CONCLUSION

Although majority of the patients presented with symptoms related with catecholamine excess, almost one third of the patients had incidental discovery. Incidence of pheochromocytoma recurrence and metastasis in our setting has been shown to be comparable with current available studies. This study has demonstrated a low rate of genetic testing likely due to limited access to the test in our setting.

PP-A-03

HYPOKALAEMIA AND COMORBIDITIES ARE COMMON AT INITIAL PRESENTATION IN PATIENTS WITH PRIMARY HYPERALDOSTERONISM

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OBJECTIVES

Primary hyperaldosteronism (PH) is the most common endocrine cause of hypertension (HTN) and is associated with end organ damage. About 30% of cases present with hypokalemia. Studies on the presentation of PH among the Indian population is lacking. This study evaluated the presenting characteristics of patients with PH from Eastern India.

METHODOLOGY

This is a retrospective study that included Saline Suppression Test (SST) confirmed PH patients.

RESULTS

The study involved seventy-eight confirmed PH patients with mean age of 55 ± 13 years and male-to-female ratio of 1.5:1. Mean duration of HTN was 13.3 ± 7.6 years and 62% had HTN more than 10 years. Mean SBP and DBP was 165.1 ± 13.5 mm Hg and 96.2±14.4 mm Hg, respectively. The mean number of anti-hypertensive medications was 3 ± 0.7. Majority presented with hypertension and hypokalemia (78%), 52% of which were spontaneous while 26% were diuretic-induced. About 14% presented with resistant HTN and 8% with adrenal incidentaloma. Overall, 64% of subjects had resistant HTN. Approximately 16.7% of patients experienced hypokalemic periodic paralysis. Mean serum sodium and potassium levels were 139.4 ± 2.3 mmol/l and 3.08 ± 0.6 mmol/l, respectively. Mean eGFR was 71.8 ± 20.8 ml/min/1.73 m^2, with 39.7% having Stage 3 CKD. Majority (95%) had comorbidities from end organ damages, with 43% having multiple comorbidities.

CONCLUSION

Our study revealed a high proportion of hypokalemia and resistant hypertension at detection of PH suggesting delayed diagnosis. A significant number of patients had comorbid illnesses due to end organ damage at presentation, highlighting the need for awareness, early screening and appropriate management of PH.

PP-A-04

IDENTIFICATION OF ALDOSTERONE E-DRIVER SOMATIC MUTATIONS IN CELL-FREE DNA FROM ADRENAL VEIN SAMPLES OF PRIMARY ALDOSTERONISM PATIENTS

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OBJECTIVES

Cell-free DNA fragments (cf-DNA) of tumour cells are often found in the blood downstream to the tumour due to the high apoptosis/necrosis rate of the cells. Primary aldosteronism (PA), a curable cause of secondary hypertension, is commonly due to an autonomous aldosterone-producing adenoma (APA) that harbours a somatic mutation in an aldosterone-driver gene. We aimed to determine the utility of cf-DNA genotyping from adrenal vein samples (AVS) for aldosterone-driver gene mutations as a biomarker for APA.

METHODOLOGY

Genotyping of cf-DNA from AVS of PA patients was performed using the Agena MassARRAY platform. In this study, six samples of cf-DNA from three PA patients were interrogated.

RESULTS

Of the three PA patients, two had unilateral APA and one had bilateral APA. Of the six cf-DNA samples, two samples from the same patient (right adrenal and left adrenal) were found to have a mutation in an aldosterone-driver gene. Genotyping of the cf-DNA of the right AVS yielded a CTNNB1 S45P mutation whereas the cf-DNA of the left AVS had a KCNJ5 G151R mutation.