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Research Article

**ANALYSIS OF FREQUENCY OF PRIMARY
HYPERALDOSTERONISM IN YOUNG HYPERTENSIVES IN
PAKISTAN**¹Dr Hasnain Aslam, ²Dr Hassam Ahmed, ³Dr Navaira Hye¹Medical Officer in Rural Dispensary chak no. 151, Chiniot, ²Medical Officer at BHU Dagger Qureshi, Bhakkar, ³Women Medical Officer at DHQ Hospital, Khanewal**Abstract:**

Introduction: Aldosterone, the major and most powerful mineralocorticoid hormone in humans, is synthesized from cholesterol in the adrenal cortex¹ and governs the physiological control of renal electrolyte balance. **Objectives of the study:** The main objective of the study is to analyze the frequency of primary hyperaldosteronism in young hypertensives in Pakistan. **Material and methods:** This cross sectional study was conducted at BHU Dagger Qureshi, Bhakkar and DHQ Hospital, Khanewal during October 2018 to December 2018. There were 100 hypertensive patients who were selected for this study. We reviewed the clinical records of each patient, and we considered hypertensive those patients with a diastolic blood pressure (DBP) >90 mm Hg and a systolic blood pressure (SBP) >140 mm Hg on at least 2 occasions on different days who were not taking antihypertensive drugs at the time of diagnosis. All patients underwent a clinical examination and serum determinations of creatinine, calcium, urea, glucose, and hepatic profile. **Results:** The data were collected from 100 patients of both genders. The mean age of the patients is 54.1±11.2. The mean systolic BP was 156.1±15.8 and mean diastolic BP was 96.6±9.1. Twenty-one of 37 patients met all criteria for PA because they had high SA values (16.5 to 41.0 ng/dL), low levels of PRA (<0.5 ng · mL⁻¹ · h⁻¹), and a high SA-PRA ratio (>50) on at least 2 determinations. In the remaining 16 of 37 patients, the SA values were between 9 and 16 ng/dL. All patients confirmed as having PA had a baseline SA value >9 ng/dL; patients with SA levels lower than this always tested negative on the confirmatory FST, independent of the magnitude of the SA-PRA ratio. **Conclusion:** It is concluded that SA-PRA ratio is a useful screening method in the diagnosis of PA, because most patients are normokalemic. Frequency of primary hyperaldosteronism was found to be 10%, emphasizing on the fact that it is not very uncommon in young hypertensives.

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INTRODUCTION:

Aldosterone, the major and most powerful mineralocorticoid hormone in humans, is synthesized from cholesterol in the adrenal cortex and governs the physiological control of renal electrolyte balance. In healthy individuals, one of the effects of aldosterone is to maintain an optimal balance, which helps to preserve a healthy vascular system [1]. However, when this balance is upset, the beneficial effects of aldosterone are lost and those that are detrimental to the vascular system predominate, leading to organ dysfunction³ and hypertension (HT). Currently, the prevalence of primary hyperaldosteronism (PHA) is much higher than the previously accepted rate, less than 1% of the hypertensive population, and in the specialist setting, may be over 10% [2].

Recent studies have demonstrated that primary aldosteronism (PA) is the most common form of secondary hypertension when determinations of serum aldosterone (SA), plasma renin activity (PRA), and the SA-PRA ratio are used as screening tools and the fludrocortisone, saline infusion, or captopril tests are used to confirm the diagnosis [3].

The different prevalence of PA could be explained by the method used to screen for the disease. The most recent investigations have used the SA-PRA ratio, and the classic studies used the presence of hypokalemia [4]. Because hypokalemia is variably present in patients with PA, the true prevalence could be underestimated. We have reported that hypokalemia is present only in 16% of PA patients and probably reflects the most severe forms of the disease. Another factor that could also explain the differences in PA prevalence might be related to the characteristics of hypertensive disease, such as hypertension severity or the difficulty to reach adequate blood pressure control [5]. PA is clinically suspected as part of "secondary hypertension," characterized by moderate to severe hypertension in young people and/or refractory hypertension. However, clinical and biochemical features vary widely, and normotensive PA patients have been described [6].

Objectives of the study

The main objective of the study is to analyze the frequency of primary hyperaldosteronism in young hypertensives in Pakistan.

MATERIAL AND METHODS:

This cross-sectional study was conducted at BHU Dagger Qureshi, Bhakkar and DHQ Hospital, Khanewal during October 2018 to December 2018. There were 100 hypertensive patients who were selected for this study. We reviewed the clinical records of each patient, and we considered hypertensive those patients with a diastolic blood pressure (DBP) >90 mm Hg and a systolic blood pressure (SBP) >140 mm Hg on at least 2 occasions on different days who were not taking antihypertensive drugs at the time of diagnosis. All patients underwent a clinical examination and serum determinations of creatinine, calcium, urea, glucose, and hepatic profile. With this screening, we excluded those with renal disease, diabetes mellitus, hepatic failure, hypercalcemia, clinical Cushing's syndrome, or acromegaly.

Patients were evaluated between 8 and 9 AM after a 12-hour fast. Because most patient were taking antihypertensive drugs at the time of the study, we instituted a washout period of at least 15 days for any drugs that could affect the renin-angiotensin system, such as β -blockers, angiotensin-converting enzyme inhibitors, angiotensin II receptors blockers, diuretics, spironolactone, and aspirin.

Ethical Considerations

This study was approved by the ethical committee of hospital.

STATISTICAL ANALYSIS:

The data obtained were transferred to computer for analysis using SPSS version 15.00 (SPSS, Inc., Chicago, IL, USA) software. A p value <0.05 was considered significant. Numbers, percentages, and mean plus standard deviation were used at analysis.

RESULTS:

The data were collected from 100 patients of both genders. The mean age of the patients is 54.1 ± 11.2 . The mean systolic BP was 156.1 ± 15.8 and mean diastolic BP was 96.6 ± 9.1 . All the demographic values are presented in table 01.

TABLE 1: Clinical and Laboratory Finding in Hypertensive Patients

Parameters	Hypertensives
Age, y	54.1±11.2
Gender, M/F	21/88
Blood pressure at study, mm Hg	
Systolic	156.1±15.8
Diastolic	96.6±9.1
SA, ng/dL	9.67±6.93
PRA, ng · mL ⁻¹ · h ⁻¹	2.3±5.4
SA/PRA ratio	12.32±21.25

Twenty-one of 37 patients met all criteria for PA because they had high SA values (16.5 to 41.0 ng/dL), low levels of PRA (<0.5 ng · mL⁻¹ · h⁻¹), and a high SA-PRA ratio (>50) on at least 2 determinations. In the remaining 16 of 37 patients, the SA values were between 9 and 16 ng/dL. All patients confirmed as having PA had a baseline SA value >9 ng/dL; patients with SA levels lower than this always tested negative on the confirmatory FST, independent of the magnitude of the SA-PRA ratio.

Table 02: Biochemical parameters of selected patients

Parameter	Positive Patients (n=37)	Negative Patients (n=25)	P
Basal SA, ng/dL	16.94±6.75 (9–41)	10.02±4.1 (5.2–20)	<0.01
Post FST SA, ng/dL	9.6±4.61 (5–23)	2.6±1.1 (2–4.1)	<0.01
PRA, ng · mL ⁻¹ · h ⁻¹	0.30±0.17 (0.1–0.9)	0.31±0.21 (0.1–0.6)	NS
SA/PRA ratio	69.23±48.8 (25.7–260)	37.83±15.03 (25.7–74.5)	<0.01
SBP, mm Hg	163.7±11.9	154±12.9	<0.01
DBP, mm Hg	101.6±9.9	96.4±10.4	NS
SK, mEq/L	4.2±0.3	4.3±0.7	NS

DISCUSSION:

The hypertensive population that should be evaluated to exclude PHA is a matter of debate. According to some experts, certain groups of patients have a higher prevalence of PHA and should undergo investigation: those with moderate/severe HT, resistant HT, HT associated with spontaneous or diuretic-induced hypokalemia [7], HT associated with a family history of early-onset HT or stroke at an early age (under 50 years), and the first-degree relatives of patients diagnosed with PHA [8].

Despite the increase in the prevalence of PHA, in Spain, the index of suspicion continues to be low, which is reflected in the low percentage of patients (4.6%) referred from primary care to the HTU with suspected PHA. This circumstance could be explained by the difficulty of carrying out systematic screening in primary care, variability in the clinical presentation of PHA, the high cost of the study, and the general acceptance of spironolactone as the fourth most widely administered drug in patients with resistant HT, the

use of which would lead to control of PHA in many cases [9].

The increase in the prevalence of PA according to the severity of hypertensive disease has never been published. However, several authors have recommended that the possibility of PA be investigated in cases of severe hypertension or resistant disease [10]. The present study would support this recommendation, because the prevalence of PA is higher in hypertensives in stage 2 and 3 of the JNC VI classification, and patients with PA needed more drugs than did non-PA, EH patients to achieve adequate BP control [11]. A recent study by Calhoun et al supports our results, providing data on subjects with resistant hypertension and in whom the prevalence of PA is even higher, reaching 20% of studied subjects. Thus, the spectrum of PA in EH seems to be continuous from low frequencies in mild hypertensives, similar to those found in normotensive subjects, to very high frequencies in severe or resistant hypertension [12].

CONCLUSION:

It is concluded that SA-PRA ratio is a useful screening method in the diagnosis of PA, because most patients are normokalemic. The low frequency of CT scan abnormalities in the absence of hypokalemia suggests that most PA patients likely have idiopathic hyperaldosteronism. Frequency of primary hyperaldosteronism was found to be 10%, emphasising on the fact that it is not very uncommon in young hypertensives.

REFERENCES:

1. Lim PO, Dow E, Brennan G, Jung RT, MacDonald TM. High prevalence of primary aldosteronism in the Tayside hypertension clinic population. *J Hum Hypertens.* 2000; 14: 311–315.
2. Funder JW, Carey RM, Fardella C, Gomez-Sanchez CE, Mantero F, Stowasser M, et al. Case detection, diagnosis, and treatment of patients with primary aldosteronism: an endocrine society clinical practice guideline. *J Clin Endocrinol Metab.* 2008;93:3266-81.
3. Sabio JM, Mediavilla-García JD, Jaén F, Fernández-Torres C, Aliaga L, Jiménez-Alonso J. Hiperaldosteronismo primario: análisis de una serie de 54 pacientes. *Med Clin (Barc).* 2005;124:765-8.
4. Morillas P, Castillo J, Quiles J, Núñez D, Guillén S, Bertomeu-González V, et al. Prevalencia del hiperaldosteronismo primario y afección cardíaca en el paciente hipertenso. *Rev Esp Cardiol.* 2008;6:418-21.
5. Young WF. Primary aldosteronism: renaissance of a syndrome. *Clin Endocrinol.* 2007;66:607-18.
6. Bunda S, Liu P, Wang Y, Liu K, Hinek A. Aldosterone induces elastin production in cardiac fibroblasts through activation of insulin-like growth factor-I receptors in a mineralocorticoid receptor-independent manner. *Am J Pathol.* 2007;171:809-19.
7. Whaley-Connell A, Johnson MS, Sowers JR. Aldosterone: role in the cardiometabolic syndrome and resistant hypertension. *Prog Cardiovasc Dis.* 2010;52:401-9.
8. Sowers JR, Whaley-Connell A, Epstein M. Narrative review: the emerging clinical implications of the role of aldosterone in the metabolic syndrome and resistant hypertension. *Ann Intern Med.* 2009;150:776-83.
9. Rossi GP, Pessina AC, Heagerty AM. Primary aldosteronism: an update on screening, diagnosis and treatment. *J Hypertens.* 2008;26:613-21.
10. Mosso L, Carvajal C, González A, Barraza A, Avila F, Montero J, et al. Primary aldosteronism and hypertensive disease. *Hypertension.* 2003;42:161-5.
11. Labinson PT, White WB, Tendler BE, Mansoorbpg GA. Primary hyperaldosteronism associated with hypertensive emergencies. *Am J Hypertens.* 2006;19:623-7.
12. Scheuner MT, Setodji CM, Pankow JS, Blumenthal RS, Keeler E. General Cardiovascular Risk Profile identifies advanced coronary artery calcium and is improved by family history: the multi-ethnic study of atherosclerosis. *Circ Cardiovasc Genet.* 2010;3:97-105.