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

https://doi.org/10.15605/jafes.039.S1.014

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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

https://doi.org/10.15605/jafes.039.S1.015

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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.

The patient was started on IV hydrocortisone 50 mg QID. He responded well to treatment with amelioration of symptoms and normalization of sodium levels. Steroids were then tapered to oral hydrocortisone with the lowest replacement dose of 20 mg/10 mg daily (double the usual physiological dose) given the ongoing use of rifampicin. The patient was started on hydrocortisone tablet 20/10 mg daily and with no further dose reduction planned while concurrently on rifampicin. The hydrocortisone dosage will be gradually reduced to the standard physiological dose upon the patient's completion of rifampicin treatment.

CONCLUSION

Prompt identification of drugs that can affect cortisol metabolism is essential to for patients on hydrocortisone replacement therapy. Close monitoring, multidisciplinary collaboration, personalized dose adjustments and careful tapering of hydrocortisone with biochemical and clinical correlation are paramount in navigating the challenges posed by rifampicin-hydrocortisone interaction.

EP_A005

A DECADE OF INITIAL EXPERIENCE IN ADMINISTRATION OF METAIODOBENZYLGUANIDINE THERAPY FOR ADVANCED STAGE PARAGANGLIOMA AND PHEOCHROMOCYTOMA

https://doi.org/10.15605/jafes.039.S1.016

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INTRODUCTION/BACKGROUND

Metaiodobenzylguanidine (MIBG) labeled with radioactive iodine can be utilised for imaging and therapy in advanced stage paraganglioma and pheochromocytoma. Our centre became a local pioneer and started to offer MIBG therapy in 2013. Patients received 200 mCi of Iodine-131 MIBG for each therapy session. We present a case series to highlight the clinical complexity of these rare endocrine neoplasms and our early experience with MIBG therapy.

CASE

The first case involves a 57-year-old male with a large, right pheochromocytoma diagnosed in 2013. Recurrence was noted post-debulking surgery and chemoembolisation. He had 2 MIBG therapies between 2015 and 2016. Surveillance showed a stable underlying tumour and decreasing urine metanephrine level. However, he developed a metastatic pleural nodule and multiple abdominal nodes in 2021. The third MIBG therapy was given in October 2022. Stable disease was noted on a follow-up MIBG scan in April 2023 with markedly decreasing serum Chromogranin A (CgA).

For the second case, a 74-year-old male diagnosed with retroperitoneal paraganglioma in 2002 underwent surgery but presented back with metastatic lesions involving the liver and right ilium in 2012. He received 3 cycles of MIBG therapy between 2015 and 2017. Unfortunately, he deteriorated over the subsequent 18 months due to progressive multiple liver, abdominal nodes, lungs and skeletal metastases.

The third case is a 50-year-old male with subhepatic paraganglioma diagnosed in 2017. Transarterial embolisations were done as surgery was deemed infeasible. He had 3 MIBG therapies between 2018 and 2020. Surveillance in 2021 showed a stable, focal upper abdomen lesion and decreasing CgA level. However, he developed metastatic disease progression with rebound CgA elevation in February 2022. Fourth MIBG therapy was given in September 2022. A MIBG-avid subhepatic mass was seen with extensive skeletal and cervical, thoracic and abdominal node metastases.

CONCLUSION

MIBG therapy may offer potential palliative benefit in pheochromocytoma and paraganglioma as seen among cases with a solitary large lesion or oligometastasis. However, advanced stage diseases especially those with bone metastasis show a poorer prognosis.

EP_A006

ANCIENT SCHWANNOMA: A GREAT MIMICKER OF ADRENAL TUMOUR

https://doi.org/10.15605/jafes.039.S1.017

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INTRODUCTION/BACKGROUND

Retroperitoneal schwannoma is a benign neoplasm arising from the neural crest cells. Pre-operative diagnosis of this rare tumour is often difficult due to its enormous size at the time of presentation and the lack of distinctive imaging phenotypes. We share a case of an incidentally discovered huge right periadrenal ancient schwannoma in an elderly patient who suffered from an underlying nasopharyngeal carcinoma.