The commonest symptoms were reduced GCS (20/31), respiratory depression (11/31), seizures (10/31) and tachycardia (8/31). Most commonly, cases required 3–5 different managements. These were: continuous oximetry PLUS oxygen PLUS ECG monitoring (68%), GCS <12 AND frequent GCS monitoring (68%), cardiovascular monitoring (65%), and invasive ventilation (48%).

Conclusions Despite relatively small numbers, our study shows that accidental poisoning is a preventable condition. Significant poisoning can cause serious symptoms requiring various treatments, but rarely results in death. Our study demonstrates that adult medications that are currently not subject to child resistant packaging laws are causing significant harm to children. This highlights that further legislation is necessary on all medications to prevent harm and even death in children. In all ages, a large proportion of episodes involved illicit drug use suggesting further public health medicine and drug safety campaigns are required to educate both young people and also families around the risks and dangers of drug use.

Down Syndrome Medical Interest Group

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PROFILE OF THYROID DISORDERS IN CHILDREN WITH DOWN SYNDROME

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Background The prevalence of thyroid disorders in children with Down syndrome is 6–10%. Guidelines on thyroid disorders in children and young people with Down syndrome: surveillance and when to initiate treatment in April 2020 were published by the Down Syndrome Medical Interest Group U.K & Ireland in 2020. The spectrum of thyroid disorders in children with Down syndrome includes congenital hypothyroidism and autoimmune thyroid disorders.

Objectives We undertook a retrospective study to review the profile of thyroid disorders of children with Down syndrome currently served by our child development centre serving a child population (0–19 years) of 64,500 children.

Methods A retrospective electronic case notes review was undertaken of 69 children with Down syndrome attending the child development centre, to identify children with a diagnosis of thyroid disorder and assess their biochemical and clinical presentation

Results Electronic case notes of 69 children with Down syndrome were reviewed. One infant (male) was diagnosed with congenital hypothyroidism. Six children were diagnosed with autoimmune hypothyroidism. Prevalence rate 8.8 percent. The median age at diagnosis was 6.6 years. The gender ratio was 3 female: 4 male. Two of these children had a borderline TSH for prolonged period before formally receiving a diagnosis of hypothyroidism, this ranged between 8 months and 45 months, the thyroid function was monitored every 6–12 months during this period. At the time of the initial raised TSH levels the TPO antibodies were normal and increasing to 997 and >1300. Two children have free T4 levels above the normal range (21) despite their TSH levels being above the local reference range and good compliance with medication. There was a rise in BMI at the time of diagnosis in six children (data not available for remaining children). Symptoms noted at diagnosis of thyroid disorder were weight gain,

tiredness and sleep disturbance particularly in female patients. None of the children were recorded to have goitre. Two additional children were noted to have persistently raised TSH levels currently undergoing close monitoring, interestingly both these children have a slight rise in their TPO levels but less than 100 and a marginal increase in their BMI at the time of the initial rise in TSH levels, both sets of parents declined repeat serum thyroid testing within 1–5 days as recommended in the updated guidelines. There were no children diagnosed with hyperthyroidism or Graves' disease.

Conclusions Thyroid disorders in children who have Down syndrome appear to follow a more insidious course with borderline or subclinical hypothyroidism being more commonly present than the general population. It is important to closely monitor the thyroid function to prevent additional disability. With the introduction of earlier thyroid surveillance at 4–6 months as per the updated DSMIG guidelines, it is important to undertake large-scale prospective population studies to evaluate the developmental outcomes in children with Down syndrome and subclinical hypothyroidism.

British Paediatric Neurology Association

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IT'S NOT ALWAYS ABOUT HONEY

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Background Infantile Botulism (IB) is a rare, but potentially fatal disorder typified by flaccid paralysis in infants. It is caused by toxins released by gram-positive anaerobic bacteria, *Clostridium botulinum* - soil organisms that exist as spores. These botulinum spores are consumed (directly or through contaminated foods such as honey or home-canned foods) and they colonise the large intestine of infants before releasing the botulinum toxins that bind irreversibly to the neuromuscular junction causing flaccid paralysis. Paralysis especially of the muscles of respiration place the affected infant at risk of death if respiratory support is not given

Objectives To review a case of IB and its management.

Methods Case report.

Results A 6-month-old male infant with no significant past medical, family, and birth history, presented to the Children's Assessment Unit with cough, decreased feeding, and lethargy at the outset of neurologic symptoms, and respiratory distress. Further anamnesis revealed no sick contacts, honey ingestion, recent travels, and indoor pet exposure. His review of systems was negative.

Upon initial assessment the infant's vital signs were normal and there were no notable findings on systemic examination apart from bilateral crackles and wheeze. However, his respiratory status worsened in less than 24 hours after admission, resulting in his mechanical ventilation and PICU admission.

The patient's PICU course was notable for the development of a broad differential consequent of his unexplainable and deteriorating clinical status. The diagnosis of infantile botulism was eventually entertained on the heels of an unremarkable extensive lab and imaging testing. POSITIVE Murine micro-assay for botulinum toxin was confirmatory.

The infant was treated with botulinum immunoglobulin (Baby BIG) on day 13 of his admission with remarkable