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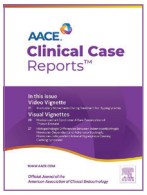

Editorial

Editorial for September/October Issue of AACE Clinical Case Reports

Dear Colleagues,

Welcome to another issue of *AACE Clinical Case Reports* (ACCR)! The current issue includes interesting and educational cases to share. We will provide a summary of some of those cases below. For more details, please access ACCR online journal available at https://www.aaceclinicalcasereports.com/

Under the Pituitary-Gonadal-Adrenal Access in this issue, a rare case of ectopic Cushing reported a presentation that featured excess cortisol and elevated androstenedione levels despite normal dehydroepiandrosterone sulfate and testosterone in a 59-year-old woman and reviewed challenges in diagnosis.[1]

Another report discussed the dramatic and favorable clinical and biochemical response of functioning mesenteric paraganglioma, presented with hypertensive crisis to octreotide treatment and proposed consideration of this treatment option.[2]

A case reviewed unique presentation of pituitary stalk duplication with a single pituitary gland, a very rare entity, in a pediatric patient, highlighting the need for proper evaluation for hypopituitarism, particularly the growth hormone and gonadotrophins deficiency, and also screen for associated neurologic and ocular abnormalities.[3]

On Diabetes, Lipids, and Metabolism, we share a clinical case highlighting the importance of considering insulin autoimmune syndrome (IAS), a rare cause of hypoglycemia, in patients with recurrent hypoglycemia if no clear explanation is found and in the absence of apparent risk factors.[4]

Another case highlighted the challenges in treating cystic fibrosis-related diabetes and proposed some tips.[5]

An interesting visual vignette described a skin rash seen in patient with type 1 diabetes mellitus due to elevated triglyceride levels.[6] You can probably figure what the rash might be?

In the field of Thyroid Disease, a unique case of thyroid cancer who developed immune checkpoint inhibitors (CPI)-induced thyroiditis during treatment for a non-thyroid malignancy and had a subsequent regression of a coexisting untreated primary papillary thyroid cancer, suggesting CPI-induced thyroiditis, an immune response against thyroid tissue, may also reflect increased immune response against thyroid cancer cells leading to tumor regression.[7]

An interesting Video Vignette describes the rare neurological presentation of chorea in hyperthyroid young patient.[8]

In the area of Bone and Calcium Disorders, Primary hyperphosphatemic tumoral calcinosis has been shown to be caused by pathogenic variants in the genes encoding FGF23, GALNT3, and KLOTHO. A case described massive tumoral calcinosis associated with phosphatonin resistance due to heterozygous alterations in the sterile alfa motif domain-containing protein-9 gene (SAMD9), alfa 2-Heremans-Schmid glycoprotein gene (AHSG), FSHD region gene 2-family member-C gene (FRG2C), and fibroblast growth factor receptor-4 gene (FGFR4).

The case highlights that the simultaneous occurrence of alterations in several genes critical in phosphate homeostasis may trigger massive tumoral calcinosis despite their heterozygosity.[9]

On related disease entity, a Visual Vignette describes imaging features of familial hyperphosphatemic tumoral calcinosis, an autosomal recessive disorder presented with hyperphosphatemia because of increase in proximal tubular phosphate reabsorption.[10]

Another interesting case described the potential benefit of Denosumab, a monoclonal antibody that inhibits bone resorption, in treating symptomatic Paget disease of the skull with symptoms improvement.[11]

As always, I truly appreciate all contributing authors, reviewers, editors, and staff that help improve our journal and create an educational platform to our readers to help best manage our patients. We welcome your educational cases.

Thank you again for your interest in ACCR. I welcome all feedback, questions, and comments from our readers. Please feel free to reach us at publications@aace.com.

Warmest regards,

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