EP_A003

'AN IMPENDING DOOM:' RARE CASE OF RUPTURED PHEOCHROMOCYTOMA PRESENTING AS PHEOCHROMOCYTOMA CRISIS

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INTRODUCTION/BACKGROUND
Spontaneously ruptured pheochromocytoma presenting as pheochromocytoma crisis is a rare complication and is associated with high morbidity and mortality.

CASE
We present a case of a 46-year-old female with left pheochromocytoma, diagnosed following a total abdominal hysterectomy for uterine leiomyoma. 24-hour urine catecholamines were elevated- norepinephrine: 2.6 x ULN [1241 nmol/L], epinephrine: 12.8 x ULN [1468.3 nmol/L], dopamine <424 nmol/L. Plasma normetanephrine and metanephrines were also elevated. CT scan showed a large adrenal mass measuring 4.98 x 5.4 x 5.5 cm. Medications were oral Prazosin 3 mg five times daily and Labetalol 100 mg twice daily. She presented four weeks later with acute onset of abdominal pain, persistent vomiting, chest discomfort, vasovagal syncope, headache and sweating. She was restless, pale and hypotensive with a BP of 80/40 mmHg and HR 104 beats per minute. Abdominal examination revealed generalised tenderness, guarding and a palpable mass over the left lower quadrant. Blood pressure increased subsequently ranging from systolic 150 to 200 mmHg and diastolic 90 to 100 mmHg. ECG showed widespread deep T inversion suggestive of Wellen syndrome with raised Troponin I (1950 ng/mL). Abdominal CT revealed a ruptured left pheochromocytoma measuring 5.0 x 6.6 x 7.1 cm with a large intra-abdominal hematoma. She was started on Prazosin 2 mg three times daily and Labetalol 100 mg three times daily for blood pressure control. She was given an insulin infusion for hyperglycemia. Packed cells were also transfused. She underwent laparotomy and adrenalectomy following adequate alpha- and beta-blockade. The postoperative course was uneventful. HPE of the left adrenal mass confirmed pheochromocytoma.

CONCLUSION
Pheochromocytoma crisis resulting from a large release of catecholamines from a ruptured pheochromocytoma is associated with high mortality. Prompt resuscitation and blood pressure control are the mainstays of treatment prior to surgical intervention.

EP_A004

UNMASKING PRIMARY ALDOSTERONISM IN A PATIENT WITH END STAGE RENAL DISEASE: A CASE REPORT

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INTRODUCTION/BACKGROUND
Managing hypertension in a patient with end-stage renal disease (ESRD) requires a combination of antihypertensive medications and volume control. It is common to encounter refractory hypertension in ESRD. Investigation of primary aldosteronism (PA) as a cause of refractory hypertension in ESRD is potentially difficult. These patients are on multiple antihypertensive medications that cannot be discontinued, thus complicating the interpretation of aldosterone-renin ratio.

CASE
We present a patient with ESRD on peritoneal dialysis with refractory hypertension and hypokalaemia investigated for primary aldosteronism.

The patient is a 72-year-old male, known hypertensive for 40 years, with poorly controlled blood pressure for the past 20 years. The presence of refractory hypertension and hypokalaemia prompted an investigation for primary aldosteronism ten years ago, where the patient tested negative. Over the next ten years, his eGFR deteriorated, and he was initiated on peritoneal dialysis a year ago. Despite peritoneal dialysis, his BP remained poorly controlled while on six antihypertensive medications, including furosemide and spironolactone.

Despite being on six confounding antihypertensive medications, his plasma aldosterone was not suppressed and instead, elevated at 1229 pmol/L with a normal direct renin level (9 mU/L). Adrenal CT revealed bilateral adrenal adenomas. Further assessment with adrenal vein sampling was done. Surgery was explored, but the patient was not keen. Spironolactone dose was optimized which led to improvement of blood pressure control and reduction of other antihypertensive medication doses without occurrence of hyperkalemia.