CONCLUSION

This case report demonstrated the importance of early detection of hypercalcaemia in a younger population, the need to obtain a comprehensive family history and the significance of proceeding with early germline CDC73 mutation detection for optimal clinical management of pHPT. Genetic counselling and surveillance of family members who are at risk of developing CDC73-related disorders must also be emphasized.

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MICROANGIOPATHIC HEMOLYTIC ANEMIA IN A CASE OF PHEOCHROMOCYTOMA CRISIS

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Shireen Lui Siow Leng, Oh Kok Ming, Tee Hwee Ching, Serena Khoo Sert Kim
Endocrinology Unit, Department of Medicine, Queen Elizabeth II Hospital, Kota Kinabalu, Sabah, Malaysia

INTRODUCTION

Pheochromocytomas are rare neuroendocrine tumours of the adrenal medulla. Pheochromocytoma crisis, a potentially fatal presentation, is characterized by severe hypertension or hypotension with end organ damage occurring spontaneously or precipitated by a stressor such as surgery, stress or medications such as anaesthetic agents, corticosteroids, and metoclopramide. A cascade of life threatening events may occur including myocardial infarction, acute pulmonary oedema, stroke, paralytic ileus, thrombosis, renal failure, diabetic ketoacidosis, and lactic acidosis. Microangiopathic hemolytic anemia (MAHA), an uncommon sequelae of pheochromocytoma crisis or malignant hypertension can occur.

CASE

We report a case of pheochromocytoma crisis presenting with microangiopathic hemolytic anemia. A 58-year-old female with diabetes mellitus, hypertension, and dyslipidemia presented with recurrent vomiting and epigastric pain for 9 days. On arrival, her blood pressure was 177/133 mmHg with a pulse rate of 146 beats/minute. It worsened to 214/146 mmHg following the administration of intravenous metoclopramide. She developed acute kidney injury (AKI) (urea 14.5 mmol/L, creatinine 321.8 umol/L) and MAHA with Coomb's test negative anemia. Her hemoglobin decreased from 10.6 g/dl to 8.1 g/dl in 2 days, with increased schistocytes from 2.7% to 3.0%, accompanied by reticulocytosis of 4.8%, thrombocytopenia with platelet of 141 k, and raised lactate dehydrogenase of 482 U/L. She received 1-unit packed cell transfusion. Her MAHA and her AKI resolved subsequently following adequate blood pressure control with alpha blockade. Twenty four hour urine metanephrine was elevated with abdominal imaging demonstrated a right adrenal mass (4.0x 4.5x 4.0 cm) supporting the diagnosis of a right adrenal pheochromocytoma. She underwent a right open adrenalectomy with symptoms resolution and normotension after surgery.

CONCLUSION

This case highlights the awareness of detecting a pheochromocytoma crisis and avoiding medication that may induce a pheochromocytoma crisis. High index of suspicion, prompt diagnosis and early initiation of treatment of pheochromocytoma crisis is essential to minimise complications and improve clinical outcomes.