deemed to be likely dystrophic calcification. During her stay on the medical floors, patient was found to be unresponsive and hypotensive, after a bout of agitation. She had to be urgently intubated and started on stress doses of steroids (Hydrocortisone 100 mg every 8 hours) and was upgraded to the Intensive Care Unit (ICU). Patient was eventually successfully weaned off the ventilator and steroid doses were slowly tapered. During her hospital course she was noted to have gradually decreasing Calcium levels, down to a corrected Calcium level of 7.6. Further workup for the hypocalcemia revealed a Vitamin D Level of 12, and Parathyroid Hormone (PTH) level of 0. Patient was subsequently started on adequate Calcium and Vitamin D supplementation for the same. After a few days when the family was located and contacted through social work support, and a more thorough history was obtained, it was found that one out of the patient's three sisters had a similar constellation of deficiencies. Patient had previously been diagnosed with a polyendocrine syndrome, however she was irregular with her medication compliance and follow-up outpatient with her endocrinologist due to her persistent psychiatric issues and poor social support.
APS-1 is an autosomal recessive disorder caused by mutation in AIRE, the autoimmune regulator gene which is hypothesized to be playing an important role in the generation of regulatory T cells. Although the complete pathogenesis is unclear, mutation in generation of these regulatory cells leads to autoantibody formation. Hypoparathyroidism or Chronic persistent Mucocutaneous Candidiasis is usually the first manifestation seen during adolescence and adrenal insufficiency usually manifests later. A variation of other autoimmune syndromes can be observed, with Hypothyroidism, Type-1 Diabetes Mellitus and Primary Hypogonadism being a few of them. Treatment primarily involves replenishment of the hormones of the underperforming gland. Management of a complex syndrome like APS-1 in a patient with psychiatric disabilities can be challenging and needs a multi-disciplinary approach involving the endocrinologist, the primary care physician and the psychiatrist.

Thyroid

THYROID NEOPLASIA AND CANCER

SPOCK1 Promotes the Progression of Papillary Thyroid Cancer ViaPI3K/Akt Signaling Activation

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With the increasing incidence, thyroid cancer as one of the most common malignancy, has got widespread attention during the past few years. Papillary thyroid cancer (PTC) is the most common thyroid cancer type. Understanding the underlining molecular mechanisms of PTC is of great interest. The oncogenic role of SPARC/osteonectin, cwcv, and kazal-like domain proteoglycan 1 (SPOCK1) has been demonstrated in several cancers, however, the clinical and functional significance of SPOCK1 in PTC are largely unknown. Here, we found that the expression of SPOCK1 was upregulated in PTC tissues when comparing with the adjacent normal thyroid tissues. The overexpression of SPOCK1 was associated with the clinicopathological characteristics of the patients with PTC. We demonstrated that the proliferation of PTC cells was significantly promoted and the apoptosis of PTC cells was significantly inhibited in cells with overexpression of SPOCK1. Furthermore, we showed that knockdown of SPOCK1 arrested the cell cycle in G0/G1 phase and promoted the apoptosis in PTC cell lines. Importantly, our data suggested that SPOCK1 promoted the progression of PTC cell via regulating the PI3K/Akt signaling pathway. Taking together, our findings demonstrate that SPOCK1 enhances the activation of PI3K/Akt signaling pathway, thereby promoting the proliferation and inhibiting the apoptosis of PTC cells.

Pediatric Endocrinology

PEDIATRIC GROWTH AND ADRENAL DISORDERS

Height Improvement with Recombinant Human Growth Hormone Therapy at Terminal Stage of Growth

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SAT-102

Recombinant growth hormone therapy (rhGH) is known to improve the final adult height in various conditions. The effectiveness of treatment depends on the status of patients, but it is generally known that the younger age the treatment starts, the better effects obtained. The authors experienced that final adult height was dramatically improved with rhGH at the very end stage of deceleration period of growth, so we would like to share our unusual experience. A boy of 13 years and 11 months visited our clinic with cessation of growth. He was 163cm (54%) tall and 62.6kg (78%), and showed Tanner stage IV-V development. There have been no unusual findings in past history and family history. His Mid Parental Height was calculated as 170.5cm (Father: 172cm, Mother: 156cm). After experiencing an acute growth acceleration around 20cm over the past two years, growth rate was abruptly decreased, with 1cm growth in the last nine months. Bone age was 17 years by TW3 method, and pelvic AP x-ray showed Risser stage 4, which is compatible to 17 years in boy. As a result of the above findings, additional height gain was expected to less than 1-2cm and medical intervention for more height gain was expected to impossible. However, the patient and his parents strongly requested rhGH treatment. So, we decided to try rhGH treatment for only three months and then discuss whether or not to extend the treatment after checking up the result. Follow up was conducted every three months, and the height of the patient continued to grow and the closure of the remaining growth plates was confirmed to be delayed. During the one-and-a-half year treatment, no side effects appeared in patient, reaching 173 centimeters, the equivalent of the Korean male standard height. In this case, we experience that the use of Growth hormone can improve the final adult height significantly, even in the end stage of growth, without any other intervention. So, we may consider attempting limitedly to treat growth hormones for