and symptoms.

British Society for Rheumatology

1753 REVIEW OF CORTICOSTEROID INDUCTION PROTOCOLS USED FOR CHILDREN WITH A NEW DIAGNOSIS OF POLYARTICULAR COURSE JIA (PJIA) IN AN EAST OF ENGLAND RHEUMATOLOGY SERVICE

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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.

Objectives
1. Describe corticosteroid regimens in children newly diagnosed with pJIA.
2. Compare disease activity at diagnosis, with follow-up review after treatment with corticosteroids.

Methods 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). Modified JADAS-27 created using active joint count (AJC), C-reactive protein (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) =47.

Results Sixteen children were diagnosed with pJIA between January 2019 and December 2020, (male = 5, 31%). Eleven children (69%) had polyarticular RF-negative JIA (RF-), 3 (19%) polyarticular RF-positive JIA (RF+), 1 (6%) Psoriatic-JIA (PsA) and 1 (6%) HLA-B27 positive enthesitis-related arthritis (ERA). All children were commenced on non-steroidal anti-inflammatory drugs (Naproxen, n=11; Ibuprofen, n=5) and subcutaneous Methotrexate (15mg/m2) alongside corticosteroids.

A three-day course of intravenous methylprednisolone (ivMP) was the initial corticosteroid of choice in 12/16 (75%) children. Six children were given a dose of 30mg/kg (maximum 1gram), two children 20mg/kg and four 500mg (weight 30.9–44.2 kg; two children were on oral prednisolone (POPred) prior to admission for ivMP; one child had T1DM). Following 3-days of ivMP, all 12 children were commenced on POPred.

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. AJC ranged from 5–12.

Starting dose of POPred following 3-days of ivMP ranged from 7.5–40mg, maximum dose 1mg/kg. Weaning instructions varied from 5mg/week (n=6), 2.5mg/week (n=4) or stay on low dose (<0.25mg/kg) until review (n=6).

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 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.

Conclusions 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.