revealed abnormal gonadal architecture in all cases. All intra-abdominal gonads demonstrated streak gonads or dysgenetic testicular tissue.

CONCLUSION

45,X/46,XY mosaicism demonstrates a broad phenotypic spectrum with frequent genital ambiguity, growth impairment, and abnormal gonadal development. The high prevalence of gonadal dysgenesis underscores the importance of careful surveillance and individualized multidisciplinary management guided by clinical, endocrine, and histopathological findings.

EP_P003

HEIGHT VELOCITY AND BODY MASS INDEX CHANGES DURING GONADOTROPIN-RELEASING HORMONE AGONIST THERAPY IN CHILDREN WITH CENTRAL PRECOCIOUS PUBERTY (CPP)

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INTRODUCTION

Precocious puberty is increasingly recognized in children. Effect of gonadotropin-releasing hormone agonist (GnRHa) on height velocity (HV) and body mass index (BMI) during treatment of Central Precocious Puberty (CPP) is not well-documented.

METHODOLOGY

Records of all patients treated with GnRH for CPP who attended the paediatric endocrine clinic over a 5-year period (2021–2025) [N1.1] were reviewed retrospectively for HV and BMI changes for 3 consecutive years. Basal luteinizing hormone levels ≥0.3 IU/L or stimulated levels ≥5 IU/L are considered diagnostic of CPP.

RESULTS

Twenty patients were reviewed and median age at the start of study was 7.8 years (4.3–10.3). Majority were female (n = 18, 90%) with median Tanner Stage 3 and of Malay origin (n = 18, 90%[N2.1]). Median chronological age was 7.8 years (4.3–10.3) with bone age 10.3 years (5.8–13.4).

Causes of CPP were idiopathic (n = 16, 80%), CNS lesion (n = 2, 10%) and poorly controlled congenital adrenal hyperplasia (n = 2, 10%). Nine (82%) of the 11 patients who had MRI brain done had normal findings.

After 1 year of treatment, the median HV SDS was −0.83 (range −2.45 to 2.42), and BMI SDS was 0.45 (−1.94 to 2.15). After 2 years (n = 12), HV SDS declined to −1.3 (−3.44 to 0.69), with BMI SDS of 0.13 (−1.16 to 1.79). After 3 years (n = 9), HV SDS declined further to −1.51 (−3.52 to 3.13), and BMI SDS was 0.16 (−0.12 to 1.25).

After 1 year of treatment, 20% (n = 4) of patients were overweight and 10% (n = 2) were obese. By 3 years, 22% (n = 2) remained overweight, and none were obese.

CONCLUSION

Premature growth plate senescence induced by prior estrogen exposure may lead to HV decline. Although obesity is a risk factor for the onset of CPP, whether GnRHa treatment directly increases BMI remains controversial as our cohort's BMI change reveals otherwise.

EP_P004

A SPECTRUM OF THYROID DYSFUNCTION IN CHILDREN WITH DOWN SYNDROME: A MALAYSIAN TERTIARY CENTRE EXPERIENCE

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INTRODUCTION

Endocrine abnormalities are frequently observed in children with Down syndrome, with thyroid dysfunction being the most common. The distribution and patterns of thyroid disorders vary between populations.

METHODOLOGY

Retrospective data were collected from the electronic medical records of children with Down syndrome who attended the paediatric clinic at University Malaya Medical Centre from the year 2000 to 2025. Data were analyzed to determine the frequency and distribution of thyroid disorders in this cohort.

RESULTS

The study included 57 patients, the majority of whom were identified through routine screening (n = 56, 98.2%). Only one child (1.8%) was diagnosed following a symptomatic presentation of diarrhea. Within the cohort, 51 patients were diagnosed with hypothyroidism and six with hyperthyroidism.

Among those with hypothyroidism, the mean TSH was 22.5 mIU/L, with a median age at presentation of 61 days (IQR: 19–211.5). Notably, 25% of the cohort presented within the