CONCLUSION
Chromosomal analysis with adequate cells is crucial in identifying subtypes of DSD. When mosaicism is suspected, a larger number of cells (at least 30) should be analyzed to accurately detect and characterize these conditions.

EP_P036

WHEN WATER BECOMES A FRENEMY: A CASE SERIES ON THIRSTY CHILDREN AND LITERATURE REVIEW

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INTRODUCTION
Polydipsia is defined as excessive thirst causing the consumption of large amounts of fluids, more than 2 liters/m^2/day in children, with consequential polyuria. It is of paramount importance to distinguish between diabetes insipidus (DI) and primary polydipsia as treatment differs, and inappropriate use of desmopressin can be detrimental in patients with primary polydipsia.

CASE
We present 3 children referred to the Paediatric Endocrine Clinic who exhibited a long history of excessive drinking.

Case 1. A 9-year-old male presented with an unquenchable thirst, drinking 6 to 8 L per day that required him to wake up 3-4 times nightly to drink water. A water deprivation test was performed, yielding inconclusive results, hence needed further investigation.

Case 2. A 9-year-old male's excessive drinking during school hours concerned his teachers, prompting an investigation. A subsequent water deprivation test confirmed primary polydipsia.

Case 3. A 2-year-old toddler presented with a progressive history of excessive drinking. Although his water deprivation test showed equivocal findings, his cranial MRI confirmed the diagnosis of central DI.

Fortunately, our patients did not demonstrate any red flags, such as dehydration, visual field loss, recurrent vomiting, headache or altered consciousness. Our school-going patients denied a history of school bullying or truancy. None of the children were on medication and there was no family history of similar symptoms.

CONCLUSION
These cases underscore the importance and limitations of a water deprivation test in diagnosing polydipsia and polyuria in children. Inconclusive results must be interpreted with caution and necessitate further investigation, as baseline clinical and biochemical variables cannot substitute for the water deprivation test.

EP_P037

THYROID CHANGES IN INFANTS OF MOTHERS WITH GRAVES' DISEASE: A CASE SERIES

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INTRODUCTION
Maternal Graves' disease (GD) can affect neonatal thyroid function. Maternal factors such as timing of diagnosis, TSH-receptor Ab (TRAb) titre, anti-thyroid medications and prior radioiodine therapy will affect outcome.

CASE
We describe six infants born to mothers with GD (2 mothers diagnosed before pregnancy and 4 mothers during pregnancy) in Hospital Sultanah Bahiyah in 2023-2024. All mothers had elevated TRAb, from 3.34 IU/L to >40 IU/L, taken at 16-35 weeks of gestation. Five were treated with carbimazole (10-40 mg daily). Four started treatment during pregnancy and one prior to pregnancy. One mother had RAI before pregnancy and her infant had negative TRAb. Two (2/6) neonates had low birth weight and four (4/6) were premature. One neonate had fetal goiter and required elective LSCS via EXIT procedure by paediatric ORL. This neonate's goitre resolved following L-thyroxine initiation and was extubated within 3 days. Four neonates had elevated TRAb ranging 11.2I U/L to 39.5I U/L. Within 1st week, five had hyperthyroidism, of whom, one was symptomatic for moderate tachycardia. Two required low dose carbimazole for 4-6 weeks. The highest fT4 was 61.24 pmol/L. One patient with no thyrotoxicosis initially developed central hypothyroidism by 1-month-old. Of those with initial transient hyperthyroidism, three (3/5) developed central hypothyroidism thereafter requiring L-thyroxine. Two of them (2/3) had transient central hypothyroidism that resolved between 2-month-old and 1-year-7-month-old. By the time of report, three (3/6) infants still require L-thyroxine of whom two (2/3) had central hypothyroidism with prior hyperthyroidism. All these infants have appropriate growth and development during follow-up.

CONCLUSION
It is crucial to ensure mothers with GD have early diagnosis and adequate monitoring during pregnancy to prevent neonatal complications. Infants of maternal GD should be monitored closely and at regular intervals, to detect alteration of thyroid function which is important for brain development.

EP_P038

THE ECLIPSE HAS PASSED

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INTRODUCTION
Primary adrenal insufficiency (PAI) can be misdiagnosed as other life-threatening conditions. Clinical signs of PAI are based on the deficiency of both glucocorticoids and mineralocorticoids. We report an infant with generalised hyperpigmentation and PAI.

CASE
A 10-month-old female, born via spontaneous vaginal delivery, with poor Apgar Score ($1^{1}3^{5}5^{10}$), was admitted for severe hypoxic-ischemic-injury (HIE). She had multiple episodes of seizures and required cooling therapy. Her parents are non-consanguineous. Clinically, she had generalised skin hyperpigmentation and normal female genitalia.

She had severe metabolic acidosis (pH: 6.7, $cHCO_3$: 8.3 mmol/L, lactate: 14.1 mmol/L). She developed an adrenal crisis at day 5 of life with lowest sodium: 125 mmol/L (134-142) and highest potassium: >7 mmol/L (3.5-5.6). The lowest blood glucose was 3.2 mmol/L.

Investigations at day 4 of life revealed low serum cortisol: 37.6 nmol/L (NV: 185-624), detectable testosterone: 0.9 nmol/L, normal 17-hydroxyprogesterone (17-OHP): 1.89 nmol/L (NV: <19.1), inappropriately low aldosterone: <103 pmol/L (NV: 471-4272) with high renin: >550 mU/L (NV: 4.00-89.00). The karyotype was 46 XX and the inborn error of metabolism study was non-diagnostic. Adrenocorticotropic hormone (ACTH) was normal at 5.16 pmol/L (NV: 1.60-13.90) but was done after initial doses of hydrocortisone. Pelvic ultrasonography (USG) showed Mullerian structures and cranialUSG was normal.

She was treated with stress dose of hydrocortisone 2.5 mg *qid* (45 mg/m^2/day), then weaned to oral hydrocortisone 1.5 mg *tid* (16 mg/m^2/day), fludrocortisone 150 mcg *od* and sodium chloride 0.5 grams *bid*. On follow-up, she had markedly reduced skin pigmentation. She had serum renin <1.80 mU/L and normal renal profiles. She was thriving with a weight of 7.4 kg (25-50th percentile), length of 65 cm (5-10th percentile) with appropriate developmental milestones.

CONCLUSION
Early diagnosis is crucial for effective management of PAI. Hyperpigmentation is a pathognomonic physical examination finding because ACTH shares the same affinity with α-melanocyte-stimulating hormone (MSH). Our patient's normal ACTH level was misleading due to hydrocortisone suppression.

EP_P039

WHEN THYROID STIMULATING HORMONE AND FREE THYROXINE MISMATCH: A CASE REPORT

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INTRODUCTION
Thyroid hormone resistance (THR) is characterized by lack of end-organ responsiveness to thyroid hormone with high serum free thyroxine (FT4) with inappropriately high thyroid stimulating hormone (TSH).

CASE
We report a 3-month-old male, born term via spontaneous vaginal delivery was referred to us at day 13 of life for inappropriately high TSH: 7.69 mIU/L (NV: 0.39-7.0) and high FT4: 57.5 mIU/L (NV: 8.7-16.2). His birth weight was 3.5 kg. Antenatally, his mother has no thyroid disorder. He is the only child of non-consanguineous parents.

The cord TSH was 22.01 mIU/L. At day 3 of life, TSH was 15 mIU/L and FT4 was insufficient. At day 23 of life, thyroid function test (TFT) revealed TSH: 6.29 mIU/L, FT4: 47.9 pmol/L and free triiodothyronine (FT3): 6.6 pmol/L (NV: 3.1-10.6). Clinically he had persistent left parieto-occipital swelling and tachycardia. He was started on carbimazole (0.1 mg/kg/day) at 2 months of life.

Thyroid autoantibodies were negative with anti-TSH receptor (TRAb) <0.8 IU/L (NV: <1.75), anti-thyroid peroxidase <9 IU/ml (NV: <35) and antithyroglobulin 14 IU/mL (NV: <115). Pituitary hormones taken on day 76 of life revealed prolactin 1,553.2 mIU/L (NV: 70.81-566.64) with mini puberty (testosterone: 4.3 nmol/L, FSH: 1.8 IU/L