Cardiac Arrest Induced by Anti-hypertensive and NSAIDS Drug Abuse Uses due to their Role Effect on Electrolytes and Aldosterone Levels in Hypertensive Patients with Renal Insufficiency

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Abstract

The present study aims to investigate the role of the drugs like angiotensin converting enzyme inhibitors (ACEI), non-steroidal anti-inflammatory drugs NSAIDs and their effects in the contributions of electrolyte fluctuation levels, as well as in an abuse uses. Forty four cardiac arrest subjects cases of hypertensive with renal failure (renal insufficiency) were taken, and forty participants as a healthy control without any heart and kidney problems with same of mean age (58±5). Done in Hilla city/Iraq were enrolled in this study. Results that collected from the present study were a significant in reduction of serum aldosterone levels, with very clear raises in plasma potassium and urinary sodium levels due to the action of hypertension drugs. Also results shown a significant decreases in the urinary potassium, otherwise plasma sodium levels within the normal elevated malady in patients, when compared with healthy control. There were significance results with moderately increases levels of the patient serums urea and creatinine, as when correlated with healthy control investigations. The current study found that BMI does not differ in patient groups (with cardiac arrest) when compared with the normal healthy control group and each were within the BMI range for normal weight of a person both in males and females. It was concluded that uses of antihypertensive drugs and NSAIDs lead to lowering aldosterone levels that relates hypertension treatments and inflammatory diseases.

Keywords: *ACEI, Aldosterone, NSAIDs, Potassium, Sodium*

Introduction

Nowadays, Blood pressure is the force that is exerted by the arterial blood to against vessels walls. Blood pressure forces depends on the heart work and the blood vessels resistance. The guideline of medicine was defined hypertension as a blood pressure higher than 130 over 80 millimeters of mercury (mmHg), according to guidelines issued by the American Heart Association (AHA) in November 2017 [(1)] . A high blood pressure can cause a damage of small blood vessels like in the kidneys and reducing their ability for properly work and may result in high stretch of blood vessels. Eventually, this stretching caused scars and weakens may including the blood vessels in the kidneys [(2)].

Historically a great relationship between hypertension and renal diseases. Renal failure may induced with hypertension as well as renal damage. Patients with uncomplicated obvious hypertension were separated into the distinct clinical and histological patterns like nephrosclerosis may compact with renal insufficiency symptoms [(3)]. The vascular lesions of hyaline arteriosclerosis develop slowly without overt proteinuria. Hypertension is the second leading cause of kidney failure in the United States after diabetes [(4)]. So the rate of kidney failure due to high blood pressure increased 7.7 percent [(5)]. The relative risk of serious

renal damage in patients with uncomplicated essential hypertension is less common, when its compared with other presented in cardiovascular complications [6]. An increased of a cardiovascular morbidity and mortality, that shown a risk of sudden cardiac death, has been presented in patients with rheumatoid arthritis (RA), that associated with heart rate variability and ventricular repolarization abnormality [7]. The interplay of these parameters and the inflammation for example RA made a known to exist that the inflammation induced and growing interest [8]. Whilst a higher incidence about world in a prevalence of the ischemic heart disease in many cases with rheumatoid arthritis, some authors have shown the increased of heart diseases that cannot be explained by traditional risk factors alone [9], so their interest in the role of inflammation as novel risk factor for atherosclerosis. Where potassium concentrations raised in the extracellular will be modify a for myocytes contractility, from -85mV to -65mV and -40mV in some cases [10]. Anti-hypertensive drugs likes Angiotensin-converting enzyme inhibitors, such as ramipril and enalapril inhibit the conversion of angiotensin I to angiotensin II and at the end lowering of aldosterone secretion, that's of the main role were blood pressure reduction by sodium reabsorption inhibition in the renal tubules, as well as the sodium was already losses due to renal failure [11]. When the nonsteroidal anti-inflammatory drugs (NSAIDs) administration are presence the clinical signs and symptoms were more obviously due to inhibition of renin secretion. So the hypoaldoserinisim was correlated with hyporeininmic that caused hyperkalemia. Whether remains inhibition of the aldosterone metabolism role by non-selective NSAIDs is a casual or causal factor in NSAID-induced cardiovascular toxicity [12].

Materials and Methods

Study Design

The study design was a case control study which was carried out between November 2017 to April 2018. Sample collection and examination was carried out at the Department of Biochemistry, College of Medicine, University of Babylon and Merjan Medical Teaching Hospital in Hilla City, Iraq. Sample size was calculated according to Daniel sample size formula equation with Iraqi disease prevalence. A total of 45 patients with hypertensive renal insufficiency were enrolled in this study.

Exclusion and Inclusion Criteria

The inclusion criteria included many cases of cardiac arrests and hypertensive patients with and renal failure (insufficiency) in presence of inflammatory diseases like rheumatoid and osteoarthritis. Only those with rheumatoid arthritis were selected. Patients who were under treatments where also taken. Participant who were smokers and patient who received antihypertensive treatment without NSAIDs or use angiotensin II receptor blocker like losartan and candesartan were excluded from the present study.

Measurements

Body mass index of patients and healthy controls were calculated using a mathematical equation, in which the weight in kilogram was divided by the square high measured by meters [13] . Determination of serum Aldosterone levels in patient and control groups were achieved using enzyme linked immunosorbent assay ELISA kit (Demeditec Diagnostics) Germany, and according to manufacturer instructions. Determination of Potassium and Sodium was examined using a spectrophotometric manual kit, blood collected without using tourniquet. Urine potassium and sodium was examined using a fully automated biochemistry mindray device. Urine sample were collected randomly and in a 1 litre volume. Determination serum urea was achieved using a spectrophotometeric manual prepared kits.

Results and Discussion

Table 1: The Number and Age of All Participants

Groups	Total	Male	Female	Mean ±SD Age (years)	
				Male	Female
Patients	45	25	20	58±5	55±5
Controls	40	20	20	55±2	50±2
P value				0.877	0.909

Table 2: Aldosterone ng/dl of Patients and Healthy Control Groups According to Sex, (normal 7.5ng/dl), P.value < 0.05.

Parameter	Gender	Patient Aldosterone Level	Healthy Control Aldosterone Level	P Value
		Mean ±SD	Mean ±SD	
Aldosterone ng/dl	Male	1.6±0.60	7.3±0.18	1.52 X10-9
	Female	1.2±0.53	7.1±0.38	9.12 X10-7

Table 3: Mean Difference of Plasma, urine and serum Parameters of Patients and Healthy Control Groups

	Parameter	Patient	Healthy Control	Normal Value
		Mean ±SD	Mean ±SD	
Plasma estimations Parameter	Na+2	1.6mmol/L	2.25mmo/L	2-2.6mmol/L
	K+	7.8mmol/L	4.3mmol/L	3.5-5.2mmol/L
Urinary estimations Parameter	Na+2 K+	18mEq/L 12.5mEq/L	50mEq/L 35mEq/L	40 - 220 m Eq/L 20 - 60 m Eq/L
Serum estimations Parameter	Urea Creatinine	77.5mg/dl 2.32mg/dl	43.75mg/dl 1.1mg/dl	45-50mg/dl 0.6-1.2mg/dl

Table 4: The Body Mass Index (kg /m2) of Participants.

Subjects	Total Number (Male)/ (Female)	Mean ±SD	
		Male	Female
Patients group	45 (25/20)	22.19±2.5	22.96±2.5
Controls group	40 (20/20)	21.55±1	20.92±1.5
P value		0.994	0.118

Cases with cardiac arrest were recruited, that are associated with a score level of symptoms and investigations. Disease severity in the current study found there was 45 patients with a significantly worsened hypertension and renal insufficiency, under treatment complexes such as NSAIDs and angiotensin converting enzyme inhibitors (ACEI). The other 40 healthy controls had no treatments or problems (table 1) .

The results of the present study revealed a significant decreases (P> 0.05) in serum aldosterone levels in patients with cardiac arrest when compared with healthy control (table 2) . A study by struthers [14] shown magnesium loss caused by aldosterone and by diuretics could contribute to coronary artery spasm like cardiac arrest and arrhythmias. The results were non-confined with a study by Chrysostomou [15] which depended on a Double-Blind Placebo-controlled or a triple mod therapy.

The Current study showed a significant decrease in the levels of plasma parameters sodium and potassium in patients groups when compared to healthy controls groups (table 3) . A similar result were revealed by Graudal and colleagues [16] and gu and colleagues [17] which showed the role mechanism of aldosterone in the sodium homeostasis and the reduction of aldosterone for reduce Na^+ in hypertension and in hypernatremia cases, as well as its correction level with the Serbian study by Tasic and colleagues [18].

The results of the present study included a significant decrease in the levels of urinary parameters potassium and in patients groups when compared to healthy controls groups, due to the reduction of aldosterone, similar results were revealed by [19] who showed the ability to distinguish hypovolemic by using the urine sodium alone was reasonably high (the accuracy with a cutoff value of 50 mEq/L was 82%). So the elevation of urine sodium levels up to 50 mEq/L depend on clinically meaningful response to isotonic saline infusion. And with experimental study [20] characterized high fat-fed mice which contribution of excretion of Na concentrations were mediated Na+ reabsorption in the presence of insulin resistance obesity and, so the high amount of fat-fed demonstrated impaired Na^+ excretion as well as elevated blood pressure. Our study agrees with the results of ueda and colleagues [21] where urinary potassium levels excretion reduced according to impairment in renal function, so the urinary potassium excretion mostly affected by urinary sodium excretion and estimated GFR in patients with chronic kidney disease. A study by weir and colleagues shows similar results to our study [22] that concluded the same results with our potassium studied. Although these results disagreed with a study by ghazi and team [23] were remembered that the antihypertensive treatments effect in spironolactone were positively related to urinary sodium excretion regardless of aldosterone status.

The present and other studies investigates the serum creatinine and urea levels, and found a clear elevation in these normal values due to the impairment renal function, and these results were in agreements with a study by Tesfaye& Jedlickova [24] have shown the nephrotoxic drugs contributions in the clinical state of spinal cord injury with chronic renal impairment , while the study by Córdova-Sánchez and team [25] showed elevation in creatinine results. The current study shows urea levels similar to those presented by Mackenzie & Chacko [26] who showed initiate in renal replacement therapy by treating acute kidney injury in the intensive care setting but without clinically significant uremia symptoms absence. The results of our study were in disagreement with a study by tujjar and colleagues [27] who showed patients who survived cardiac arrest were independent prognostic factors like age, epinephrine dose, cumulative fluid balance and presence of shock were independent predictors of the development of acute kidney injury in this population. Renal failure and neurological has no clear correlation.

The current study found that BMI does not differ in patient groups (with cardiac arrest) when compared with the normal healthy control group and each were within the BMI range for normal weight of a person both in males and females (table 4) . This was statistically insignificant between these groups and was coherent with a study by Gil and colleagues [28] who showed limited data between BMI and cardiac arrest.

Conclusion

Uses of antihypertensive drugs and NSAIDs lead to lowering aldosterone levels that relates hypertension treatments and inflammatory diseases. Hyperkalemia may contribute in the cardiac arrest pathogenesis . In the same time heart activity will be altered by abnormal depolarization like sudden cessation may occur.

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Conflict of Interest : Nil

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