Primary Hyperaldosteronism Presenting as Severe Symptomatic Hypocalcemia in a Postpartum Woman: A Case Report Highlighting its Variable Presentations and the Effects of Pregnancy

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Abstract

Background: Non-suppressible secretion of aldosterone is an important, underdiagnosed cause of secondary hypertension that typically presents with a triad of hypertension, hypokalemia, and metabolic alkalosis. However, its impact on calcium, magnesium, and parathyroid metabolism, as well as renal handling of these electrolytes, remains less documented. Additionally, descriptions of the variable presentation of primary hyperaldosteronism in pregnancy remain limited.

Objective: To highlight the broader clinical spectrum of primary aldosteronism beyond the classic triad of hypertension, hypokalemia, and metabolic alkalosis, and its varied presentation in pregnancy.

Method: We describe a case of a postpartum woman who presented with severe symptomatic hypocalcemia and grade 2 hypertension, whose evaluation diagnosed primary hyperaldosteronism.

Results: A 24-year-old female, para 4 + 0, presented one month postpartum with difficulty breathing and sudden spasms of her jaw, neck, and limb muscles, along with stiff posturing of her hands, without associated loss of consciousness. These symptoms interfered with her ability to care for her newborn. Her past medical history was significant for chronic hypertension diagnosed during her third pregnancy, associated with severe symptomatic hypocalcemia post-delivery. Following treatment with anti-hypertensives, Vitamin D, and calcium supplementation, she discontinued all her medications approximately a year later, with no further evaluation or follow-up documented. Her blood pressure, while off treatment, was within normal limits during her antenatal follow-up for her most recent pregnancy. There was no family history of hypertension or any other chronic illness. On general examination, her blood pressure was elevated at 175/65 mmHg, she was tachycardic at 122 b/min, afebrile, diaphoretic, in respiratory distress, and in pain. Both Chvostek's and Trousseau's signs were positive. The rest of her systemic examination was non-contributory. Targeted laboratory test revealed severe hypocalcemia (1.59mmol/L), hypokalemia (1.47mmol/L), hypomagnesemia (0.33 mmol/L), and severe metabolic alkalosis with respiratory compensation (pH=7.64, pCO2=57.6 mmHg, HCO3=64.8 mmol/L). Her urinalysis was normal. A 2D-echo found heart failure with reduced ejection fraction (left ventricular ejection fraction of 35-40%) and left ventricular hypertrophy. A CT coronary angiogram did not show any evidence of coronary arterial disease. Further evaluation revealed an elevated plasma Aldosterone: Renin Ratio (ARR) of 54.45, against a plasma renin activity of 1.75 ng/ml/h, suggestive of non-suppressible hyperaldosteronism. An adrenal protocol CT scan demonstrated a 3.3 x 2.0cm left adrenal mass consistent with a lipid-poor adenoma. The free plasma metanephrines and serum cortisol levels were within the normal range. Management included intravenous electrolyte supplementation and initiation of triple-agent antihypertensive therapy, with close monitoring in our high dependency unit. She awaits a planned laparoscopic adrenalectomy.

Conclusions: Our case underscores the diverse clinical presentation of primary aldosteronism. Recognition of electrolyte abnormalities beyond hypokalemia, including hypocalcemia and hypomagnesemia, is crucial in the evaluation

of secondary hypertension due to primary aldosteronism. Pregnancy may conceal the clinical symptoms of primary aldosteronism, presenting as unexpected severe hypertension in the postpartum period. Increased awareness of these variable presentations would hopefully lead to early diagnosis, intervention, and mitigation of the increased cardiovascular morbidity and mortality associated with untreated hyperaldosteronism.