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Primary aldosteronism diagnosis in the intensive care unit: resistant alkalosis and hypokalemia during severe sepsis with hyperlactatemia: a case report

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Abstract

Background Primary aldosteronism screening indications include hypertension (resistant, severe, early onset, with stroke/other comorbidities/sleep apnea), hypokalemia, adrenal incidentaloma, and primary aldosteronism first-degree relatives. We report rare diagnosis of primary aldosteronism in intensive care unit setting, characterized by resistant alkalosis and hypokalemia during severe sepsis with hyperlactatemia.

Case presentation A 50-year-old Asian-Indian male patient with 18-year history of hypertension (blood pressure 166/104 mmHg) presented with acute septicemia and septic shock following an outpatient urethral dilatation. Despite aggressive management, including intravenous fluids, inotropes, antibiotics, and potassium supplementation, he exhibited severe alkalosis and resistant hypokalemia. Initial laboratory findings showed blood pressure 90/70 mmHg, heart rate 109 beats per minute, pH 7.49, serum lactate 123 mmol/L, sodium 141–144 mmol/L, potassium 2.7–2.9 mmol/L, and creatinine 1.2–1.54 mg/dL (106.1–136.1 µmol/L). Abdominal imaging revealed left adrenal adenoma (20 mm × 19 mm). Patient improved with supportive care and was discharged on day 10 with reinstituted antihypertensive medications.

Post-hospitalization, endocrine evaluation confirmed primary aldosteronism with plasma renin activity 0.62 ng/mL/hour, serum aldosterone 43.2 ng/dL (1.20 nmol/L), and aldosterone–renin ratio 69.7. After initiation of spironolactone, blood pressure significantly improved (currently 122/76 mmHg).

Conclusion Severe sepsis and septic shock in the intensive care unit typically present with metabolic acidosis. This case highlights an atypical presentation of paradoxical, resistant hypokalemia and alkalosis during severe sepsis, leading to a diagnosis of primary aldosteronism. Does the "inbuilt" tendency to metabolic alkalosis in primary aldosteronism confer survival advantage during intercurrent episodes of sepsis and metabolic acidosis? Given the high prevalence of renin-independent aldosterone production and benefits of mineralocorticoid receptor antagonists, universal primary aldosteronism screening for newly diagnosed hypertension appears meritorious and cost-effective.

Keywords Primary aldosteronism, Hypertension, Sepsis, Hyperlactatemia, Metabolic alkalosis, Hypokalemia

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Background

Primary aldosteronism (PA) is recognized as the most common cause of secondary hypertension, contributing significantly to the overall burden of cardiovascular disease. Despite its prevalence, PA is frequently underdiagnosed due to its heterogeneous clinical presentations. The pathophysiology of PA involves the autonomous overproduction of aldosterone from the adrenal glands, leading to hypertension and hypokalemia. This condition results in a range of clinical manifestations from mild hypertension to severe, resistant forms of the disease, often complicated by hypokalemia and metabolic alkalosis. The condition is implicated in causing more end-organ damage than essential hypertension, including a higher risk of cardiovascular morbidity such as heart failure, stroke, myocardial infarction, and atrial fibrillation [1, 2].

Current recommendations for screening PA are based on a variety of clinical scenarios in which the likelihood of PA is increased. Indications for screening for primary aldosteronism include: 1. resistant hypertension (RH; defined as blood pressure that remains above goal despite the use of three antihypertensive medications of different classes, including a diuretic; patients who require four or more antihypertensive agents to control blood pressure); 2. hypertension with hypokalemia [spontaneous hypokalemia (serum potassium < 3.5 mmol/L); hypokalemia induced by low-dose diuretics]; 3. severe hypertension (blood pressure > 150/100 mmHg on three separate measurements on different days); 4. hypertension and an adrenal mass [incidental discovery of an adrenal mass (incidentaloma) during imaging studies in hypertensive patients]; 5. early onset hypertension (hypertension diagnosed before the age of 30 years without a family history of hypertension or obesity); 6. family history of early onset hypertension or stroke (particularly if there is a history of hypertension or cerebrovascular accidents/strokes at a young age, < 40 years); 7. first-degree relatives of patients with primary aldosteronism (screening may be considered for first-degree relatives, especially if they have hypertension or other signs suggestive of PA); 8. hypertension with sleep apnea (patients with confirmed obstructive sleep apnea and hypertension may have an increased prevalence of primary aldosteronism); and 9. hypertension with other comorbidities (presence of cardiovascular diseases, such as left ventricular hypertrophy, atrial fibrillation, or stroke, particularly if hypertension is difficult to control). Despite these guidelines, PA remains underdiagnosed, in part due to the complexity of its presentations and the lack of widespread awareness among clinicians.

Pathophysiology and diagnosis

PA can be broadly categorized into unilateral aldosterone production, typically from an aldosterone-producing adenoma (APA) and bilateral adrenal hyperplasia (BAH). Both conditions result in excessive aldosterone production, but their pathophysiological mechanisms and clinical implications differ. APAs are more straightforward to diagnose and treat surgically, whereas BAH, characterized by bilateral adrenal nodular hyperplasia, presents more diagnostic challenges and typically requires medical management [3–5].

The diagnostic process for PA usually begins with the measurement of the aldosterone–renin ratio (ARR), a screening test that detects inappropriate aldosterone production relative to plasma renin activity. Confirmatory tests, such as saline infusion test, oral sodium loading test, or captopril challenge test, are used to establish the diagnosis. Cross-sectional imaging, typically with computed tomography (CT) or magnetic resonance imaging (MRI), is employed to visualize adrenal morphology and identify potential adenomas [1, 3, 6].

In this manuscript, we present an unusual clinical situation, involving a patient with paradoxical, resistant, and enigmatic hypokalemia and alkalosis during severe septicemia and hyperlactatemia, leading to diagnosis of primary aldosteronism, in an intensive care unit.

Case presentation

A 50-year-old Asian-Indian male with a history of hypertension [since age 32 (blood pressure, BP, 166/104 mmHg); on bisoprolol (5 mg/day), amlodipine (5 mg/day; olmesartan (40 mg/day); hydrochlorothiazide (12.5 mg/day)], dyslipidemia (age 45), prediabetes (age 50), and chronic urethral stricture (age 24), presented with fever, acute septicemia, and septic shock approximately 2–3 hours after an outpatient urethral dilatation procedure for chronic urethral stricture, which had recently led to acute urinary retention. Initial vital signs showed BP at 90/70 mmHg, heart rate (HR) at 109 beats per minute, and oxygen saturation (SpO2) at 96%. Arterial blood gas (ABG) analysis indicated a pH of 7.49, pCO2 of 30.9 mmHg (4.12 kPa), pO2 of 53.8 mmHg (7.17 kPa), and bicarbonate (HCO3) at 23 mmol/L. Laboratory findings included serum lactate at 123 mmol/L (reference range 0.5–1), sodium 141–144 mmol/L, potassium 2.7–2.9 mmol/L, chloride at 109 mmol/L, serum creatinine at 1.2 mg/dL (106.1 µmol/L), white blood cell (WBC) count at 1610/µL (acute bone marrow suppression), thrombocytopenia 107×10^9/L, and C-reactive protein (CRP) at 7.3 mg/dL (695 nmol/L) (reference range 0.3–1 mg/dl; 28.5–95.2 nmol/L) (Table 1).

The presence of respiratory alkalosis (indicated by a high pH and low pCO2) along with severely elevated

lactate levels (indicating lactic acidosis) suggested a mixed acid–base disorder. In this case, the respiratory alkalosis was likely compensatory for underlying hypoxemia or shock, while the extremely high lactate indicated severe lactic acidosis, pointing to a coexisting metabolic acidosis.

Despite aggressive management with intravenous fluids, triple inotropes, oxygen, intravenous antibiotics, and potassium supplementation, the patient exhibited severe metabolic alkalosis and resistant hypokalemia. These imbalances persisted from days 2–4, with fluctuating pH levels (7.34–7.53), low pCO2 (22.4–29.7 mmHg) (3.01–3.96 kPa), and persistently high serum lactate levels. Procalcitonin levels were significantly elevated (33.8 to > 90 µg/L; reference range < 0.1 µg/L), indicating ongoing severe infection. (Table 1).

The complicated clinical course included severe acute leukopenia at sepsis onset (toxic bone marrow suppression), progressive thrombocytopenia, cystitis, bilateral pyelonephritis, septic shock, acute kidney injury (serum creatinine 1.89 mg/dl; 167.1 µmol/L), and acute liver dysfunction (serum bilirubin 3.9 mg/dl; 66.7 µmol/L), N-terminal pro b-type natriuretic peptide (NT proBNP) 5880 pg/mL (695 pmol/L) (reference range < 125 pg/ml; < 14.78 pmol/L)—multiorgan failure. Blood culture grew ESBL *E. coli.*

The patient's mixed acid–base imbalance, characterized by dominant metabolic alkalosis and severe resistant hypokalemia, prompted suspicion of undiagnosed PA as a potential underlying cause. Abdominal imaging (CT) revealed a left adrenal adenoma measuring 20 mm × 19 mm (15 Hounsfield units, HU), indicative of a possible aldosterone-producing adenoma (Fig. 1).

Following supportive care, the patient's condition improved, and he was discharged on day 10 with reinstituted antihypertensive medication (amlodipine 5 mg/day) and oral antibiotics.

Post-hospitalization endocrine evaluation confirmed PA with plasma renin activity of 0.62 ng/mL/hr, serum aldosterone of 43.2 ng/dL (1.20 nmol/L), and an aldosterone–renin ratio of 69.7. MRI of the adrenals indicated left adrenal adenoma measuring 22 mm × 14 mm × 21 mm. His serum sodium was 135 mEq/L, serum potassium 3.3 mEq/L, and serum creatinine 0.77 mg/dl (68.1 µmol/L). Upon initiation of spironolactone (50 mg/day), the patient experienced significant improvement in BP, reducing to 106/81 mmHg. A total of 2 months later, spironolactone was changed to eplerenone (75 mg/day) due to bilateral mastalgia. His urine albumin–creatinine ratio (UAC) was 926 µg/mg of creatinine. (Table 1; Figs. 1 and 2).

His current (2024) blood pressure is 120–126/70–80 mmHg and pulse rate 60–70 beats per minute on telmisartan (40 mg/day), amlodipine (5 mg/day), and eplerenone (75 mg/day). His serum sodium is 139 mEq/L, serum potassium 4.1 mEq/L, and serum creatinine 0.73 mg/dl (64.5 µmol/L). Repeat CT abdomen indicates stability of the left adrenal adenoma 23 mm × 16 mm × 22 mm (5.5 HU) (Table 1). The patient and family have opted for medical management of PA currently.

There was a very strong family history of hypertension in father, mother, brother, two paternal uncles, two maternal uncles, and three maternal aunts. None of the family members had a history of diabetes mellitus.

Discussion and conclusion

This case illustrates an example of atypical/unusual presentation (paradoxical, resistant hypokalemia, and alkalosis during severe sepsis), which led to the diagnosis of PA in the intensive care unit. Recognizing such unusual clinical scenarios is crucial for timely diagnosis and treatment, ultimately improving patient outcomes, and reducing the risk of cardiovascular and renal complications.

Complexity of acid–base disorders in sepsis and septic shock

Acid–base disorders in sepsis and septic shock are complex, reflecting a dynamic interplay of systemic responses to infection. Metabolic acidosis, primarily lactic acidosis, is the most common disturbance due to tissue hypoxia and anaerobic metabolism. This condition is often accompanied by hyperchloremic acidosis, resulting from chloride-rich fluid resuscitation and respiratory alkalosis, driven by hyperventilation in response to systemic inflammation. These disorders are pivotal in clinical assessment, offering insights into disease severity and guiding therapeutic interventions.

Mixed acid–base disturbances are also prevalent. A mixed acid–base disorder is the simultaneous coexistence of two or more primary acid–base disorders in the same patient. Mixed acid–base disorders may be suspected on the basis of findings obtained from the medical history, physical examination, serum electrolytes and chemistries, and anion gap. Combined metabolic acidosis and respiratory alkalosis frequently occur, indicating severe systemic disturbances. Metabolic alkalosis, though rarer, can arise from diuretic use or gastrointestinal losses. Renal dysfunction exacerbates these disturbances, contributing to non-anion gap metabolic acidosis. Understanding these disorders is crucial for timely and effective treatment, impacting patient outcomes significantly. Monitoring and managing acid–base balance is essential in the critical care of patients with sepsis, highlighting the need for tailored therapeutic strategies on the basis of individual pathophysiological responses [7–10].

Table 1 Journey of a 50-year-old man with delayed and missed (and subsequent serendipitous) diagnosis of primary aldosteronism (2004–2022–2024): summary of clinical and biochemical observations in the case under discussion

Year Date	Clinical events	Hypertension medicines	pH	PaO2	PaCO2	HCO3	Blood lactate	Serum Na	Serum K	Serum creatinine	BP	WBC count	Platelet count	Serum procalcitonin	Serum CRP
				mmHg (kPa)	mmHg (kPa)	mEq/L (mmol/L)	mmol/L	mEq/L (mmol/L)	mEq/L (mmol/L)	mg/dl (µmol/L)	mmHg	/µL	10^3/µL	ng/mL (µg/L)	mg/dL (nmol/L)
Ref range			7.35–7.45	75–100 (9.99–13.33)	35–45 (4.66–5.99)	22–26 (22–26)	0.5–1	136–145 (136–145)	3.5–5.1 (3.5–5.1)	0.72–1.18 (63.7–104.3)		4000-10,000	150–400	< 0.1	0.3–1 (29–95)
2004–2017	Hypertension; nasal bleed; dyslipidemia, pre-diabetes; chronic urethral stricture	Bisoprolol Amlodipine Olmesartan Hydrochlorothiazide						141	3.7	0.85 (75.1)	166/104	6110	194		
2021		Same						140	3.5	0.87 (76.9)	142/88	7490	229		1.4 (133)
2022	September	Admission													
September 10	Urinary retention; urethral dilatation; acute septicemia; septic shock; leukopenia, thrombocytopenia; prediabetes	Nil	7.49	53.8 (7.17)	30.9 (4.12)	23.0	123	141	2.9	1.20 (106.1)	90/70	1610	107	5.7	
September 11		Nil	7.41	71.9 (9.59)	22.6 (3.01)	14.1	86	144	2.7	1.54 (136.1)	92/66	4500	78	64.2	7.3 (695)
September 12		Nil	7.34	25.0 (3.33)	29.7 (3.96)	15.6	58	138	3.5	1.89 (167.1)	94/62		58	> 90.0	142 (13523)
September 12		Nil	7.53	48.8 (6.51)	27.3 (3.63)	23.3	51		3.5	1.54 (136.1)	90/60	19840	48	33.8	
September 13	Serum bilirubin 3.9 mg/dl	Nil	7.48	76.0 (10.13)	22.4 (2.99)	16.6		141	3.8	1.39 (122.9)	92/63	22900	35		301 (28666)
September 13	Multiorgan failure, acute kidney injury; liver dysfunction	Nil	7.48	58.4 (7.79)	27.5 (3.66)	20.4	29	140	3.3	1.05 (92.8)	90/60	17250	27		
September 14		Nil	7.46	60.1 (8.01)	26.7 (3.56)	18.7	26	136	3.9	0.90 (79.6)	94/62	17530	16	16.8	209 (19904)
September 15		Nil	7.40	32.2 (4.29)	37.0 (4.93)	23.4	18	136	4.2	0.85 (75.1)	100/72	13930	39	9.7	
September 16	CT abdomen: left adrenal adenoma, 20 mm × 19 mm (15 HU)	Nil	7.45	36.1 (4.81)	33.0 (4.40)	22.9		136	4.1	0.76 (67.2)	102/73	18910	52	5.6	
September 17		Nil						140	3.5	0.69 (60.9)	120/74	23190	76		
September 18		Nil													
September 19		Amlodipine						141	3.5	0.69 (60.9)	134/86		166		
2022	September	Discharge													

Table 1 (continued)

Year Date	Clinical events	Hypertension medicines	pH	PaO2 mmHg (kPa)	PaCO2 mmHg (kPa)	HCO3 mEq/L (mmol/L)	Blood lactate mmol/L	Serum Na mEq/L (mmol/L)	Serum K mEq/L (mmol/L)	Serum creatinine mg/dl (μmol/L)	BP mmHg	WBC count /μL	Platelet count 10^3/μL	Serum procalcitonin ng/mL (μg/L)	Serum CRP mg/dL (nmol/L)
October	Primary aldosteronism; ARR: 69.7	Telmisartan Amlodipine Bisoprolol (silodosin)						135	3.3	0.77 (68.1)	130/80	9870			2.5 (238)
October	MRI abdomen: left adrenal adenoma, 22 mm × 14 mm × 21 mm	Spironolactone: added						139	3.1	0.76 (67.2)	132/81 106/81				
October–December	Mastalgia bilateral	Changed to eplerenone						139	4.1	0.77 (68.2)	126/78	9730	200		
Follow-up	2023–2024														
2023								140	4.0	0.81 (71.6)	128/72	5670			
2024	CT abdomen: left adrenal adenoma, 23 mm × 16 mm × 22 mm (5.5 HU)	Telmisartan Amlodipine Eplerenone						139	4.1	0.73 (64.5)	122/76				

Endocrine evaluation confirmed PA: plasma renin activity 0.62 ng/ml/hour and 0.62 μg/L/hour; serum aldosterone 43.2 ng/dL; 1.20 nmol/L; aldosterone: renin ratio of 69.7

BP blood pressure, *CRP* C-reactive protein

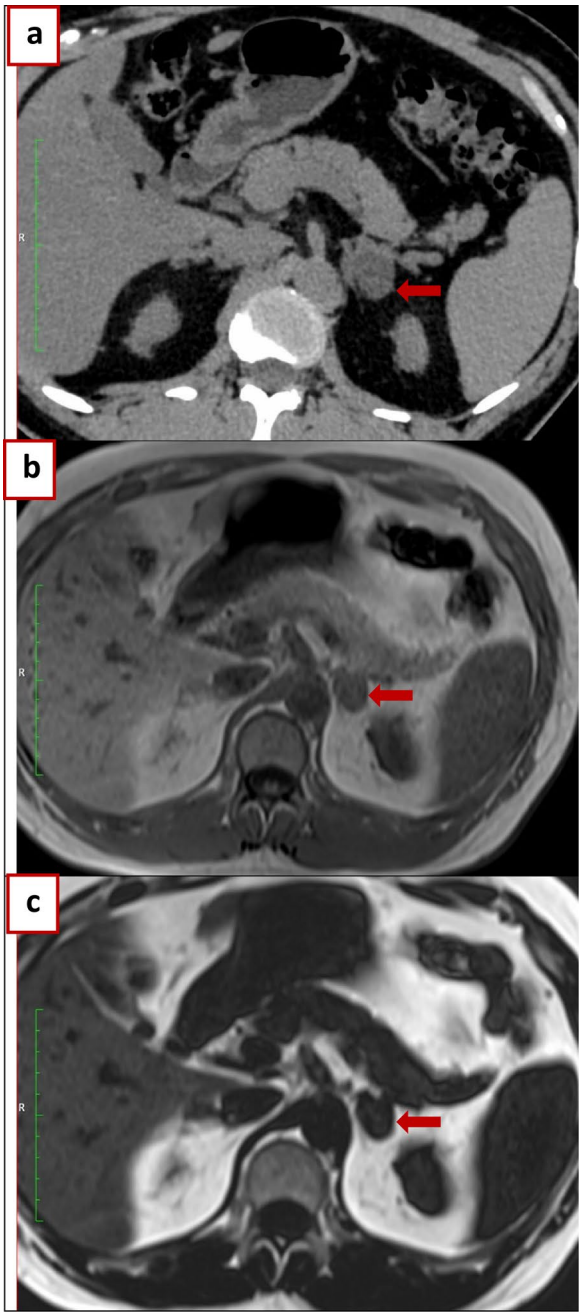

Fig. 1 Abdominal imaging: **a** computed tomography scan: left adrenal gland shows an isodense soft tissue nodule (red arrow), indicating left adrenal adenoma, measuring 20 mm × 19 mm (15 Hounsfield units). **b** and **c** Magnetic resonance imaging scan: right adrenal gland is normal in size and signal intensity. Left adrenal gland shows a well-defined round to oval T1-weighted/T2-weighted iso- to hypointense lesion epicentered in the medial limb of the gland (red arrows), measuring 22 mm × 14 mm × 21 mm, indicating left adrenal adenoma. The lesion shows few areas of signal drop on outphase images. The lesion is slightly hyperintense on DWI (Diffusion-Weighted Imaging)

Does the "inbuilt" tendency to metabolic alkalosis in primary aldosteronism provide survival advantage during intercurrent sepsis and metabolic acidosis?

The commonest acid–base abnormality in sepsis is acidosis. Specifically, metabolic acidosis is frequently observed due to lactic acid accumulation from anaerobic metabolism, decreased tissue perfusion, and impaired oxygen utilization. Respiratory acidosis can also occur if there is respiratory failure. However, metabolic acidosis is the predominant issue.

The most common acid–base abnormality in primary aldosteronism is metabolic alkalosis. This condition arises due to the excess production of aldosterone, which leads to increased reabsorption of sodium and excretion of potassium and hydrogen ions in the kidneys. The loss of hydrogen ions results in an increase in blood pH, causing metabolic alkalosis.

A recent retrospective study from Taiwan explored the incidence of sepsis and all-cause mortality in patients with PA, presenting intriguing findings that prompt further discussion. This study, by Chan *et al.*, suggests a lower incidence of sepsis and reduced mortality in patients with PA following targeted treatments. Our hypothesis is that the "inbuilt" tendency to metabolic alkalosis in PA may confer a survival advantage during episodes of sepsis and metabolic acidosis. (Also please refer to: Supplementary Discussion).

The study by Chan *et al.* utilized Taiwan's National Health Insurance Research Database to identify 2448 patients with PA, including those with aldosterone-producing adenoma (APA) and bilateral adrenal hyperplasia (BAH). The main outcomes were the incidence of sepsis and all-cause mortality post-diagnosis and treatment. The findings indicated that patients with APA had a lower incidence of sepsis (HR 0.29, $P < 0.001$) and lower all-cause mortality (adjusted HR 0.31, $P < 0.001$) compared with patients with matched essential hypertension (EH).

While the study is informative, several limitations must be considered. Categorization: the analysis could have been more comprehensive with an additional category for patients without APA. Treatment effects: the effects of different treatments [adrenalectomy versus mineralocorticoid receptor antagonist (MRA)] were not segregated. Timing: the study did not specify the time intervals between diagnosis, treatment initiation, and sepsis episodes. Despite these limitations, the study raises important questions about the mechanisms underlying the observed protective effects.

Hypothesis: metabolic alkalosis of PA as a protective mechanism. We hypothesize that the tendency toward metabolic alkalosis in PA might confer a survival advantage during sepsis, which typically induces metabolic

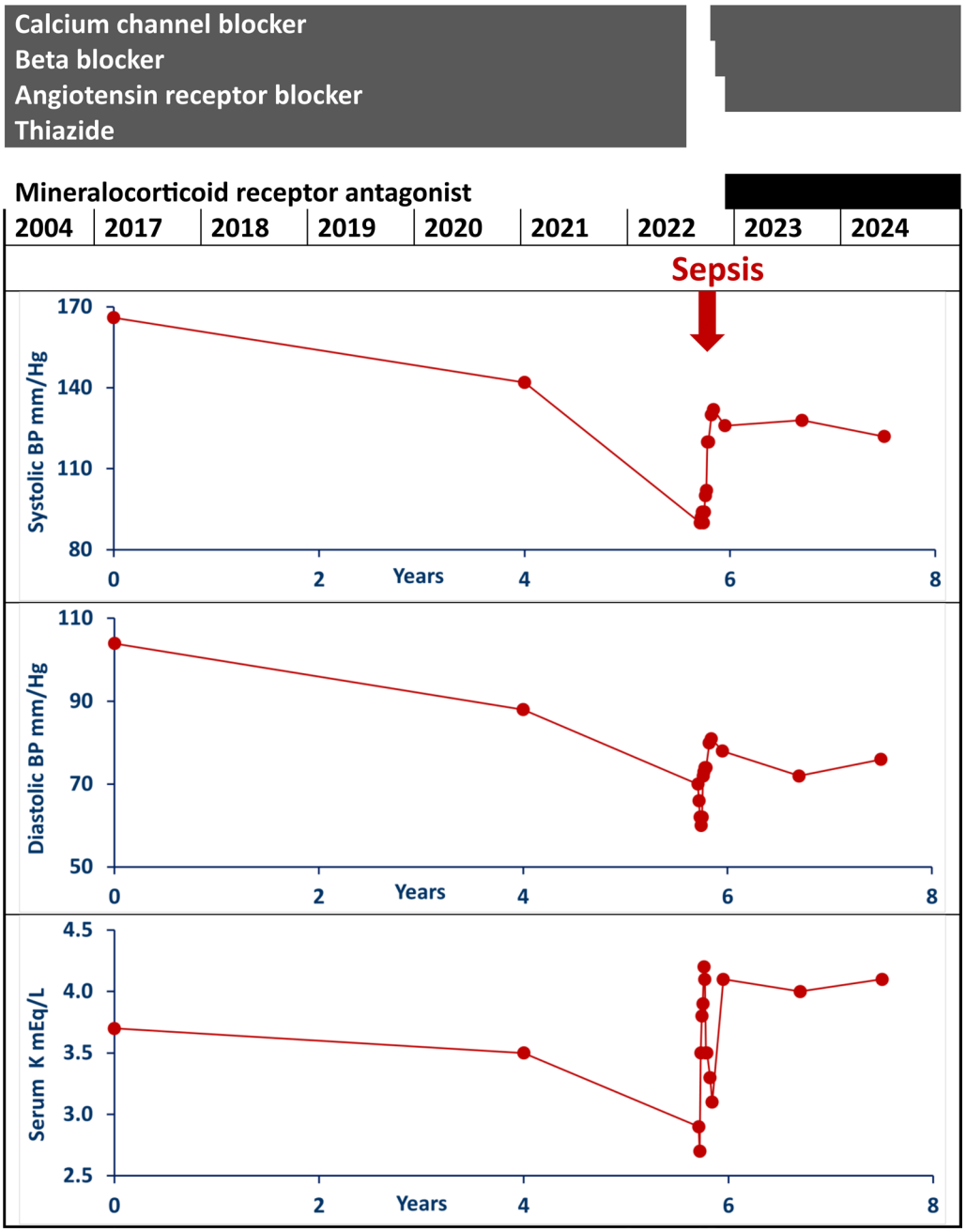

Fig. 2 Journey of the 50-year-old man with delayed and missed (and subsequent serendipitous) diagnosis of primary aldosteronism: 2004–2022–2024; serial salient clinical (systolic and diastolic blood pressure) and biochemical (serum potassium) features. Hypertension diagnosis in 2004. Red arrow marks September 2022 (emergency intensive care unit admission with events of urinary retention, urethral dilatation, acute septicemia, septic shock, hypotension, hypokalemia, leukopenia, and thrombocytopenia)

acidosis. This could be an example of "one disease providing a protective effect against another." Examples from other medical conditions include (a) sickle cell trait and malaria: the trait provides a protective advantage against *Plasmodium falciparum*; (b) glucose-6-phosphate dehydrogenase (G6PD) deficiency and malaria: G6PD deficiency offers protection against malaria by limiting the parasite's access to the host's red blood cells; (c) cystic fibrosis and tuberculosis: the thick mucus in cystic fibrosis can limit *Mycobacterium tuberculosis* colonization; and (d) other examples (also please refer to: Supplementary Discussion).

Possible mechanisms of PA-associated protection in sepsis: metabolic compensation. The chronic metabolic alkalosis in PA could buffer the acidosis that occurs during sepsis, mitigating the severity of the acidosis and its complications. Adrenalectomy benefits: the reduced sepsis incidence and mortality post-adrenalectomy could be due to the surgical removal of the aldosterone-producing tumor, reducing hyperaldosteronism's adverse effects on the immune system. Immune function: PA and its treatments might modulate immune responses, potentially reducing sepsis risk. However, the interactions between hyperaldosteronism, glucocorticoid co-secretion in PA, and immune dysfunction require further exploration [11].

Spectrum or continuum of "renin-independent aldosterone production": suggestion of universal screening ("pre-primary" aldosteronism) in newly diagnosed hypertension

The pathogenesis of PA involves autonomous aldosterone secretion from the adrenal glands, independent of renin-angiotensin signalling. This condition is typically classified into unilateral aldosterone-producing adenoma (APA) and bilateral adrenal hyperplasia (BAH). However, recent advances in immunohistochemistry and molecular genetics have revealed a broader spectrum of aldosterone overproduction, including subtle forms such as aldosterone-producing cell clusters (APCCs) and microadenomas. These milder forms often go undetected with current screening thresholds, leading to underdiagnosis and suboptimal management of patients with hypertension [4, 12].

Renin-independent aldosterone production (RIAP) represents a spectrum of pathophysiological conditions ranging from mild to severe. This continuum includes various forms of primary aldosteronism (PA), characterized by excessive aldosterone production despite low renin levels. Recognizing the high prevalence of RIAP and its significant health implications, there is a prudent argument for universal screening for RIAP in all newly diagnosed hypertension cases [13–15]. However, the cost-effectiveness, practicality, and validity of this recommendation should come from randomized control trials.

PA is associated with significant cardiovascular and renal morbidity, including an increased risk of heart failure, myocardial infarction, stroke, and chronic kidney disease. Early detection and treatment with mineralocorticoid receptor antagonists (MRAs) such as spironolactone or eplerenone can substantially mitigate these risks. MRAs not only improve blood pressure control, but also offer protective effects on the heart and kidneys, reducing long-term healthcare costs associated with uncontrolled hypertension and its complications.

Current guidelines for PA screening are based on specific clinical indicators such as resistant hypertension (RH), spontaneous or diuretic-induced hypokalemia, and adrenal incidentalomas. However, these criteria may miss a significant proportion of patients with milder forms of PA. Given the continuum nature of RIAP, it is plausible that many patients with newly diagnosed hypertension may harbor undetected aldosterone excess, contributing to their hypertensive state and associated risks [6, 16–18].

The proposed concept of "subclinical" or "pre-primary" aldosteronism, analogous to prediabetes or prehypertension, suggests that individuals with slightly elevated aldosterone–renin ratios (ARR), even within the currently accepted normal range, could benefit from early intervention. Screening all newly diagnosed hypertensive patients for PA using a lower ARR threshold could identify those at risk for developing more severe forms of PA. This proactive approach could facilitate timely initiation of MRA therapy, potentially preventing the progression of cardiovascular and renal disease.

Implementing universal screening for PA in newly diagnosed hypertensive patients involves practical challenges, including the need for widespread availability of reliable ARR testing and standardized diagnostic protocols. However, the potential benefits in terms of improved patient outcomes and reduced healthcare costs justify these efforts. Additionally, advancements in diagnostic technologies and increased awareness among healthcare providers can streamline the screening process and ensure timely identification and management of PA. Future research should focus on refining screening strategies and exploring the full spectrum of PA to optimize patient care.

Prevalence of primary aldosteronism in the general population and in hypertensive patients

PA was historically considered a rare cause of hypertension, but contemporary research has redefined it as the most common etiology of secondary hypertension. Epidemiological studies estimate its prevalence at approximately 20% among patients with RH, 10% in those with severe hypertension, and 6% in individuals with otherwise uncomplicated hypertension. Despite these significant figures, PA remains vastly underdiagnosed and undertreated, with a screening rate reported at a mere 2.1% in patients with RH [19].

Severe sepsis and septic shock in the intensive care unit (ICU) typically present with metabolic acidosis. This case highlights an atypical presentation of paradoxical, resistant hypokalemia and alkalosis during severe sepsis, leading to a diagnosis of PA. Does the "inbuilt" tendency to metabolic alkalosis in PA confer

survival advantage during intercurrent episodes of sepsis and metabolic acidosis? The importance of maintaining a high index of suspicion for PA in diverse clinical situations is reemphasized. Given the high prevalence of renin-independent aldosterone production and benefits of mineralocorticoid receptor antagonists, universal PA screening for newly diagnosed hypertension appears meritorious and cost-effective.

Abbreviations

PA	Primary aldosteronism
APA	Aldosterone-producing adenoma
BAH	Bilateral adrenal hyperplasia
ARR	Aldosterone–renin ratio
ABG	Arterial blood gas
EH	Essential hypertension
MRA	Mineralocorticoid receptor antagonist
RIAP	Renin-independent aldosterone production
RH	Resistant hypertension

Supplementary Information

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Supplementary material 1.

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Author contributions

All authors contributed to clinical care, data collection, analysis and interpretation of results, and manuscript preparation. All authors reviewed and approved the final draft.

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Availability of data and materials

The datasets analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Ethical Committee of Samatvam Trust (Science for Health; 2024-07-10) approved this presentation.

Consent for publication

Written informed consent was obtained from the patient and patient's next-of-kin for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

The authors declare that they have no competing interests.

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