EP_A106

A MULTIMODAL APPROACH USING CALCITONIN, DENOSUMAB, AND HEMODIALYSIS FOR THE MANAGEMENT OF REFRACTORY HYPERCALCEMIA IN MALIGNANCY

Nur Haziqah Baharum[1], Mohd Hazriq A.[1], Aimi Fadilah M.[1], Nur Aini Eddy Warman[1], Fatimah Zaherah MS[1], Rohana A.G.[1]
[1]Endocrine Unit, Faculty of Medicine, University Technology MARA (UiTM)

INTRODUCTION
Severe hypercalcemia is a life-threatening metabolic emergency that necessitates prompt initiation of systemic therapy due to the risk of cardiac arrhythmias. It is frequently linked to squamous cell carcinoma through the production of parathyroid hormone–related protein, which mediates the development of humoral hypercalcemia of malignancy.

CASE
This is a case of a 38-year-old male who was diagnosed 1 year ago with locally advanced poorly differentiated basaloid squamous cell carcinoma of the lower anterior mandibular alveolus involving cortical, medullary bone, and perineural invasion. He underwent extensive tumor resection with reconstruction, tracheostomy, and bilateral neck dissection, followed by multiple revision surgeries for postoperative complications. He completed adjuvant chemoradiotherapy.

He presented with acute confusion without other systemic symptoms. His Glasgow Coma Scale was E4V4M5. Neurological and systemic examinations were unremarkable, and oral cavity assessment showed no evidence of recurrence.

Investigations revealed severe hypercalcemia (5.94 mmol/L) with normal phosphate (1.16 mmol/L) associated with shortened QTc. Other tests were unremarkable, with no evidence of infection, uremia, liver dysfunction, or alternative metabolic causes. Lumbar puncture was unremarkable. His parathyroid hormone level was suppressed at 0.50 pg/mL. Computed tomography brain showed no evidence of meningoencephalitis, hydrocephalus, cerebral oedema, or metastasis. Aggressive hydration was initiated alongside subcutaneous calcitonin, which was subsequently titrated. However, there was no clinical or biochemical improvement after 1 day, with persistent confusion and calcium remaining at 5.87 mmol/L. Subcutaneous denosumab was then administered, and hemodialysis was initiated on alternate days due to refractory hypercalcemia. This resulted in improvement of calcium levels to 3.2–3.6 mmol/L and resolution of confusion.

CONCLUSION
Refractory hypercalcemia may represent a late manifestation of advanced squamous cell carcinoma and is an ominous prognostic indicator, necessitating prompt evaluation and oncologic management.

EP_A107

THE PLACENTA AS A PARATHYROID: PREGNANCY-INDUCED NORMALIZATION OF REFRACTORY POSTSURGICAL HYPOPARATHYROIDISM

Dinehs Rao[1], Siti Sanaa Wan Azman[1], Dorothy Maria Anthony Bernard, Foo Siew Hui[1]
[1]Endocrine Unit, Medical Department, Hospital Selayang

INTRODUCTION
Permanent hypoparathyroidism is a recognized complication of total thyroidectomy, with an estimated incidence of 10.47%. Standard treatment involves supplementation with calcium and activated vitamin D. However, some patients remain refractory to conventional therapy and fail to achieve normocalcemia. During pregnancy, significant physiological changes occur in calcium-regulating hormones. Notably, the placenta increases the production of parathyroid hormone-related peptide (PTHrP), which acts as a calcitropic hormone, mimicking the effects of parathyroid hormone.

CASE
A 38-year-old female with a history of total thyroidectomy 14 years ago for follicular thyroid carcinoma presented with permanent hypoparathyroidism. Her condition was refractory to high-dose replacement therapy, which included calcium carbonate 3 g BD, alfacalcidol 4 mcg ON, calcitriol 1 mcg BD, and cholecalciferol 100,000 IU OD. Despite compliance, her serum calcium levels fluctuated between 1.7 and 1.93 mmol/L, necessitating intermittent intravenous calcium infusions. Laboratory investigations showed a phosphate level of 1.63 mmol/L and 24-hour urine calcium of 4.81 mmol/L. Her thyroid function remained stable on levothyroxine 150 mcg daily (T4: 12.0 pmol/L; thyroid-stimulating hormone: 2.93 mIU/L). While off-label use of teriparatide was being considered, she became pregnant. As the pregnancy progressed, her calcium levels stabilized. Calcitriol was discontinued at 26 weeks' gestation when her calcium reached 2.5 mmol/L. By 30 weeks, the alfacalcidol dose was reduced to 3.5 mcg daily, with cholecalciferol dosing reduced to 50,000 IU OD. Her corrected calcium levels remained between