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exogenous steroid use but had successfully discontinued oral hydrocortisone after an adequate Synacthen test response 2 years ago. Four days after starting valsartan, she presented to the Emergency Department (ED) with dizziness and vomiting. Laboratory results revealed severe hypotonic hyponatremia (serum sodium 110 mmol/L, serum osmolality 259 mOsm/kg, urine osmolality 247 mOsm/kg, urine Na 71 mmol/L) and hyperkalemia (serum potassium 7.0 mmol/L). In the ED, she was given a lytic cocktail and 150 cc of 3% saline. Prior to starting valsartan, her serum sodium at the PC clinic was 135 mmol/L. She denied using any over-the-counter or traditional medications. Her blood pressure and blood glucose levels were normal throughout hospitalization, making adrenal insufficiency less likely. Further investigations, including morning serum cortisol (500.4 nmol/L) and TSH (0.54 mIU/L, NR 0.4-4.0 mIU/L), were normal. Thus, the diagnosis of severe hypotonic hyponatremia secondary to valsartan was made. After withholding valsartan, her symptoms resolved, the serum sodium and potassium normalized, and she was discharged well 4 days later.

CONCLUSION

ARBs can lead to severe hyponatremia by blocking the angiotensin II receptor, which inhibits renal tubular sodium reabsorption. This effect is particularly pronounced in the elderly and individuals on concomitant thiazide therapy. Although rare, ARB-associated hyponatremia should be considered in patients with hypotonic hyponatremia when other causes have been ruled out.

EP_A052

A SILENT THREAT: LARYNGEAL INVOLVEMENT IN PAGET'S DISEASE LEADING TO AIRWAY COMPROMISE

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INTRODUCTION/BACKGROUND

Paget's disease of bone (PDB) is a chronic skeletal disorder characterized by disorganized bone remodelling, often affecting the skull, spine, pelvis and long bones. While complications such as fractures, arthritis and hearing loss are well-documented, laryngeal involvement leading to acute airway obstruction is exceptionally rare. To our knowledge, no previous case reports have described PDB affecting the thyroid and arytenoid cartilages, resulting in airway compromise.

CASE

A 45-year-old male with hypertension and eczema was diagnosed with a variant of PDB (normal alkaline phosphatase) in 2018, following an evaluation for right knee and ankle pain that began in 2016. Extensive investigations, including a bone biopsy, revealed nonspecific sclerosis, normal ALP, and mildly elevated bone formation markers (P1NP). A Tc-99m MDP bone scan showed multiple hot spots involving the skull, clavicles, ribs, L5, right elbow, both knees and both ankles. He was initiated on yearly intravenous zoledronate (4 mg). In 2022, he sustained a low-impact distal third right ulna fracture, necessitating a locking plate. The fracture site biopsy confirmed Paget's disease.

In 2024, he presented with acute upper airway obstruction. A CT neck scan revealed expansile lytic lesions involving thyroid and arytenoid cartilages, causing significant airway narrowing. An emergency tracheostomy was performed to secure his airway. A repeated Tc-99m MDP bone scan demonstrated disease progression, with worsening involvement of the thyroid and cricoid cartilages.

CONCLUSION

This case highlights a rare and potentially fatal complication of PDB, with airway obstruction due to expansile lytic lesions of the laryngeal cartilages. Given the absence of prior reports on this manifestation, clinicians should remain vigilant for atypical presentations of PDB, particularly in patients with progressive disease. Early diagnosis and intervention are critical to preventing life-threatening outcomes.

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MARINE-LENHART SYNDROME: A RARE CASE OF AUTOIMMUNE HYPERTHYROIDISM AND FUNCTIONAL THYROID NODULE

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INTRODUCTION/BACKGROUND

Marine-Lenhart syndrome is a rare thyroid disorder characterized by the presence of Graves' disease and autonomously functioning thyroid nodules. This dual

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pathology poses a diagnostic and therapeutic challenge as it combines features of autoimmune hyperthyroidism and toxic multinodular goiter. The prevalence is estimated to be 0.8-4.1% among patients with Graves' disease. Patients with Marine-Lenhart Syndrome generally have lower remission rates with thionamide therapy, unlike typical Graves' disease, and frequently require definitive treatment such as radioiodine ablation or total thyroidectomy.

CASE

A 31-year-old female presented with persistent tremors, palpitations, heat intolerance and an unintended 5 kg weight loss over three months. She also reported progressive bilateral eye bulging over the past year. Upon physical examination, tachycardia, fine tremors, a diffusely enlarged thyroid gland with palpable nodules, and mild exophthalmos with lid lag were observed. Laboratory evaluations revealed suppressed thyroid-stimulating hormone level (<0.01 mIU/L; reference range 0.35-4.94) and elevated free thyroxine level (>64.35 pmol/L; reference range 0.70-1.48). The thyroid receptor antibody level was significantly elevated at 11.1 U/L, indicating Graves' disease. Thyroid ultrasound showed a diffusely enlarged, hyperplastic gland with multiple mixed cystic and solid nodules bilateral (TI-RADS 1) and solid nodule size 1 x 0.9 x 0.8 cm in the right thyroid (TI-RADS 3). Thyroid scintigraphy demonstrated diffusely increased uptake (35.3%; normal 1-5%) with multiple hot nodules. The presence of autoimmune hyperthyroidism alongside functioning nodules confirmed the diagnosis of Marine-Lenhart syndrome. She was initially treated with thiamazole, propranolol, selenium and ocular lubricants. She underwent radioiodine ablation as definitive treatment.

CONCLUSION

This case highlights the importance of considering a diagnosis of Marine-Lenhart syndrome in patients presenting with hyperthyroidism and thyroid nodules. Delay or failure to recognize it may lead to misdiagnosis and inadequate treatment, potentially extending symptoms and increasing the risk of complications. A comprehensive clinical, biochemical and imaging examination is needed for accurate diagnosis and proper management.

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BEYOND OSMOTIC DIURESIS: DIAGNOSING ARGININE VASOPRESSIN DEFICIENCY (AVP-D) IN A PATIENT WITH UNCONTROLLED DIABETES

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INTRODUCTION/BACKGROUND

Polyuria and polydipsia in patients with poorly controlled diabetes mellitus are often attributed to osmotic diuresis. However, concurrent (AVP-D) is a rare but critical differential diagnosis that requires careful evaluation.

CASE

A 57-year-old female with hypertension and poorly controlled diabetes mellitus, with HbA1c 10.6%, presented with polyuria, polydipsia and significant weight loss. She had no history of fever, infective symptoms, head surgery or head trauma. She denied any pertinent family history. There was no evidence of hyper- or hypopituitarism symptom-wise. She was hemodynamically stable, and systemic examinations were unremarkable. Her initial investigations showed sodium 138 mmol/L, potassium 4.1 mmol/L, creatinine 56 umol/L, random blood sugar 18.3 mmol/L, corrected calcium 2.5 mmol/L and phosphate 1.15 mmol/L. Chest X-ray, KUB ultrasound and brain CT were unremarkable. In the ward, the patient was commenced on insulin therapy to optimize blood glucose control. Nevertheless, despite controlling the blood glucose, she had persistent polyuria up to 9L/day, with hypernatraemia 149 mmol/L and low urine SG 1.000, even with urine glucose 4+. Hence, modified water deprivation test was performed, revealing an inappropriately low urine osmolality of 97 mOsm/kg despite elevated plasma osmolality of 319 mOsm/kg, with a significant increase of urine osmolality to 426 mOsm/kg post-desmopressin, confirming AVP-D. Pituitary MRI showed a normal posterior pituitary bright spot without structural abnormalities. The patient was initiated on oral desmopressin, which resulted in marked improvement in clinical symptoms. Her pituitary hormonal assessment showed FSH 42.4 IU/L, LH 24.7 IU/L, estradiol 96.0 pmol/L, TSH 0.54 mIU/L, FT4 12.46 pmol/L, AM cortisol 217 nmol/L, prolactin 93.3 uIU/mL. Her tumor markers, beta HCG, Mantoux test and anti-TPO antibodies were negative, which would be mostly idiopathic AVP-D.

CONCLUSION

AVP-D should be considered in patients with diabetes who have persistent polyuria and polydipsia despite glucose