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Primary Aldosteronism Presenting as Chronic Hypokalemia in a Normotensive Patient

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Introduction: Conn first described the hallmark features of primary aldosteronism (PA) as a state of aldosterone excess in the setting of hypertension and hypokalemia. Recent work has shed light on a wide spectrum of disease that likely includes normotensive patients with elevated aldosterone and suppressed renin. Mineralocorticoid excess appears to be the main driver of disease progression; however, it is not known whether individual differences in intra-arterial plasticity and effective sodium potassium exchange alter the overt clinical presentation. Here, we describe a patient with chronic hypokalemia and normal blood pressure with the diagnosis of a unilateral aldosterone-producing adenoma.

Clinical Case: A 47-year-old woman with no significant medical history was referred to endocrinology for evaluation of isolated hypokalemia since 2012. Her potassium was 2.8mEq/L at its lowest a month prior to her visit and had numerous other values less than 3.5mEq/L over the past 10 years. She did not carry a diagnosis of hypertension nor was she hypertensive at the time of her initial and subsequent evaluations. Results on initial endocrinology evaluation demonstrated a potassium of 3.2mEq/L (3.5-5.0mEq/L), Plasma Aldosterone Concentration (PAC) of 29ng/dL (≤21ng/dL), Plasma Renin Activity (PRA) of 0.42ng/mL/h (0.25-5.82ng/mL/h), and Aldosterone Renin Ratio (ARR) of 69. PA was suspected based on hypokalemia with an ARR >30 and PAC >20ng/dL. Further work-up was pursued to confirm the diagnosis as the patient did not have hypertension. She underwent 3-day oral sodium loading. 24-hour urine collection at the conclusion revealed adequate sodium loading with a urine

sodium content of 208mEq/24hr (>200mEq/24hr) and aldosterone secretion of 41.3mcg/24hr (<12mcg/24hr). She subsequently underwent a CT abdomen demonstrating a 1.3×0.8 cm right adrenal nodule. Adrenal venous sampling was performed (continuous cosyntropin infusion with simultaneous adrenal venous sampling) which revealed a cortisol-corrected aldosterone lateralization ratio (right to left) of 406 at -5 minutes, 368 at 0 minutes, 33 at 10 minutes and 85 at 15 minutes. Results were consistent with strong lateralization to the right with contralateral suppression. Diagnosis of a right-sided aldosterone-producing adenoma was confirmed and she was referred to surgery for right adrenalectomy.

Conclusion: This case highlights one of the many presentations of PA. The persistent hypokalemia for a decade suggests a history of longstanding untreated PA prior to eventual diagnosis, yet the patient has remained normotensive. Though normotensive PA is a recognized entity, most patients develop hypertension within 5 years. The ability to remain normotensive despite intravascular volume expansion may highlight the patient's highly compliant vasculature and kidneys that have likely adapted to excrete high sodium loads in the setting of sodium excess. This case emphasizes that isolated hypokalemia in patients without hypertension should prompt consideration of PA and that patients with PA may maintain normotension for multiple years.

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