no malignant features. She awaits total thyroidectomy. Her kindred were advised to undergo screening for MEN 2, albeit the lack of genetic study due to financial constraints.

CONCLUSION

Genetic testing for RET proto-oncogene would be useful to guide management and screening in MEN 2. Medullary thyroid carcinoma is the most common manifestation of MEN 2 with 100% penetrance and should be actively sought for in patients suspected of having MEN 2.

EP_A003

RIFAMPIN-INDUCED ADRENAL CRISIS

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INTRODUCTION/BACKGROUND

Rifampicin is an essential first-line anti-tuberculosis drug. It is crucial for medical practitioners practicing in countries such as Malaysia where tuberculosis is endemic to recognize that rifampicin, an enzyme inducer, can have serious drug-drug interactions and needs to be used cautiously.

CASE

We describe a case of a 30-year-old male who sustained a mild traumatic brain injury with cerebrospinal fluid leakage in 2022. His injury was complicated by panhypopituitarism and secondary adrenal insufficiency, which required hydrocortisone 10mg/5mg BD and desmopressin replacement. He was compliant to hormonal replacement and remained asymptomatic throughout regular follow-up. In February 2024, he presented with submandibular swelling that turned out to be tuberculous lymphadenitis with pulmonary tuberculosis. He was started on first-line antituberculosis medications (Akurit-4), containing rifampicin, isoniazid, pyrazinamide and ethambutol with his usual dose of hydrocortisone. Three days after the initiation of anti-tuberculosis medication, the patient presented with vomiting, fever with postural dizziness without polyuria. Blood pressure was 102/64 mmHg, with postural hypotension and hypoglycaemia. The patient was diagnosed with adrenal insufficiency secondary to rifampicin.

The patient was started on intravenous hydrocortisone 50 mg QID. Laboratory investigations revealed serum cortisol of <27 nmol/L with adrenocorticotropic hormone level of 0.36 pmol/L. After adequate hydrocortisone replacement, the patient had polyuria with a gradual reduction of serum sodium to 125 mmol/L, unmasking the presence of central diabetes insipidus. Desmopressin was started and the patient clinically improved with normalisation of serum sodium.

CONCLUSION

In patients with pre-existing adrenal insufficiency, initiation of an anti-tuberculosis regimen containing rifampicin may increase the metabolism of cortisol by inducing cytochrome CYP3A4 activity and precipitate an adrenal crisis. Before initiation of anti-tuberculosis medications, drug-drug interaction should be reviewed. In such cases, dose adjustment of hydrocortisone is necessary to prevent adrenal insufficiency. Increasing the hydrocortisone dose gradually and close monitoring of the patient's biochemical and clinical state are important to reduce the risk of adrenal crisis and mortality.

EP_A004

THE RIFAMPICIN RED FLAG

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INTRODUCTION/BACKGROUND

Rifampicin is an essential first-line anti-tuberculosis (TB) drug which exhibits potent hepatic enzyme-inducing properties. It has significant drug interactions with an array of other medications, including hydrocortisone as we report in this case.

CASE

A 65-year-old male, HIV positive, treatment-naive, with concurrent primary adrenal insufficiency (Synacten done: Cortisol 0 hour 247.8 nmol/L, 60 minutes 316 nmol/L, and normal ACTH 7.76 pmol/L) on hydrocortisone 10 mg/5 mg replacement for 4 months was admitted for prolonged fever and lethargy. He was diagnosed to have extrapulmonary TB by urine lipoarabinomannan (LAM) test and was started on isoniazid, rifampicin, pyrazinamide plus ethambutol – HREZ regime.

On Day 12 of HREZ, he exhibited hypoglycaemia, postural hypotension, and hyponatremia. Serial monitoring of his sodium levels showed a decreasing trend from a normal level initially of 135 mmol/L to a nadir of 116 mmol/L on day 21 of rifampicin. A diagnosis of adrenal insufficiency secondary to rifampicin was made. Rifampicin accelerates cortisol metabolism resulting in low levels of serum cortisol.