Case Series

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Primary Aldosteronism: A Series of 11 Cases

Internal Medicine Section

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ABSTRACT

Primary Aldosteronism (PA) should be suspected in patients with resistant hypertension, early-onset hypertension, hypertension with hypokalaemia, and metabolic alkalosis. The diagnosis is often missed due to a lack of awareness and cumbersome investigations. Eleven cases of PA who attended the Endocrinology Outpatient Department (OPD) and were also admitted to the hospital over the last three years (January 2020 to January 2023) were studied, and data were collected and analysed. All 11 patients had hypertension, and five out of 11 had resistant hypertension and 36.36% (four patients) had a family history of hypertension. In this cohort, nine out of 11 patients presented with hypokalaemia (81.81%), and five of them had hypokalaemic paralysis at presentation. In 54.54% of patients, plasma aldosterone was in the range of 20-40 ng/dL, with a mean plasma Aldosterone conc. of 31.7 ng/dL. Plasma Renin Activity (PRA) was suppressed in all eight patients. In nine out of 11 patients, PAC/PRA was >20. A left-sided adrenal adenoma was observed in 54.54% of cases. Six patients (54.54%) received surgical management (laparoscopic adrenalectomy), and the remaining five patients were doing well with medical management (antihypertensives and spironolactone). This case series illustrates the need for screening of PA in patients with resistant hypertension, specifically those with hypokalaemia. Elevated PAC is diagnostic, especially in the context of suppressed PRA. Computed Tomography (CT) is the modality of choice for localising the adenoma in most cases. Clinicians can make decisions regarding the choice of treatment based on these two investigations.

Keywords: Adrenal, Adenoma, Aldosterone, Hypertension, Hypokalaemia, Renin

INTRODUCTION

PA is a disorder of the adrenal gland, caused by the hypersecretion of aldosterone. PA is the most common cause of secondary hypertension, with a prevalence of over 20% in resistant hypertension [1,2] and 10% in individuals with severe hypertension (Systolic Blood Pressure (SBP) ≥180, Diastolic Blood Pressure (DBP) ≥110 mmHg) [3,4]. PA increases cardiovascular morbidity and mortality even after blood pressure is controlled [5,6]. Therefore, early identification and specific treatment of PA are essential, although only a small fraction of patients with PA are diagnosed and treated [7].

PA should be suspected in patients with resistant hypertension, hypertension with hypokalaemia, or early-onset hypertension with metabolic alkalosis. The presence of hypokalaemia alongside hypertension should immediately raise suspicion of PA, although hypokalaemia may be absent in many PA patients [8,9]. A plasma aldosterone level >20 ng/dL with suppressed PRA strongly suggests PA, and confirmatory tests like saline suppression are unnecessary [9]. This case series highlights the recent expansion of knowledge about PA and provides a practical approach to its diagnosis and treatment. To the best of our knowledge, this is the first case series on PA from Eastern India. This case series demonstrates the utility of properly conducted PAC/PRA ratio, along with absolute values of PAC, in the diagnosis of PA. Hypokalaemia, including hypokalaemic paralysis, is a useful but important hint for the diagnosis of PA.

CASE SERIES

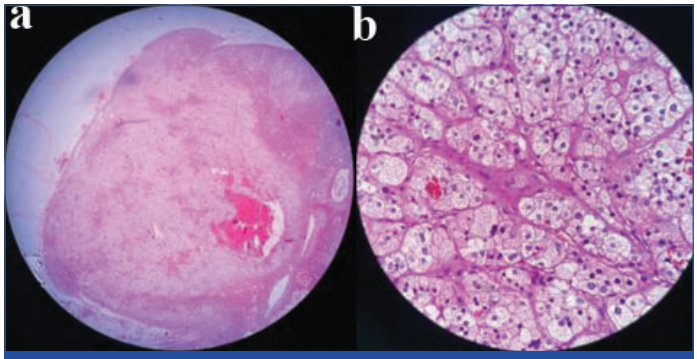
A consecutive series of 11 PA cases, who attended the endocrinology OPD or were admitted to the hospital over the last three years (January 2020 to January 2023) were studied. Data was collected and analysed regarding their demographic profile, clinical and biochemical features, and imaging characteristics. Patients were managed conservatively or surgically based on treatment guidelines and patient preferences. Prior to surgery, patients provided their consent as per the institutional protocol. The mean age in this population was 46 years [Table/Fig-1], and the majority were female (72.72%). All patients had hypertension, with 45.45% (5 out of 11) had resistant hypertension and 36.66% (four patients) having a family history of hypertension. Nine out of 11 of patients presented with hypokalaemia, and 45.45% (five patients) had hypokalaemic paralysis at presentation. In 54.54% of patients, plasma aldosterone levels ranged from 20-40 ng/dL, with a mean PAC of 31.7 ng/dL. PRA was suppressed in eight patients. Adrenal adenomas were identified in all 11 patients by imaging (Contrast Enhanced Computed Tomography (CECT) abdomen with adrenal protocol), with 54.54% of them being left-sided. The mean maximum dimension of the adrenal adenomas was 1.64 cm. Six patients (54.54%) received surgical management (laparoscopic adrenalectomy), and the remaining five patients were doing well with medical management (antihypertensives and spironolactone). In four out of the six patients who received surgical management, antihypertensive medications were stopped during follow-up visits as blood pressure was well controlled. The summary of all 11 patients is given in [Table/Fig-2]. [Table/Fig-3-7] shows histological and radiological images of cases.

Variables	Mean±SD	N
PAC (ng/dL)	31.7836±12.42667	11
Age (years)	46.18±14.49	11
Nodule size (Cm)-Max dimension	1.640±0.4648	10
Potassium at presentation (meq/L)	2.7000±0.99899	11
Duration of hypertension (years)	6.91±0.99899	11

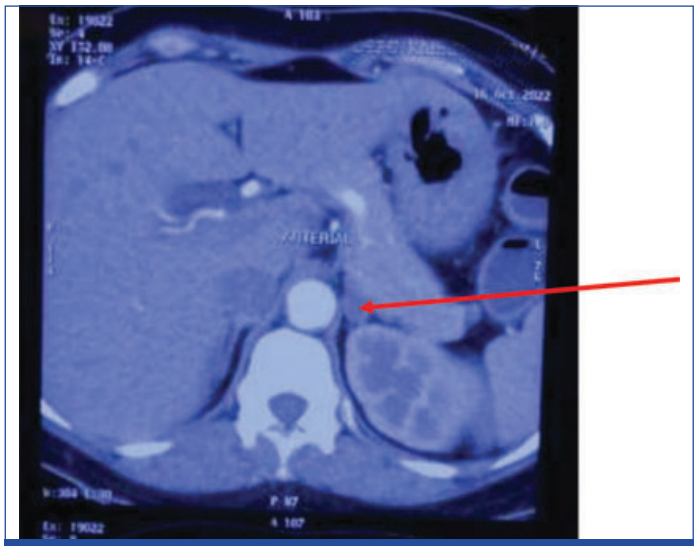
[Table/Fig-1]: Shows the mean and standard deviation of study variables of the study cohort.
PAC: Plasma aldosterone concentration

Case No.	Age-Sex-Current antihypertensive medication	Diagnosis	Clinical features and physical examination	Biochemical investigations	Imaging	Treatment, follow-up and prognosis
1	Female 34 years on Amlodipine 5 mg, Telmisartan 40 mg and Torsemide 10 mg	Primary aldosteronism (Right adrenal adenoma)	Sudden onset quadriparesis and muscle cramps Known HTN for three years BP on admission-150/90 mmHg, muscle power on admission-B/L UL 3/5 B/L LL 2/5 Deep Tendon Reflexes (DTR)-absent	Na/K-145 meq/L/1.1 meq/L, Creat-0.6 mg/dL, Post Op Na/K-132 meq/L/4.5 meq/L, PAC-39.5 ng/dL, PRA-3.33 ng/mL/hr, DRC 3.5 mIU/mL (4.4-46)	USG W/A-Cholelithiasis CECT W/A-Right adrenal small (16×11 mm) hypodense rounded lesion involving anterior limb and body. On contrast enhancement with early washout. No calcification	Laparoscopic right adrenalectomy done. On amlodipine 2.5 mg OD at the time of discharge. Three months after surgery BP was 130/80 mmHg with Amlodipine 2.5 mg and serum Potassium was 3.5 meq/L. Amlodipine was stopped on subsequent visits and there was no further episodes of quadriparesis till now.
2	Female 55 years on Amlodipine 5 mg, Telmisartan 40 mg and Torsemide 10 mg	Primary aldosteronism (Right adrenal adenoma)	Resistant HTN for 10 years Family history of HTN BP on admission 146/88 mmHg GI system-Hepatomegaly present Other organ system-WNL	Na/K-136 meq/L/2.7 meq/L, Na/K-143 meq/L /3.7 meq/L, (At the time of dsischarge), Creat-0.47mg/dL, PAC-22.9 ng/dL, PRA-0.11 ng/mL/hr, PAC/PRA 208.18	CT W/A-Liver enlarged (17.5 cm), tiny cystic lesion in segment VI of Liver. Small hypodense 12×10 mm lesion in lateral limb of right adrenal (AW 77 %, RW 75%)	On medical management Verapamil (120 mg OD) Prazosin (2.5 mg OD). At 3 months follow-up BP 136/86 mmHg with above mentioned medical management. Verapamil was reduced to 40 mg BD.
3	Male 67 years on Metoprolol 50 mg, Telmisartan 40 mg and Hydrochlorthiazide 12.5 mg	Primary aldosteronism (Right adrenal adenoma)	HTN for five years, Incidentally detected right adrenal adenoma during evaluation of dyspepsia. BP on admission 160/90 mmHg, GI examination-No organomegaly, P/A-No tenderness. Other organ system-WNL	NA/K-141 meq/L/4.4 meq/L, Creat-1.6 mg/dL, PAC 10 ng/dL, PRA <0.1 ng/mL/hr	CECT W/A-24×20 mm lesion in right adrenal unenhanced 12 HU	On medical management Verapamil (120 mg BD) Prazosin (10 mg BD) at the time of discharge. BP 140/86 mmHg at three months follow-up with above medical management. Prazosin was reduced to 10 mg OD and Verapamil reduced to 40 mg TDS 6 months after discharge.
4	Male 73 years on Amlodipine 10 mg, Telmisartan 80 mg, Chlorthalidone 6.25 mg and Spironolactone 50 mg	Primary aldosteronism (Right adrenal adenoma)	HTN for three years. H/O AMI followed by LVF. Persistent hypokalemia. BP on admission 144/100 mmHg CVS examination-No added sound. S1, S2 audible. Power on admission-B/L UL 5/5 B/L LL 5/5	NA/K-131 meq/L/3.85 meq/L, Creat-1.5 mg/dL, PAC 33.6 ng/dL, PRA 1.18 ng/mL/hr, PAC 25.3 ng/dL (after saline suppression)	CT W/A-23×13 mm mildly thickened medial limb of right adrenal	On medical management Amlodipine (5 mg) Spironolactone (25 mg). BP after 3 months 130/80 mmHg with Amlodipine 5 mg and spironoloctone 25 mg. Serum potassium was 3.9 meq/L.
5	Female 34 years on Amlodipine 20 mg, Telmisartan 80 mg and Hydrochlorthazide 6.25 mg	Primary aldosteronism (Left adrenal adenoma)	Refractory HTN since 10 years with H/O HPP seven years back. BP on admission 160/100 mmHg, Power on admission-B/L UL 5/5 B/L LL 5/5 DTR-present	Na/K-136 meq/L/3.08 meq/L, Creat 0.9 mg/dL, PAC-44.42 ng/dL, PRA 0.72 ng/mL/hr	CT W/A-Lipid rich adenoma in medial limb of left adrenal (19×11 mm) <1O HU (Unenhanced)	Left-sided laparoscopic adrenalectomy done. Amlodipine 2.5 mg OD given at the time of discharge. Three months after surgery BP was 120/80 mmHg and serum potassium was 3.8 meq/L at follow-up. Amlodipine was stopped at follow-up (one year after discharge).
6	Female 28 years on Cilnidipine 20 mg, Losartan 50 mg and Hydrochlorthiazide 6.25mg	Primary aldosteronism (Left adrenal adenoma)	HTN for two years with H/O recurrent quadrparesis. Family H/O HTN. BP on admission 140/90 mmHg Power on admission-B/L UL 5/5 B/L LL 5/5 DTR-present	Na/K-146.8 meq/L/2.7 meq/L, Creat-0.8 mg/dL, PAC-47.8 ng/dL, PRA-0.4 ng/mL/hr, PAC 27.8 (after saline suppression)	CT W/A-small 1.4×1.2 well-defined hypodense lesion in left adrenal	Laparoscopic left adrenalectomy done. Prazosin 5 mg BD (at the time of discharge). Three months after surgery BP was 120/84 mmHg and Prazosin was reduced to 5 mg OD. Serum potassium was 3.5 meq/L after three months. No further episodes of quadriparesis till now (one year after discharge).
7	Female 42 years on Metoprolol 25 mg, Losartan 50 mg and Hydrochlorthiazide 6.25 mg	Primary aldosteronism (Left adrenal adenoma)	HTN for 12 years (difficult to control). BP on admission 160/96 mmHg Other organ system-WNL	Na/K-141 meq/L/2.5 meq/L, Creat-0.48 mg/dL, PAC-37.8 ng/dL, PRA-0.1 ng/mL/hr	CT W/A-well defined nodular hypodense lesion 1.1×0.6 cm non contrast-3 HU (lipid rich adenoma)	Laparoscopic left adrenalectomy done. Amlodipine 5 mg OD given (at the time of discharge). 3 months after discharge BP was 126/80 mmHg and Amlodipine was stopped at follow-up after 9 months.
8	Female 34 years on Cilnidipine 20 mg, Telmisartan 80 mg and Hydrochlorthiazide 6.25 mg	Primary aldosteronism (Right adrenal adenoma)	Resistant HTN for eight years Sudden onset quadriparesis. BP on admission 170/100 mmHg. Power on admission-B/L UL 2/5 B/L LL 2/5 DTR-diminished	Na/K-143 meq/L/1.45 meq/L, PAC-45.8 ng/dL, PRA-1.55 ng/mL/hr PAC-19.7 ng/dL (Post saline suppression) AVS-inconclusive	CECT W/A-Right adrenal-18×13×17 mm hypodense rounded lesion enhancement in arterial phase, rapid washout in delayed phase, AW 64%, RW 43%, Left adrenal mildly enlarged body (10×9 mm)	Laparoscopic right adrenalectomy done. Amlodipine 5 mg OD (At the time of discharge). B.P 120/80 mmHg three months after surgery and Amlodipine was reduced to 2.5 mg. Serum potassium was 3.4 meq/L on after 9 months.
9	Male 44 years on Amlodipine 10 mg, Losartan 50 mg and torsemide 10 mg	Primary aldosteronism (Left adrenal adenoma)	Recent acute onset flaccid paralysis one month back with HTN for 20 years. BP on admission 140/90 mmHg Power on admission-B/L UL 5/5 B/L LL 5/5 Other organ system-WNL	Na/K-142 meq/L/1.72 meq/L, Creat- 1.0 mg/dL, PAC-21.2 ng/dL, PRA-2.07 ng/mL/hr, PAC 9.8 ng/dL (Post Saline suppression)	CT W/A-Left-side adrenal adenoma with slightly thickened right adrenal	On medical management Prazosin 5 mg BD Amlodipine 10 mg OD Spironolactone 50 mg OD Potklor 10 mL TDS BP and serum potassium was normal with above medical management on follow-up after three months. Potklor was stopped on follow-up visits after one year.
10	Female 43 year on Amlodipine 5 mg, Telmisartan 40 mg and Chlorthalidone 6.25 mg	Primary aldosteronism (Left adrenal adenoma)	HTN for four years, History of muscle spasm, previous admission with carpopedal spasm, investigations revealed hypokalemic metabolic alkalosis. BP on admission 150/90 mmHg Power on admission-B/L UL 5/5 B/L LL 5/5 DTR-present	Na/K-141 meq/L/2.8 meq/L, Creat-0.6 mg/dL, PAC 26.7 ng/dL, PRA <0.1 ng/mL/hr, Na/K-138 meq/L/3.6 meq/L (at the time of discharge)	CECT W/A-Left adrenal adenoma 11×9 mm, AW 163%, RW-46% AVS inconclusive	Laparoscopic left adrenalectomy done. Amlodipine 5 mg and Prazosin 5 mg at discharge. BP was 110/70 mmHg three months after surgery, both Amlodipine and Prazosin were stopped and maintaining BP six months after discharge.
11	Female 54 years on Amlodipine 5 mg, Atenolol 25 mg and Hydrochlorthiazide 6.25 mg	Primary aldosteronism (Left adrenal adenoma)	Incidentally detected adrenal SOL during evaluation of pain abdomen, HTN for two years, Hypokalemic metabolic alkalosis on investigations. BP on admission 150/100 mmHg Other organ system-WNL	Na/K-143 meq/L/3.4 meq/L, Creat-0.7 mg/dL, PAC 19.9 ng/dL, PRA <0.1 ng/mL/hr	CECT W/A-16×13 mm SOL in left adrenal gland, Non contrast +2 HU, On enhancement Venous +74 HU, Portal+68 HU, 15 min delayed +32 HU AW-54%, RW-52% AVS Ipsilateral ratio 2.23	On medical management-verapamil 120 mg BD Prazosin 10 mg OD Spironolactone 50 mg BD. BP was 130/80 mmHg three months after discharge and Verapamil was reduced to 40 mg TDS and Prazosin reduced to 5 mg OD.

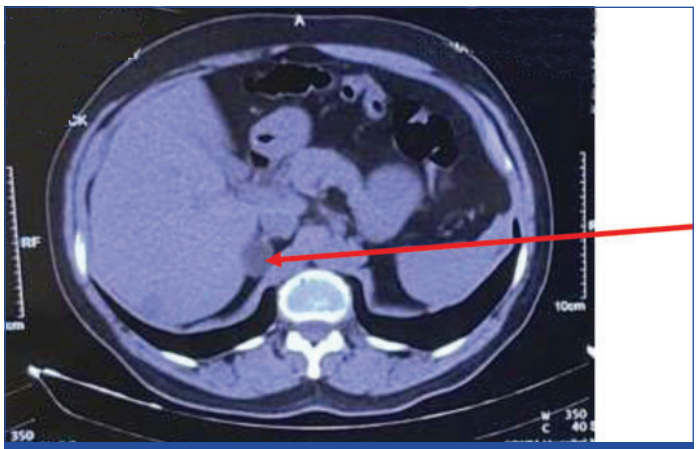
[Table/Fig-2]: Shows the demographic, clinical, biochemical and imaging characteristics of the study cohort (n=11).
DRC: Direct renin concentration; HTN: Hypertension; BP: Blood pressure; USG: Ultrasound; W/A: Whole abdomen; CECT: Contrast enhanced computed tomography; GI: Gastrointestinal; WNL: Within normal limits; AMI: Acute myocardial infarction; CVS: Cardiovascular; SOL: Space occupying lesions



[Table/Fig-3]: Microscopic findings from left adrenal sol in a patient with primary aldosteronism (case 10). It shows large polygonal cells with abundant clear cytoplasm, condensed nuclei with inconspicuous nucleoli. (a) 40X image; (b): 400X image (Light Microscope).



[Table/Fig-4]: CECT Abdomen (Axial section) of a patient with primary aldosteronism (case10) showing left-sided adrenal adenoma.

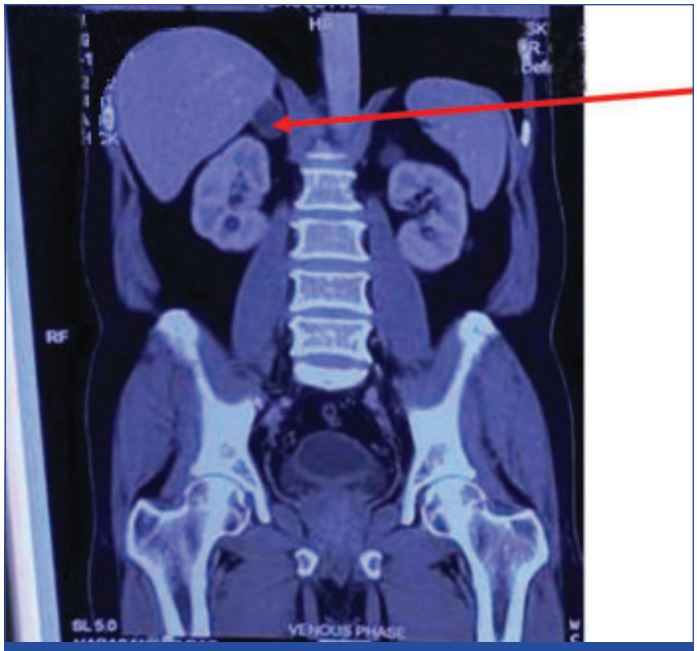


[Table/Fig-5]: CECT abdomen (Axial section) of patient with primary aldosteronism (case 3) showing right-sided adrenal adenoma.

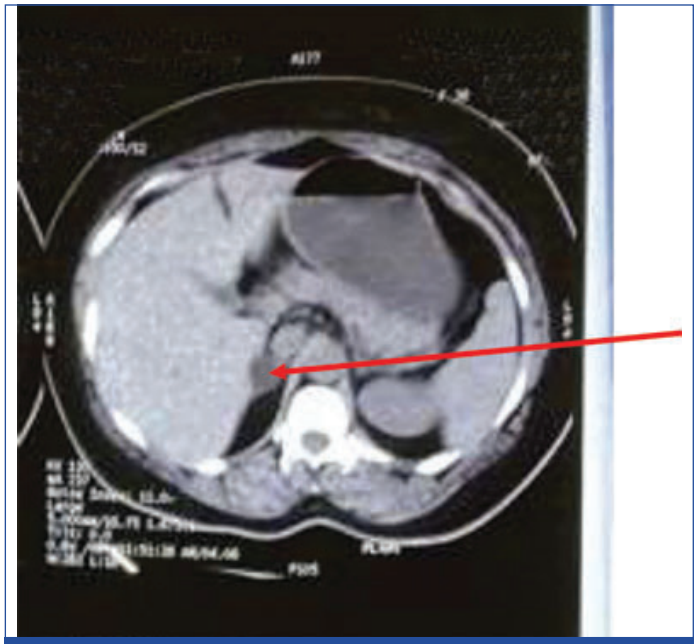
DISCUSSION

Dr. Litynski reported PA in 1953, which involves the overproduction of the hormone aldosterone that suppresses renin and inhibits the influx of sodium and the efflux of potassium, resulting in hypertension and hypokalaemia [8]. PA should be considered in patients with severe hypertension or drug-resistant hypertension when taking three or more antihypertensives, including a diuretic, or in those with a family history of early-onset hypertension before the age of 40 years, and always in patients with an adrenal adenoma and HTN [9].

PA usually manifests between 30-50 years of age. The mean age in this case series was 46 years. Alam S et al., studied 202 patients with young-onset HTN and got 36 cases (17.8%) of PA. The mean age in their study was 43.9±10.9 years [10]. Late-onset disease has also been described in the literature [11].



[Table/Fig-6]: Coronal section of CECT abdomen of the (case 3) showing right-sided adrenal adenoma.



[Table/Fig-7]: CECT abdomen (Axial section) of patient with primary aldosteronism (case 4) showing right-sided adrenal adenoma.

In this case series, 81.81% of patients presented with hypokalaemia, and 45.45% presented with acute onset quadriparesis, signifying the severity of hypokalaemia. The frequency of hypokalaemia in PA varies widely in different studies, ranging from 9-37% [12]. Burrello J et al., studied 5100 patients with HTN and 804 patients were identified with hypokalaemia (15.8%). PA was diagnosed in 226 of these 804 patients (28.1%). 8.3% had an adrenal adenoma, and 15.3% were diagnosed with bilateral PA [13].

Bilateral Adrenal Hyperplasia (BAH) and Aldosterone-Producing Adenoma (APA) are the most frequent causes of PA, with a prevalence of 70% and 30% respectively [14]. Surprisingly, all patients in the current case series had unilateral involvement, with five of them on the right side and the remaining six on the left side. Sometimes, aldosterone-producing microadenomas and most bilateral lesions are undetectable with computed tomography or magnetic resonance imaging because of poor accuracy [15,16].

The majority of patients (81.81%) had PAC levels above 20.0 ng/dL, with only two had PAC <20.0 ng/dL. One patient had a PAC of only 10 ng/dL, signifying that a fixed cut-off should always be combined with clinical acumen as well as other investigation modalities.

PRA was suppressed in eight cases. The Endocrine Society guidelines reinforce the specificity of elevated aldosterone levels (>20 ng/dL) in the presence of hypokalaemia and suppressed renin [17]. In this case series, nine out of 11 patients had an Aldosterone Renin ratio (ARR) >20. PA cases are screened based on an ARR >20 (using PAC in ng/dL and PRA in ng/mL/hr) [17]. ARR can vary widely in different populations, and no single ARR threshold is recommended for clinical decision-making [18]. Adrenal Venous Sampling (AVS) was done in two patients with age more than 40 years but the results were inconclusive. AVS was not performed in other eligible patients due to the clear diagnosis and the technical difficulty of the procedure [19]. CT scan of the abdomen was used as the imaging procedure. Most adenomas were more than 1 cm and hypodense with a washout of more than 50%, consistent with the literature [20]. Six patients (54.54%) underwent unilateral adrenalectomy, resulting in normalisation of potassium levels and a reduction of more than 50% in blood pressure. In a worldwide cohort of 380 patients with PA, complete and partial success rates of 30% and 48%, respectively, were achieved in patients undergoing unilateral adrenalectomy according to the PA surgical outcomes consensus [21].

CONCLUSION(S)

PA is an important cause of hypertension that often goes undiagnosed. Hypokalaemia is common and is often associated with acute onset paralysis of the limbs. This case series highlights the need to screen for PA in patients with hypertension and hypokalaemia or resistant hypertension. A plasma aldosterone level more than above 20 ng/dL with suppressed plasma renin activity confirms the diagnosis in most cases. Most lesions are small and unilateral. It is important to diagnose PA early, as it is a curable condition. Once PA is diagnosed, an adrenal CT scan with adrenal protocol should be performed, followed by AVS in eligible cases to localise the adenoma and determine whether surgical or pharmacological treatment with mineralocorticoid receptor antagonists is appropriate.

Ethical issues: This case series included patients who were seen in the Endocrinology OPD or admitted to the IPD under the Endocrinology Department over the past three years and were managed as per standard protocols. No active intervention was taken for research purposes during their admission, and patient identities were not disclosed. Patients provided consent in accordance with institutional protocols.

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