hyperparathyroidism often remits but bone disease progresses. Bisphosphonate therapy such as pamidronic acid has been described in those with recurrent fractures in later life.

EP_P005

CASE REPORT: VAN WYK-GRUMBACH SYNDROME: HYPOTHYROIDISM PRESENTING AS PRECOCIOUS PUBERTY

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INTRODUCTION/BACKGROUND
Acquired causes of hypothyroidism such as Hashimoto's thyroiditis is mostly insidious and often goes undetected, unless there is concomitant thyroid gland enlargement or profound hypothyroid symptoms. Precocious puberty in girls is a rare cause of acquired hypothyroidism.

CASE
We report an 8 year-3-month-old female of Chinese-Indian descent, who presented with precocious puberty (bilateral breast budding, axillary hair, and pubic hair) about 6 months prior, with the mother's concern of poor learning in school. There were no other symptoms of puberty such as vaginal discharge or growth acceleration. She was adopted at 6 months old, thus, there is uncertainty about her biological family history. Her height was at the 25th centile and weight at 75th centile. She had a single café-au-lait spot at the right thigh, with Tanner stage 2 breasts, axillary hair, and pubic hair. She also had mild scoliosis with no other skeletal deformities. She had no thyroid gland enlargement but her facial expression was dull. Her blood investigations revealed pre-pubertal levels of gonadotrophins with undetectable estradiol, normal prolactin and negative b-HCG screen. Her thyroid function revealed markedly increased TSH (>100mIU/L) with severely low fT4 (<5.4 pmol/L). Her thyroid peroxidase antibody (anti-TPO) level was 131 IU/ml (<35). Ultrasound of the thyroid showed features in keeping with autoimmune thyroiditis with incidental thyroglossal duct cyst. Upon further questioning, the mother did recall prominent neck swelling since the past 2 years. Following L-thyroxine initiation, her thyroid function normalized and she showed significant improvement in height (she grew 10 cm/year) with progression of puberty. Her last bone age was 10 years old (CA: 9 year and 6 months).

CONCLUSION
Van Wyk-Grumbach syndrome is a relatively uncommon cause of pseudo-precocious puberty that often skips detection. Thyroid assessment is recommended in a girl presenting with precocious puberty, even in the absence of goiter. Timely diagnosis and treatment with L-thyroxine normalizes thyroid function and significantly improves linear growth.

EP_P006

CASE REPORT: EXOGENOUS CUSHING SYNDROME IN A GROWING CHILD FOLLOWING CHRONIC TOPICAL STEROIDS FOR FAMILIAL PSORIASIS

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INTRODUCTION/BACKGROUND
Cushing syndrome is relatively uncommon in young children but exogenous Cushing syndrome is increasingly seen due to both prescribed and surreptitious intake of steroids.

CASE
We report a 6 year-10-month-old female, referred from Dermatology for obesity. She had itchy and scaly red plaques about 1 year prior. Both her mother and elder sister had been diagnosed with psoriasis and treated elsewhere. Even without a doctor's advice, she was given over-the-counter topical betamethasone dipropionate by her mother 1 year before consult. The mother applied the steroid generously over the child's whole body including the face and inguinal region twice a day for every flare, which usually occurred around 2 episodes every month. Since then, her daughter gained weight and stopped growing. She had florid Cushingoid features with emotional lability (easily tearing), truncal obesity, thick violaceous purple striae over the trunk, neck, upper, and lower limbs, and extensive erythematous scaly psoriatic plaques (BSA~80%). She was hypertensive with blood pressures ranging from 130-150/88-100 mmHg. She did not have proximal muscle weakness. Her eye assessment was negative for glaucoma or cataract, or hypertensive retinopathy changes. She had a pre-pubertal Tanner stage with no virilisation or hirsutism. She had persistently suppressed 8am cortisol level (<27.6 nmol/L) and low ACTH level (1.10 pmol/L, reference range 1.6-13.9) with normal 17OHP and DHEAS screen. She had borderline HbA1c (5.7%), dyslipidaemia, and non-alcoholic fatty liver disease based on an abdominal ultrasound. Her topical steroids for psoriasis were stepped down and she was initiated on steroid-sparing UV

phototherapy in the ward. Her BP stabilized. She was given physiologic oral hydrocortisone replacement (6 mg/m^2/day). Six months later, her weight reduced with resumption of linear growth, and improved metabolic control.

CONCLUSION

Exogenous Cushing syndrome resulting from topical medications has been described well especially among young infants. Potent topical steroids particularly for young children should ideally be administered with doctor's prescription.

EP_P007

STEROID-RESPONSIVE ENCEPHALOPATHY ASSOCIATED WITH AUTOIMMUNE THYROIDITIS: THE OUTCOME OF NEUROLOGICAL AND THYROID STATUS

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INTRODUCTION/BACKGROUND

Steroid-Responsive Encephalopathy Associated with Autoimmune Thyroiditis (SREAT), also termed as Hashimoto's Encephalopathy is a neuroendocrine disorder characterized by a triad of subacute onset of encephalopathy, elevated anti-thyroid antibodies, and neurological improvement following steroid therapy. It is a rare but more likely under-diagnosed condition in patients presenting with encephalopathy.

CASE

A 7-year-old Chinese female was referred for headache, seizures, and mood changes for 2 months. She was found to have hyperthyroidism when she presented with frequent hunger and weight loss 4 months ago. She was started on carbimazole 2.5 mg twice a day and she became euthyroid clinically and biochemically. She was otherwise a brilliant child with no other medical illness. She had no family history of thyroid or autoimmune disorders. Examination revealed an irritable child with upper motor neuron signs. Her cerebrospinal fluid analysis for viral PCR and neuronal antibodies were negative. Her cranial MRI and EEG were reported as normal. Her thyroid function was normal (TSH 3.65 uIU/mL, T4 8.5 pmol/L). Her thyroid antibody levels were all significantly elevated (thyroid stimulating immunoglobulins 3.1 IU/L, anti-thyroid peroxidase antibodies 731.8 IU/ml, anti-thyroglobulin antibodies 642.9 IU/ml). A diagnosis of SREAT was made. She received intravenous methylprednisolone 30 mg/kg/day for 5 days followed by a course of prednisolone for a month. She made a complete recovery. She remained clinically and biochemically euthyroid without medication. A year later, her symptoms recurred, and she was treated similarly as her previous presentation. She recovered but had persistent seizures and needed anti-seizure medication. Her seizure frequency was once a month until 4 years later wherein her seizure frequency increased to weekly without other symptoms. She was given a course of oral prednisolone for 3 months. She was seizure-free for 2 months before her seizures resumed monthly. She was also found to have hypothyroidism (TSH 28.69 uIU/ml, T4 6.65 pmol/L) on screening. She was started on L-thyroxine and became euthyroid after 2 months of treatment.

CONCLUSION

This case report illustrates that epilepsy is a clinical sequela of SREAT despite being a steroid-responsive condition. Thyroid status does not determine seizure control; hence it reflects an association rather than causation of the encephalopathy.

EP_P008

TRIPLE-A SYNDROME: A RARE PRESENTATION OF ADRENAL INSUFFICIENCY

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INTRODUCTION/BACKGROUND

Triple-A syndrome or Allgrove syndrome is a rare autosomal recessive congenital disorder. It is characterized by Addisonianism, achalasia and alacrima. It is a progressive disorder that can take years to develop the full-blown clinical picture.

CASE

We report 2 individuals with Triple-A syndrome who initially presented with recurrent hypoglycemic seizures at about 4 years old. They also had faltering growth with short stature. Both had significant hyperpigmentation, without ambiguous genitalia or neurological abnormality. Hormonal assay confirmed glucocorticoid deficiency, with sparing of mineralocorticoid involvement. Both were subsequently started on hydrocortisone replacement.