Alterations in Hepatic Enzymes and Hormonal Biomarkers in Hypertensive Men: A Cross-Sectional Comparative Study

Ikhlas Abdel Fattah
King Saud University, Riyadh, Saudi Arabia
Email: Iklasabed20000@gmail.com

Received: 10 Sep 2025 | Accepted: 25 Dec 2025 | Published: 1 Jan 2026

Abstract

Hypertension is a chronic condition characterized by persistently elevated arterial blood pressure and is associated with significant metabolic and hormonal disturbances affecting multiple organ systems. This study aimed to evaluate liver enzyme activity and selected hormonal biomarkers in hypertensive patients compared with normotensive individuals. A cross-sectional study was conducted on 60 adult male participants, including 35 hypertensive patients attending outpatient clinics and 25 apparently healthy normotensive controls. Venous blood samples (5 mL) were collected under standardized conditions. Serum levels of aldosterone, cortisone, and creatinine were measured using ELISA (Human Diagnostics, Germany), while liver enzymes (ALT and AST) were assessed using standard biochemical methods. The results revealed significantly higher systolic and diastolic blood pressure in hypertensive patients compared to controls ($p \leq 0.01$). In addition, hypertensive patients exhibited significantly elevated levels of aldosterone, cortisone, ALT, AST, and creatinine. These findings indicate that hypertension is associated with significant alterations in hormonal balance, liver function, and renal parameters, reflecting its systemic nature. The combined evaluation of these biomarkers may provide useful insights for early detection and risk assessment in hypertensive patients. Further studies are warranted to clarify the underlying mechanisms linking hormonal dysregulation and hepatic function to hypertension.

Keywords: Hypertension; Liver enzymes; Aldosterone; Cortisone; Creatinine; Biomarkers

Introduction

Hypertension is a major systemic disorder characterized by sustained elevation of arterial blood pressure and associated with profound disturbances in vascular function and cardiac workload. Rather than being a simple hemodynamic abnormality, it reflects a complex interaction between cardiac output, peripheral resistance, and multiple regulatory pathways influenced by neurohormonal and metabolic factors [1]. Its asymptomatic progression often delays diagnosis, allowing structural and functional damage to accumulate over time, which justifies its classification as a "silent" but high-impact disease.

Diagnostic criteria for hypertension are well established, with optimal blood pressure levels ranging between 90/60 and 120/80 mmHg, while persistent values ≥140/90 mmHg confirm the diagnosis [2]. The condition is strongly associated with modifiable and non-modifiable risk factors, including obesity, smoking, and a history of cardiovascular events such as myocardial infarction and stroke [3]. Globally,

hypertension remains a leading contributor to mortality, accounting for approximately 9.4 million deaths annually, and plays a central role in the progression of chronic kidney disease and other long-term complications that impose significant healthcare burdens [4,5].

The development of hypertension involves a multifaceted pathophysiological process driven by genetic predisposition, aging, sex-related differences, and environmental influences such as diet, physical inactivity, and psychosocial stress [1,4]. These factors collectively disrupt vascular homeostasis and promote persistent elevations in arterial pressure, highlighting the need for a more integrated understanding of disease mechanisms.

A central component in blood pressure regulation is the renin–angiotensin–aldosterone system, which governs fluid balance and vascular tone. Aldosterone, the principal mineralocorticoid hormone, plays a critical role in sodium retention and potassium excretion; however, its effects extend beyond electrolyte regulation to include vascular remodeling, cardiac hypertrophy, and progressive renal damage such as proteinuria and glomerulosclerosis [6–8]. Dysregulation of aldosterone is clinically evident in primary hyperaldosteronism, a well-recognized cause of secondary hypertension associated with increased cardiovascular risk [9].

Beyond endocrine mechanisms, hypertension has been increasingly linked to alterations in liver function, suggesting a broader systemic involvement. Elevated hepatic enzymes may reflect underlying metabolic stress, oxidative damage, or inflammatory processes accompanying hypertensive states. Therefore, simultaneous evaluation of hormonal and hepatic biomarkers may offer a more comprehensive perspective on disease progression.

Accordingly, this study aimed to investigate the relationship between aldosterone and cortisone levels and selected renal and hepatic function parameters, and to assess their potential value as early indicators of hypertension in non-diabetic individuals.

Materials and Methods

A cross-sectional study was conducted between October 2024 and March 2025 in Texas, United States. The study population consisted of 80 male participants aged 40–60 years, including 45 patients diagnosed with hypertension and attending outpatient clinics, and 35 apparently healthy normotensive individuals recruited from academic staff and students, who served as the control group.

Venous blood samples (5 mL) were collected from the antecubital vein of each participant under aseptic conditions using sterile disposable syringes. The collected samples were transferred into clean, sterile tubes and centrifuged at 3000 rpm for 15 minutes to obtain serum. The separated sera were subsequently used for biochemical analysis. Serum concentrations of aldosterone, cortisone, and creatinine were measured using enzyme-linked immunosorbent assay (ELISA) kits (Human Diagnostics, Germany), following the manufacturer's instructions.

Arterial blood pressure was measured using a calibrated standard sphygmomanometer. All participants were seated and allowed to rest for at least 10 minutes prior to measurement. Three consecutive blood pressure readings were recorded at five-

minute intervals, and the average of these readings was considered for statistical analysis.

Data were expressed as mean ± standard deviation (SD). Statistical comparisons between hypertensive patients and normotensive controls were performed using an independent samples Student's t-test. A p-value of less than 0.05 was considered statistically significant.

Results and Discussion

The present study provides a comprehensive evaluation of hemodynamic, hormonal, hepatic, and renal alterations associated with hypertension. As presented in Table 1, no significant difference in age was observed between hypertensive patients and normotensive controls ($p > 0.05$), indicating appropriate group comparability. In contrast, both systolic and diastolic blood pressure values were significantly elevated in hypertensive patients ($p \leq 0.01$), confirming the clinical classification and validity of the selected study population.

The marked elevation in blood pressure reflects the underlying pathophysiological imbalance between cardiac output and peripheral vascular resistance, which is a hallmark of hypertension [1]. These findings are consistent with established diagnostic criteria and epidemiological data indicating that sustained elevations in arterial pressure are central to disease progression and its associated complications [2,4].

Table 1. The age, systolic and diastolic blood pressure in hypertensive patients and controls

Parameters	Control	Patients	P value
Age (years)	49.1 ± 8.1	51.4 ± 4.3	NS
Systolic Bp (mmHg)	118 ± 3	159 ± 7	0.01
Diastolic blood pressure (mmHg)	80.5 ± 1.5	96 ± 4	0.01

Biochemically, hypertensive patients demonstrated significantly increased levels of liver enzymes (AST and ALT) compared with controls ($p \leq 0.01$), as shown in Table 2 and Figure 1. This elevation may indicate subclinical hepatic involvement in hypertensive individuals. The increase in transaminases can be attributed to metabolic stress, oxidative damage, and low-grade inflammation, which are frequently associated with hypertension. These findings support the growing body of evidence suggesting that hypertension extends beyond vascular dysfunction to involve systemic metabolic disturbances affecting multiple organs, including the liver.

In addition to hepatic alterations, significant elevations in aldosterone and cortisone levels were observed in hypertensive patients ($p \leq 0.01$). The increase in aldosterone is particularly relevant, as this hormone plays a central role in the regulation of blood pressure through sodium retention and fluid balance [6]. However, beyond its classical function, aldosterone has been implicated in vascular remodeling, myocardial hypertrophy, and progressive renal injury [7,8]. The elevated aldosterone

levels observed in this study are consistent with previous reports highlighting the contribution of the renin–angiotensin–aldosterone system (RAAS) to the pathogenesis of hypertension. Moreover, excessive aldosterone secretion, as seen in conditions such as primary hyperaldosteronism, is strongly associated with increased cardiovascular risk [9].

Similarly, the significant rise in cortisone levels suggests an additional endocrine component contributing to hypertension. Cortisone, as a glucocorticoid-related hormone, influences vascular tone, metabolic regulation, and stress responses. Elevated levels may enhance vascular sensitivity to vasoconstrictors and promote sodium retention, thereby contributing to increased blood pressure. These findings highlight the importance of hormonal imbalance in the development and maintenance of hypertensive states.

Furthermore, serum creatinine levels were significantly higher in hypertensive patients compared with controls ($p \leq 0.01$), indicating possible early renal impairment. This observation aligns with the well-documented relationship between hypertension and kidney dysfunction, where prolonged elevation in blood pressure leads to structural and functional damage to renal tissues, including glomerulosclerosis and reduced filtration capacity [5,8].

Table 2: Levels of liver enzymes, cortisone, and aldosterone in the blood of people with high blood pressure and healthy controls.

Parameters	Control	Patients	P value
AST (U/L)	20.5 ± 2.3	36.4 ± 3.5	0.01
ALT (U/l)	21.5± 3.8	33.5 ± 3.4	0.01
Aldosterone (pg/ml)	25.1 ± 7.3	106.8 ± 18.2	0.01
Cortisone (ng/ml)	6.44 ± 2.7	14.3 ± 3.5	0.01
Creatinine (mg/dl)	0.398 ± 0.03	0.45 ± 0.05	0.01

Overall, the combined elevation of liver enzymes, hormonal biomarkers, and creatinine suggests that hypertension is a systemic disorder involving interconnected pathways of endocrine dysregulation, metabolic disturbance, and organ dysfunction. The findings of this study reinforce the concept that hypertension should not be viewed solely as a cardiovascular condition but rather as a multisystem disease with broad biochemical implications.

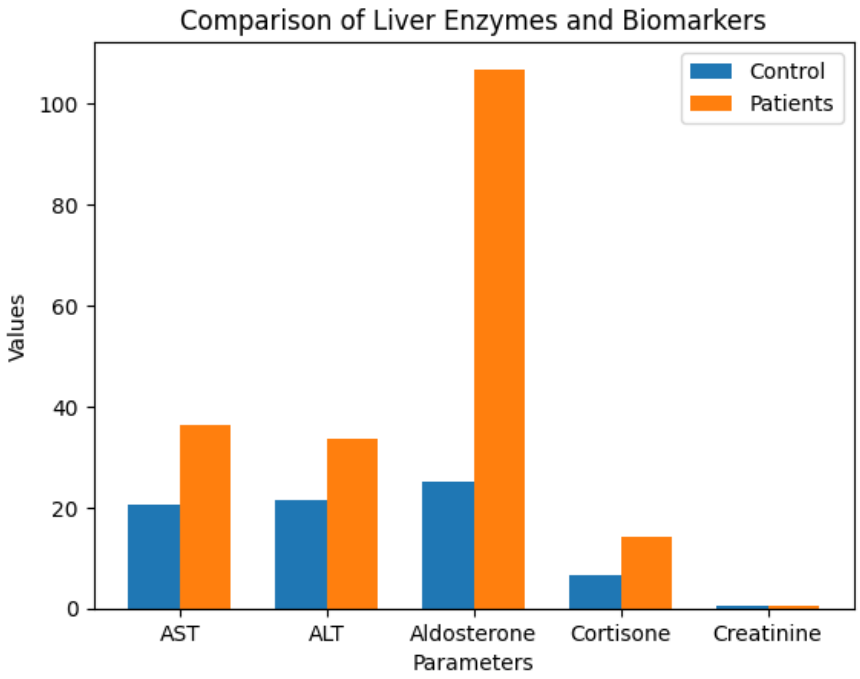

Figure 1. Comparison of liver enzymes and hormonal biomarkers between hypertensive patients and normotensive controls. Hypertensive patients exhibited significantly higher levels of AST, ALT, aldosterone, cortisone, and creatinine compared to controls ($p \leq 0.01$).

Importantly, the integration of hormonal (aldosterone and cortisone) and biochemical (liver enzymes and creatinine) parameters may provide valuable insights for early detection and risk stratification in hypertensive patients. These biomarkers could serve as potential indicators of disease severity and progression, supporting more comprehensive clinical assessment and management strategies.

Conclusion

The findings of the present study demonstrate that hypertension is associated with significant alterations in hormonal, hepatic, and renal biomarkers. Hypertensive patients exhibited markedly elevated levels of aldosterone and cortisone, alongside increased liver enzyme activity (AST and ALT) and serum creatinine, compared with normotensive controls. These changes reflect the multifactorial nature of hypertension, involving endocrine dysregulation, metabolic disturbance, and early organ dysfunction.

The observed elevation in aldosterone supports the central role of the renin–angiotensin–aldosterone system in the pathogenesis of hypertension, while increased cortisone levels highlight the contribution of stress-related hormonal pathways. Concurrent increases in liver enzymes suggest potential hepatic involvement, possibly linked to oxidative stress and inflammatory processes. In addition, elevated creatinine levels may indicate early renal impairment associated with sustained high blood pressure.

Collectively, these findings emphasize that hypertension should be considered a systemic disorder rather than a purely cardiovascular condition. The combined assessment of hormonal and biochemical markers may provide valuable insights for

early detection, risk stratification, and improved clinical management of hypertensive patients. Further large-scale studies are recommended to clarify the mechanistic relationships between endocrine alterations, liver function, and hypertension progression.

References

1. Espejo M, Magabo S, Rivera-Castro A, Faiz M, et al. Qualitative study of knowledge, perception, and behavior related to hypertension and cardiovascular disease risk reduction among hypertensive African-Americans in urban inner city of South Bronx, New York. *J Racial Ethn Health Disparities*. 2019;6:197–206.
2. Ona DID, Jimeno CA, Jasul GV Jr, Bunyi MLE, et al. Executive summary of the 2020 clinical practice guidelines for the management of hypertension in the Philippines. *J Clin Hypertens*. 2021;23:1637–1650.
3. Castillo RR, Atilano AA, David-Ona DI, Napiza-Granada C, et al. May Measurement Month 2017: an analysis of blood pressure screening in the Philippines—South-East Asia and Australasia. *Eur Heart J Suppl*. 2019;21(Suppl D):D92–D96.
4. World Health Organization (WHO). Global brief on hypertension: silent killer, global public health crisis. World Health Day 2013. Available from: https://ish-world.com/downloads/pdf/global_brief_hypertension.pdf
5. Petrie JR, Guzik TJ, Touyz RM. Diabetes, hypertension, and cardiovascular disease: clinical insights and vascular mechanisms. *Can J Cardiol*. 2018;34:575–584.
6. Roldán J, Morillas P, Castillo J, Andrade H, et al. Plasma aldosterone and glomerular filtration in hypertensive patients with preserved renal function. *Rev Esp Cardiol*. 2010;63:103–106.
7. Yoshimoto T, Hirata Y. Aldosterone as a cardiovascular risk hormone. *Endocr J*. 2007;54:359–370.
8. Ibrahim HN, Hostetter TH. Aldosterone in renal disease. *Curr Opin Nephrol Hypertens*. 2003;12:159–164.
9. Wenzel U. Aldosterone and progression of renal disease. *Curr Opin Nephrol Hypertens*. 2008;17:44–50.
10. Vasan RS, Evans JC, Larson MG, Wilson PW, Meigs JB, Rifai N, et al. Aldosterone and the incidence of hypertension in nonhypertensive persons. *N Engl J Med*. 2004;351:33–41.
11. Shibata S, Nagase M, Yoshida S, Kawachi H, Fujita T. Podocyte as the target for aldosterone: roles of oxidative stress and Sgk1. *Hypertension*. 2007;49:355–364.
12. Rossier MF, Feldman D. Aldosterone and blood pressure regulation: recent milestones on the long and winding road from electrocortin to KCNJ5, GPER, and beyond. *Hypertension*. 2014;63:19–21.
13. Zennaro MC, Rickard AJ, Boulkroun S. Genetics of mineralocorticoid excess: an update for clinicians. *Eur J Endocrinol*. 2013;169:R15–25.
14. Armanini D, Andrisani A, Ambrosini G. Role of adrenocorticotropic hormone in essential hypertension and primary aldosteronism. *Clin Hypertens*. 2017;19:287–289.

15. Kobayashi H, Haketa A, Takahiro U, et al. Plasma adrenocorticotropic hormone but not aldosterone is correlated with blood pressure in patients with aldosterone-producing adenomas. *J Clin Hypertens (Greenwich)*. 2016;19:280–286.
16. El Ghorayeb N, Bourdeau I, Lacroix A. Role of ACTH and other hormones in the regulation of aldosterone production in primary aldosteronism. *Front Endocrinol (Lausanne)*. 2016;7:72.
17. Kennon B, Ingram MC, Friel EC, et al. Aldosterone synthase gene variation and adrenocortical response to sodium status, angiotensin II and ACTH in normal male subjects. *Clin Endocrinol (Oxf)*. 2004;61:174–181.
18. Pooja, Mittal Y. Serum creatinine level in hypertensive patients: a study from Uttarakhand, India. *Int J Pharm Sci Res*. 2014;5:2955–2959.
19. Nagah AA, Mohammed H, Hamad HM. Serum creatinine, albumin, and urine protein in hypertensive patients. *Indian J Basic Appl Med Res*. 2012;1:292–295.
20. Sarkar D, Latif SA, Aich J, Udin MM. Studies on serum creatinine and creatinine clearance in hypertensive patients. *J Bangladesh Soc Physiol*. 2006;1:19–26.
21. Yadav SC. Evaluation of serum creatinine in hypertensive patients. *J Nobel Med Coll*. 2016;5:56–59.
22. Chambergo-Michilot D, Rodrigo-Gallardo PK, Huaman MR. Hypertension and histopathology severity of non-alcoholic fatty liver disease among adults with obesity: a cross-sectional study. *Clin Exp Gastroenterol*. 2023;16:129–136.
23. Ciardullo S, Monti T, Sala I, Grassi G, Mancia G, Perseghin G. Nonalcoholic fatty liver disease and advanced fibrosis in US adults across blood pressure categories. *Hypertension*. 2020;76:562–568.
24. Bioletto F, Bollati M, Lopez C, Arata S, Procopio M, Ponzetto F, et al. Primary aldosteronism and resistant hypertension: a pathophysiological insight. *Int J Mol Sci*. 2022;23:4803.
25. Hammer F, Stewart PM. Cortisol metabolism in hypertension. *Best Pract Res Clin Endocrinol Metab*. 2006;20:337–353.