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

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Chiew Ken Seng, Lee Ling Wei, Khaw Chong Hui
Hospital Pulau Pinang, Malaysia

INTRODUCTION

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

METHODOLOGY

A 29-year-old female with no prior history of endocrine or autoimmune diseases, presented with a week of palpitations, heat intolerance and excessive sweating three days after her second dose of Vaxzevria. She did not experience these symptoms after her first dose which she received two months earlier. Her father and sister have Graves' disease. She had a diffuse goiter with no orbitopathy. Thyroid Stimulating Hormone (TSH) was <0.01 mIU/L (normal range: 0.27-4.2) with a markedly elevated free T4 of >100 pmol/L (normal range: 12-22). TSH receptor antibody was positive at >40.00 IU/L (Normal range: <1.75). Ultrasonography revealed a hypervascular, diffusely enlarged goiter. She was started on oral carbimazole and propranolol. Five months later, her free T4 had normalized at 18 pmol/L though her TSH was still undetectable. To date, she remains hesitant for her booster dose.

RESULTS

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 current prevailing theory is that the adjuvants in the vaccines can trigger an autoimmune event, also called "autoimmune/inflammatory syndrome induced by adjuvants" (ASIA).

CONCLUSION

Physicians need to be aware of thyroid dysfunction after SARS-CoV-2 vaccination, especially in those with a strong family history of autoimmune disease. Nevertheless, it is also important to note that the benefit of vaccination far outweighs this uncommon potential risk. More studies are required to establish a causal relationship.

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A RARE CASE OF METASTATIC PHEOCHROMOCYTOMA IN MULTIPLE ENDOCRINE NEOPLASIA TYPE 2A (MEN 2A)

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Chiew Ken Seng, Shanty A/P Velaiutham, Khaw Chong Hui
Hospital Pulau Pinang, Malaysia

INTRODUCTION

In MEN 2A, there is a heritable predisposition to medullary thyroid carcinoma (MTC), pheochromocytoma and hyperparathyroidism. MEN2A-associated pheochromocytoma is typically benign. We report a case of malignant pheochromocytoma in MEN2A.

METHODOLOGY

A 45-year-old female was diagnosed with MEN 2A when she was 19-years-old as part of a family screening in the year 1996. Genetic analysis revealed a mutation in codon 634 of the RET proto-oncogene (cysteine-to-tyrosine substitution, C634Y). She underwent total thyroidectomy for MTC and bilateral adrenalectomy for bilateral pheochromocytoma in 1997. She developed recurrence of her pheochromocytoma and underwent laparoscopic left adrenalectomy in 2005. At that time, she also developed primary hyperparathyroidism and underwent total parathyroidectomy.

However, her urinary catecholamines remained elevated. Iodine-131 meta-iodobenzylguanidine (I-131 MIBG) scan in 2007 revealed increased uptake in her left adrenal, liver, mediastinum, skull and upper abdomen. Since she was asymptomatic, she declined systemic therapy. Her disease subsequently progressed. She now experiences daily paroxysms with rising levels of urine metanephrines/normetanephrines. Latest 131-MIBG and Gallium-68 PET/CT scans showed progression with disease activity at her left adrenal bed and with regional node involvement and metastasis to lung, liver and bones. She is now agreeable for further therapy.

RESULTS

In patients with RET codon 634 mutations, pheochromocytoma occurs in 52% by the age of 50 years old. It is almost always benign. Metastatic pheochromocytoma in MEN2A is rare. Case series suggest a prevalence of 3-5%, in contrast to the prevalence of more than 10% in sporadic pheochromocytomas.

CONCLUSION

While MTC is the usual culprit of metastatic disease in MEN2A, it may rarely be due to malignant pheochromocytoma. Multidisciplinary approach to management is optimal.