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VIPoma: A Rare Cause of Chronic Diarrhoea with Hypokalemia, Metabolic Acidosis and Pancreatic Mass

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Ilham I,[1] Waye Hann K,[2] Nor Azmi K,[1] Ian C,[3] Azyani Y,[4] Lizawati RH[4]
[1]*Department of Medicine, PPUKM, Malaysia*
[2]*Department of Medicine, University Tunku Abdul Rahman, Malaysia*
[3]*Department of Surgery, PPUKM, Malaysia*
[4]*Department of Pathology, PPUKM, Malaysia*

INTRODUCTION

Vasoactive intestinal peptide tumour or VIPoma is a rare pancreatic neuroendocrine tumour (PNET) with the incidence of 1 in 10 million population. The characteristic presentation of VIPomas are profuse diarrhoea with hypokalaemia and metabolic acidosis.

We report a case of VIPoma who presented to our centre and was thoroughly investigated for his chronic diarrhoea.

CASE

A 40-year-old gentleman had 7-months history of chronic watery diarrhoea and severe weight loss despite multiple courses of antibiotics. Clinically he was cachexic looking with no other abnormalities on examination. Upon presentation there was hypokalaemia, hypophosphataemia, metabolic acidosis and acute kidney injury. He was thoroughly investigated for infections which were negative, while his endoscopies showed only mild gastritis and colitis. Despite intensive fluid replacements, anti-emetics and anti-diarrhoeals his symptoms persisted. CT Abdomen revealed a large pancreatic tail mass with hypodense necrotic centre and calcification. Classical tumour markers for pancreatic adenocarcinoma (CA-19-9 and CEA) were negative. A diagnosis of VIPoma was made based of the clinical features but serum VIP levels were not sent due to patient's financial constraint. There was also a remarkable response to a trial of somatostatin analogue with symptoms resolving upon administration of octreotide, subcutaneously. Distal pancreatectomy and splenectomy were performed and patient's gastrointestinal symptoms resolved immediately. Histopathological examination (HPE) confirmed a grade 1 (pT3 N1 Mx) PNET but unfortunately immunohistochemical staining for VIP is not available in our centre. Post-operatively, patient is recovering well and is scheduled for a 68-Ga-DOTATATE PET/CT scan.

CONCLUSION

This case illustrated a patient diagnosed as VIPoma based on (i) characteristic diarrheagenic symptoms, (ii) typical biochemical features, (iii) radiological evidence of a distal pancreatic mass, (iv) positive response to somatostatin analogue and (v) HPE findings of a NET.

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A Case of Resistant Hypertension with Hypokalaemia due to Co-Secreting Cortisol and Aldosterone in a Patient with Bilateral Adrenal Adenomas

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Muhammad Aizat A, Muhammad Shukri J, Har Kiran Kaur Deol KS, Nurul Hakimmah AM, Nurul Ainna KA, Jami'atul Ezma MA, Yek Ying C, May Ching T, Chiew Yee C, Nurul Izah A, Elliyyin K
Medical Department, Hospital Kajang, Malaysia

INTRODUCTION

Aldosterone and cortisol co-secreting adrenal tumours are rare. We report a case of subclinical Cushing's syndrome (CS) with co-secreting aldosterone in a patient with bilateral adrenal adenomas.

CASE

A 53-year-old Indonesian lady with hypertension and diabetes for 15 years, presented to our hospital with hypertensive urgency and symptomatic hypokalaemia. Clinically there were skin-tags and acanthosis nigricans but no pathognomonic features of CS. Fundoscopy showed narrowing of retinal arteries and silver wiring. Urine microscopy showed macroalbuminuria. Her ECG showed left ventricular hypertrophy which was confirmed on echocardiogram. She was discharged with metformin, a DPP4-inhibitor, four antihypertensives and potassium supplements of 7.2 g/day. During workup for hypertension and hypokalaemia, she demonstrated a positive screening test for primary aldosteronism during which her creatinine was 112 umol/L with eGFR of 57 mls/min/1.73 m^2. A 24-hour urinary cortisol was within normal. The patient underwent a 1-mg overnight dexamethasone suppression test during which her serum cortisol was elevated; 429 nmol/L. Subsequently, a low dose dexamethasone suppression test showed inability to decrease cortisol level below the cut off value to rule out CS; 290 nmol/L. Pheochromocytoma was excluded with normal 24-hour urinary catecholamine levels. A confirmation test for primary aldosteronism was not done in view of worsening renal function to stage-4 CKD. A CT scan of the adrenals revealed benign features of bilateral adrenal adenomas with rapid wash-out, measuring 2.6 cm and 1.5 cm by the largest diameter of the right and left adenomas respectively. The patient opted for conservative management. Her hypertension and hypokalaemia status improved while on spironolactone 75 mg BD. All potassium supplements were stopped and her BP was much easier controlled on three medications.

CONCLUSION

This interesting case illustrates that adrenal adenomas might be capable of secreting both aldosterone and cortisol without clinical features of CS.