COLLEGE OF CHEMICAL PATHOLOGISTS OF SRI LANKA

"Integrating Laboratory and Clinical Systems"

3rd Annual Academic Sessions

2018

15th to 17th March 2018

at Hotel Galadari, Colombo.

CASE REPORTS

CR 8

Pseudohypoaldosteronism-II in a Patient with Renal Impairment and on Losartan; a Diagnostic Challenge

Senarathne UD[1,2], Dayanath BKTP[2], Siyambalapitiya S[3], Tilakarathne PMYI[4]
[1]Department of Biochemistry, Faculty of Medical Sciences, University of Sri Jayewardenepura, Nugegoda, Sri Lanka
[2]Department of Chemical Pathology, Colombo North Teaching Hospital, Ragama, Sri Lanka
[3]Endocrinology Unit, Colombo North Teaching Hospital, Ragama, Sri Lanka
[4]Department of Medicine, Faculty of Medicine, University of Kelaniya, Ragama, Sri Lanka
udara.senarathne@sjp.ac.lk

Introduction

Pseudohypoaldosteronism-II (PHA-II) is a rare genetic disorder of renal tubular electrolyte handling due to defective sodium and potassium channels. These patients classically present with hypertension, hyperkalaemia (despite normal glomerular filtration), metabolic acidosis and hyperchloremia. A similar biochemical abnormality can occur in patients with chronic kidney disease and on angiotensin receptor blockers. However; metabolic derangements in PHA-II, characteristically respond to thiazides if due to overactive NaCl co-transporter (NCCT) at distal convoluted-tubules (DCT).

Case Presentation

A 70-year-old female presented with generalised body weakness for one month and difficulty in walking for one week duration. She had a history of hypertension (>20 years) for which she was recently started on losartan. Two years back, she had an episode of hyperkalemic periodic paralysis.

On this admission, her blood pressure was 160/100 mmHg and there was mild weakness in all four limbs. Her investigations revealed hyperkalaemia (8.9mmol/L), hyperchloremia (110 mmol/L), metabolic acidosis (pH 7.277, HCO_3^- 15.1 mmol/L), and moderately tall T waves in ECG. Her serum creatinine was 154 µmol/L and aldosterone was 452 pmol/L.

A trial of HCT was able to correct above metabolic derangements (K^+ 4.8 mmol/L, pH 7.392, HCO_3^- 25.8 mmol/L) with evidence of increased fractional excretion of potassium. During follow-up a week later, she remained normokalaemic on HCT and frusemide.

Discussion

In PHA-II, overactivity of NCCT increases sodium, water reabsorption at DCT leading to hypertension and reduced Na^+ delivery to collecting ducts (CD). This retards the development of a potential gradient through Na^+ reabsorption, along which K^+, H^+ are excreted at CD. By inhibiting Na^+ reabsorption at DCT, thiazides facilitate aldosterone regulated K^+, H^+ excretion at CD, correcting hyperkalaemia and acidosis.

Considering the response to HCT, diagnosis of PHA-II was made in this patient, which awaits conformation by genetic studies. Although rare, channelopathies should be considered in patients presenting with electrolyte abnormalities.

Keywords

Pseudohypoaldosteronism, hyperkalaemia, hypertension, renal tubular acidosis, channelopathies