PP-T-07

NEW ONSET GRAVES' DISEASE AFTER SARS-CoV-2 VACCINATION

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BACKGROUND
There are increasing number of reports of thyroid dysfunction after severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) vaccination. We report a case of new-onset Graves' disease following vaccination with adenovirus-vectored Vaxzevria (Oxford-AstraZeneca).

CASE
A 29-year-old female with no prior history of endocrine or autoimmune disease presented with a week of palpitations, heat intolerance and excessive sweating starting three days after her second dose of Vaxzevria. She was asymptomatic after her first dose given two months earlier. Her father and sister have Graves' disease. She had a diffuse goitre and no orbitopathy. Tests showed suppressed TSH [<0.01 mIU/L, normal range (NR) 0.27-4.2], elevated free T4 (>100 pmol/L, NR 12-22) and positive TSH receptor antibody (>40.00 IU/L, NR <1.75). Ultrasonography revealed a hypervascular, diffusely enlarged goitre. Oral carbimazole and propranolol were commenced. Five months later, free T4 normalized and TSH remained undetectable. To date, she remains hesitant to receive her booster dose.

SARS-CoV-2 infection and vaccination have been associated with subacute thyroiditis and autoimmune thyroid disease. While there are reports of new-onset Graves' disease after mRNA and adenovirus-vectored vaccines, it has not been associated with inactivated virus vaccines. The prevailing postulation is that the adjuvants in the vaccines can trigger an autoimmune event.

CONCLUSION
Physicians should be aware of the possibility of thyroid dysfunction after SARS-CoV-2 vaccination, especially in those with strong family history. More studies are required to establish causal relationship.

PP-T-08

LONG-TERM FOLLOW-UP OF THREE PATIENTS WITH PERIODIC PARALYSIS

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BACKGROUND
Periodic paralysis (PP) is a disorder of muscle ion channels, often precipitated by heavy exercise, fasting or high-carbohydrate meals.

CASES
Case 1: A 28-year-old male with hyperthyroidism had severe lower extremity weakness progressing to the upper extremities after high-intensity exercise, with serum K 1.7 mmol/L. Examination showed normal vitals, palpable thyroid and no neurological deficit. Methimazole, propranolol and KCl wer given. KCl was discontinued after 4 weeks with normalization of serum K. He developed hypothyroidism three months later and was prescribed levothyroxine. He is currently euthyroid without recurrent PP for six years.

Case 2: A 29-year-old male presented with severe exertional muscle weakness for 1.5 years. Examination showed normal vitals, thyroid, muscle and neurologic exam. Results revealed normal TFT, serum K, creatine kinase and decreased amplitude of compound muscle action potential with reduced motor unit recruitment or electrical silence on electromyography. Genetic testing found CACNA1S mutation consistent with hypokalemic PP. He was prescribed eplerenone and acetazolamide with resolution of symptoms for ten years.

Case 3: A 72-year-old male presented with episodes of flaccid paralysis, occurring an hour post-dinner, with serum K 3.2 mmol/L. Symptoms resolved after taking potassium. Examination during the episode showed stage 1 hypertension, proximal muscle weakness and hyporeflexia. Results revealed normal TSH (2.03 μIU/mL) and K (4.3 mmol/L). He continued to perform normal daily activities without any PP symptoms for nine years.

CONCLUSION
Limited information is available regarding the long-term follow up of patients with PP. Our patients illustrate a relatively benign long-term course with appropriate treatment.