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

https://doi.org/10.15605/jafes.038.S2.136

Jen Ern Tan,[1] Joyce Soo Synn Hong,[2] Sau Wei Wong[1]
[1]*Neurology Unit, Department of Paediatrics, Hospital Pakar Kanak-Kanak, Universiti Kebangsaan Malaysia*
[2]*Endocrinology Unit, Department of Paediatrics, Hospital Pakar Kanak-Kanak, Universiti Kebangsaan Malaysia*

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

https://doi.org/10.15605/jafes.038.S2.137

Muhamad Ideris Othman,[1] Song Hai Lim,[1] Janaeni Vethaguru[2]
[1]*Department of Paediatrics, Sabah Women and Children Hospital, Kota Kinabalu, Malaysia*
[2]*Department of Paediatrics, Hospital Duchess of Kent, Sandakan, Malaysia*

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.