ORIGINAL ARTICLE

Perspective of Cardiac Troponin and Membrane Potential in People Living with Hypertension

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ABSTRACT

Background: Hypertension is an event in which the force of the blood against the artery walls is too high leading to severe health complications and increases the risk of heart disease, stroke, and sometimes death. **Aim:** This study was carried out to determine the levels of cardiac troponin 1 and membrane potential in hypertensive subjects in Owerri, Imo state. **Materials and Methods:** A total of 120 subjects within the age 30–70 years were recruited for this study. The study consists of 60 subjects who were diagnosed of hypertension and 60 were apparently healthy individuals who served as controls subjects of the same age bracket. The levels of cardiac troponin 1 and membrane potential were analyzed using enzyme-linked immunosorbent assay technique. Data were assessed using SPSS version 20, the mean value with $P < 0.05$ was considered statistically significant. **Results:** The result revealed that the levels of cardiac troponin 1 in hypertension were significantly increased when compared with control subjects while the levels of membrane potential were significantly decreased when compared to control at $P < 0.05$. **Conclusion**: The increased serum level of cardiac troponin 1 and decreased membrane potential in hypertensive subjects may contribute some risk factors in patients with hypertension.

Key words: Cardiac troponin, hypertension, membrane potential

INTRODUCTION

Hypertension is a leading cause of mortality and is when the blood pressure (BP) is above 140/90. It will be considered as severe if the pressure is above 180/120. It often has no symptoms. Over time, if untreated, it can lead to ill health conditions, such as heart disease and stroke.[1] The eating of a healthier die, exercising the body regularly and taking drugs as well as taking less salt leads to lower BP.[2]

Hypertension is associated with abnormal heart conditions leading to high BP (HBP). The heart working under increased pressure causes various heart disorders. It can cause serious ill health as well as death. In fact, the heart disorders linked with HBP relate to the heart's arteries and muscles. The coronary arteries transport blood to the heartarteries and muscle. When HBP results in narrowing the blood vessels, the blood flow to the heart can be slowed or stopped.[3]

Hypertension makes it not easy for the heart to pump blood. Like other muscles in the body, regular hard work causes the heart muscles to thicken and grow. This changes the way the heart performs its role.[4]

The risk of hypertension may increase as a result of overweight and lack of exercise. Furthermore, smoking and eating food

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high in fat and cholesterol jack up the hypertensive risk. It can increase as a result of genetic inheritance.[5]

Symptoms of hypertension vary depending on the severity of the situation and progression of the disease. There may be no symptoms or symptoms linked with hypertension. They include: Chest pain (angina), tightness or pressure in the chest, shortness of breath, fatigue, pain in the neck, back, arms, or shoulders, persistent cough, loss of appetite, and leg or ankle swelling.[6]

Hypertension seems to affect the status of cardiac troponin and membrane potential. Troponin is a form of protein that resides in the muscles of the heart. Troponin normally is not seen in the blood. However, when heart muscles are injured, troponin goes into the bloodstream. As heart injures, elevation at greater amounts of troponin is released in the blood.[7]

On the other hand, membrane potential is due to difference in concentration and permeability of important ions across a membrane. Due to unequal concentrations of ions across a membrane, the membrane has an electrical charge.[8] Membrane potential simply is the difference between the electric potential in the inside and outside matrices of the cell when it is not excited.[9] Every cell of the body possess its own membrane potential, but only excitable cells – nerves and muscles – are capable to change it and generate an action potential.[10]

In the light of the above, it will be very necessary to determine cardiac troponin and membrane potential in hypertension to provide information for better diagnosis and management.

MATERIALS AND METHODS

Subjects

A total of 120 subjects between the ages 30 and 70 years were recruited for this study. Sixty were hypertensive subjects who had been attending cardiology clinic for not <3 months diagnosed of hypertension consisting of 30 males and 30 females. Sixty were apparently healthy individuals who served as controls subjects of the same age limits and sex who had no record of any other ailment.

Blood collection

Venous blood samples (5 ml) were collected aseptically by venipuncture from each of the subjects using a 5 ml sterile disposable syringe and needle. The whole blood samples were dispensed into a pre-labeled plain dry specimen container and allowed to clot. The clotted samples were centrifuged at 3000 rpm for 5 min to separate and obtain the serum for analysis. Informed consent of the participants was obtained and was conducted in line with the ethical approval of the hospital.

Biochemical assay

The serum cardiac troponin 1 was determined by enzyme-linked immunosorbent assay technique[11] while membrane potential was determined by Nernst Equation.[12]

Statistical analysis

The results were expressed as mean ± standard deviation (SD). The statistical evaluation of data was performed using independent students.

RESULTS

The result in Table 1 revealed that the levels of cardiac troponin 1 in hypertension were significantly increased when compared with control subjects while the levels of membrane potential were significantly decreased when compared to control at $P < 0.05$.However, there is no significant difference in the levels of cardiac troponin 1 and membrane potential in Table 2.

DISCUSSION

Hypertension is a major health disorder throughout the world as a result of its high prevalence and its association with increased risk of cardiovascular disease. Advances in the diagnosis and treatment of hypertension have played a relevant role in recent dramatic declines in coronary heart disease and stroke mortality in industrialized countries.[13] Hypertension is dangerous because the heart works harder to move blood through the blood vessels. HBP contributes to the hardening of the arteries, decrease of blood and oxygen flow to the heart. When left untreated, HBP can lead to heart attack, heart failure, stroke, erectile dysfunction, kidney failure, and irregular heartbeat.[14]

The result from this study showed that the serum cardiac troponin 1 in hypertension was significantly increased when compared with the controls. This elevation of troponin in hypertension could be linked to the necrosis of cardiac muscles as a result of cardiac myopathies.[15] This leads to spillage of troponin into the blood stream. Similarly, it could be associated with associated with microvascular and macrovascular complications as this causes myocardial injury.[16] This is in line with the work of Eggers *et al*.,[17] in which there an elevation of troponin in coronary disease.

Table 1: Mean±SD values of serum cardiac troponin 1 and membrane potential in hypertensive subjects

Parameters	Hypertensive (*n*=30)	Controls Subjects (*n*=30)
Troponin 1 (µg/ml)	0.083±0.01*	0.02±0.01
Membrane potential(J)	145.64±12.27*	262.74±13.42

*Statistically significant compared with control ($P < 0.05$);
SD: Standard deviation

Table 2: Mean±SD values of serum troponin1 and membrane potential in male and female hypertensive subjects

Parameters	Female: Hypertension subjects (*n*=15)	Male: Hypertension subjects (*n*=15)
Troponin (µg/ml)	0.081±0.014	0.084±0.014
Membrane potential(µg/ml)	145.6±12.51	146.04±12.74

SD: Standard deviation

In the same vein, the increase in cardiac troponin 1 in hypertension could be associated with ion transport disorder. In which there are production of circulating Na+ transport inhibitors that act on renal epithelial cells and vascular smooth muscle cells.[18] This results to the idea that, in response to salt loading, the kidney produces a circulating substance that causes both natriuresis and vasoconstriction. The increased intracellular Na+, acting on a plasma membrane Na+-Ca2+ exchange system, would result to increased intracellular Ca2+. Increased vascular smooth muscle cell Ca2+, by its well-known action on the contractile machinery, would in turn lead to contraction of vascular smooth muscle cells or increased responsiveness to vasoconstrictors.[19] Increased vascular tone would raise BP, contributing to the desired natriuresis. This is in agreement with the report of Eggers *et al*.,[17] in which increase in cardiac troponin activate the development of hypertension. These substances may not have been appreciated in the past because of their failure to circulate significantly. An excel-lent example of this is the endothelium-derived relaxing factor. This agent, whose chemical identity is probably nitric oxide, 81 is released from endothelial cells in response to a various hormonal and physical factors.[20]

Furthermore, hypertension remains the number one silent killer in this generation, claiming thousands of lives all over the world. The level of membrane potential was significantly decreased in hypertension when compared with the control. This could be linked to sodium potassium pump dysfunction, in which the ion transport is highly hindered. This tends to affect the microvascularity of the cell in hypertension. Hence, it affects the contractibility of the diastolic and systolic system of the body. This is in line with the work of Ashcroft and Rorsman,[21] in which the membrane potential was adversely affected in diabetes. The defects in the transport systems play an important role in the pathogenesis of hypertension due to the cellular handling of Na+ and Ca2+ions in vascular smooth muscle cells. Na+ pump inhibitors would also be expected to affect vascular smooth muscle function. In these cells, the inhibitors would cause cell Na+ and volume to rise and membrane potential to fall.[22] However, when the Na+ intake is matched by Na+ excretion at a plasma volume and BP very close to normal, there may not be alteration in the membrane potential, and the vascular architecture is intact.[23] However, in hypertensive individual, there is failure to excrete the Na+ load at an adequate rate; hence, there is volume overloaded, which will continuously produce the natriuretic or hypertensive substance which interfere with the integrity of the membrane potential.[24] The membrane potential is the main area of cellular function and cell-to-cell signaling, and ultimately body functions.[25]

CONCLUSION

The increase in cardiac troponin and decrease in membrane potential may probably indicate errors, defects or abnormality in the ion transport system in hypertension.

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