causes have also been excluded. Intact parathyroid hormone (iPTH) yielded a low result of 1.61 pmol/L (1.95-8.49 pmol/L). Overall features point to non-iPTH dependent hypercalcemia. Corticosteroid therapy with IV hydrocortisone 50 mg TDS together with subcutaneous calcitonin 100 iU TDS were initiated. Bisphosphonate therapy consisting of one dose of intravenous zoledronic acid 4 mg was added to the therapeutic regimen the next day. After 3 days of treatment, hypercalcemia resolved with corrected calcium ranging from 1.7-2.3 mmol/L. The patient unfortunately succumbed to overwhelming sepsis with multiorgan involvement.

CONCLUSION

Granuloma-induced-hypercalcemia remains a diagnostic challenge in persons with TB due to its uncommon occurrence. However, there must be a high index of suspicion to facilitate early intervention with calcium lowering drugs to avoid morbidity and mortality in such patients.

EP_A007

BEYOND THE SORE THROAT: UNVEILING THE THYROID'S HIDDEN TURMOIL

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INTRODUCTION

Subacute thyroiditis (SAT) is an inflammatory thyroid disorder often presenting with anterior neck pain and transient thyroid dysfunction. This case highlights the diagnostic challenges and evolving thyroid function tests (TFT) in a patient initially referred for prolonged throat pain.

CASE

A 50-year-old female presented with anterior neck pain persisting for two weeks, initially treated with antibiotics for suspected infection. She had no dysphagia, odynophagia, or overt hyperthyroid symptoms but reported a significant weight loss of 6 kg over a year, early satiety, and loss of appetite. Examination revealed tenderness over the thyroid gland with no palpable goiter or lymphadenopathy. Initial TFT showed suppressed TSH <0.01 mIU/L with elevated free T4 of 56.4 pmol/L.

Inflammatory markers were raised, with an ESR of 77 mm/hr and CRP of 71.5 mg/L, supporting an inflammatory process. Tumor markers, including CEA, AFP, CA-125, CA 15-3, and CA 19-9, were not elevated, reducing the suspicion for the presence of malignancy.

Follow-up TFT showed a rapid transition from hyperthyroidism (TSH <0.01, T4 56.4) to euthyroidism (TSH 1.39, T4 8.6), and subsequently, hypothyroidism (TSH 9.9, T4 7.1). The dynamic TFT pattern, absence of thyroid autoantibodies, and recent upper respiratory symptoms supported the diagnosis of subacute thyroiditis.

The patient was started on L-thyroxine 50 mcg OD due to hypothyroid progression, with plans for TFT reassessment in 4 weeks. Referral to gastroenterology for early satiety was deferred, considering the likelihood of thyroid-related symptoms.

CONCLUSION

This case underscores the importance of recognizing evolving TFT trends in subacute thyroiditis in order to avoid unnecessary interventions. A systematic approach to thyroid dysfunction in medical care is crucial for timely diagnosis and management, bridging the gap between knowledge and clinical practice.

EP_A008

POST-COVID-19 CHRONIC FATIGUE SYNDROME WITH ACUTE PANCREATITIS AND TRANSIENT HYPERZINCEMIA

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INTRODUCTION/BACKGROUND

Chronic fatigue syndrome (CFS) often follows viral infections, including COVID-19. Long COVID is increasingly recognized as a cause of post-viral fatigue. While rare, hyperzincaemia has been linked to acute pancreatitis. This case explores the interplay between post-viral fatigue, metabolic disturbances, autonomic dysfunction, and transient hyperzincaemia in acute pancreatitis.

CASE

A 61-year-old male with hypertension, benign prostatic hyperplasia, hepatosteatosis, and gallstone-induced acute pancreatitis presented with persistent fatigue

post-COVID-19. Clinical evaluation included metabolic, endocrine, and nutritional markers, focusing on zinc, copper, ceruloplasmin, thyroid, and adrenal function. Autonomic function and post-exertional fatigue patterns were assessed.

The patient developed persistent fatigue following his third and most severe COVID-19 infection, which required hospitalization. Fatigue worsened with exertion and was not relieved by rest. Gallstone-related acute pancreatitis revealed transient hyperzincaemia (serum zinc: 153 mcg/dL, reference: 60-106 mcg/dL) with normal copper, ceruloplasmin, and adrenal function (AM cortisol: 196 nmol/L).

Possible mechanisms include transient zinc release from pancreatic tissue due to acinar cell destruction, reduced zinc excretion resulting from impaired clearance due to the presence of hepatic dysfunction and potential renal impairment, gallstone-related factors such as the presence of cholestasis leading to decreased biliary excretion, altered zinc distribution due to systemic inflammation, exogenous sources leading to contamination or artifacts arising from measurement errors.

Fatigue improved with nil by mouth but recurred post-discharge. Blood pressure fluctuations during this period suggest possible autonomic or even beginning adrenal dysfunction. Hyperzincaemia resolved with dietary modifications.

CONCLUSION

Post-COVID-19 fatigue requires a thorough metabolic, endocrine, and autonomic evaluation. This case highlights transient hyperzincaemia in acute pancreatitis and the need for cautious interpretation of trace element abnormalities. Understanding zinc metabolism and autonomic dysfunction may offer insights into post-viral fatigue syndromes.

EP_A009

THE FIRST CASE OF GUSELKUMAB-INDUCED THYROID STORM IN A YOUNG WOMAN WITH PLAQUE PSORIASIS

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INTRODUCTION/BACKGROUND

Guselkumab is a biologic agent used to treat moderate to severe plaque psoriasis by targeting interleukin-23 (IL-23). While effective in managing psoriasis, the impact of Guselkumab on thyroid function is not well-documented. Autoimmune thyroid diseases such as Graves' disease can be triggered by several factors, including immune-modulating therapies. This case report aims to highlight a rare but severe adverse reaction of Guselkumab in a young female with a predisposition to autoimmune diseases.

CASE

We report a 20-year-old Malay female, a medical student, with plaque psoriasis on Guselkumab therapy. Her elder sister has psoriasis, Graves' disease with severe orbitopathy. Following the patient's first injection of Guselkumab, she developed a moderate-sized diffuse goiter with tenderness. Despite this, she continued with two more doses of Guselkumab over the next six months at three-month intervals.

Approximately two weeks after the fourth dose of Guselkumab, she experienced symptoms of palpitations, hand tremors, low-grade fever, and generalized malaise. She was admitted to the hospital for treatment of severe thyrotoxicosis. Serum free T4 levels were found to be three times above the upper limit of normal, T4: 59.1 pmol/l and a TSH level of <0.01 mIU/l with borderline high anti-TPO antibodies. Despite good compliance with carbimazole 30 mg daily and propranolol 60 mg three times daily for one month, her condition worsened.

Development of signs of thyroid storm, including anxiety, hyperdefecation, hand tremor, low-grade fever (37.6 °C), and sinus tachycardia (150 beats per minute) prompted consult at the emergency department, where she was found to have Burch-Wartofsky Point Scale of 45. Acute phase reactants showed a CRP level of 5, which made subacute thyroiditis unlikely. Due to the severity of her condition,