

Case Report

Primary Hyperaldosteronism in a 38 Year Old Woman from Pakistan: A Case Report on Conn's Syndrome

Sajjad Ali Khan, Zafar Aleem Suchal, Muhammad Saleem, Kaleemullah Badini, Aisha Shaikh

Department of Medicine, Aga Khan Medical College, AKUH, Karachi, Pakistan

Abstract

Conn's syndrome refers to excess production of aldosterone by adrenal glands leading to an increase in blood pressure of the patient. There are often various other associated signs and symptoms due to which a patient may present often due to fluid overload and/or electrolyte abnormality. A 38 year old married female presented to the clinical with a raised blood pressure of 150-170 mmHg systolic and 100-120 mmHg diastolic. She also had a history of raised blood pressure during pregnancy with the first pregnancy being aborted and the second and third having markedly raised blood pressures. There was no associated history of palpitations, headache or anxiety. Her workup was done with a high aldosterone concentration and a low serum renin being detected. Her electrolytes were found to be normal and on ultrasound her kidneys were also normal. The patient was started verapamil to lower her blood pressure and a diagnosis of primary hyperaldosteronism was reached as the Plasma aldosterone to Renin ratio was raised with absence of any other cause.

Keywords: Hyperaldosteronism, Pakistan

How to cite this:

Khan SA, Suchal ZA, Saleem M, Badini K, Shaikh A. Primary Hyperaldosteronism in a 38 Year Old Woman from Pakistan: A Case Report on Conn's Syndrome. J Pak Soc Intern Med. 2022;3(1):63-65

Corresponding Author: Dr. Zafar Aleem Suchal

Email: sufna.foundation@gmail.com

Introduction

Primary Hyperaldosteronism or Conns Syndrome characterized by either idiopathic adrenal hyperplasia or adrenal adenoma leading to an excess production of aldosterone in the body.¹ Excess of this mineralocorticoid can lead to a plethora of problems including a suppression of renin, sodium and fluid retention leading to hypertension, loss of potassium causing hypokalemia and hypomagnesemia. The first case of primary aldosteronism was reported by Dr Litynski in 1953.² The diagnosis of different types of primary aldosteronism can be challenging as although bilateral adrenal hyperplasia and aldosterone secreting adenoma are the most common causes it can also manifest as rarer unilateral adrenal hyperplasia or glucocorticoid responsive aldosteronism.³

The hypertension and electrolyte imbalance in primary aldosteronism can complicate a patient's condition and can cause severe problems especially in a patient who is pregnant. It is a well-known fact that 8% of all pregnancies are made complicated because of hypertension. Despite 10% of these cases being due to hyperaldosteronism little data is available regarding it.⁴

Case

A 38 year old, married female, Para 1 Gravida 1+1, who was a housewife, belonging to a middle class family in Karachi with reported hypertension during previous pregnancies presented to the endocrine clinic at Aga Khan Hospital with the primary complain of raised blood pressure at home ranging from 150-170/100-110 mmHg. She did not have any history of steroid intake, anxiety, palpitations or headache. She had hypertension in both her previous pregnancies with the first pregnancy in 2016 being aborted due to hemodynamic instability. In 2017 she had raised blood pressure during third trimester of her second pregnancy and needed a preterm caesarian delivery following raised blood pressure. Now in her third pregnancy she has raised blood pressure from the first trimester with no other current complain.

During clinical examination she was found to have a BMI of 26.8kgm⁻² and a blood pressure of 140/90 mmHg. No other abnormality was found and the rest of general physical, CVS and respiratory examination was unremarkable. She was started on Calan SR (verapamil), a calcium channel blocker and was advised to

get a workup done to rule out the causes of hypertension.

The patient returned after a week with her reports. The patient had metanephrines and normetanephrines in normal range being 28 and 164 uIU/ml respectively. Her sodium was 134 and potassium was 3. Her serum TSH was 0.54, Cr was 0.6 and renin was 4. She had a raised aldosterone level of 28 uIU/ml and an elevated plasma aldosterone to renin ratio. An ultrasound of the kidneys was also done which showed normal functioning kidneys with no gross abnormality. Her hypertension was being managed with the medication given with now her average blood pressure at home being 125-130/80-90 mmHg.

A diagnosis of primary aldosteronism during pregnancy was reached because of the raised aldosterone and plasma aldosterone to renin ratio in presence of hypokalemia and the patient was managed over the duration of her pregnancy with verapamil.

Discussion

To understand the pathophysiology of primary aldosteronism it is necessary to recognize that there deviation from the normal mechanism of aldosterone synthesis which primarily depends on renin-angiotensin-aldosterone system (RAAS). In a normal and healthy human the juxtaglomerular system recognizes a decrease in either blood flow or the concentration of serum sodium causing the enzyme renin to be released which in turns activates angiotensinogen into angiotensin I which is further converted into angiotensin II by the enzyme, angiotensin converting enzyme (ACE) in the body. This angiotensin II acts to increase aldosterone synthesis in the glomerulosa layer of the adrenal cortex by the enzyme aldosterone synthase. Apart from the RAAS system aldosterone synthesis is also promoted by ACTH from the anterior pituitary and increase in serum potassium level (hyperkalemia).^{5,6} In our patient it was seen that the serum renin level wasn't raised and the renal ultrasound revealed no abnormality ruling out an increased renin as a cause of hyperaldosteronism. Furthermore there was no hyperpigmentation nor were there any features of Cushing Syndrome and the serum potassium level was also low thus increasing the suspicious for primary aldosteronism.

A number of studies have shown the effect primary aldosteronism can have on the cardiovascular system as a result of hypertension an electrolyte imbalance. In comparison with essential hypertension, primary aldosteronism is associated with higher risk of coronary artery disease,⁷ atrial fibrillation alone or with other heart disease⁸, stroke⁹, left ventricular hypertrophy or heart failure¹⁰, kidney disease¹¹ and decrease bone density and increase risk of fracture.¹²

Primary aldosteronism can negatively affect a women and the fetus in pregnancy. The effects of hypertension on pregnancy have been well documented (4) and an iatrogenic increase in aldosterone can lead to several potential problems in pregnancy. Morton et al reviewed 5 pregnancies in four women reaching the conclusion that cases of primary aldosteronism are underdiagnosed in patients.¹³ A high plasma aldosterone as well as a high aldosterone to renin ratio is needed for the diagnoses of primary aldosteronism as was the case with our patient.

As seen in previous studies the treatment of primary aldosteronism in pregnancy required the use of drugs that lower blood pressure and are also safe to be administered in pregnancy.¹⁴ Our patient showed decrease in blood pressure following the use of verapamil thus it was continued throughout the duration of pregnancy.

Conclusion

Primary aldosteronism can present as high blood pressure in a pregnant female, thus making it necessary for it to be ruled out in a patient with elevated blood pressure. For the diagnosis of primary aldosteronism a raised serum aldosterone level with an elevated plasma aldosterone to renin ratio is required. An antihypertensive agent which is safe during pregnancy such as a calcium channel blocker can be considered a viable option for the management of such patients.

Conflict of Interest: *None*

Funding Source: *None*

References

1. Lee FT, Elaraj D. Evaluation and Management of Primary Hyperaldosteronism. *Surg Clinics North Am.* 2019;99(4):731-45.
2. Kucharz EJ. Michał Lityński--a forgotten author of the first description on primary hyperaldosteronism. *Pol Arch Med Wewn.* 2007;117(1-2):57-8.
3. Stewart PM. Mineralocorticoid hypertension. *Lancet.* 1999;353(9161):1341-7.
4. Landau E, Amar L. Primary aldosteronism and pregnancy. In *Annales d'endocrinologie.* 2016; 77(2):148-60.
5. Vaidya A, Mulatero P, Baudrand R, Adler GK. The expanding spectrum of primary aldosteronism: implications for diagnosis, pathogenesis, and treatment. *Endocrine reviews.* 2018;39(6):1057-88.
6. Kronenberg H, Melmed S, Polonsky K, Larsen P. *Williams Textbook of Endocrinology.* 11th ed. Philadelphia, PA: Saunders; 2008
7. Catena C, Colussi G, Nadalini E, Chiuch A, Baroselli S, Lapenna R, Sechi LA. Cardiovascular outcomes in patients with primary aldosteronism after treatment. *Arch Intern Med.* 2008;168(1):80-5.

8. Monticone S, Burrello J, Tizzani D, Bertello C, Viola A, Buffolo F, Gabetti L, Mengozzi G, Williams TA, Rabbia F, Veglio F. Prevalence and clinical manifestations of primary aldosteronism encountered in primary care practice. *J Am Coll Cardiol*. 2017;69(14):1811-20.
9. Hundemer GL, Curhan GC, Yozamp N, Wang M, Vaidya A. Cardiometabolic outcomes and mortality in medically treated primary aldosteronism: a retrospective cohort study. *Lancet Diab Endocrinol*. 2018;6(1):51-9.
10. Murata M, Kitamura T, Tamada D, Mukai K, Kurebayashi S, Yamamoto T, Hashimoto K, Hayashi RD, Kouhara H, Takeiri S, Kajimoto Y. Plasma aldosterone level within the normal range is less associated with cardiovascular and cerebrovascular risk in primary aldosteronism. *J Hypertens*. 2013;35(5):1079-85.
11. Sechi LA, Novello M, Lapenna R, Baroselli S, Nadalini E, Colussi GL, Catena C. Long-term renal outcomes in patients with primary aldosteronism. *JAMA*. 2006;295(22):2638-45.
12. Salcuni AS, Palmieri S, Carnevale V, Morelli V, Battista C, Guarnieri V, Guglielmi G, Desina G, Eller-Vainicher C, Beck-Peccoz P, Scillitani A. Bone involvement in aldosteronism. *J Bone Min Res*. 2012;27(10):2217-22.
13. Morton A. Primary aldosteronism and pregnancy. *Pregnancy Hypertension*. *Int J Women's Cardiovasc Health*. 2015;5(4):259-62.
14. Corsello SM, Paragliola RM. Evaluation and Management of Endocrine Hypertension During Pregnancy. *Endocrinology and metabolism clinics of North America*. 2019;48(4):829-42.